113993

APPENDIX F, SECTION 1

FISH TISSUE SAMPLING
FIELD TRIP REPORT
ROY F. WESTON, INC.

MEMORANDUM

To: Project File
From: Andrew Frebowitz
Project Manager
B&R Environmental
Date: December 8, 1997
c: Keystone Sanitation Landfill
Remedial Investigation Report for OU-2
Appendix F

In the report presented in Appendix F (Fish Tissue Sampling Field Trip Report by Roy F. Weston), Pond 1 and Pond 2 have been substituted by B&R Environmental in place of local residents' names. These locations are consistent with all other references to Pond 1 and Pond 2 throughout the RI report.

TRIP REPORT

Keystone Sanitation Landfill NPL Site
Hanover Township, Adams County, Pennsylvania

TDD No. 9605-23
Contract No. 68-S5-3002

1.0 INTRODUCTION

On 17 May 1996, the Roy F. Weston, Inc. (WESTON®), Site Assessment Technical Assistance (SATA) Team was directed by the U.S. Environmental Protection Agency (EPA) Remedial Project Manager (RPM) Christopher Corbett to conduct a fish tissue sampling event at the Keystone Sanitation Landfill NPL Site located in Hanover Township, Adams County, Pennsylvania.

2.0 BACKGROUND

2.1 Site Description

The Site is an inactive landfill owned by the Keystone Sanitation Company and is located on Clouser Road, Hanover Township, Adams County, Pennsylvania (see Figure 1-Site Location Map). The landfill operated from 1966 to 1990 and was permitted by the Pennsylvania Department of Environmental Protection (PADEP) to receive household and municipal wastes, and certain types of industrial and construction debris. The landfill was constructed without a liner (SATA, 1995).

The Keystone Sanitation Landfill Site was placed on the National Priorities List (NPL) in July 1987. EPA issued a Record of Decision (ROD) on 30 September 1990 (SATA, 1995). The ROD established the Site remedial design that is to be completed in two phases, Operable Unit #1 (OU1) and Operable Unit #2 (OU2). OU1 included the capping of the landfill area gas collection system, and the installation of a pump and treat system. Currently, the OU1 R.D. is 90% complete. OU2 required an off-site contaminant migration investigation.

Continuing operations at OU2, the EPA Region III Alternative Remedial Contracts Strategy (ARCS) contractor, Halliburton NUS Corporation, sampled monitoring and residential wells in January and in the fall of 1995. Elevated lead concentrations were detected in three of the residential wells sampled.

3.0 SITE ACTIVITIES

On 4 and 5 June 1996, an EPA sampling activity, led by RPM Christopher Corbett, was initiated to collect fish tissue samples and information to supplement an Ecological Risk Assessment performed by Halliburton NUS. The following representatives were present on site: EPA RPM Christopher Corbett; EPA Public Relations representative, Larry Brown; Maryland Department of Environment (MDE), Michelle Mosco-Lascuola and James Gravette; SATA members [redacted], and [redacted]; WESTON employee, [redacted]; and Baltimore Sun reporter, Donna Engels.

3.1 Meteorological Conditions

The ambient meteorological conditions during the 4 and 5 June 1996 sampling event are summarized below:

Table 1
Meteorological Conditions

Temperature	75°F
Winds	15 mph
Conditions	sunny and clear
Humidity	40%

3.3 Sampling Activities

SATA collected 45 fish tissue samples during the 4 and 5 June 1996 sampling event. Nineteen of the 45 samples were analyzed as whole fish samples and 26 of the 45 samples were analyzed as fillet samples for mercury. The Llaing, I, and Bloom, N, Determination of Total Mercury by Single Stage Gold Amalgamation with Cold Vapor Atomic Spectrometric Detection provided in the *Journal of Analytical Atomic Spectrometry*, 1993, was used. Sampling locations are identified in Figure 2-Sampling Location Map. All samples were handled and packaged in accordance with the sampling plan and were shipped via Federal Express to Brooks Rand Limited in Seattle, Washington for analysis.

3.3.1 Pond 1

On 4 June 1996, SATA members sampled Pond 1 using a seine and an electroshocker to obtain composite samples of a predator species and a bottom-dwelling species. The objective, as stated in the sampling plan, was to analyze fish tissue samples to investigate the possibility of mercury bioaccumulation in the fish. Due to the absence of a bottom-dwelling fish species, *Lepomis macrochirus* (bluegill) and *Micropterus salmoides* (largemouth bass), the only two species of fishes in the pond, were retrieved for the samples. Since bass are consumed by humans and bass prey on bluegill, mercury bioaccumulation can be observed. Therefore, the absence of the bottom-dwelling species will not affect the objective as stated in the sampling plan. A total of 15 bluegills and 8 bass were collected. The following table represents each of the four composite samples collected at Pond 1

Table 2
Composite Samples Collected from Pond 1

Sample Identification							
BrP1(W)-Bluegill		BrP2(F)-Bluegill		BrP3(F)-Bass		BrP4(W)-Bass	
Length	Weight	Length	Weight	Length	Weight	Length	Weight
191 mm	139.1 g	190 mm	117.4 g	415 mm	650.0 g	245 mm	162.5 g
170 mm	94.1 g	204 mm	138.0 g	307 mm	311.7 g	240 mm	153.9 g
176 mm	104.7 g	197 mm	134.1 g	230 mm	134.7 g	242 mm	145.3 g
181 mm	98.9 g	188 mm	127.5 g	433 mm	780.0 g	252 mm	173.3 g
196 mm	124.1 g	192 mm	143.2 g	---	---	---	---
---	---	195 mm	124.8 g	---	---	---	---
---	---	201 mm	126.6 g	---	---	---	---
---	---	192 mm	123.9 g	---	---	---	---
---	---	194 mm	126.0 g	---	---	---	---
---	---	192 mm	112.1 g	---	---	---	---

W Whole Fish Sample
F Fillet Fish Sample
mm millimeters
g grams

Each fish had its spines clipped and were individually wrapped in aluminum foil. Samples were placed in water-tight plastic bags and stored on dry ice. Observations made by SATA at Pond 1 l are as follows:

- According to the topographic map, the pond is a tributary to Pine Creek.
- The pond is approximately 10% shaded.
- The watershed is 80% open and 100% hills.
- The area surrounding the pond consists of wood and grass.
- The bank consists of mud and grass.
- The pond is approximately 110 feet by 150 feet.
- The pond is green and full flow.
- There are no odors, oil sheens, or deposits.
- The bottom of the pond is 100% clay.
- Carex and Cyperex plants line the shore in addition to *Gomphii dae* (dragon flies).
- There is an algae bloom on the surface of the pond.
- Dissolved oxygen readings at the surface were 11.0 mg/L and at the bottom were 10.8 mg/L.
- The water temperature was 23°C, pH was 8.2, and the conductivity was 190µS.
- The center of the pond was 7 feet deep.

The numbers of each species of fish caught by the seine are listed in Table 3. The fish were not diseased or deformed and no parasites were located on their bodies. SATA could not obtain complete samples, both fillet and whole, for the bass, using the seine. Five bass were collected using the electroshocking sampling technique. Electroshocking entailed the use of pulsating electric currents to stun the fish long enough to collect them with a long handle dip net.

Table 3
Species Caught at Pond 1

First Seine		Second Seine		Third Seine		Fourth Seine		Fifth Seine	
Bass	1	Bass	1	Bass	0	Bass	0	Bass	1
Bluegill	53	Bluegill	75	Bluegill	15	Bluegill	69	Bluegill	200

3.3.2 Pond 2

On 5 June 1996, SATA members sampled Pond 2 using a seine to collect composite samples of bass and bluegill. Due to the absence of a bottom-dwelling species in Pond 2 l, the bluegill and bass were the sample species. The objective for the sampling event was not compromised using the same reasoning as previously noted in subsection 3.3.1. A total of 12 bluegills and 10 bass were collected. The following table represents each of the four composite samples collected at Pond 2

Table 4
Composite Samples Collected from Pond 2

Sample Identification							
RuP1(F)-Bluegill		RuP2(W)-Bluegill		RuP3(F)-Bass		RuP4(W)-Bass	
Length	Weight	Length	Weight	Length	Weight	Length	Weight
228 mm	251.3 g	220 mm	211.5 g	292 mm	293.1 g	244 mm	162.8 g
215 mm	190.5 g	214 mm	214.5 g	265 mm	214.9 g	247 mm	169.9 g
226 mm	215.4 g	219 mm	201.8 g	273 mm	241.0 g	220 mm	128.8 g
219 mm	225.9 g	216 mm	201.7 g	262 mm	210.5 g	200 mm	101.0 g
217 mm	209.6 g	215 mm	236.0 g	247 mm	176.5 g	230 mm	134.7 g
242 mm	275.9 g	---	---	---	---	---	---
223 mm	232.1 g	---	---	---	---	---	---

W Whole Fish Sample
F Fillet Fish Sample
mm millimeters
g grams

Each fish had its spines clipped and were individually wrapped in aluminum foil. Samples were placed in water-tight plastic bags and stored on dry ice. Observations made by SATA at Pond 2 are as follows:

- According to the topographic map, the pond is a tributary to Pine Creek.
- The pond is approximately 350 feet by 250 feet with green water.
- The bank of the pond is 30% shaded and is mostly mud and grass.
- The watershed is 80% open, and the area around the pond is 100% hills.
- The bottom of the pond is 50% clay and 50% silt.
- There are no odors, deposits, or oil sheens.
- The flood plain is 50% wood and 50% grass.
- The center of the pond is approximately 4.8 feet deep.
- Dissolved oxygen at the surface was 13.4 mg/L and at the bottom 14 mg/L.
- The water temperature was 22°C, pH was 7.1, and the conductivity was 150 µS.
- The bottom of the pond was covered with Potamegetan (pond weed).
- Sagittania and Carex plants were on the bank of the pond , in addition to dragon flies and damsel flies..
- Snails, bull frogs, and green frogs were found in the pond.

The number of each species of fish caught by the seine are listed in Table 5. One bluegill had a portion of one fin missing and some parasites, but the rest of the fish caught in the seine were not deformed or diseased.

Table 5
Species Caught at Pond 2

First seine		Second seine		Third seine		Fourth seine	
Bass	4	Bass	6	Bass	24	Bass	5
Bluegill	45	Bluegill	71	Bluegill	152	Bluegill	68

4.0 DATA SUMMARY

A summary of the results is listed in Table 6.

Table 6
Summary of Results

Sample Identifier	BTAG* Value (mg/kg)	RBC** Value (mg/kg)	FDA*** Action Level (mg/kg)	Concentration of mercury in sample (mg/kg)		Sample Type	Species
				Dry weight	Wet weight		
BrP1(W)	0.1	0.41	1.0	0.175	0.044	Whole	Bluegill
BrP2(F)	0.1	0.41	1.0	0.251	0.059	Fillet	Bluegill
BrP3(F)	0.1	0.41	1.0	1.370	0.285	Fillet	Bass
BrP4(W)	0.1	0.41	1.0	0.234	0.055	Whole	Bass
RuP1(F)	0.1	0.41	1.0	0.286	0.070	Fillet	Bluegill
RuP2(W)	0.1	0.41	1.0	0.233	0.053	Whole	Bluegill
RuP3(F)	0.1	0.41	1.0	0.502	0.107	Fillet	Bass
RuP4(W)	0.1	0.41	1.0	0.250	0.055	Whole	Bass

* Biological Technical Assistance Group

** Risk-Based Concentration Table, July-December 1995 by Roy L. Smith, PhD. Value represents the Risk-Based concentration of mercury (inorganic) in fish.

*** Food and Drug Administration

The RBC value can be compared to the concentration of mercury, dry weight. The bass fillet samples from Pond 1 and Pond 2 have exceeded the RBC value for fish indicating that there is a risk for human consumption of the bass from both ponds. The FDA Action Level can be compared to the concentration of mercury, wet weight. The FDA Action Level is not exceeded. The more conservative FDA Action Level of .5 mg/kg used for women of child-bearing years and children is also not exceeded. The BTAG value for considering an environmental risk is compared to the concentration of mercury, wet weight. The bass fillet samples from each pond exceeds this value. A threat to humans exist should the bass be consumed; an environmental threat may exist should an animal higher on the food chain consume the fish.

5.0 FUTURE ACTIONS/RECOMMENDATIONS

Future actions will be contingent on the RPM's direction. SATA recommends posting signs in the area informing people not to eat the fish. In reference to ecological or environmental risks, SATA recommends a more extensive biological assessment of the area if an assessment has not already been conducted. The assessment should include information on the animals in the area that could be affected by consuming contaminated fish. SATA further recommends contacting BTAG, EPA Toxicologists, Maryland Department of the Environment, and the Fish and Boat Commission for their individual recommendations.

6.0 PHOTOGRAPH LOG

SATA members photographed the 4 and 5 June sampling event. The photographs' numbers and descriptions are provided below. See Appendix A for photographs of the site along with dates.

Photograph	Description
1	SATA removing the spines from fish at Pond 1
2	SATA measuring the length of fish at Pond 1
3	SATA preparing to electroshock pond at Pond 1
4	SATA electroshocking Pond 1
5	SATA electroshocking Pond 1
6	Electroshock equipment
7	Pond 2
8	SATA calibrating monitoring equipment at Pond 2
9	SATA preparing the seine at Pond 2
10	SATA deploying the seine at Pond 2
11	SATA deploying the seine at Pond 2
12	SATA pulling in seine at Pond 2

7.0 REFERENCES

SATA (Site Assessment Technical Assistance). 1995. Keystone Sanitation Landfill Site Sampling Plan from October 1995 Sampling Event. Delran, NJ.

WESTON MANAGEMENT DESIGNERS/CONSULTANTS FEDERAL PROGRAMS DIVISION	Keystone Sanitation Landfill NPL Site Hanover, Adams Co., PA	TDD #: 9605-23 PCS #: 2428

Figure 1: Site Location Map

AR309477

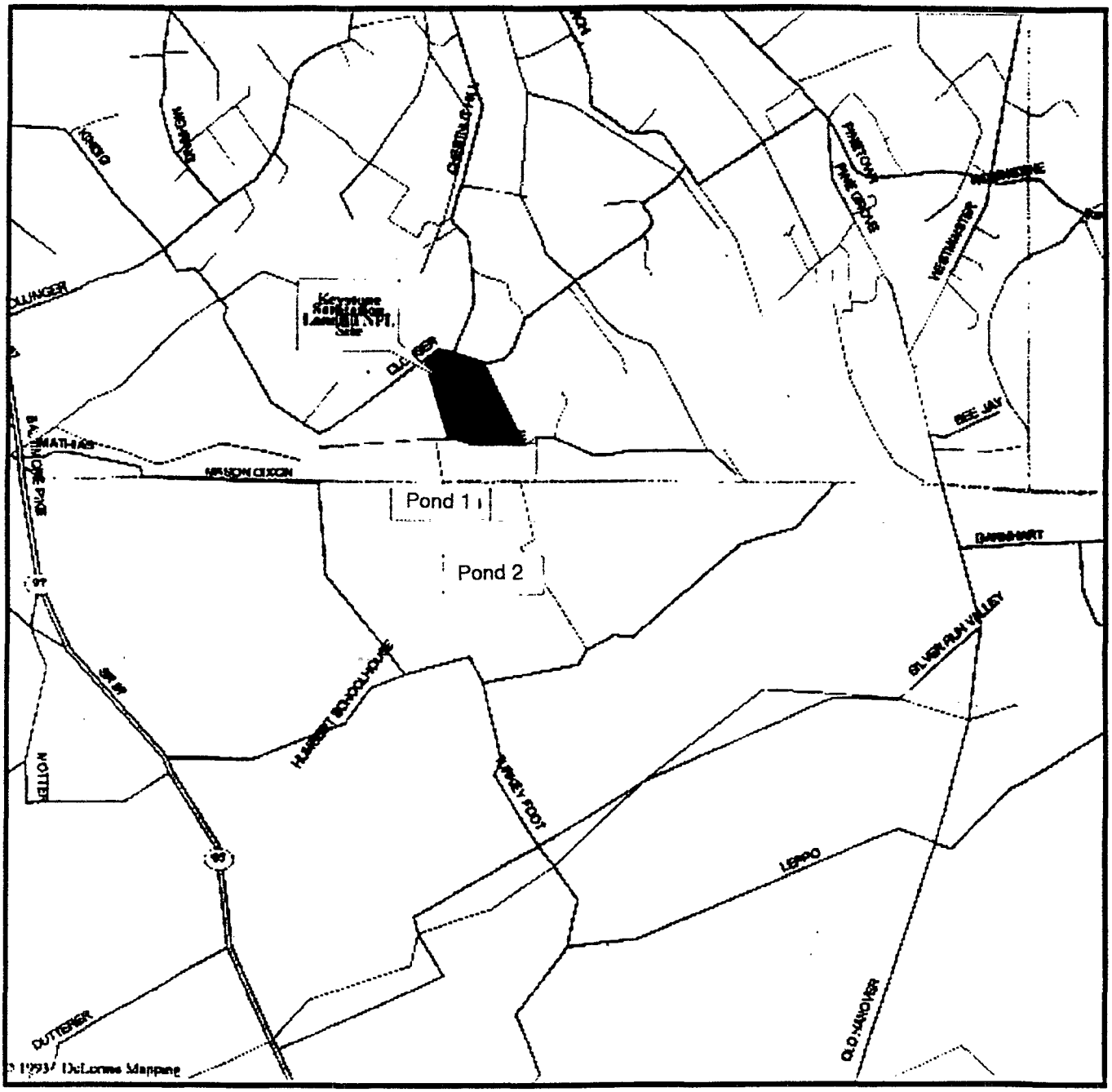
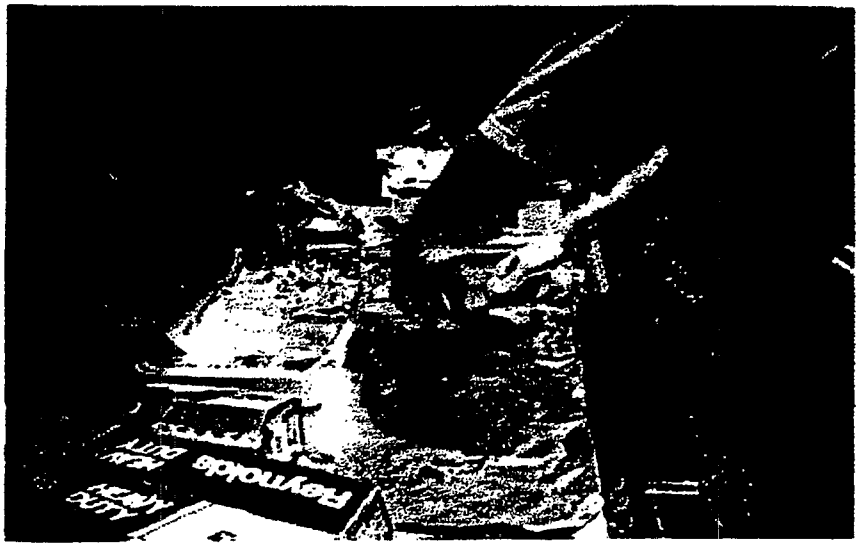


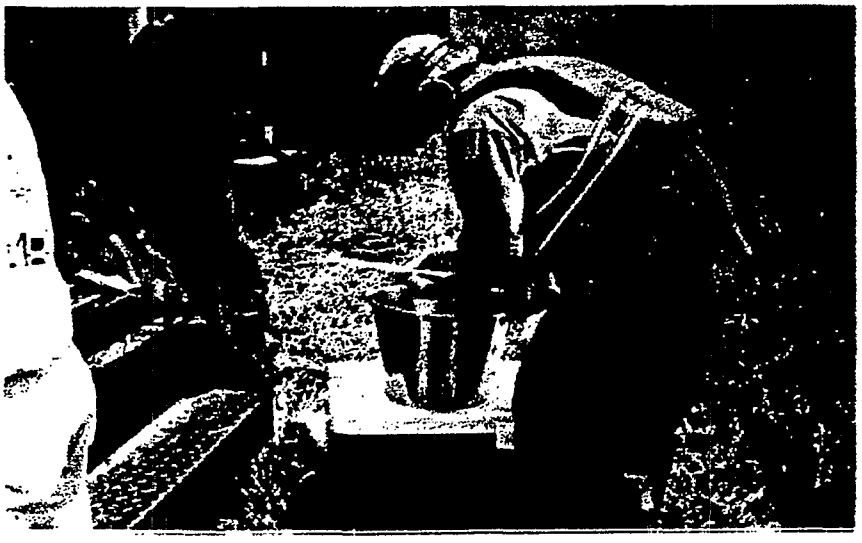
Figure 2: Sampling Location Map

Appendix A
Photograph Log

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania

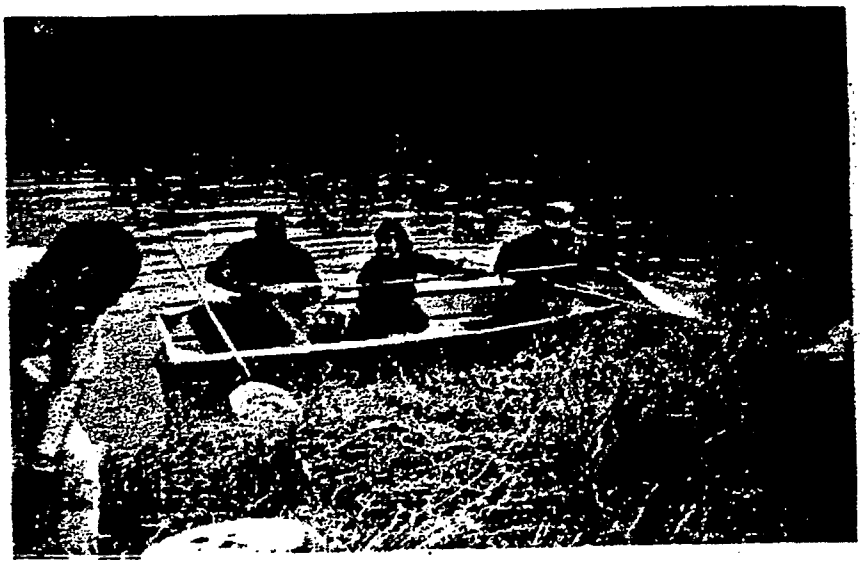


Date: 4 June 1996
Photo 1 - SATA removing the spines from fish at Pond 1

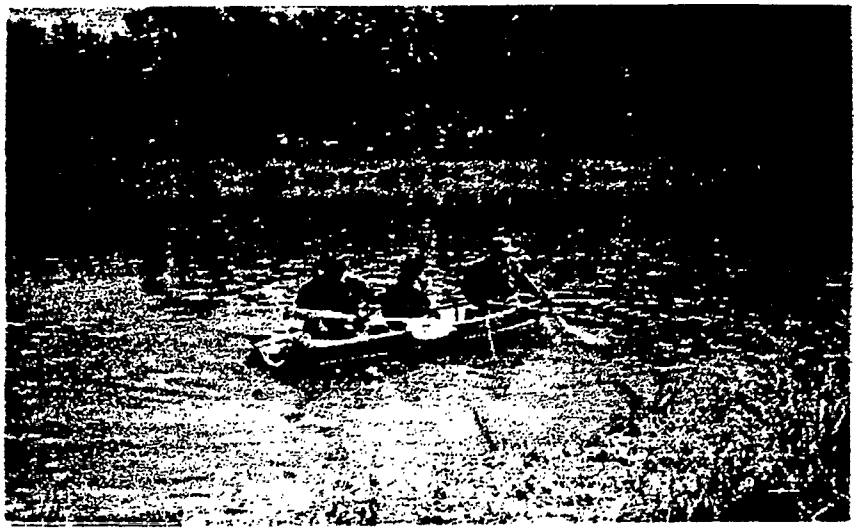


Date: 4 June 1996
Photo 2 - SATA measuring the length of fish at Pond 1

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania

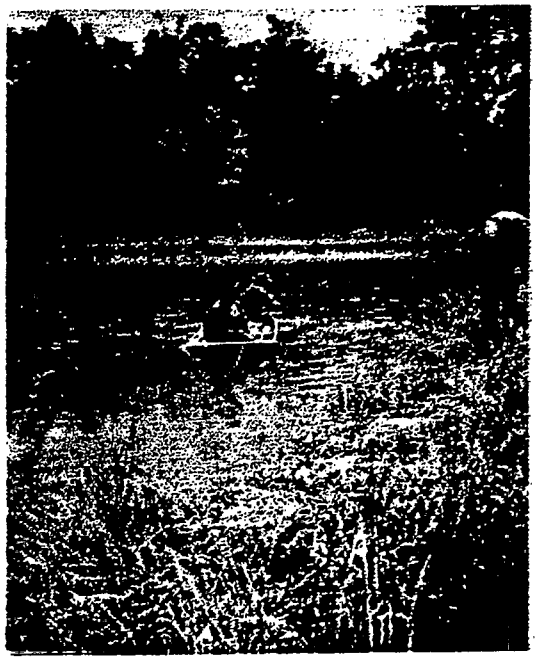


Date: 4 June 1996
Photo 3 - SATA preparing to electroshock pond at Pond 1

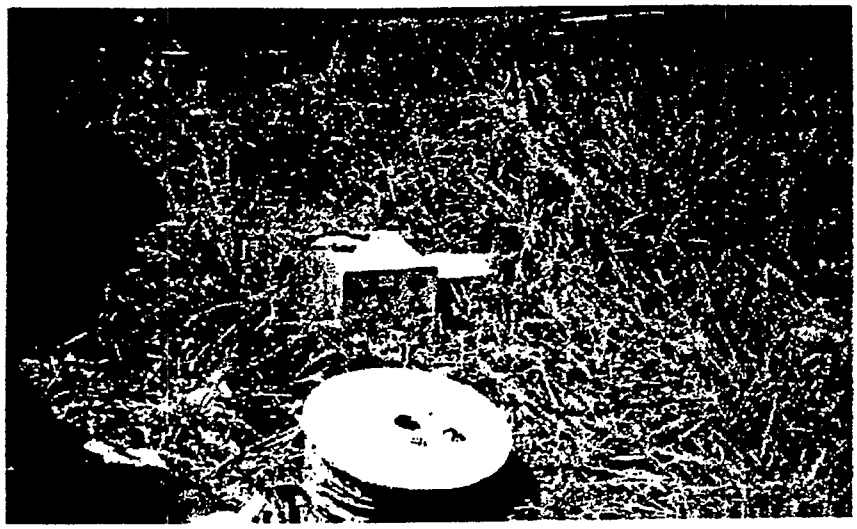


Date: 4 June 1996
Photo 4 - SATA electroshocking Pond 1

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania



Date: 4 June 1996
Photo 5 - SATA electroshocking Pond 1



Date: 4 June 1996
Photo 6 - Electroshock equipment

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania

Date: 5 June 1996
Photo 7 - Pond 2

Date: 5 June 1996
Photo 8 - SATA calibrating monitoring equipment at Pond 2

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania

Date: 5 June 1996
Photo 9 - SATA preparing the seine at Pond 2

Date: 5 June 1996
Photo 10 - SATA deploying seine at Pond 2

Photograph Log
Keystone Sanitation Landfill Site
Hanover, Adams County, Pennsylvania

Date: 5 June 1996
Photo 11 - SATA deploying seine at Pond 2

Date: 5 June 1996
Photo 12 - SATA pulling in seine at Pond 2

APPENDIX F, SECTION 2

TOXICOLOGICAL EVALUATION OF MERCURY LEVELS IN FISH TISSUE SAMPLES FROM THE KEYSTONE LANDFILL SITE PONDS MARYLAND DEPARTMENT OF THE ENVIRONMENT

MDE
MARYLAND DEPARTMENT OF THE ENVIRONMENT
2500 Broening Highway • Baltimore, Maryland 21224
(410) 631-3000

Parris N. Glendening
Governor

Jane T. Nishida
Secretary

October 21, 1996

Mr. Christopher Corbett
Remedial Project Manager
General Remedial Section
U.S. Environmental Protection Agency
Region III (3HW24)
841 Chestnut Building
Philadelphia PA 19107

RE: Keystone Landfill Site - Mercury in Fish Tissue Samples from Carroll County Ponds

Dear Mr. Corbett:

In reference to our phone conversation on September 12, 1996, the Maryland Department of the Environment's (MDE) Technical and Regulatory Services Administration (TARSA) has prepared a Toxicological Evaluation of the Mercury Levels in Fish Tissue Samples from the Pond 1 and Pond 2 Carroll, County, Maryland. TARSA has concluded that mercury levels in fish from the above referenced ponds do not pose unacceptable health risks to fish consumers.

Enclosed for your review is a copy of the TARSA report. If you have any questions concerning this matter, please contact me at (410) 631-3440.

Sincerely,

Michele Mosco-Lascuola

Michele Mosco-Lascuola
Remedial Project Manager
Federal/NPL Superfund Division

Enclosure

cc: Mr. Richard Collins
Mr. Robert DeMarco

DEPARTMENT OF THE ENVIRONMENT
Technical and Regulatory Services Administration
Environmental Risk Assessment Program

MEMORANDUM

TO: Robert DeMarco, Program Administrator
Environmental Restoration and Redevelopment Program

FROM: Deirdre Murphy, acting Program Administrator
Environmental Risk Assessment Program

DATE: October 8, 1996

SUBJECT: Keystone Landfill - Mercury in Fish from Carroll County Ponds

****

As you requested, Michael Sivak and I have evaluated mercury levels measured in bluegill and bass taken from ponds in Carroll County near Keystone Landfill (enclosure). The levels detected are similar to those observed in our Statewide monitoring program for these two species. We also performed quantitative risk calculations, assuming consumption of .44 lb/wk and 0.1 lb/wk for 50 weeks per year for adults and children, respectively. These consumption rates correspond to adults each consuming 58 average size bass or blue gill from the ponds per year (17 fish/yr for children), which appear to be high estimates for yearly consumption from small ponds.

The conclusion is that the mercury levels in fish from these ponds do not pose unacceptable health risks to fish consumers.

Please contact me or Michael Sivak (3906) with any questions regarding this evaluation.

/DLM

enclosure
cc: Michael Sivak, MDE
Michele Mosco-Lascuola, MDE
Gail Godfrey, ATSDR

Keystone Landfill

Mercury is a naturally occurring metal found in several forms. The most toxic to fish is methylmercury, which, due to its slow elimination, is readily bioaccumulated in fish tissue (1). Therefore, concentrations in fish tissue will increase with the age of the fish. Additionally, methylmercury is accumulated in fat tissue, and larger fish are consequently found to contain higher concentrations of mercury (1). MDE monitors mercury in fish tissue as part of a statewide monitoring program (2).

On June 6, 1996, samples of bass and bluegill were collected from Pond 1 and Pond 2 Pond. For these fish, both fillet and whole fish samples were analyzed for total mercury, which includes methylmercury, as well as inorganic mercury. A summary of the fish sampling and results is as follows:

Table 1. Mercury Concentrations in Fish from Carroll County Lakes

Species	# Fish/Sample	Total Weight, g	Fillet/Whole Fish	Mercury (Wet Weight), ppm
Bluegill	5	560.9	Whole	0.0441
Bluegill	10	1273.6	Fillet	0.05899
Bass	4	1876.4	Fillet	0.28496
Bass	4	635.0	Whole	0.05522
Bluegill	7	1600.7	Fillet	0.06978
Bluegill	5	1065.5	Whole	0.05312
Bass	5	1136	Fillet	0.10693
Bass	5	697.2	Whole	0.05325

The Food and Drug Administration (FDA) has set a limit of 1.0 ppm as the maximum level of methylmercury in the edible portion of seafood products (3). Comparison of these data to the FDA Action Level, which has been established based only on methylmercury, is a conservative approach because the total mercury in the fish tissue is compared to the methylmercury-based guideline. For the fish tissue collected at this site, no fillet sample exceeds this FDA Action Level.

The USEPA Region III Risk-Based Concentration (RBC) Table presents screening levels (4) for organic and inorganic forms of mercury in fish. The most sensitive or these screening levels is 0.14 mg/kg for methylmercury, which was derived assuming ingestion of 54 grams of fish per day (0.8 lb/week for 50 week per year). The USEPA Region III RBC Table is a conservative way to screen site-specific data to determine if further investigation into these data are necessary. For this use, whole fish data are not appropriate when evaluating a risk to human health through fish consumption because these data include parts of the fish which are not eaten (the head, tail, and bone). Fillet data are more appropriate in evaluating ingestion of fish tissue, as this is the portion of the fish which is actually eaten (5). Comparing fillet data to the RBC level of 0.14 mg/kg shows that three of the four results do not exceed this value. Only one bass sample, at 0.284 ppm, exceeds this conservative screen. This sample is a composite of four fish of individual weights of 650.0, 311.7, 134.7, and 780.0 grams. Three of the fish from this sample were the three largest bass collected for the study, which indicates they would have accumulated methylmercury at concentrations among the highest found. Quantification of risk through ingestion of contaminants in fish tissue is based on an average of concentrations from all samples, and not individual concentrations or a maximum concentration. The average concentration of mercury in the fish tissue of the two bass samples is 0.196 mg/kg, which also slightly exceeds the methylmercury RBC, indicating that further evaluation (which follows) is appropriate.

Maryland monitors contaminant levels in fish throughout the state (2). This program divides the state into three groups and collects resident species from each group every three years. These species include both bluegill and smallmouth bass and Table 2 presents concentrations of mercury (total) found in these fish. The table includes the number of samples collected, whether they were analyzed whole or fillet, and the minimum and maximum concentrations detected in the sample. For both species, all fillet and whole tissue concentrations are below the FDA action level of 1.0 ppm. Based on these data, the mercury concentrations found in Carroll County are shown to be typical of those mercury concentrations found in fish throughout Maryland.

Table 2. Mercury Concentrations/Maryland Fish Tissue Monitoring Program

Species	# Samples	Fillet/Whole Fish	Minimum Concentration (mg/kg)	Maximum Concentration (mg/kg)	Mean Mercury Concentration (mg/kg)
Bluegill	11	Whole Fish	0.011	0.176	0.077
Bluegill	1	Fillet	0.027	0.027	0.027
Bass	29	Whole Fish	0.007	0.141	0.068
Bass	21	Fillet	0.008	0.269	0.089

Using the average mercury concentrations found in fillet samples from the 2 ponds, quantitative risk calculations were performed for 3 types of fish consumers. An evaluation of the possible health risks associated with consumption of mercury in fish includes several populations. The three populations selected for this estimation of potential risk are adult male, adult female, and child (less than 9 years old). As a comprehensive study of consumption of fish from Maryland waters has not been performed, data from surveys of recreationally caught fish from angler studies were evaluated to obtain a high estimate of recreational fish consumption in Maryland. The intake assumptions (5, 7) used in the calculations for these three populations are as follows:

Adult Male:	70 kg body weight, 29.2 g of fish per day (0.45 lb per week, or approximately 58 average size bass or bluegill from the ponds per year), 350 days per year exposure frequency, 30 years exposure time;
Adult Female:	60 kg body weight, 29.2 g of fish per day (0.45 lb per week, or approximately 58 average site bass or bluegill from the ponds per year), 350 days per year exposure frequency, 30 years exposure time;
Child: (< 9 years old)	22 kg average body weight, 6.98 g of fish per day (0.1 lb per week, or approximately 17 average size bass or bluegill from the ponds per year), 350 days per year exposure frequency, 9 years exposure time.

Also, all populations were assumed to ingest only the fillet portion of the fish. For the quantitative assessment, mercury concentrations from both fillet samples for each species were averaged as the site concentration of mercury in fish tissue.

Table 3. Noncarcinogenic Risks of Mercury in Fish Tissue to Human Populations

	Average Daily Dose (mg/kg/day)	Reference Dose (mg/kg/day)	Hazard Index
BASS			
Child	6E-05	1E-04	0.60
Adult Male	8E-05	1E-04	0.78
Adult Female	9E-05	1E-04	0.92
BLUEGILL			
Child	2E-05	1E-04	0.20
Adult Male	3E-05	1E-04	0.26
Adult Female	3E-05	1E-04	0.30

The quantitative risk estimate is evaluated for systemic risks only. Mercury, including methylmercury, is not considered carcinogenic (4). USEPA Region III has recommended a safe dose, or exposure, for mercury. This safe dose, the oral reference dose (RfD) is an estimate of a daily exposure to the general public, including sensitive subpopulations, that is likely to be without an appreciable risk of adverse effects (5). The adverse effects of concern for chronic exposure to methylmercury are developmental and neurological (3, 6). For mercury, the RfD is 1E-04 mg/kg/day (4). The average daily dose for each population is summarized in Table 3. This intake is then compared to the RfD, and this ratio is the hazard index (HI). A HI value less than or equal to 1 is indicative of a safe exposure. The HI for all 3 populations ingesting either species is less than 1.

It is concluded that mercury in the fish from Pond 1 and Pond 2 does not pose an unacceptable risk to human consumers. The mercury levels in the sampled fish are similar to those observed in those species throughout Maryland.

References:

1. USEPA. 1984. Ambient Water Quality Criteria for Mercury. Office of Water, Regulations, and Standards. Criteria and Standards Division. EPA 440/5-84-026.

2. Maryland Department of the Environment. 1996. CORE Fish Tissue Collection Guide. Environmental Risk Assessment Program.

3. ATSDR. 1994. Toxicological Profile for Mercury (Update). Atlanta, Georgia.

4. USEPA, Region III. April, 1996. Risk-Based Concentration Table, January - June 1996.

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

6. USEPA. 1994. Guidance for Assessing Chemical Contaminant Data For Use In Fish Advisories Volume II Risk Assessment And Fish Consumption Limits. Office of Water. EPA 823-B-94-004.

7. USEPA. 1991. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure Factors: Interim Final. Office of Emergency and Remedial Response. OSWER Directive: 9285.6-03.

APPENDIX F, SECTION 3

REVIEW OF KEYSTONE MERCURY [FISH TISSUE] DATA
MEMORANDUM DATED OCTOBER 28, 1996
EPA REGION III PROJECT TOXICOLOGIST

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
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SUBJECT: Review of Keystone Mercury Data **DATE:** 10/28/96

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TO: Christopher Corbett, RPM
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The validated mercury results for fish samples from June of 1996 were reviewed. MDE's letter of October 21, 1996 was also consulted.

As MDE points out, the following issues affect interpretation of the data:

Wet weight results are generally more appropriate than dry weight when the population is not known to use unusual cooking techniques such as smoking and drying the fish.

Fillets are more appropriate than whole-body samples to assess exposure through human consumption.

Because results were reported as total mercury, the amount of methylmercury in the fish is unknown. However, if a conservative estimate is desired, 100% can be assumed when estimating risks. Mercury in fish tissue is likely to be in the methylated form.

Because mercury is not classified as a carcinogen, it is evaluated using the Hazard Index. Adverse effects are generally not expected below a Hazard Index of 1.

MDE's calculations are correct for the exposure assumptions they give, using the averages of fillets. Additional information about risks for various exposure scenarios is provided below.

If one uses the Superfund default factors (a 70-kilogram adult consuming 54 g/day, 350 days/year), the target level of methylmercury in fish for a Hazard Index of 1 would be 0.14 ppm. This is the RBC, as noted by MDE. An ingestion rate of 54 g/day for 350 days/year is equivalent to approximately 3/4 lb. fish per week.

If a 70-kilogram adult consumes the bass fillet with the maximum mercury concentration for 30 years, the adult should consume no more than 26 g/day, 350 days/year, for a methylmercury Hazard Index of 1. This is equivalent to

consuming 20 lbs./year of locally caught fish, or a little more than 1/3 lb. per week.

If a 15-kilogram child consumes the bass fillet with the maximum mercury concentration for 6 years, the child should consume no more than 5.5 g/day, 350 days/year, for a methylmercury Hazard Index of 1. This is equivalent to consuming approximately 4.2 lb/year, or approximately 1/3 lb. per month.

Sources of uncertainty in these estimations include the following:

Some of the mercury may be inorganic mercury, such that these risks may be biased high.

Concentrations of mercury in other fish may be higher or lower than those sampled to date. Direction of bias may be high or low.

Toxicity factors, such as the reference doses for mercury, may include sources of uncertainty due to interspecies and intraspecies variability, extrapolation of data from animals to humans, and use of high-dose, short-term studies to estimate low-dose, chronic effects. For this reason, reference doses include modifying and uncertainty factors to attempt a conservative bias.

People may react differently to mercury, and may be exposed to other sources of mercury. Individual weights and consumption rates vary. These are sources of individual variability.

The method of preparing the fish can affect the ultimate intake of mercury (e.g., skinning, frying, grilling, baking, etc.).

It is unknown whether the existing fish population could support the fishing and consumption rates cited herein.

If you have any questions concerning this review, please contact me at x3328.

cc: Eric Johnson (3HW41)

APPENDIX G

TOXICOLOGICAL PROFILES

APPENDIX G

SUMMARY OF TOXICOLOGICAL PROFILES FOR POTENTIAL CHEMICALS OF CONCERN KEYSTONE SANITATION LANDFILL

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G.1 ALDRIN/DIELDRIN (Clement 1985)

G.1.1 Pharmacokinetics

Both aldrin and dieldrin are carcinogens, causing increases in a variety of tumors in rats at low but not at high doses and producing a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation; while teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic; the oral LD_{50} is around 50 mg/kg, and the dermal LD_{50} is about 100 mg/kg.

G.1.1 Non-carcinogenic Toxicity

Chronic feeding with aldrin induced evidence of degeneration of the liver in rats. (EPA 1997). The EPA (1997) presented a verified chronic oral RfD of 0.03 ug/kg/day based on a LOAEL for liver effects in rats and an uncertainty factor of 1000. The principal target organ of aldrin is the liver.

G.1.2 Carcinogenicity

The EPA (1997) classifies aldrin in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient animal data. The human data consist of epidemiologic studies that had results which were statistically insignificant. Animal studies associated treatment with liver tumors in male and female mice.

G.2 ALUMINUM

G.2.1 Non-carcinogenic Toxicity

Aluminum is not generally regarded as an industrial poison. Inhalation of finely divided powders has been reported as a cause of pulmonary fibrosis. Aluminum in aerosols has been implicated in Alzheimers disease. EPA has not published an inhalation reference dose for aluminum, but the provisional RfD is based on developmental effects on the nervous system.

G.2.2 Carcinogenicity

Aluminum is not classified as a carcinogen by EPA.

G.3 ANTIMONY

G.3.1 Pharmacokinetics

Ingested antimony is absorbed slowly and incompletely from the gastrointestinal (GI) tract (Iffland 1988). Within a few days of acute exposure, highest tissue concentrations are found in the liver, kidney, and thyroid. Organs of storage include skin, bone, and teeth. Highest concentrations in deceased smelter workers (inhalation exposure) occurred in the lungs and skeleton. Excretion is largely via the urine or feces, although some is incorporated into the hair.

G.3.2 Noncancer Toxicity

Acute intoxication from ingestion of large doses of antimony induces GI disturbances, dehydration, and cardiac effects in humans (Iffland 1988). Chronic effects from occupational exposure include irritation of the respiratory tract, pneumoconiosis, pustular eruptions of the skin called "antimony spots," allergic contact dermatitis, and cardiac effects, including abnormalities of the electrocardiograph (ECG) and myocardial changes. Cardiac effects were also observed in rats and rabbits exposed by inhalation for six weeks and in animals (dogs, and possibly other species) treated by intravenous injection (Elinder and Friberg 1986a).

Chronic oral exposure studies in laboratory animals include two briefly reported lifetime drinking water studies in rats and mice (Kanisawa and Schroeder 1969; Schroeder et al. 1970).

G.3.3 Carcinogenicity

Data were not located regarding the carcinogenicity of antimony to humans. Antimony fed to rats did not produce an excess of tumors (Goyer 1991), but a high frequency of lung tumors was observed in rats exposed by inhalation to antimony trioxide for one year (Elinder and Friberg 1986). Antimony is classified in EPA cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1987a).

G.4 ARSENIC

G.4.1 Pharmacokinetics

Several studies confirm that soluble inorganic arsenic compounds and organic arsenic compounds are almost completely (>90 percent) absorbed from the GI tract in both animals and humans (Ishinishi et al. 1986). The absorption efficiency of insoluble inorganic arsenic compounds depends on particle size and stomach pH. Initial distribution of absorbed arsenic is to the liver, kidneys, and lungs, followed by redistribution to hair, nails, teeth, bone, and skin, which are considered tissues of accumulation. Arsenic has a longer half-life in the blood of rats, compared with other animals and humans, because of firm binding to the hemoglobin in erythrocytes.

Metabolism of inorganic arsenic includes reversible oxidation-reduction so that both arsenite (valence of 3) and arsenate (valence of 5) are present in the urine of animals treated with arsenic of either valence (Ishinishi et al. 1986). Arsenite is subsequently oxidized and methylated by a saturable mechanism to form mono- or dimethylarsenate; the latter is the predominant metabolite in the urine of animals or humans. Organic arsenic compounds (arsenilic acid, cacodylic acid) are not readily converted to inorganic arsenic. Excretion of organic or inorganic arsenic is largely via the urine, but considerable species variation exists. Continuously exposed humans appear to excrete 60 to 70 percent of their daily intake of arsenate or arsenite via the urine.

G.4.2 Non-carcinogenic Toxicity

A lethal dose of arsenic trioxide in humans is 70 to 180 mg (approximately 50 to 140 mg arsenic; Ishinishi et al. 1986). Acute oral exposure of humans to high doses of arsenic produce liver swelling, skin lesions, disturbed heart function, and neurological effects. The only non-carcinogenic effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyperpigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (Tseng 1977; Tseng et al. 1968; EPA 1995a). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico (Cebrian et al. 1983; Southwick et al. 1983). Occupational (predominantly inhalation) exposure is also associated with neurological deficits, anemia, and cardiovascular effects (Ishinishi et al. 1986), but concomitant exposure to other chemicals cannot be ruled out. The principal target organ for arsenic in humans appears to be the skin. The nervous system and cardiovascular systems appear to be less significant target organs. The skin and cardiovascular systems are considered the most sensitive target organs for evaluating chronic oral exposure. Inorganic arsenic may be an essential nutrient, exerting beneficial effects on growth, health, and feed conversion efficiency (Underwood 1977).

G.4.3 Carcinogenicity

Inorganic arsenic is clearly a carcinogen in humans. Inhalation exposure is associated with increased risk of lung cancer in persons employed as smelter workers, in arsenical pesticide applicators, and in a population residing near a pesticide manufacturing plant (EPA 1995a). Oral exposure to high levels in well water is associated with increased risk of skin cancer (Tseng 1977; EPA 1995a). Extensive animal testing with various forms of arsenic given by many routes of exposure to several species, however, has not demonstrated the carcinogenicity of arsenic (International Agency for Research on Cancer [IARC] 1980). The EPA (1995a) classifies inorganic arsenic in cancer weight-of-evidence Group A (human carcinogen), and recommends an oral unit risk of 0.00005 μg/L in drinking water, based on the incidence of skin cancer in the Tseng (1977) study. The EPA (1997) notes that the uncertainties associated with the oral unit risk are considerably less than those for most carcinogens, so that the unit risk might be reduced an order of magnitude. An inhalation unit risk of 0.0043 per $\mu g/m^3$ was derived for inorganic arsenic from the incidence of lung cancer in occupationally exposed men (EPA 1997).

G.5 BARIUM

G.5.1 Non-carcinogenic Toxicity

Barium is a naturally occurring alkaline earth metal that comprises approximately 0.04 percent of the earth's crust (Reeves 1986a). Acute oral toxicity was manifested by GI upset, altered cardiac performance, and transient hypertension, convulsions, and muscular paralysis. Repeated oral exposures were associated with hypertension. Occupational exposure to insoluble barium sulfate induced benign pneumoconiosis (ACGIH 1991). The EPA (1997) presented a verified chronic oral RfD of 0.07 mg/kg/day, based on an NOAEL of 0.21 mg/kg/day in a ten-week study in humans exposed to barium in drinking water and an uncertainty factor of 3. Barium is principally a muscle toxin. Its targets are the GI system, skeletal muscle, the cardiovascular system, and the fetus. The cardiovascular system is considered the most sensitive target organ for evaluating chronic oral exposure.

G.5.2 Carcinogenicity

The EPA (1995c) classifies barium as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity in humans). Cancer risk is not estimated for Group D substances.

G.6 BENZENE

G.6.1 Non-carcinogenic Toxicity

In humans, short-term inhalation exposure to benzene induced CNS effects such as drowsiness, dizziness, and headaches; long-term exposure induced anemia (ACGIH 1991). Oral dosing in animals induced hematopoietic effects (ATSDR 1995c). An inhalation RfD value of 0.002 mg/kg/day was derived (EPA 1995b) for benzene. The CNS and the hematopoietic system are the target organs of benzene.

G.6.2 Carcinogenicity

The EPA (1995a) classifies benzene in cancer weight-of-evidence Group A (human carcinogen) based on several studies of increased risk of non-lymphocytic leukemia associated with occupational exposure, supported by an increased incidence of neoplasia in rats and mice exposed by inhalation and gavage.

G.7 BERYLLIUM

G.7.1 Non-carcinogenic Toxicity

Beryllium has a low order of toxicity when ingested because it is poorly absorbed from the GI tract (Reeves 1986b). Occupational exposure was associated with dermatitis, acute pneumonitis, and chronic pulmonary granulomatosis (berylliosis). Berylliosis was also observed in humans living in the vicinity of a beryllium plant. Similar pulmonary effects were observed in laboratory animals subjected to inhalation exposure. The target organ for inhalation exposure appears to be the lung; a target organ is not identified for oral exposure.

G.7.2 Carcinogenicity

The EPA (1997) classifies beryllium in cancer weight-of-evidence Group B2 (probable human carcinogen) based on inadequate human (occupational) cancer data and sufficient animal data. A significant increase in lung tumors occurred in rats and in rhesus monkeys subjected to inhalation exposure or intratracheal instillation of a variety of beryllium compounds. Osteogenic sarcomas were induced in rabbits and mice, but not in rats or guinea pigs, injected intravenously with various beryllium compounds. Oral studies in animals yielded inconclusive results.

G.8 BIS(2-ETHYLHEXYL)PHTHALATE DI[2-ETHYLHEXYL]PHTHALATE)

G.8.1 Non-carcinogenic Toxicity

The acute oral toxicity of bis(2-ethylhexyl)phthalate is very low; oral $LD_{50/30}$ (lethal dose to 50 percent of population within 30 days without medical treatment) values in rats and mice were 33,800 and 26,300 mg/kg, respectively (ACGIH 1991). Repeated high-dose oral exposures were associated with decreased growth, altered organ weights, testicular degeneration, and developmental effects. The principal target organs for the toxicity of bis(2-ethylhexyl)phthalate are the liver and testis. The liver is considered the most sensitive target organ for chronic oral exposure.

G.8.2 Carcinogenicity

The EPA (1995a) classifies bis(2-ethylhexyl)phthalate in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human cancer data (one limited occupational study) and sufficient cancer data in laboratory animals.

G.9 BROMODICHLOROMETHANE

G.9.1 Non-carcinogenic Toxicity

Chronic gavage treatment with bromodichloromethane induced histopathologic evidence of degeneration of the liver and kidney in rats and mice, and hyperplastic lesions of the thyroid in the mice (EPA 1997). The principal target organs of bromodichloromethane are the liver and kidney; the thyroid may be a target in mice. The kidney is considered the most sensitive target organ for evaluating chronic oral exposure.

G.9.2 Carcinogenicity

The EPA (1997) classifies bromodichloromethane in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient animal data. The human data consist of epidemiologic studies that associate chlorination of drinking water with increased risk of several different types of cancer. Bromodichloromethane is one of several trihalogenated methanes formed from the interaction of chlorine with organic matter in water. Animal studies associated treatment with several different tumor types in rats and mice.

G.10 CARBON TETRACHLORIDE

G.10.1 Noncarcinogenicity

Carbon tetrachloride is a classic hepatotoxicant in humans and animals exposed by any route (ATSDR 1989a). High exposure levels also induced kidney effects in animals. Occupational exposure was associated with CNS and liver effects (ACGIH 1991). The principal target organs for the toxicity of carbon tetrachloride are the liver and the CNS. The liver is considered the most sensitive target organ for evaluating chronic oral or inhalation exposure. The kidney is also a target in animals exposed to high levels.

G.10.2 Carcinogenicity

Carbon tetrachloride is classified in cancer weight-of-evidence Group B2 (probable human carcinogen), based on increased incidence of liver tumors in rats, mice, and hamsters treated orally or by subcutaneous injection (EPA 1994). A verified oral slope factor of 0.13 per mg/kg/day was based on liver tumor data from gavage studies in all three species previously mentioned (EPA 1997).

G.11 CHLORDANE

Technical chlordane is a mixture of at least 50 related compounds (ATSDR 1992). The principal components of the mixture are cis- and trans-chlordane, heptachlor, cis- and trans-nonachlor, and alpha-, beta- and gamma-chlordane. Each component has its own environmental fate and transport kinetics.

G.11.1 Pharmacokinetics

Kinetic studies in rats, in which the area under the curve was compared following intravenous and oral dosing, indicate that approximately 80 percent of an oral dose of trans-chlordane is absorbed from the GI tract (Ohno et al. 1986). In animals, absorbed chlordane is distributed most rapidly to the liver and kidneys, probably because of the extensive vascularity of these organs (Ohno et al. 1986), followed by redistribution to adipose tissue (Barnett and Dorough 1974). In humans, levels of chlordane residues in adipose tissue increase with increasing duration of exposure (ATSDR 1992). Metabolism involves principally oxidation, dechlorination, and conjugation, yielding lipophilic products that accumulate in adipose tissue as well as more polar products that are excreted. Chlordane residues are excreted principally through the bile, although considerable species differences occur. Lactation is an important mechanism of excretion of chlordane residues retained in body fat.

G.11.2 Non-carcinogenic Toxicity

An acute oral lethal dose of chlordane in humans is estimated to be 25 to 50 mg/kg (ATSDR 1992). Symptoms of acute oral or inhalation intoxication in humans consistently include GI disturbances such as vomiting, cramps, and diarrhea, and neurological effects including headache, irritability, dizziness, incoordination, convulsions, and coma. Data were not located regarding symptoms or effects in humans chronically exposed by the oral route, and no noncancer effects were observed in several studies of occupationally exposed humans. Mild liver lesions were observed in chronic oral studies in rats and mice. Prenatal or early postnatal exposure of mice to chlordane damages the developing immune system and nervous system. Target organs of chlordane include the liver, nervous system, and the fetus and neonate. The liver is considered the most sensitive target organ for evaluating chronic oral exposure.

G.11.3 Carcinogenicity

The EPA (1995a) classifies chlordane in cancer weight-of-evidence Group B2, based on inadequate evidence in humans and sufficient evidence in animals. The human data consist of several epidemiologic studies of chlordane manufacturing workers and pesticide applicators. The only indication of a carcinogenic effect was a borderline significantly increased incidence of bladder cancer in one study of pesticide applicators, but chlordane exposure was not quantified and the workers were concomitantly exposed to other carcinogenic pesticides. The animal data consist of several studies in which oral exposure induced a dose-related increase in the incidence of liver tumors.

G.12 CHLOROETHANE

G.12.1 Non-carcinogenic Toxicity

Developmental inhalation studies with chloroethane displayed delayed fetal ossification in rats and mice (EPA [IRIS 1997]). The principal target organ of chloroethane is the bones.

G.12.2 Carcinogenicity

The EPA (1997) classifies chloroethane in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient animal data.

G.13 CHLOROFORM

G.13.1 Non-carcinogenic Toxicity

Oral or inhalation exposure of animals to chloroform was associated with liver and kidney damage (ACGIH 1991; EPA 1997). In humans, acute inhalation exposure to high levels induced narcosis, ventricular fibrillation, and death (ACGIH 1991). Limited occupational data associated chronic exposure to chloroform with CNS depression, digestive disturbances, and enlarged livers. Target organs for the toxicity of chloroform include the liver and kidney for oral and inhalation exposure, and the heart and CNS for inhalation exposure. The liver is considered the most sensitive target organ for evaluating chronic oral exposure and the cardiovascular system and CNS for evaluating chronic inhalation exposure.

G.13.2 Carcinogenicity

Chloroform is classified as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on increased incidence of several tumor types in rats and liver tumors in mice (EPA 1997). Human carcinogenicity data are inadequate.

G.14 CHLOROMETHANE

G.14.1 Noncancer Toxicity

Chloromethane is a natural and ubiquitous constituent of the oceans and atmosphere. It is a product of biomass combustion and is produced by wood rotting fungi. It has been detected in surface waters, drinking water, groundwater and soil. It is the dominant organochlorine species in the atmosphere. In water it is expected to volatilize rapidly with a half-life of $\geq$ 25 hours. In air it has a half life of about 1.5 years. In soil it is expected to volatilize from the surface but in a landfill will probably leach to groundwater.

Information regarding health effects of chloromethane in humans and animals is available only for the inhalation route of exposure. Before its use as a refrigerant declined about 30 or more years ago, many human deaths were reported as a result of exposure to chloromethane vapors from leaks from home refrigerators and industrial cooling and refrigeration systems. (ATSDR, 1989b).

G.14.2 Carcinogenicity

A retrospective epidemiology study of male workers exposed to chloromethane in a butyl rubber manufacturing plant produced no statistical evidence that the rates of deaths due to cancer at any sit were increased in the exposed population when compared with U. S. Mortality rates. (ATSDR, 1989b)

EPA classifies chloromethane as Group C, a possible human carcinogen, based on some evidence of carcinogenicity in animals.

G.15 CHROMIUM

G.15.1 Non-carcinogenic Toxicity

In nature, chromium (III) predominates over chromium (VI) (Langård and Norseth 1986). Little chromium (VI) exists in biological materials, except shortly after exposure, because reduction to chromium (III) occurs rapidly. Chromium (III) is considered a nutritionally essential trace element and is considerably less toxic than chromium (VI). However, chromium (VI) is more readily absorbed, and the chromium from chromium (VI) is more likely to bind to tissues (ATSDR, 1993). No effects were observed in rats consuming 1800 mg chromium (III)/kg/day in the diet for over two years (EPA 1995a).

Acute oral exposure of humans to high doses of chromium (VI) induced neurological effects, GI hemorrhage and fluid loss, and kidney and liver effects. Parenteral dosing of animals with chromium (VI) is selectively toxic to the kidney tubules.

Occupational (inhalation and dermal) exposure to chromium (III) compounds induced dermatitis (ACGIH 1991). Similar exposure to chromium (VI) induced ulcerative and allergic contact dermatitis, irritation of the upper respiratory tract including ulceration of the mucosa and perforation of the nasal septum, and possibly kidney effects.

A target organ was not identified for chromium (III). The kidney appears to be the principal target organ for repeated oral dosing with chromium (VI). Additional target organs for dermal and inhalation exposure include the skin and respiratory tract, respectively.

G.15.2 Carcinogenicity

Data were not located regarding the carcinogenicity of chromium (III). The EPA (1997) classifies chromium (VI) in cancer weight-of-evidence Group A (human carcinogen), based on the consistent observation of increased risk of lung cancer in occupational studies of workers in chromate production or the chrome pigment industry. Parenteral dosing of animals with chromium (VI) compounds consistently induced injection-site tumors. There is no evidence that oral exposure to chromium (VI) induces cancer. An inhalation unit risk of 0.012 per $\mu g/m^3$, equivalent to 42 per mg/kg/day (EPA 1997), assuming humans inhale 20 m^3/day and weigh 70 kg, was based on increased risk of lung cancer deaths in chromate production workers.

G.16 COBALT

G.16.1 Non-carcinogenic Toxicity

Acute high oral or parenteral doses of cobalt in humans or animals induced myocardial degeneration often leading to mortality, erythropoiesis, enlarged thyroid, and, in animals, renal tubular degeneration (Elinder and Friberg 1986b). Chronic ingestion from the consumption of beer containing high concentrations of cobalt was associated with "beer-drinkers cardiomyopathy," which includes polycythemia and goiter, as well as marked myocardial degeneration and mortality. The therapeutic use of 0.16 to 0.32 mg cobalt/kg/day in anemic, anephric dialysis patients for 12 to 32 weeks induced a significant, but reversible, rise in blood hemoglobin concentration (EPA 1992b).

Occupational (inhalation and dermal) exposure was associated with allergic dermatitis, chronic interstitial pneumonitis, reversibly impaired lung function, occupational asthma, and myocardial effects (ACGIH 1991). Cobalt was determined to be the etiologic factor in hard metal disease, the syndrome of respiratory symptoms, and pneumoconiosis associated with inhalation exposure to dusts containing tungsten carbide with cobalt powder as a binder (Elinder and Friberg 1986b). The lowest occupational air concentration of cobalt associated with hard metal disease was 0.003 mg cobalt/m^3 (Sprince et al. 1988). It should be noted that the workers were also exposed to tungsten and sometimes to titanium, tantalum, and niobium (Elinder and Friberg 1986b). Similar lung effects were seen in animals exposed to cobalt by inhalation.

The developmental toxicity of cobalt was tested in rodents treated orally with cobalt chloride (EPA 1992b). Maternal effects (unspecified) were reported in rats treated with 5.4 to 21.8 mg cobalt/kg/day from gestation day 14 through lactation day 21. Effects on the offspring included stunted growth at 5.4 mg cobalt/kg/day and reduced survival at 21.8 mg cobalt/kg/day. In rats treated with 6.2, 12.4, or 24.8 mg cobalt/kg/day on gestation days 6 through 15, maternal effects included reduced food consumption and body weight gain and altered hematologic parameters, although it is unclear at what dose level(s) these effects occurred. There were no effects on fetal survival, although a nonsignificant increase in fetal stunting was observed in rats treated with ≥ 12.4 mg cobalt/kg/day. Mice treated with 81.7 mg cobalt/kg/day had reduced maternal weight gain, but no fetal effects.

Cobalt is nutritionally essential as a cofactor in cyanocobalamin (vitamin B12) (EPA 1992b). Cobalt is universally present in the diet. Average daily adult dietary intakes of cobalt range from 0.16 to 0.58 mg/day (0.002 to 0.008 mg/kg/day, assuming adults weigh 70 kg) (Tipton et al. 1966; Schroeder et al. 1967). In 9- to 12-year-old children, dietary intakes of cobalt range from 0.3 to 1.77 mg/day (Murthy et al. 1971; National

Research Council 1989). Assuming an average weight for children in this age range of 28 kg (National Research Council, 1989), the dietary intakes are equivalent to 0.01 to 0.06 mg/kg/day.

The EPA recommends a provisional oral reference dose for cobalt of 0.06 mg/kg/day based on the upper range of dietary intake for children (NCEA, 1992).

Important target organs in orally exposed humans are the blood, erythrocytes, skin, and thyroid. Target organs for occupational exposure are the skin, lungs, and heart. The blood, erythrocytes, skin, and thyroid are considered the most sensitive target organs for evaluating chronic oral exposure.

G.16.2 Carcinogenicity

Data regarding the carcinogenicity of cobalt were not located.

G.17 COPPER

G.17.1 Non-carcinogenic Toxicity

Copper is a nutritionally essential element that functions as a cofactor in several enzyme systems (Aaseth and Norseth 1986). Acute exposure to large oral doses of copper salts was associated with GI disturbances, hemolysis, and liver and kidney lesions. Chronic oral toxicity in humans has not been reported. Chronic oral exposure of animals was associated with an iron-deficiency type of anemia, hemolysis, and lesions in the liver and kidneys. Occupational exposure may induce metal fume fever, and, in cases of chronic exposure to high levels, hemolysis and anemia (ACGIH 1991). The target organs for copper are the GI tract, erythrocyte, liver, and kidney, and, for inhalation exposure, the lung. The GI tract is considered the most sensitive target organ for evaluating chronic oral exposure.

G.17.2 Carcinogenicity

Copper is classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1995a). Quantitative risk estimates are not derived for Group D chemicals.

G.18 DDT (4,4'-DICHLORODIPHENYL-TRICHLOROETHANE)

G.18.1 Pharmacokinetics

Dichlorodiphenyltrichloroethane (DDT) is readily absorbed when dissolved in oils, fats, or lipid solvents, but is poorly absorbed as dry powder or aqueous suspension. Once absorbed, DDT concentrates in adipose tissue. Storage in fat is protective because it decreases the amount of chemicals at the site of toxic action, the brain. At a constant rate of intake, concentrations in adipose tissue reach a steady state and remain relatively constant. When exposure ceases, DDT is slowly eliminated. The rate of elimination is estimated to be 1 percent of stored DDT excreted per day (Gartrell 1985).

After absorption in mammals, DDT degrades by dehydrochlorination to unsaturated DDE and by substitution of hydrogen for one chlorine atom yielding DDD. DDD is further metabolized through a series of intermediates yielding DDA. DDA is relatively water soluble and excreted primarily in the urine. Ingestion studies of DDT administered to volunteers demonstrated that within 24 hours, urinary DDA excretion increased detectably. Excretion of DDT as DDA appeared to be totally dependent on preferential reductive dechlorination of DDT to DDD (rather than DDE) and then to DDA (Clayton 1981).

G.18.2 Noncancer Toxicity

The CNS is an important target organ in humans acutely exposed to DDT. Symptoms include altered sensory perception, headache, nausea, disequilibrium, confusion, tremors, and convulsions (Hayes 1982; ATSDR 1989d). Tremors and hyperirritability were observed in chronically exposed animals (NCI 1978c; Rossi et al. 1977). The liver appears to be the other important target organ, at least in animals. Liver effects include enzyme induction, increased liver weight, increased serum levels of liver enzymes, hepatocellular hypertrophy, and necrosis (ATSDR 1989d).

Dermal exposure has been associated with no illness and usually no irritation. Subcutaneous injection of colloidal suspensions of DDT in saline up to 30 ppm caused no irritation. Studies of DDT-impregnated clothing have found it to cause no irritation (Hayes 1982). The earliest symptom of acute DDT poisoning is paresthesia of the mouth and lower part of the face. This is followed by paresthesia of same areas and of the tongue and then dizziness, and tremors of extremities, confusion, malaise, headache, fatigue, and delayed vomiting. Vomiting is probably of central origin and not due to local irritation. Convulsions occur only in severe poisoning. Onset may be as soon as 30 minutes after ingestion of a large dose or as late as six hours after smaller but still-toxic doses. Recovery from mild poisoning usually is essentially complete in 24 hours, but recovery from severe poisoning requires several days (Hayes 1982).

There is no documented evidence that dietary absorption of DDT, alone or in combination with insecticides of the aldrin-toxaphene group, has caused cancer in the general population. No evidence has been presented that DDT has caused cancer among the millions of individuals (almost entirely men) who have been handling or spraying DDT (as dust, solution, and suspension) in all parts of the world and under all possible climatic conditions.

DDT is a mixture of p,p'-DDT and related compounds. One of the more important of the DDT isomers is o,p'-DDT. These agents have prominent estrogenic effects that have been well-characterized in a number of assay systems (Johnson, et al. 1988). The estrogenicity of DDT has lead to the supposition that it may adversely affect reproductive outcome by causing birth defects, increasing pregnancy complications, or affecting fertility (RTC 1990).

G.18.3 Carcinogenicity

The EPA (1997) has classified DDT in cancer weight-of-evidence Group B 2 (probable human carcinogen) based on the observation of tumors (generally of the liver) in seven studies in various mouse strains and in three studies in rats.

G.19 DIBROMOCHLOROMETHANE

G.19.1 Non-carcinogenic Toxicity

A 13-week subchronic gavage treatment with dibromochloromethane induced histopathologic evidence of degeneration of the liver in rats (EPA 1997). The EPA (1997) presented a verified subchronic oral RfD of 0.02 mg/kg/day based on an NOAEL for liver effects in rats and an uncertainty factor of 1000.

No adequate data on the teratogenic or reproductive effects of trihalomethanes are available.

G.19.2 Carcinogenicity

The EPA (1997) classifies dibromochloromethane in cancer weight-of-evidence Group C (possible human carcinogen), based on inadequate human and animal data. The human data consist of epidemiologic studies that associate chlorination of drinking water with increased risk of several different types of cancer. Dibromochloromethane is one of several trihalogenated methanes formed from the interaction of chlorine with organic matter in water.

G.20 DICHLORODIFLUOROMETHANE

G.20.1 Noncancer Toxicity

Oral exposure to dichlorodifluoromethane induces a low order of toxicity. In a two-year study, 150 mg/kg/day decreased the rate of body weight gain in female rats; no effects were observed in rats receiving 15 mg/kg/day (Sherman 1974). The method of oral dosing (diet or gavage) was unclear. No clinical signs, organ weight effects, or histopathologic alterations were observed in rats treated with 430 mg/kg/day for 10 days or in dogs treated with 90 mg/kg/day for 90 days (Clayton 1967). The liver is considered the most sensitive target organ for evaluating chronic inhalation exposure.

G.20.2 Carcinogenicity

Data were not located in EPA (1997) regarding the carcinogenicity of dichlorodifluoromethane.

G.21 1,4-DICHLOROBENZENE (para-DICHLOROBENZENE)

G.21.1 Pharmacokinetics

No data are available to quantitatively evaluate the absorption of 1,4-dichlorobenzene. Absorption via oral administration is assumed to be 100 percent since this chemical is similar in structure to benzene and smaller chlorinated aliphatic hydrocarbons. Approximately 20 percent of the compound is absorbed following inhalational exposure. The dermal absorption of 1,4-dichlorobenzene has not been studied (ATSDR, 1991b).

Once absorbed, whether through inhalation or oral exposure, 1,4-dichlorobenzene is mainly deposited in fatty tissue and the liver and kidneys to a lower extent. The major urinary metabolite of 1,4-dichlorobenzene is 2,5-dichlorophenol. This metabolite is eliminated as conjugates of glucuronic and sulfuric acids.

G.21.2 Non-carcinogenic Toxicity

Studies indicate that the liver is the primary target organ associated with toxic effects for 1,4-dichlorobenzene. Malaise, nausea, anemia, proteinuria, hematuria, as well as liver effects, were observed in humans exposed to this chemical (ATSDR 1991b).

Oral LD_{50} values for male and female rats were identified as 3,900 and 3,800 mg/kg respectively (ATSDR 1991b). No human studies are available regarding toxic effects of 1,4-dichlorobenzene from oral and dermal exposure.

G.21.3 Carcinogenicity

The U.S. EPA (1997) has classified this compound in the cancer weight-of-evidence Group C (possible human carcinogen). This classification is based on several oral exposure studies which indicate that this chemical is carcinogenic in male rats.

G.22 1,1,-DICHLOROETHANE

G.22.1 Noncancer Toxicity

CNS depression was the critical effect of oral or inhalation exposure of animals to 1,1-dichloroethane (ACGIH 1991). Kidney damage was observed in cats, but not laboratory rodents, exposed by inhalation. Inhalation exposure of humans was associated with CNS depression and respiratory tract and ocular irritation. The EPA (1995c) presented a provisional chronic oral RfD of 0.1 mg/kg/day based on an NOEL in a 13-week intermittent exposure inhalation study in rats and an uncertainty factor of 1000. A provisional subchronic oral RfD of 1 mg/kg/day was based on the same NOEL and an uncertainty factor of 100. Target organs for the toxicity of 1,1-dichloroethane are the CNS and kidney for oral exposure, and the kidney, CNS, and respiratory and ocular mucosa for inhalation exposure. The kidney is considered the most sensitive target organ for evaluating chronic inhalation exposure.

G.22.2 Carcinogenicity

EPA classifies 1,1-dichloroethane as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on no human cancer data and limited evidence of carcinogenicity in animals (EPA 1997). The data were considered to be inadequate for quantitative cancer baseline risk assessment.

G.23 1,2-DICHLOROETHANE

G.23.1 Non-carcinogenic Toxicity

Oral or inhalation exposure of humans or laboratory animals to 1,2-dichloroethane induced liver and kidney effects (ACGIH 1991). Inhalation exposure also induced pulmonary congestion or edema, and, in humans, CNS depression. The most sensitive target organs for evaluating 1,2-dichloroethane toxicity are the liver, kidney, and GI tract for chronic inhalation exposure and the kidney, CNS, and metabolic effects/weight loss for chronic oral exposure.

G.23.2 Carcinogenicity

EPA classifies 1,2-dichloroethane as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on the induction of several tumor types in rats and mice treated by gavage, and on the induction of benign lung papillomas in mice after dermal application (EPA 1997).

G.24 1,1-DICHLOROETHENE

G.24.1 Non-carcinogenic Toxicity

Chronic oral exposure of laboratory animals to 1,1-dichloroethene induced liver effects (EPA 1997). In animals, inhalation exposure induced degenerative changes in the liver and kidneys (ATSDR 1989c). No health effects were observed in a limited study of 138 exposed workers (ACGIH 1986). The liver and kidneys are the target organs for exposure to 1,1-dichloroethene. The liver is considered the most sensitive target organ for evaluating chronic oral exposure.

G.24.2 Carcinogenicity

EPA classifies 1,1-dichloroethene as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on an inadequate occupational exposure cancer study, limited data in several animal studies, its mutagenicity and ability to alkylate deoxyribonucleic acid (DNA), and its structural similarity to vinyl chloride, a known human carcinogen (EPA 1997). The eighteen available animal studies (11 by inhalation exposure, 5 by oral exposure, and 1 each by dermal application and subcutaneous injection) were limited in sensitivity by various deficiencies in design. Credible evidence that 1,1-dichloroethene was a complete carcinogen was provided only by one 12-month inhalation study in mice, in which the incidence of kidney adenocarcinomas was significantly greater in the high-dose males than in the control males.

G.25 1,2-DICHLOROETHENE, TOTAL

G.25.1 Non-carcinogenic Toxicity

Repeated oral exposure of rats to cis-1,2-dichloroethene was associated with signs of anemia (decreased hematocrit and hemoglobin) (EPA 1995a). Inhalation exposure to isomeric mixtures of 1,2-dichloroethene induced narcosis, and mixed isomers of 1,2-dichloroethene were used as an anesthetic gas (ACGIH 1991). Target organs appear to be the blood and erythrocytes for evaluating chronic oral exposure.

The oral $LD_{50/30}$ for trans-1,2-dichloroethene in rats was 1275 mg/kg; death was preceded by CNS and respiratory depression (ACGIH 1991). Histopathologic examination revealed lesions in the lungs and heart. Prolonged oral administration induced clinicopathologic evidence of mild liver damage (EPA 1995a). The target organs for inhalation exposure to trans-1,2-dichloroethene are the CNS, heart, and lungs; the liver and blood appear to be the principal target of oral exposure. The most sensitive target organ for evaluating chronic oral exposure is the blood.

The oral reference dose for total 1,2-dichloroethene is 0.009 mg/kg/day (EPA 1995c). The liver is considered to be the most sensitive target organ for evaluating chronic oral exposure.

G.25.2 Carcinogenicity

The EPA (1997) classifies cis-1,2-dichloroethene as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans), based on an absence of human or animal cancer data. Quantitative estimates of cancer risk are not derived for Group D chemicals.

Data regarding the carcinogenicity of trans-1,2-dichloroethene were not located.

G.26 1,2-DICHLOROPROPANE

G.26.1 Noncarcinogenic Toxicity

A 13-week inhalation study with 1,2-dichloropropane induced hyperplasia of the nasal mucosa in rats (EPA 1997). The principal target organ is the nasal mucosa.

G.26.2 Carcinogenicity

The EPA (1995c) classifies 1,2-dichloropropane in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient animal data. There is no human data. Animal studies associated treatment with tumors in mice.

G.27 HEPTACHLOR/ HEPTACHLOR EPOXIDE (Clement 1985)

G.27.1 Pharmacokinetics

Heptachlor and heptachlor epoxide are liver carcinogens when administered orally to mice. Results from mutagenicity bioassays suggest that these compounds also may have genotoxic activity. Reproductive and teratogenic effects in rats include decreased litter size, shortened life span of suckling rats, and development of cataracts in offspring.

Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects due to heptachlor exposure. Although heptachlor and heptachlor epoxide are absorbed most readily through the gastrointestinal tract, inhalation and skin contact are also potential routes of exposure. Acute exposure by various routes can cause development of hepatic vein thrombi and can effect the central nervous system and cause death. Chronic exposure induces liver changes, affects hepatic microsomal enzyme activity, and causes increased mortality in offspring. The oral LD_{50} in the rat is 40 mg/kg for heptachlor and 47 mg/kg for heptachlor epoxide. The liver is considered to be the most sensitive target organ for evaluating chronic oral exposure.

Although there are reports of acute and chronic toxicity in humans, with symptoms including tremors, convulsions, kidney damage, respiratory collapse, and death, details of such episodes are not well documented. Heptachlor epoxide has been found in a high percentage of human adipose tissue samples, and also in human milk samples and biomagnification of heptachlor/heptachlor epoxide occurs. This compound also has been found in the tissues of stillborn infants, suggesting an ability to cross the placenta and bioaccumulate in the fetus.

G.27.2 Carcinogenicity

EPA classifies heptachlor and heptachlor epoxide as Group B2 (probable human) carcinogens based on sufficient evidence of cancer in animals but inadequate human data.

G.28 HEXACHLOROCYCLOHEXANES (HCH) (Clement, 1985)

G.28.1 Pharmacokinetics

The alpha, beta, and gamma isomers of hexachlorocyclohexane (HCH) have all been shown to cause liver tumors in rats and mice. HCH has not been thoroughly tested for genotoxic effects but does not appear to be mutagenic. The alpha, beta, and delta isomers have not been tested for their teratogenic or reproductive toxicological potential. Lindane (gamma-HCH) has been tested and was not teratogenic, but in two studies it decreased the number of live young produced. Lindane has been associated with the development of aplastic anemia in humans. The liver is considered to be the most sensitive target organ for evaluating noncarcinogenic toxicity from chronic oral exposure.

Alpha-HCH is classified as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on increased incidence of liver tumors in rats and mice (EPA 1997). Human carcinogenicity data are inadequate. An oral slope factor of 6.3 per mg/kg/day (EPA 1997) was derived from the incidence of liver tumors in rats and mice fed alpha-HCH.

Beta-HCH is classified as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on increased incidence of liver tumors in mice (EPA 1997). Human carcinogenicity data are inadequate.

G.29 IRON

G.29.1 Non-carcinogenic Toxicity

Iron is potentially toxic in all forms and by all routes of exposure. Inorganic iron is a poison by the intraperitoneal route. The inhalation of large amounts of iron dust may result in iron pneumoconiosis or arc welders lung. Chronic exposure to excess levels of iron (>50-100 mg Iron/day) can result in pathological deposition of iron in tissues. The target organs are the blood, pancreas, liver, and GI tract (Sax and Lewis 1989). The blood, liver, and GI tract are considered to be the most sensitive target organs for evaluating chronic oral exposure.

Iron compounds are of varying toxicity. Iron oxides are a potential risk in all industrial settings. In general, ferrous compounds are more toxic than ferric compounds. Acute exposure to excessive levels of ferrous compounds can cause liver and kidney damage, altered respiratory rates and convulsions (Sax and Lewis 1989). A provisional RfD of 0.3 mg/kg/day has been recommended iron by EPA, based on dietary intake (NCEA, 1996). No inhalation RfD has been found for iron.

G.29.2 Carcinogenicity

Some iron compounds are suspected human carcinogens. Iron dust is an experimental neoplastigen and an increased incidence of lung cancer has been associated with exposure to iron dust. Iron oxide is an experimental tumorigen and a suspected human carcinogen. (Sax and Lewis 1989). EPA has not published oral or inhalation slope factors for iron.

G.30 LEAD

G.30.1 Pharmacokinetics

Studies in humans indicate that an average of 10 percent of ingested lead is absorbed, but estimates as high as 40 percent were obtained in some individuals (Tsuchiya 1986). Nutritional factors have a profound effect on GI absorption efficiency. Children absorb ingested lead more efficiently than adults; absorption efficiencies up to 53 percent were recorded for children three months to eight years of age. Similar results were obtained for laboratory animals; absorption efficiencies of 5 to 10 percent were obtained for adults and ≥50 percent were obtained for young animals. The deposition rate of inhaled lead averages approximately 30 to 50 percent, depending on particle size, with as much as 60 percent deposition of very small particles (0.03 μm) near highways. All lead deposited in the lungs is eventually absorbed.

Approximately 95 percent of the lead in the blood is located in the erythrocytes (EPA 1991a). Lead in the plasma exchanges with several body compartments, including the internal organs, bone, and several excretory pathways. In humans, lead concentrations in bone increase with age (Tsuchiya 1986). About 90 percent of the body burden of lead is located in the skeleton. Neonatal blood concentrations are about 85 percent of maternal concentrations (EPA 1990c). Excretion of absorbed lead is principally through the urine, although GI secretion, biliary excretion, and loss through hair, nails, and sweat are also significant.

G.30.2 Non-carcinogenic Toxicity

The non-carcinogenic toxicity of lead to humans has been well characterized through decades of medical observation and scientific research (EPA 1995a). The principal effects of acute oral exposure are colic with diffuse paroxysmal abdominal pain (probably due to vagal irritation), anemia, and, in severe cases, acute encephalopathy, particularly in children (Tsuchiya 1986). The primary effects of long-term exposure are neurological and hematological. Limited occupational data indicate that long-term exposure to lead may induce kidney damage. The principal target organs of lead toxicity are the erythrocyte and the nervous system (CNS) for chronic oral exposure and the CNS for chronic inhalation exposure. Some of the effects on the blood, particularly changes in levels of certain blood enzymes, and subtle neurologic behavioral changes in children, appear to occur at levels so low as to be considered non-threshold effects.

EPA (1994b) presents no inhalation RfC for lead, but referred to the National Ambient Air Quality Standard (NAAQS) for lead. The NAAQSs are based solely on human health considerations and are designed to

protect the most sensitive subgroup of the human population. The NAAQS for lead is 1.5 $\mu g/m^3$, averaged quarterly (EPA 1994b).

The EPA (1991a, 1995a) determined that it is inappropriate to derive an RfD for oral exposure to lead for several reasons. First, the use of an RfD assumes that a threshold for toxicity exists, below which adverse effects are not expected to occur; however, the most sensitive effects of lead exposure, impaired neurologic behavioral development in children and altered blood enzyme levels associated with anemia, may occur at blood lead concentrations so low as to be considered practically non-threshold in nature. Second, RfD values are specific for the route of exposure for which they are derived. Lead, however, is ubiquitous, so that exposure occurs from virtually all media and by all pathways simultaneously, making it practically impossible to quantify the contribution to blood lead from any one route of exposure. Finally, the dose-response relationships common to many toxicants, and upon which derivation of an RfD is based, do not hold true for lead. This is because the fate of lead within the body depends, in part, on the amount and rate of previous exposures, the age of the recipient, and the rate of exposure. There is, however, a reasonably good correlation between blood lead concentration and effect. Therefore, blood lead concentration is the appropriate parameter on which to base the regulation of lead.

The EPA UBK lead model is an iterated set of equations that estimate blood lead concentration in children aged 0 to 7 years (EPA 1991a; 1991c). The biokinetic part of the model describes the movement of lead between the plasma and several body compartments and estimates the resultant blood lead concentration. The rate of the movement of lead between the plasma and each compartment is a function of the transition or residence time (i.e., the mean time for lead to leave the plasma and enter a given compartment, or the mean residence time for lead in that compartment). Compartments modeled include the erythrocytes, liver, kidneys, all the other soft tissue of the body, cortical bone, and trabecular bone. Excretory pathways and their rates are also modeled. These include the mean time for excretion from the plasma to the urine, from the liver to the bile, and from the other soft tissues to the hair, skin, sweat, etc. The model permits the user to adjust the transition and residence times.

EPA guidance establishes an interim soil cleanup level for lead of 400 parts per million (ppm) to be applied at Superfund sites. This value is considered by EPA to be protective for direct contact with lead-contaminated soils in residential settings. The guidance is to be followed when current or predicted land use is residential.

G.30.3 Carcinogenicity

EPA (1995a) classifies lead in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate evidence of cancer in humans and sufficient animal evidence. The human data consist of several epidemiologic occupational studies that yielded confusing results. All of the studies lacked quantitative exposure data and failed to control for smoking and concomitant exposure to other possibly carcinogenic metals. Rat and mouse bioassays showed statistically significant increases in renal tumors following dietary and subcutaneous exposure to several soluble lead salts. Various lead compounds were observed to induce chromosomal alterations in vivo and in vitro, sister chromatid exchange in exposed workers, and cell transformation in Syrian hamster embryo cells; to enhance simian adenovirus induction; and to alter molecular processes that regulate gene expression. EPA (1995a) declined to estimate risk for oral exposure to lead because many factors (e.g., age, general health, nutritional status, existing body burden and duration of exposure) influence the bioavailability of ingested lead, introducing a great deal of uncertainty into any estimate of risk.

G.31 MANGANESE

G.31.1 Non-carcinogenic Toxicity

Manganese is nutritionally required in humans for normal growth and health (EPA 1995a) Humans exposed to approximately 0.8 mg manganese/kg/day in drinking water exhibited lethargy, mental disturbances (1/16 committed suicide), and other neurologic effects. The elderly appeared to be more sensitive than children. Oral treatment of laboratory rodents induced biochemical changes in the brain, but rodents did not exhibit the neurological signs exhibited by humans. Occupational exposure to high concentrations in air induced a generally typical spectrum of neurological effects, and increased incidence of pneumonia (ACGIH 1986).

Exposure from environmental sources of manganese is evaluated using a modified RfD. Estimated dietary exposure (in this case, 5 mg/day), is subtracted from the intake corresponding to the dietary RfD (10 mg/day), and the RfD is adjusted and an uncertainty factor is then applied in accordance with IRIS (EPA 1997). The CNS and respiratory tract are target organs of inhalation exposure to manganese. The CNS is considered the most sensitive target organ for evaluationg chronic oral exposure.

G.31.2 Carcinogenicity

The EPA (1997) classifies manganese in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans). Quantitative cancer risk estimates are not derived for Group D chemicals.

G.32 MERCURY

Mercury occurs in three forms: elemental, organic, and inorganic. Although the toxicity of all forms is mediated by the mercury cation, the extent of absorption and pattern of distribution within the body, which determines the effects observed, depends on the form to which the organism is exposed (Goyer 1991). Bacterial activity in the environment converts inorganic mercury to methyl mercury (Berlin 1986a). It is likely that either inorganic mercury or methyl mercury may be taken up by plants and enter the food chain, and this discussion will focus on inorganic and methyl mercury. Exposure to elemental mercury, which is more likely to occur in an occupational setting, is not discussed herein.

G.32.1 Pharmacokinetics

The GI absorption of inorganic mercury salts is about 2 to 10 percent in humans, and slightly higher in experimental animals (Berlin 1986a; Goyer 1991). Inorganic mercury in the blood is roughly equally divided between the plasma and erythrocytes. Distribution is preferentially to the kidney, with somewhat lower concentrations found in the liver, and even lower levels found in the skin, spleen, testes, and brain (Berlin 1986a). Inorganic mercury is excreted principally through the feces and urine, with minor pathways including the secretions of exocrine glands and exhalation of elemental mercury vapor.

Methyl mercury is nearly completely (90 to 95 percent) absorbed from the GI tract (Berlin 1986a). The concentration of methyl mercury in the erythrocytes is about 10 times that in the plasma. Methyl mercury leaves the blood slowly, showing particular affinity for the brain, particularly in primates. In rats, 1 percent of the body burden of methyl mercury is found in the brain, but in humans, 10 percent of the body burden is found in the brain. Somewhat lower levels are found in the liver and kidney. During pregnancy, methyl mercury accumulates in the fetal brain, often at levels higher than in the maternal brain. Most tissues except the brain transform methyl mercury to inorganic mercury. Excretion of methyl mercury is principally via the bile, with a half-life of 70 days in humans not suffering from toxicity. Following exposure to methyl mercury, some of the mercury in the bile exists as methyl mercury and some as the inorganic form. The inorganic form is largely passed in the feces, but methyl mercury is subject to enterohepatic recirculation. Another important excretory pathway for methyl mercury is lactation.

G.32.2 Non-carcinogenic Toxicity

Target organs for inorganic or methyl mercury include the kidney, nervous system, fetus, and neonate. The immune system is considered the most sensitive target organ for evaluating chronic oral exposure to inorganic mercury. The CNS and the peripheral nervous system are considered the most sensitive target organs for evaluating chronic inhalation exposure. Acute oral exposure to high doses of inorganic mercury causes severe damage to the GI mucosa because of the corrosive nature of mercury salts, which may lead to bloody diarrhea, shock, circulatory collapse, and death (Berlin 1986a; Goyer 1991). Acute sublethal poisoning induces severe kidney damage. Chronic exposure induces an autoimmune glomerular disease and renal tubular injury.

Acute or chronic exposure to methyl mercury leads to neurologic dysfunction (Berlin 1986a; Goyer 1991). The region of the nervous system affected is species-dependent. Methyl mercury poisoning in rats induces peripheral nerve damage and kidney effects. In humans, the sensory cortex appears to be the most sensitive. The brain of the fetus and the neonate may be unusually sensitive to methyl mercury; retarded neurologic development was observed in prenatally exposed children whose mothers showed no clinical signs of poisoning.

G.32.3 Carcinogenicity

The EPA (1997) classifies inorganic mercury in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans), based on no data regarding cancer in humans, and inadequate animal and supporting data. In an intraperitoneal injection study with metallic mercury in rats, sarcomas developed only in those tissues in direct contact with the test material (Druckrey et al. 1957). A two-year dietary study in rats with mercuric acetate (inorganic mercury) yielded no evidence of carcinogenicity (Fitzhugh et al. 1950). In mice, however, dietary exposure to high doses of mercury chloride for up to 78 weeks induced renal adenomas and adenocarcinomas (Mitsumori et al. 1981). The EPA has not yet evaluated the carcinogenicity of organic mercury. No carcinogenic effect, however, was observed in a two-year feeding study with phenylmercuric acetate in rats (Fitzhugh et al. 1950).

G.33 METHYLENE CHLORIDE

G.33.1 Non-carcinogenic Toxicity

Occupational exposure to high concentrations of methylene chloride may induce liver damage (ACGIH 1986). Liver effects were induced in animals by inhalation or oral exposure (EPA 1997). The principal target organ for methylene chloride is the liver.

G.33.2 Carcinogenicity

Methylene chloride is classified in EPA cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient evidence of carcinogenicity in animals (EPA 1997). Animal inhalation studies showed increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, mammary tumors in rats of either sex, salivary gland sarcomas in male rats, and leukemia in female rats. Oral studies were inconclusive.

G. 34 NICKEL

G.34.1 Noncancer Toxicity

In a subchronic gavage study with nickel chloride in water, clinical signs of toxicity in rats included lethargy, ataxia, irregular breathing, reduced body temperature, salivation, and discolored extremities (EPA 1994). Inhalation exposure was associated with asthma and pulmonary fibrosis in welders using nickel alloys (ACGIH 1986). Lung effects were observed in laboratory animals exposed by inhalation. Metabolic effects are considered the most sensitive critical effects for evaluating chronic oral exposure. The lung is clearly the target organ for inhalation exposure.

G.34.2 Carcinogenicity

Occupational exposure to nickel was associated with increased risk of nasal, laryngeal and lung cancer (ATSDR 1995a). Inhalation exposure of rats to nickel subsulfide increased the incidence of lung tumors. The EPA (1997) presents a cancer weight-of-evidence Group A classification (human carcinogen) for nickel via the inhalation route.

G.35 NITRATE/NITRITE

G.35.1 Noncancer Toxicity

The oral toxicity of nitrate is mediated by its reduction to nitrite by the microflora of the GI tract (EPA 1994). Nitrite induces oxidation of hemoglobin to methemoglobin, which is incapable of transporting oxygen from the lungs to the tissues. Human toxicity is generally associated with high levels of nitrate or nitrite in drinking water. The EPA (1997) adopted the chronic oral RfD for nitrite nitrogen as sufficiently protective for subchronic inhalation as well. The target tissue for the toxicity of nitrate or nitrite is the erythrocyte.

G.35.2 Carcinogenicity

Data regarding the carcinogenicity of uncombined nitrate or nitrite were not located. Nitrite can combine with secondary amines in food or other nitrogenous compounds to form nitrosamines or other N-nitroso compounds, many of which are important animal carcinogens (Menzer 1991).

G.36 PENTACHLOROPHENOL

G.36.1 Noncancer Toxicity

Acute inhalation exposure to mists or dusts of pentachlorophenol was associated with vascular damage culminating in heart failure (ACGIH 1986). Survivors suffered from impaired autonomic function, circulation, and vision. Chronic oral exposure was associated with liver and kidney lesions (EPA 1997). Target organs for the toxicity of pentachlorophenol include the circulatory and nervous systems, and the liver and kidney. The kidney and liver are considered the most sensitive target organs for evaluating chronic oral exposure.

G.36.2 Carcinogenicity

The EPA (1997) classifies pentachlorophenol in cancer weight-of-evidence Group B2 (probable human carcinogen) on the basis of inadequate human data and sufficient animal data. The animal data consisted of dietary studies in mice that show an increased incidence of liver, adrenal and vascular tumors, and studies in rats that show no carcinogenic effect. The test material used in these studies was approximately 90 percent pure, and was contaminated with tri- and tetrachlorophenol, hexachlorobenzene, PCDDs, and PCDFs.

G.37 POLYCHLORINATED BIPHENYLS (PCBs)

G.37.1 Non-carcinogenic Toxicity

Epidemiologic studies of women in the United States associated oral PCB exposure with low birth weight or retarded musculoskeletal or neurobehavioral development of their infants (ATSDR 1991a). Oral studies in animals established the liver as the target organ in all species, and the thyroid as an additional target organ in the rat. Effects observed in monkeys included gastritis, anemia, chloracne-like dermatitis, and immunosuppression. Oral treatment of animals induced developmental effects, including retarded neurobehavioral and learning development in monkeys.

Occupational exposure to PCBs was associated with upper respiratory tract and ocular irritation, loss of appetite, liver enlargement, increased serum concentrations of liver enzymes, skin irritation, rashes and chloracne, and, in heavily exposed female workers, decreased birth weight of their infants (ATSDR 1991a). Concurrent exposure to other chemicals confounded the interpretation of the occupational exposure studies. Laboratory animals exposed by inhalation to Aroclor-1254 vapors exhibited moderate liver degeneration, decreased body weight gain and slight renal tubular degeneration. Neither subchronic nor chronic inhalation RfC values were available.

PCBs tend to act on the skin, liver, fetus, and neonate.

Specific information was not available for Aroclor 1248, but would be assumed to be similar to that of Aroclor 1254.

G.37.1.1 Non-carcinogenic Toxicity for Aroclor 1254:

Monkeys that ingested 0.005-0.08 mg/kg/day doses of Aroclor 1254 exhibited ocular exudate, prominence and inflammation of the Meibomian glands and distortion in nail bed formation. Similar changes have been documented in humans for accidental oral ingestion of PCBs. Immunological assessment showed that the monkeys had a significant increase in IgM and IgG antibodies in response to sheep erythrocytes after 23 months of exposure (EPA 1997). Laboratory animals exposed by inhalation to Aroclor 1254 vapors exhibited moderate liver degeneration, decreased body weight gain and slight kidney effects. Subchronic and chronic inhalation RfC values were not available for Aroclor 1254.

G.37.2 Carcinogenicity

The EPA (1997) classifies the PCBs as EPA cancer weight-of-evidence Group B2 substances (probable human carcinogens), based on inadequate data in humans and sufficient data in animals. The human data consist of several epidemiologic occupational and accidental oral exposure studies with serious limitations, including poorly quantified concentrations of PCBs and durations of exposure, and probable exposures to other potential carcinogens.

The animal data consist of several oral studies in rats and mice with various Aroclors, kanechlors, or clophens (commercial PCB mixtures manufactured in the United States, Japan and Germany, respectively) that reported increased incidence of liver tumors in both species (EPA 1995a).

The PCB slope factors for high risk and persistence are used for Aroclors 1242, 1254, and 1260 because of chlorination of these compounds.

G.38 SELENIUM

G.38.1 Noncancer Toxicity

Selenium is a nutritionally essential trace element that is an integral part of the enzyme glutathione peroxidase and other proteins (Högberg and Alexander 1986). The National Research Council (1989) recommended dietary allowances (RDAs) for humans range from 10 to 75 mg/day. Chronic ingestion of 5 mg/day (0.071 mg/kg/day, assuming humans weigh 70 kg) induced selenosis in humans, characterized by abnormal hair and nail formation (Högberg and Alexander 1986). Effects in domestic grazing animals exposed to high levels of selenium included emaciation, lameness, and loss of hair and hooves. Occupational exposure to selenium fume or various selenium compounds was associated with intense ocular and respiratory tract irritation, chemical pneumonia, skin rashes, garlic odor to the breath, metallic taste in the mouth, and various socio-psychological effects (ACGIH 1986). The principal target organs for oral exposure to selenium are the skin, including the nails and hair, and the blood, erythrocytes, CNS, and peripheral nervous system. Targets for inhalation or dermal exposure include the skin and mucous membranes of the eyes and respiratory tract, and possibly the CNS.

G.38.2 Carcinogenicity

An impressive body of data indicates that selenium exerts an anticarcinogenic effect (Högberg and Alexander 1986). In laboratory animals, selenium supplementation decreased the incidence of chemical-induced cancers. In humans, the incidence of lymphomas and cancers of the breast, digestive tract, and lung were lower in geographic areas with high soil selenium levels. Occupational data suggest that selenium may protect against lung cancer. Several animal tests with various deficiencies in design and conduct equivocally associated exposure to selenium with cancer induction. In a well controlled oral experiment, selenium sulfide was associated with an increase in the incidence of liver tumors in rats, and with liver and lung tumors in mice. On the basis of this study, EPA (1997) classified selenium sulfide a cancer weight-of-evidence Group B2 compound (probable human carcinogen), but declined to derive quantitative risk estimates. Selenium and other selenium compounds were classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1997). Quantitative risk estimates are not derived for Group D substances.

G.39 SILVER

G.39.1 Pharmacokinetics

The GI absorption of ingested silver in animals was estimated at £10 percent; however, absorption of 18 percent was estimated for one human subject given silver acetate (Fowler and Nordberg 1986). Highest tissue levels are located in the liver; lower levels are located in the lungs, brain, spleen, bone marrow, muscle, and skin (Fowler and Nordberg 1986; Goyer 1991). Excretion is virtually entirely through the bile. The excretion kinetics appear to be species- and organ-dependent. In humans, the apparent half-life for silver in the liver is approximately 50 days. Silver in skin also appeared to have a long half-life (not quantified).

G.39.2 Noncancer Toxicity

Silver compounds have been used in dentistry, medicinally in the treatment of burns, as a local disinfectant, and as a drinking water disinfectant (Fowler and Nordberg 1986). The classical syndrome of toxicity, called argyria, is a blue-gray to nearly black discoloration of areas of the skin or the viscera resulting from deposition of microscopic granules of silver compounds in the affected tissues. Argyria results from occupational (inhalation), parenteral, or oral exposure.

G.39.3 Carcinogenicity

The EPA (1997) classifies silver in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans). The human data consist of no evidence in the literature of cancer despite frequent medical use of silver compounds. The animal data are limited to studies of implanted silver foil or injected metallic silver that provided unconvincing indications of a carcinogenic response relevant to humans.

G.40 1,1,2,2-TETRACHLOROETHANE

G.40.1 Noncancer Toxicity

Chronic oral exposure of laboratory animals to 1,1,2,2-tetrachloroethane was associated with liver and kidney effects (ATSDR 1994). Acute occupational exposure to high levels was associated with CNS effects; prolonged exposure to more moderate levels was associated with GI disturbances and liver damage (ACGIH 1986). Inhalation exposure studies in animals confirm that 1,1,2,2-tetrachloroethane is highly hepatotoxic. Neither oral nor inhalation RfD or RfC values were located. The liver, kidney, and the GI tract are considered the most sensitive target organs for evaluation of chronic oral exposure.

G.40.2 Carcinogenicity

Oral treatment with 1,1,2,2-tetrachloroethane induced a highly significant dose-related increase in hepatocellular carcinomas in rats (ATSDR 1994). Occupational data regarding carcinogenicity in humans are inadequate. The EPA (1997) classifies 1,1,2,2-tetrachloroethane as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on liver tumors in mice.

G.41 TETRACHLOROETHENE (PCE)

G.41.1 Non-carcinogenic Toxicity

Occupational (inhalation and dermal) exposure to tetrachloroethene was associated with neurologic effects, beginning with incoordination and progressing to dizziness, headache, vertigo, and unconsciousness (ACGIH 1986). The CNS is the principal target organ for inhalation exposure and the liver is the principal target organ for oral exposure to tetrachloroethene.

G.41.2 Carcinogenicity

Inhalation exposure to tetrachloroethene induced mononuclear cell leukemia in rats, and inhalation or oral exposure induced hepatocellular carcinomas in mice (ATSDR 1995b). A 1985 EPA evaluation of PCE found some evidence of carcinogenicity; the carcinogenic status of this compound is under review.

G.42 THALLIUM, SOLUBLE SALTS

G.42.1 Non-carcinogenic Toxicity

Thallium is highly toxic; acute ingestion by humans or laboratory animals induced gastroenteritis, neurological dysfunction, and renal and liver damage (Kazantzis 1986). Chronic ingestion of more moderate doses characteristically caused alopecia. Thallium was used medicinally to induce alopecia in cases of ringworm of the scalp, sometimes with disastrous results. In industrial (inhalation, oral, dermal) exposure, neurologic signs preceded alopecia, suggesting that the nervous system is more sensitive than the hair follicle. The EPA (1993a) presented verified chronic oral RfD values for several thallium salts (thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, and thallium sulfate) based on increased incidence of alopecia and increased serum levels of liver enzymes indicative of hepatocellular damage in rats treated with thallium sulfate for 90 days. Evaluation of thallium was based on RfDs for those compounds. Target organs for thallium include the GI tract (acute exposure), nervous system, skin, kidney, and liver. The kidney and liver are considered the most sensitive target organs for evaluating chronic oral exposure.

G.42.2 Carcinogenicity

Several thallium compounds (thallium oxide, thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium sulfate) were classified as cancer weight-of-evidence Group D substances (not classifiable as to carcinogenicity to humans) (EPA 1994). No weight-of-evidence classification was located for thallium alone.

G.43 1,1,1-TRICHLOROETHANE

G.43.1 Non-carcinogenic Toxicity

The toxicity of oral exposure to 1,1,1-trichloroethane is low (ACGIH 1986). Chronic ingestion by laboratory animals reduced growth rate, but produced little pathology in internal organs (ATSDR 1990). Acute inhalation exposure of humans or animals to high levels induced death due to narcosis or cardiac sensitization (ACGIH 1986). Occupational exposure was not associated with systemic effects. Target organs for inhalation exposure to 1,1,1-trichloroethane are the CNS and heart. The CNS is considered the most sensitive target organ for evaluating chronic oral exposure.

G.43.2 Carcinogenicity

The EPA (1995a) classifies 1,1,1-trichloroethane as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans). There are no reported human cancer data, and animal studies (78-week gavage studies in rats and mice, and a 12-month inhalation study in rats) were inadequate to determine the carcinogenicity of 1,1,1-trichloroethane in animals. Quantitative cancer risk estimates are not derived for Group D compounds.

G.44 1,1,2-TRICHLOROETHANE (Clement 1985)

G.44.1 Non-carcinogenic Toxicity

1,1,2-Trichloroethane was not mutagenic when tested using the Ames assay. No information was found concerning the reproductive toxicity or teratogenicity of 1,1,2-trichloroethane. No chronic studies were found on the toxicity of 1,1,2-trichloroethane but single doses as low as 400 mg/kg caused liver and kidney damage in dogs. The oral LD_{50} value for 1,1,2-trichloroethane in rats is 835 mg/kg. The liver and blood are considered the most sensitive target organs for evaluating chronic oral exposure.

G.44.2 Carcinogenicity

1,1,2-Trichloroethane induced hepatocellular carcinomas and pheochromocytoma of the adrenal gland in mal and female mice but did not produce a significant increase in tumor incidence in male or female rats (NCI 1977). EPA classifies this chemical as a Group C (possible human) carcinogen, based on animal data.

G.45 TRICHLOROETHENE (TCE)

G.45.1 Non-carcinogenic Toxicity

Little is known about the toxicity of prolonged oral exposure to trichloroethene. Acute inhalation exposure to high levels induced anesthesia, tachypnea, and ventricular arrhythmias (ACGIH 1986). Occupational exposure was associated with headache, dizziness, lassitude, and other CNS effects. Prolonged inhalation exposure of animals affected the liver and kidneys. The EPA has published an oral RfD of 0.006 mg/kg/day (1995b) for trichloroethene. The principal target organs for trichloroethene are the CNS and heart, and, to a lesser extent, the liver and kidney. The CNS and liver are considered the most sensitive target organs for evaluating chronic oral toxicity.

G.45.2 Carcinogenicity

Carcinogenicity studies in laboratory animals showed increased incidence of hepatocellular carcinomas (gavage exposure) and malignant lymphomas (inhalation exposure) in mice and increased incidence of renal adenocarcinomas in male rats (gavage) (EPA 1988d). Cancer studies in humans were inadequate. Interpretation of the data regarding the carcinogenicity of trichloroethene is controversial, and the EPA (1992c) has not adopted a final position on a cancer weight-of-evidence classification or quantitative risk estimates for trichloroethene. For this reason, trichloroethene was removed from the IRIS and the 1992 HEAST (EPA 1992b). Currently, EPA believes the weight-of-evidence to be on the C-B2 continuum (possible-probable human carcinogen), and offers provisional slope factors of 0.011 per mg/kg/day for oral exposure and 0.006 per mg/kg/day (EPA 1995b) for inhalation exposure as being useful.

G.46 VINYL CHLORIDE

G.46.1 Non-carcinogenic Toxicity

Data were not located regarding oral exposure of humans to vinyl chloride (ATSDR 1995d). In rats, lifetime dietary ingestion of vinyl chloride slightly but significantly increased mortality and induced mild histopathologic effects in the liver. Several early occupational studies associated vinyl chloride exposure with a syndrome known as vinyl chloride disease, which includes acroosteolysis (dissolution of the ends of the distal phalanges of the hands), circulatory disturbances in the extremities, Raynaud syndrome (sudden, recurrent bilateral cyanosis of the digits), scleroderma, hematologic effects, effects on the lungs, and impaired liver function and liver damage. Mild neurologic effects were also associated with occupational exposure. Long-term inhalation studies in rats and mice identified elevated relative liver weight as a sensitive indicator of liver effects. Neither inhalation RfC values nor oral RfD values for vinyl chloride were located. The principal target organs for vinyl chloride appear to be the CNS and the liver.

G.46.2 Carcinogenicity

The EPA (1993a) lists vinyl chloride as an EPA cancer weight-of-evidence Group A compound (human carcinogen). Vinyl chloride has been associated with unusual liver tumors such as angiosarcomas (ATSDR 1993).

G.47 ZINC

G.47.1 Pharmacokinetics

Zinc is a nutritionally required trace element. Estimates of the efficiency of GI absorption of zinc in animals range from <10 to 90 percent (Elinder 1986c). Estimates in normal humans range from approximately 20 to 77 percent (Elinder 1986c; Goyer 1991). The net absorption of zinc appears to be homeostatically controlled, but it is unclear whether GI absorption, intestinal secretion, or both are regulated. Distribution of absorbed zinc is primarily to the liver (Goyer 1991), with subsequent redistribution to bone, muscle, and kidney (Elinder 1986c). Highest tissue concentrations are found in the prostate. Excretion appears to be principally through the feces, in part from biliary secretion, but the relative importance of fecal and urinary excretion is species-dependent. The half-life of zinc absorbed from the GI tracts of humans in normal zinc homeostasis is approximately 162 to 500 days.

G.47.2 Non-carcinogenic Toxicity

Humans exposed to high concentrations of aerosols of zinc compounds may experience severe pulmonary damage and death (Elinder 1986c). The usual occupational exposure is to freshly formed fumes of zinc, which can induce a reversible syndrome known as metal fume fever. Orally, zinc exhibits a low order of acute toxicity. Animals dosed with 100 times dietary requirement showed no evidence of toxicity (Goyer 1991). In humans, acute poisoning from foods or beverages prepared in galvanized containers is characterized by GI upset (Elinder 1986c). Chronic oral toxicity in animals is associated with poor growth, GI inflammation, arthritis, lameness, and a microcytic, hypochromic anemia (Elinder 1986c), possibly secondary to copper deficiency (Underwood 1977). The blood and erythrocytes are considered the most sensitive target organs for chronic oral exposure.

G.47.3 Carcinogenicity

The EPA (1995a) classifies zinc in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) based on inadequate evidence for carcinogenicity in humans and animals. The human data consist largely of occupational exposure studies not designed to detect a carcinogenic response, and of reports that prostatic zinc concentrations were lower in cancerous than in noncancerous tissue. The animal data consist of several dietary, drinking water, and zinc injection studies, none of which provided convincing data for a carcinogenic response.

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APPENDIX H

LEAD IEUBK MODEL PRINTOUTS

APPENDIX H, SECTION 1

HISTOGRAMS FOR RESIDENTIAL WELL NO. 1
IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 16.00 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

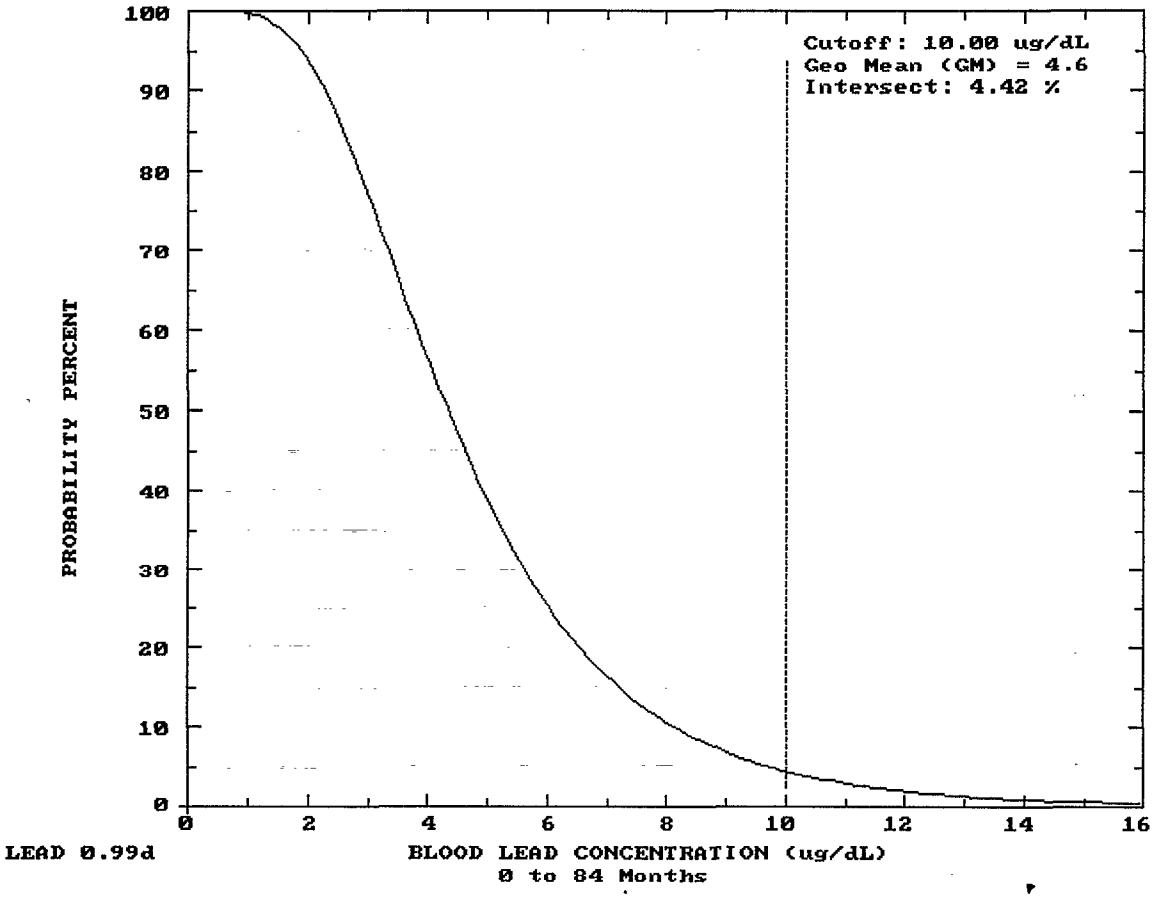
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

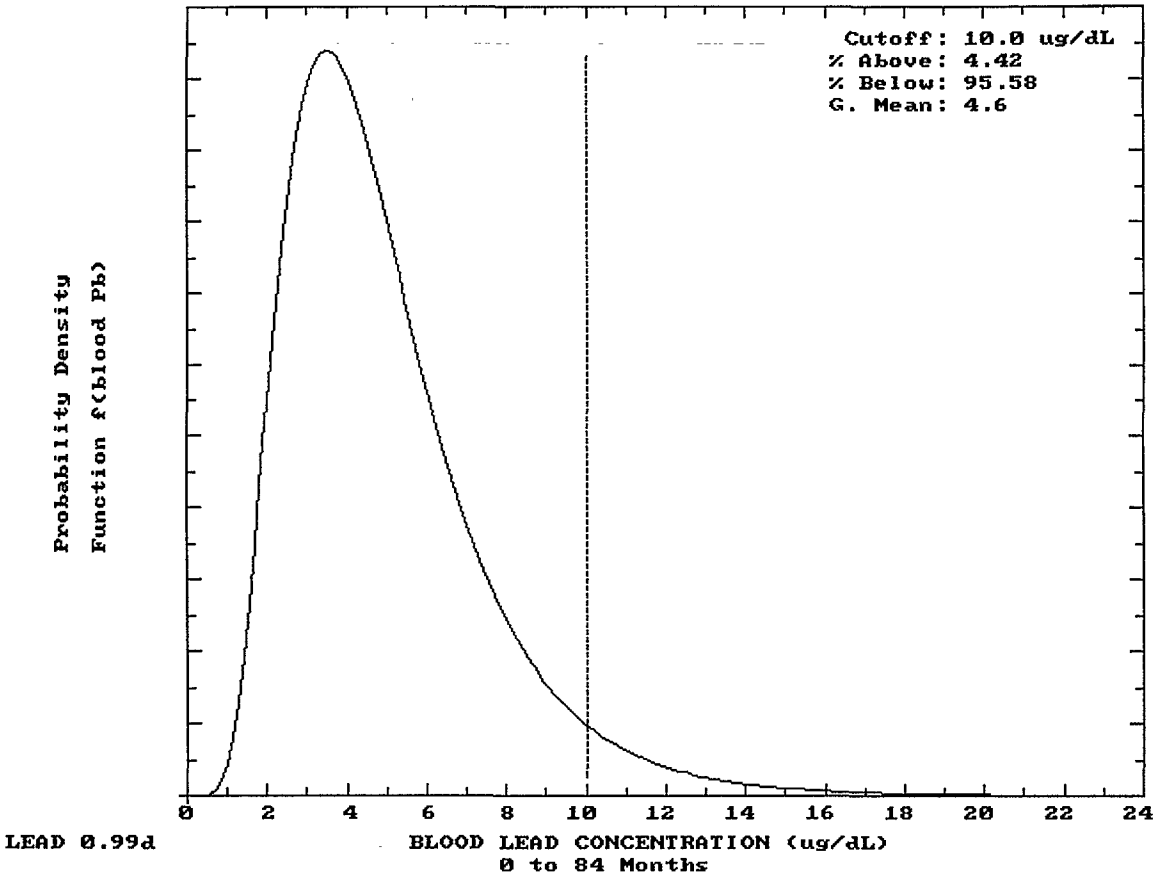
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.6	8.60	4.62
1-2:	5.5	13.35	7.20
2-3:	5.2	14.02	7.29
3-4:	5.0	14.19	7.40
4-5:	4.3	12.58	5.60
5-6:	3.9	12.53	5.08
6-7:	3.6	12.68	4.82

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)

0.5-1:	2.51	1.45	0.00	0.02
1-2:	2.57	3.55	0.00	0.03
2-3:	2.92	3.75	0.00	0.06
3-4:	2.85	3.87	0.00	0.07
4-5:	2.80	4.11	0.00	0.07
5-6:	2.98	4.37	0.00	0.09
6-7:	3.31	4.46	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 4.6
Intersect: 4.42 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
2
4
6
8
10
12
14
16
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 4.42
% Below: 95.58
G. Mean: 4.6
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
20
22
24
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months

APPENDIX H, SECTION 2

HISTOGRAMS FOR RESIDENTIAL WELL NO. 20
IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 18.80 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

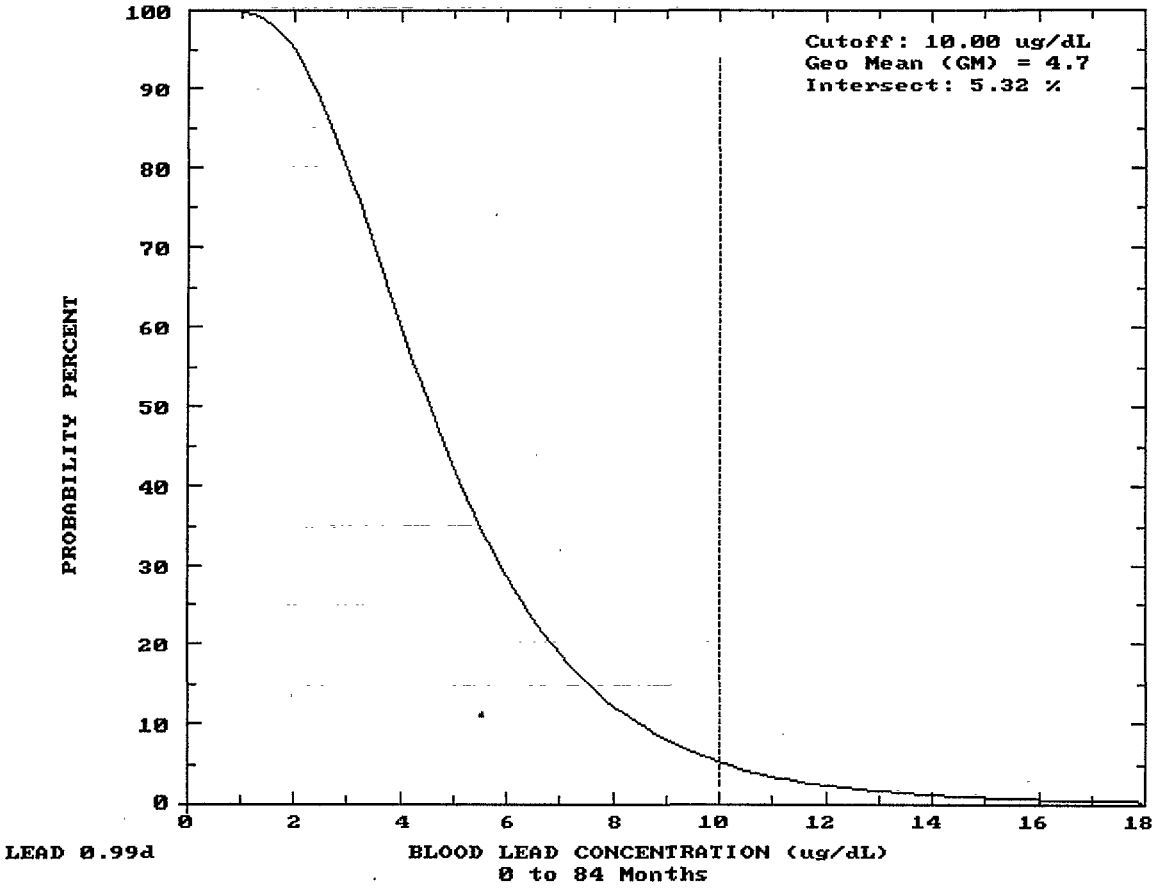
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

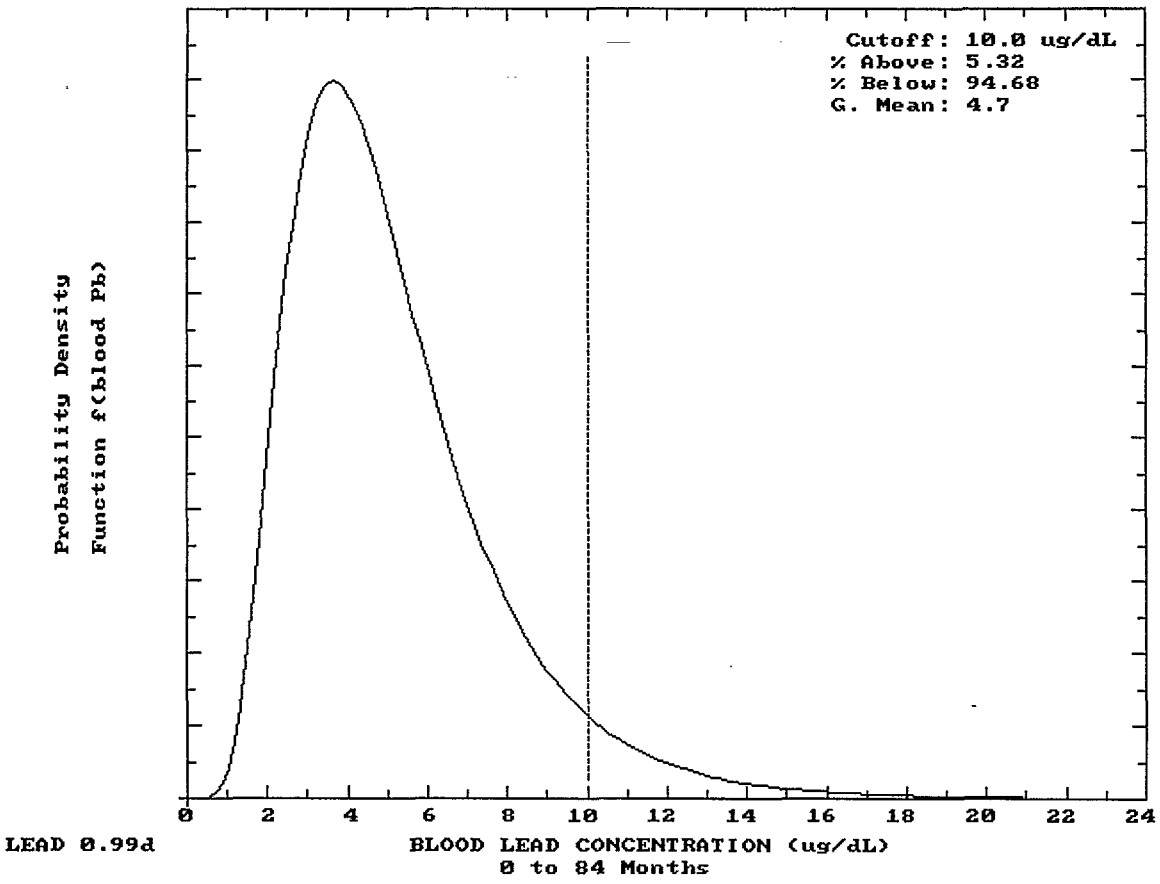
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.8	8.83	4.61
1-2:	5.7	13.91	7.16
2-3:	5.4	14.61	7.26
3-4:	5.2	14.81	7.37
4-5:	4.6	13.25	5.58
5-6:	4.1	13.24	5.06
6-7:	3.8	13.42	4.80

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)

0.5-1:	2.50	1.70	0.00	0.02
1-2:	2.56	4.16	0.00	0.03
2-3:	2.91	4.38	0.00	0.06
3-4:	2.84	4.53	0.00	0.07
4-5:	2.79	4.81	0.00	0.07
5-6:	2.97	5.11	0.00	0.09
6-7:	3.30	5.22	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 4.7
Intersect: 5.32 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 5.32
% Below: 94.68
G. Mean: 4.7
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
20
22
24
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months

APPENDIX H, SECTION 3

HISTOGRAMS FOR RESIDENTIAL WELL NO. 60
IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 17.00 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

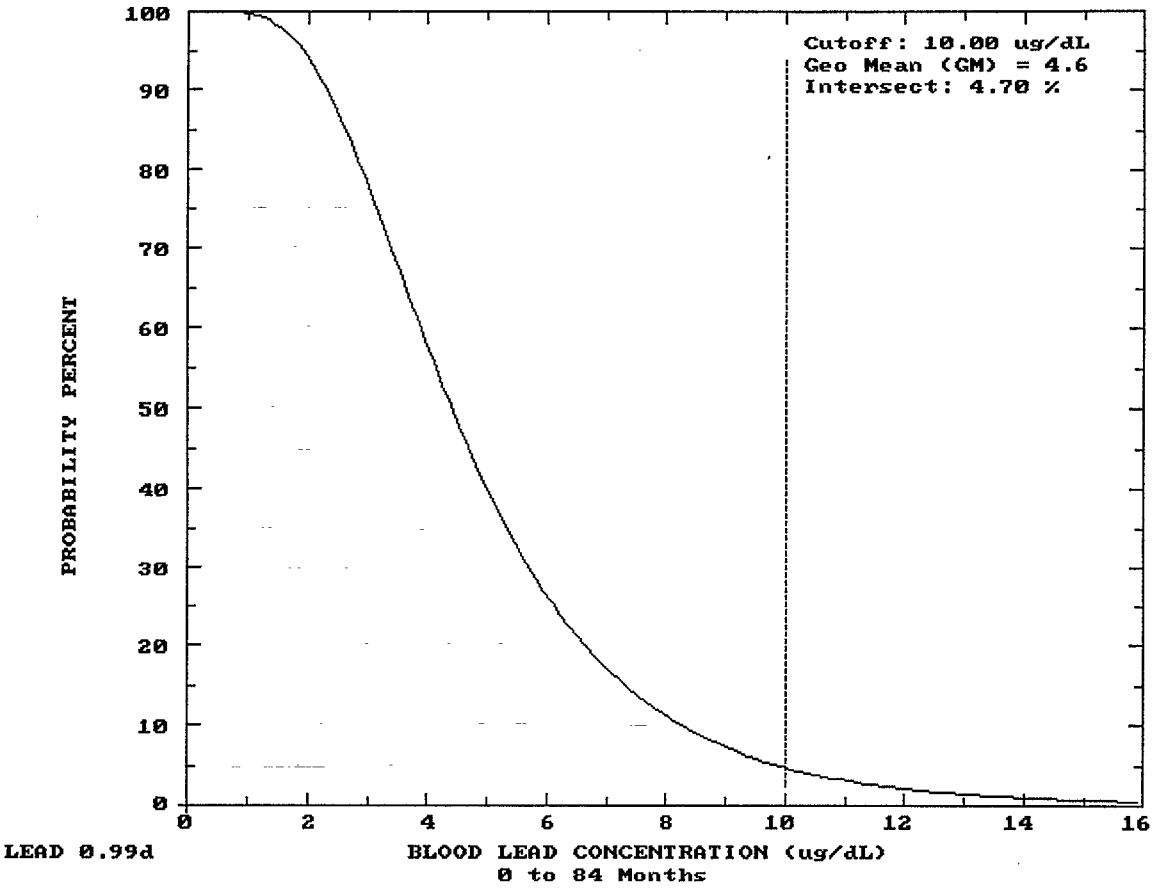
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

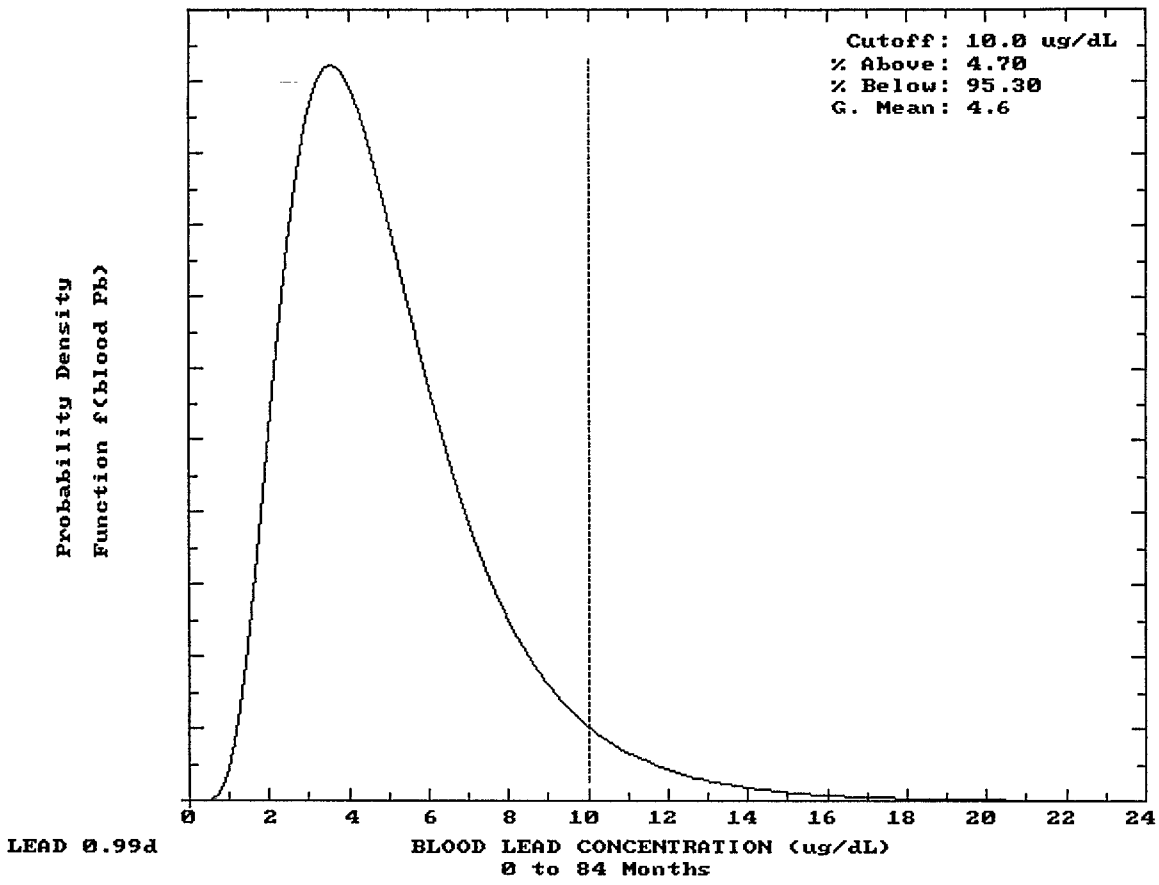
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.7	8.68	4.62
1-2:	5.5	13.55	7.18
2-3:	5.3	14.23	7.28
3-4:	5.0	14.41	7.39
4-5:	4.4	12.82	5.59
5-6:	4.0	12.78	5.08
6-7:	3.7	12.95	4.82

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)

0.5-1:	2.50	1.54	0.00	0.02
1-2:	2.56	3.77	0.00	0.03
2-3:	2.92	3.97	0.00	0.06
3-4:	2.85	4.11	0.00	0.07
4-5:	2.80	4.36	0.00	0.07
5-6:	2.98	4.63	0.00	0.09
6-7:	3.30	4.73	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 4.6
Intersect: 4.70 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
2
4
6
8
10
12
14
16
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 4.70
% Below: 95.30
G. Mean: 4.6
Probability Density
Function f(Blood Pb)
0
2
4
6
8
10
12
14
16
18
20
22
24
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
0 to 84 Months

APPENDIX H, SECTION 4

HISTOGRAMS FOR
CONEWAGO CREEK, KEYSTONE TRIBUTARY SURFACE WATER
IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 99.8 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 266.00 ug Pb/L 0.22 % 0.22% estimated as follows:

$$\left(\frac{0.065\ L/day\ IR_{SW}}{0.5625\ L/day\ TOTAL\ DW\ IR}\right) \times \left(\frac{7\ days/yr\ EF_{SW}}{365\ days/yr\ EF_{DW}}\right) \times 100\ \%$$

(IEUBK average age 3 to 7)

WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

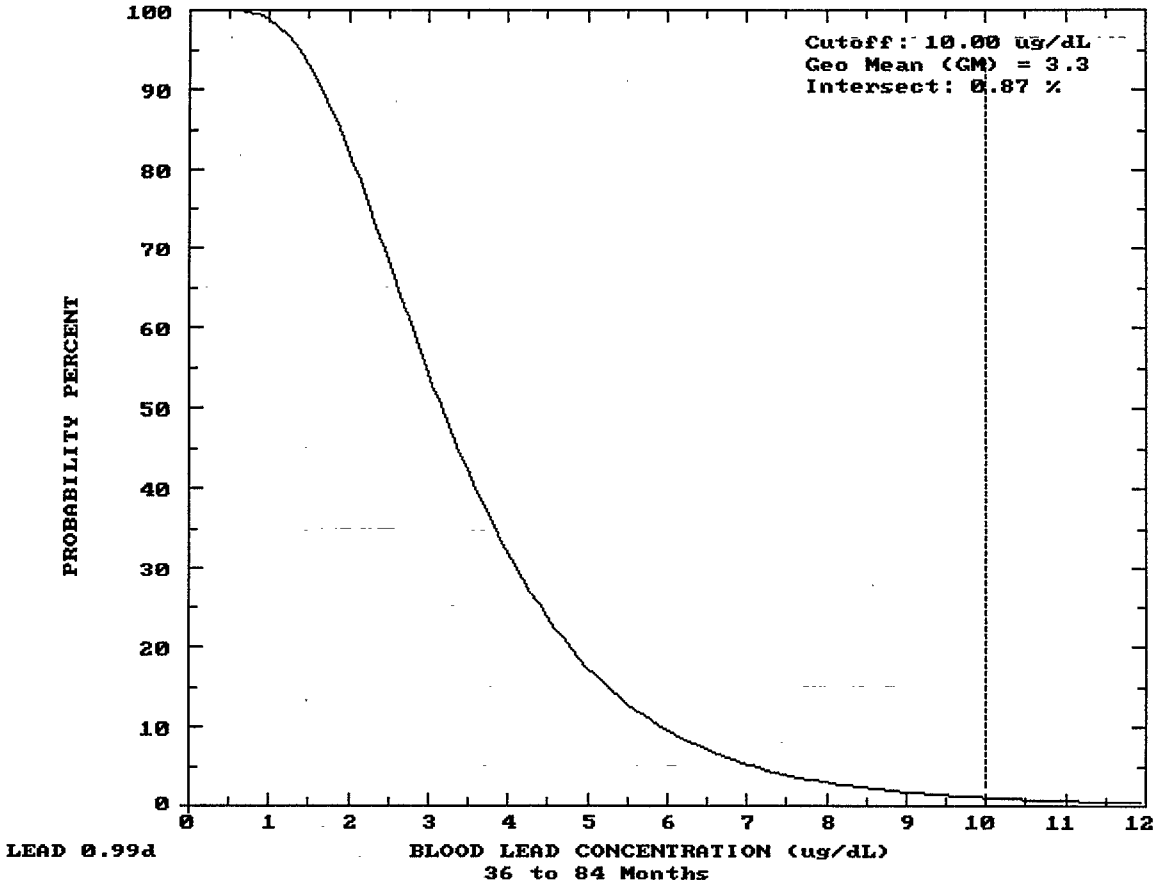
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

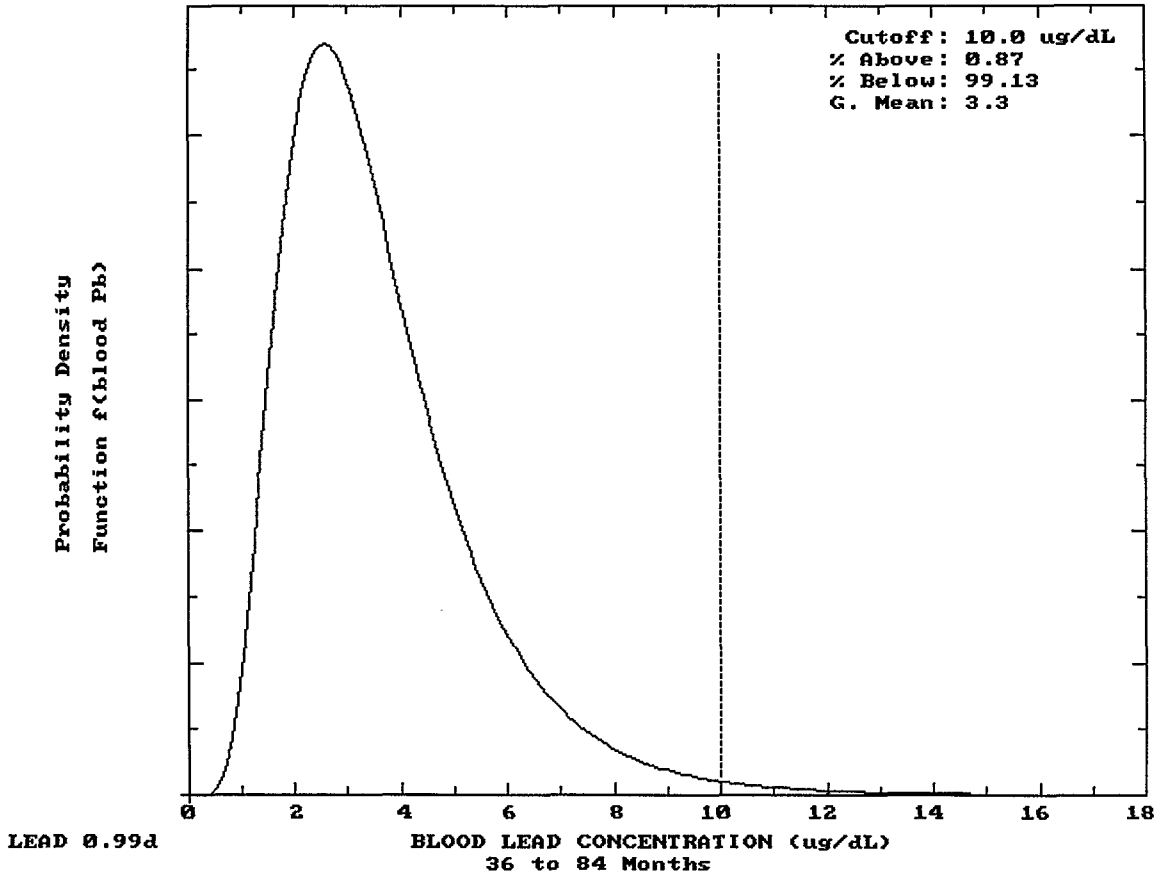
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.65	4.67
1-2:	4.6	11.05	7.35
2-3:	4.3	11.56	7.43
3-4:	4.1	11.62	7.52
4-5:	3.4	9.80	5.69
5-6:	3.0	9.55	5.16

6-7:	2.7	9.63	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.53	0.42	0.00	0.02
1-2:	2.62	1.04	0.00	0.03
2-3:	2.98	1.09	0.00	0.06
3-4:	2.90	1.13	0.00	0.07
4-5:	2.85	1.19	0.00	0.07
5-6:	3.03	1.27	0.00	0.09
6-7:	3.35	1.30	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.87 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
5
6
7
8
9
10
11
12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.87
% Below: 99.13
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

LEAD 0.99d

APPENDIX H, SECTION 5

HISTOGRAMS FOR
PINEY CREEK, BOUNDARY TRIBUTARY SURFACE WATER
IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 99.8 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 43.00 ug Pb/L 0.22 % 0.22% estimated as follows:

WATER Consumption: DEFAULT $\left(\frac{0.065\ L/day\ IR_{SW}}{0.5625\ L/day\ \text{TOTAL D.W. IR}}\right) \times \left(\frac{7\ days/yr\ EF_{SW}}{365\ days/yr\ EF_{DW}}\right) \times 100\%$

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

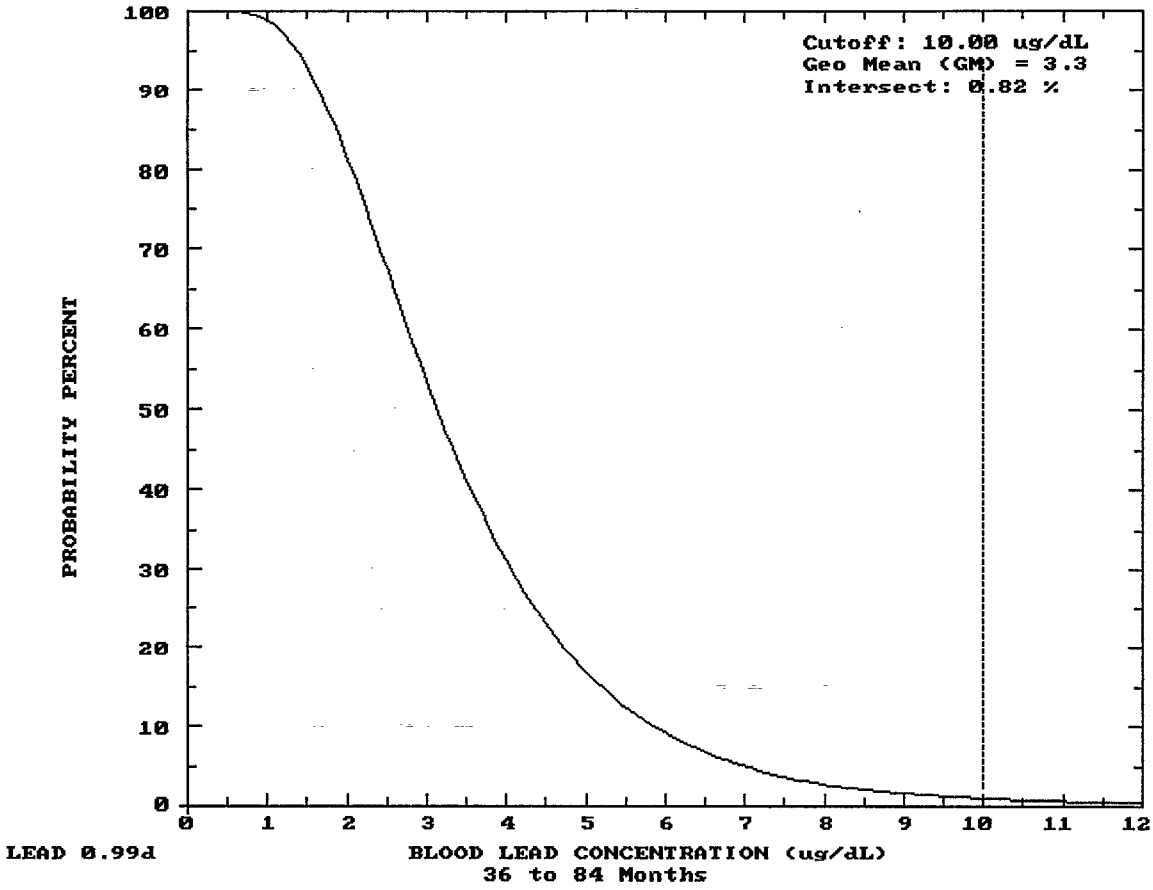
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

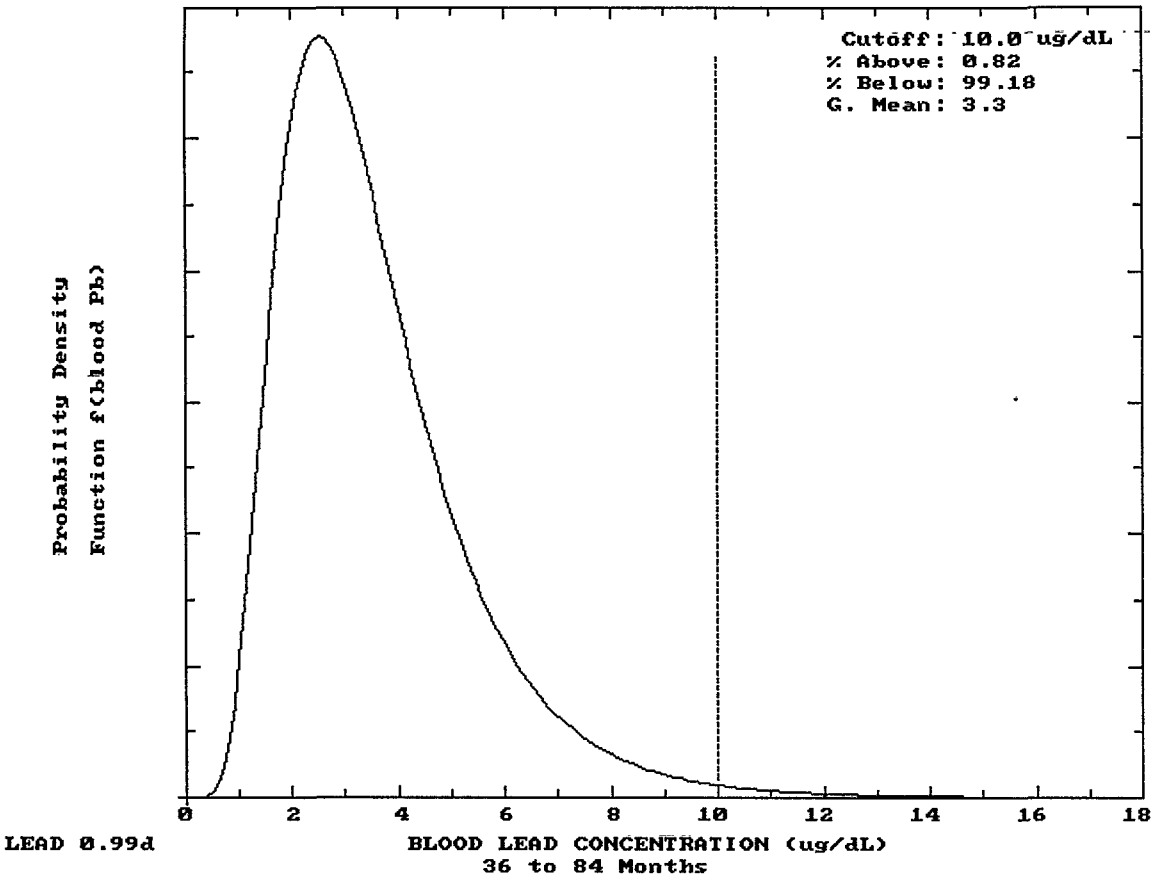
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.61	4.68
1-2:	4.5	10.94	7.36
2-3:	4.3	11.45	7.44
3-4:	4.0	11.50	7.53
4-5:	3.4	9.67	5.69
5-6:	3.0	9.41	5.16

6-7:	2.7	9.49	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.62	0.93	0.00	0.03
2-3:	2.98	0.97	0.00	0.06
3-4:	2.90	1.00	0.00	0.07
4-5:	2.85	1.06	0.00	0.07
5-6:	3.03	1.13	0.00	0.09
6-7:	3.36	1.15	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.82 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
5
6
7
8
9
10
11
12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.82
% Below: 99.18
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

APPENDIX H, SECTION 6

HISTOGRAMS FOR SEEP NO. 1 SURFACE WATER IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 100.0 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 18.40 ug Pb/L 0.022% 0.022% estimated as follows:

$$\left(\frac{0.001\ L/day\ IR_{SW}}{0.5625\ L/day\ Total\ DW_{IR}}\right) \times \left(\frac{45\ days/yr\ EF_{SW}}{365\ days/yr\ EF_{DW}}\right) \times 100\ \%$$

WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

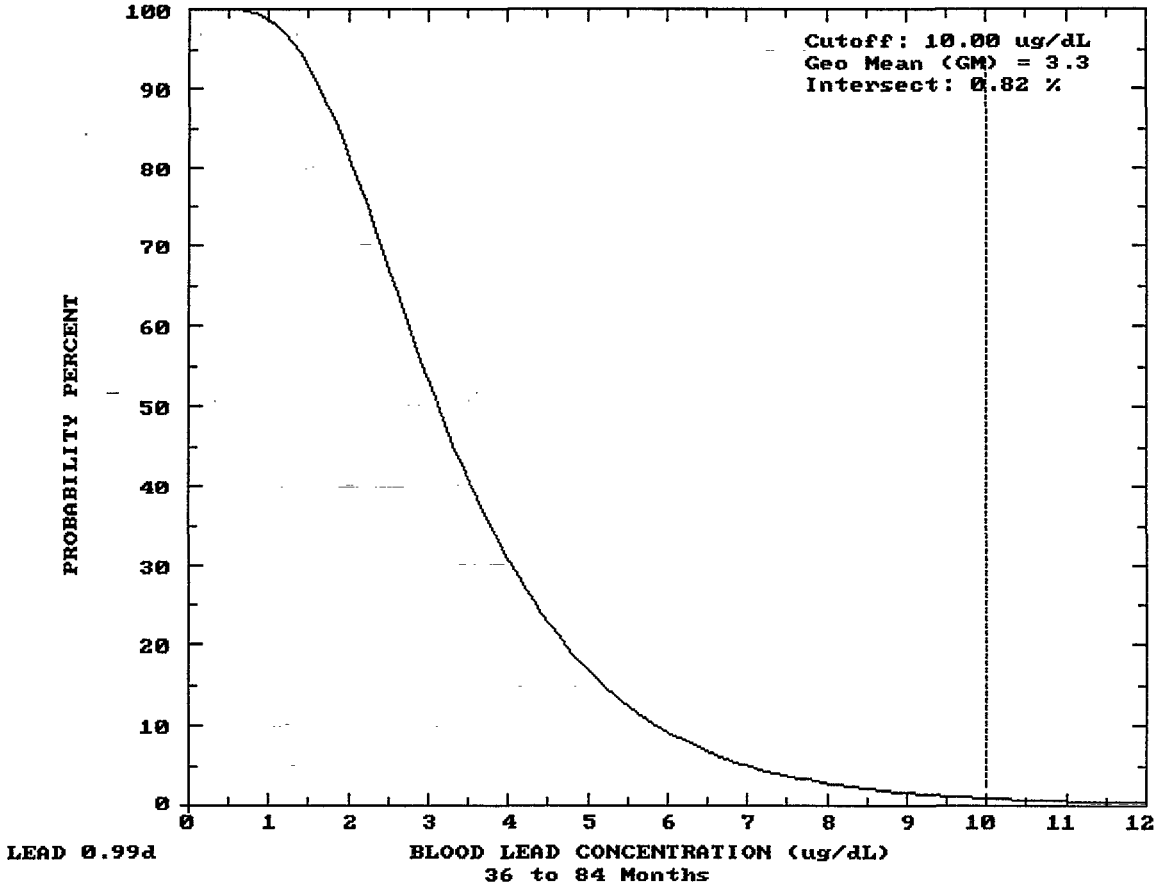
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

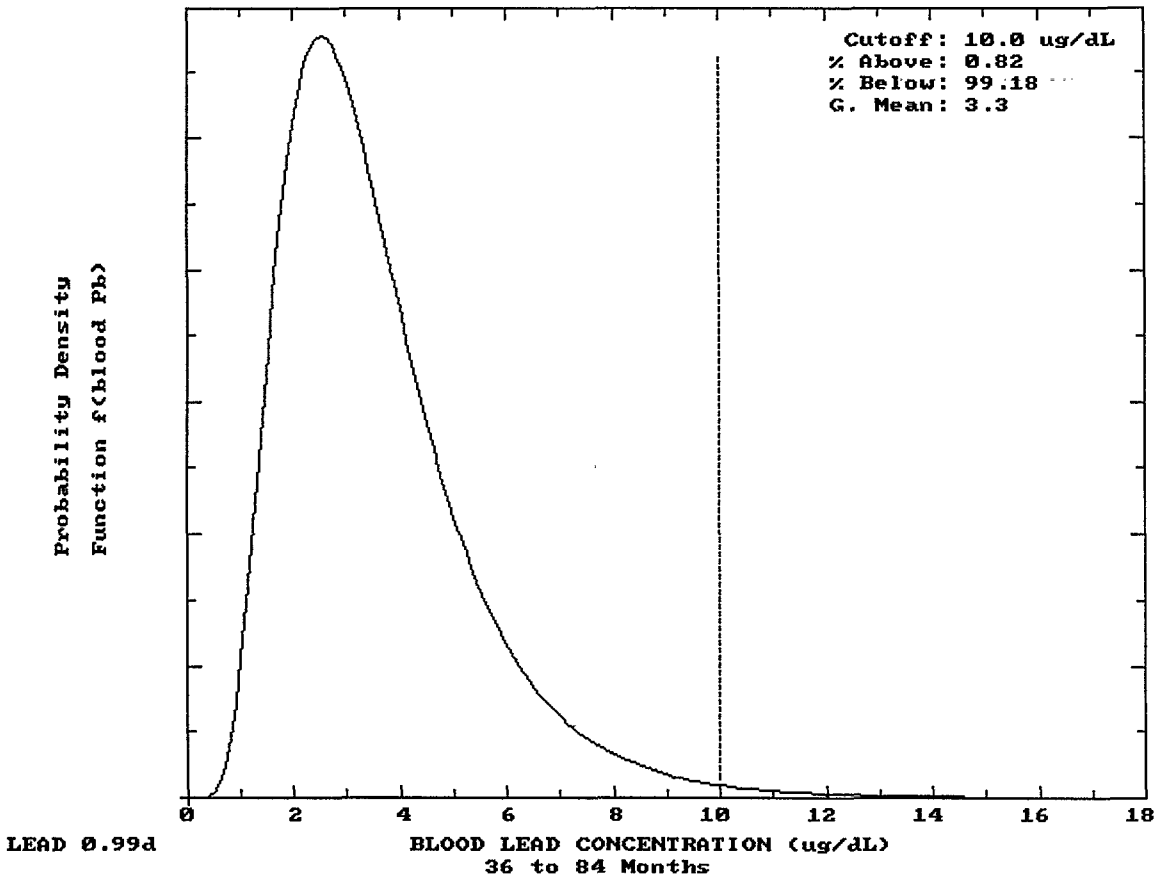
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.60	4.68
1-2:	4.5	10.93	7.36
2-3:	4.2	11.44	7.44
3-4:	4.0	11.48	7.53
4-5:	3.4	9.65	5.69
5-6:	3.0	9.39	5.16

6-7:	2.7	9.47	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.63	0.91	0.00	0.03
2-3:	2.98	0.96	0.00	0.06
3-4:	2.90	0.99	0.00	0.07
4-5:	2.85	1.04	0.00	0.07
5-6:	3.03	1.11	0.00	0.09
6-7:	3.36	1.13	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.82 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
5
6
7
8
9
10
11
12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.82
% Below: 99.18
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

APPENDIX H, SECTION 7

HISTOGRAMS FOR SEEP NO. 2 SURFACE WATER IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 100.0 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 48.90 ug Pb/L 0.022% 0.0229% estimated as follows:

$$\left(\frac{0.001\ L/day\ SWIR}{0.5625\ L/day\ total\ DW_{IR}}\right) \times \left(\frac{45\ days/yr\ EF_{SW}}{365\ days/yr\ EF_{DW}}\right) \times 100\%$$

WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

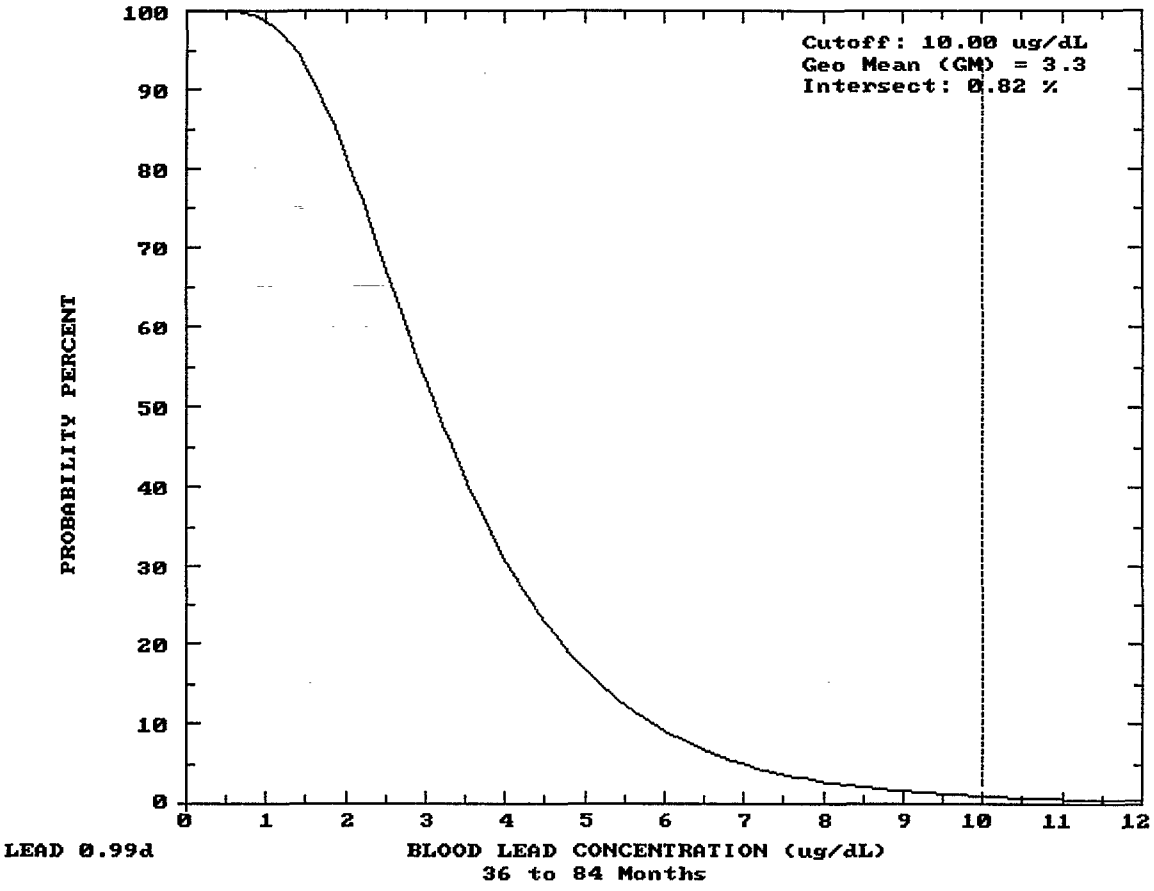
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

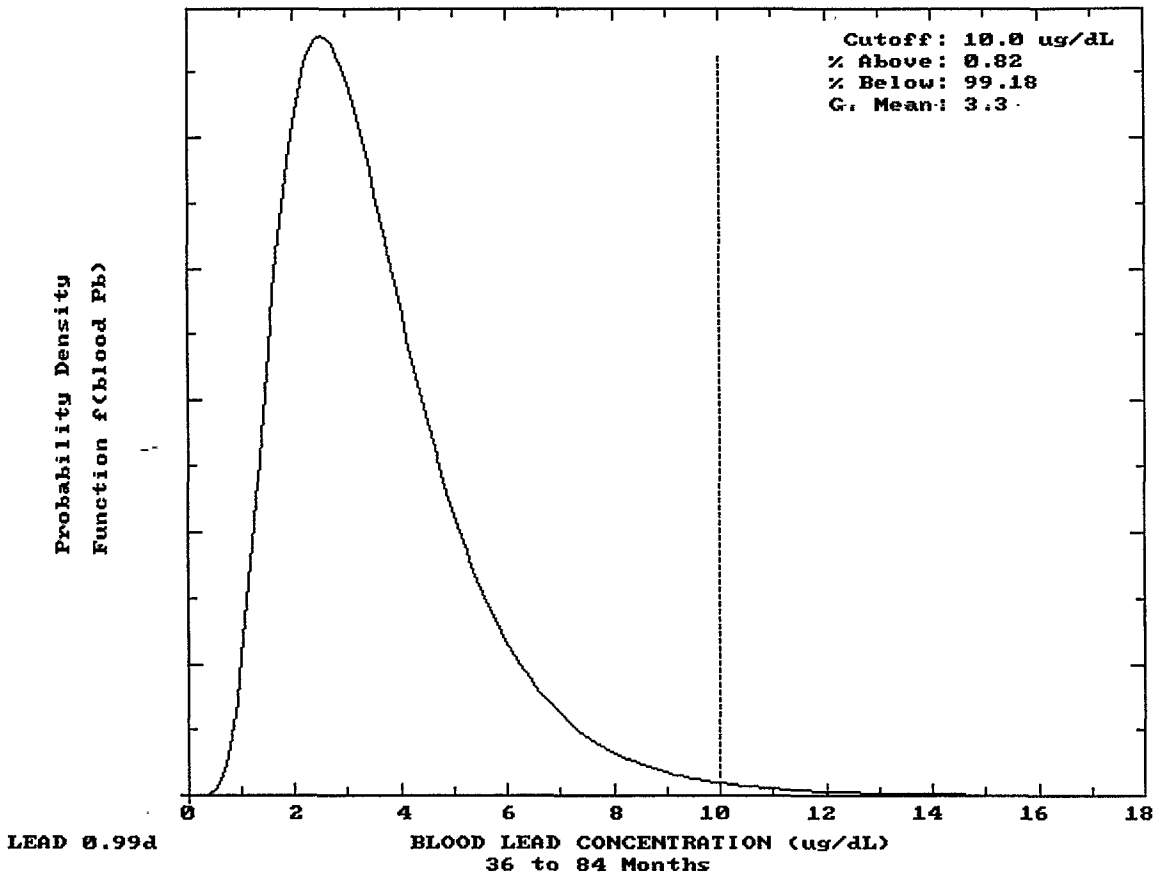
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.60	4.68
1-2:	4.5	10.93	7.36
2-3:	4.2	11.44	7.44
3-4:	4.0	11.49	7.53
4-5:	3.4	9.66	5.69
5-6:	3.0	9.40	5.16

6-7:	2.7	9.48	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.63	0.91	0.00	0.03
2-3:	2.98	0.96	0.00	0.06
3-4:	2.90	0.99	0.00	0.07
4-5:	2.85	1.05	0.00	0.07
5-6:	3.03	1.11	0.00	0.09
6-7:	3.36	1.13	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.82 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
5
6
7
8
9
10
11
12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.82
% Below: 99.18
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

APPENDIX H, SECTION 8

HISTOGRAMS FOR SEEP NO. 5 SURFACE WATER IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 100.0 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 22.20 ug Pb/L 0.022% 0.022% estimated as follows:

$$\left(\frac{0.001\ \text{L/day SW}_{IR}}{0.5625\ \text{L/day Total DW}_{IR}}\right) \times \left(\frac{45\ \text{days/yr EF}_{SW}}{365\ \text{days/yr EF}_{DW}}\right) \times 100\%$$

WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

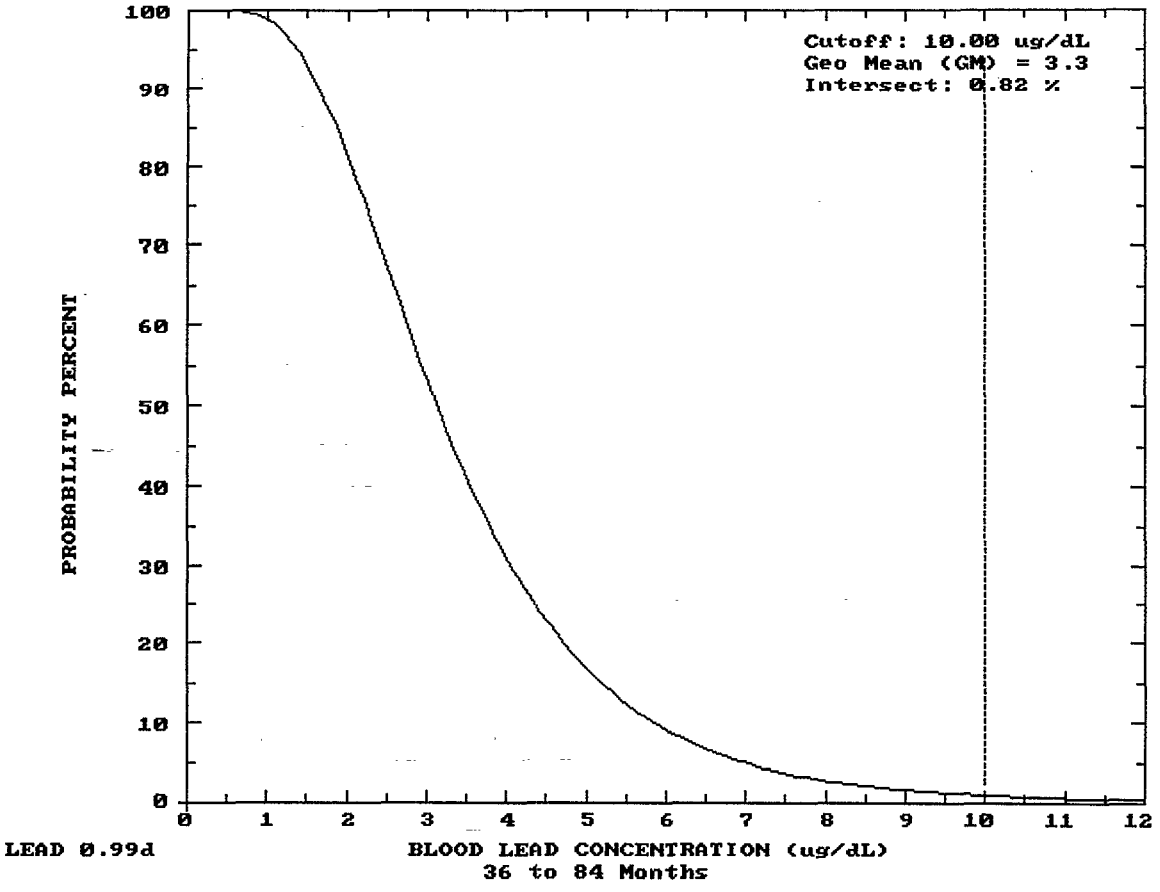
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

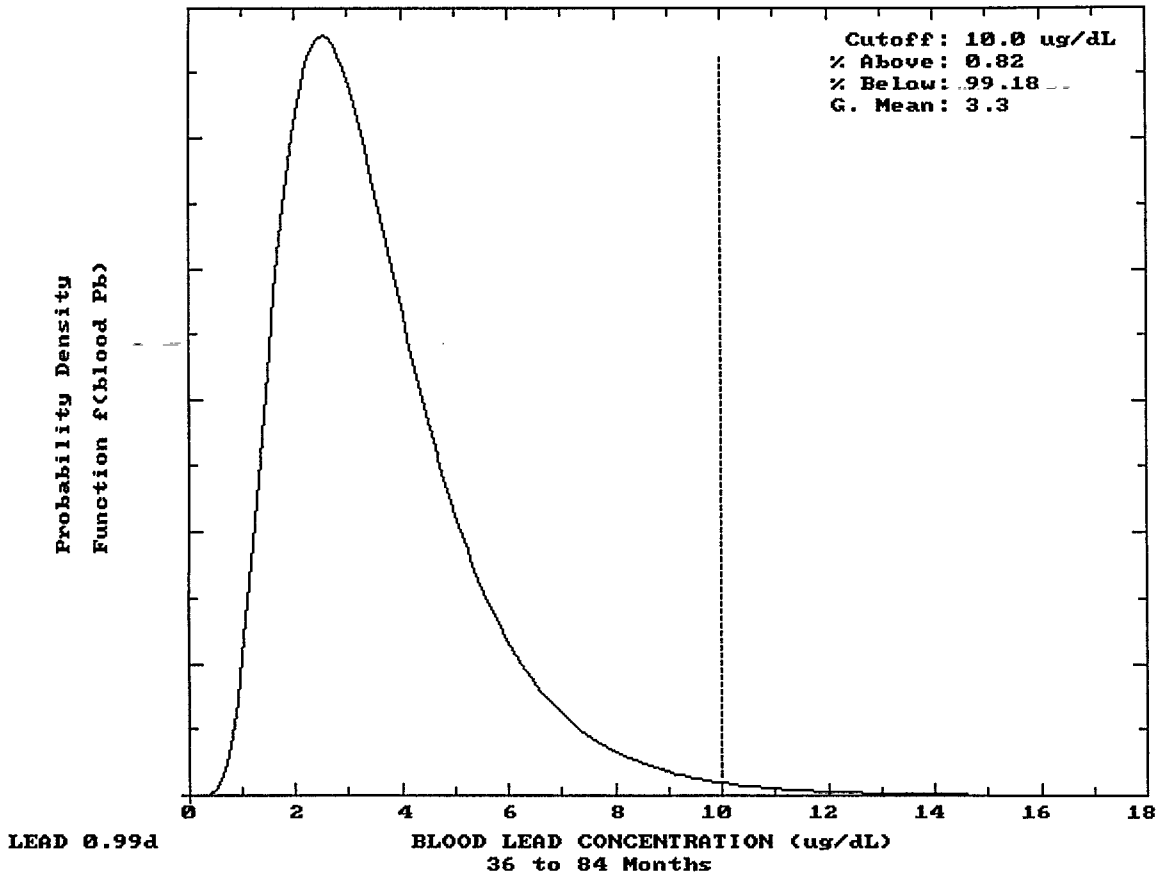
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.60	4.68
1-2:	4.5	10.93	7.36
2-3:	4.2	11.44	7.44
3-4:	4.0	11.48	7.53
4-5:	3.4	9.65	5.69
5-6:	3.0	9.39	5.16

6-7:	2.7	9.47	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.63	0.91	0.00	0.03
2-3:	2.98	0.96	0.00	0.06
3-4:	2.90	0.99	0.00	0.07
4-5:	2.85	1.04	0.00	0.07
5-6:	3.03	1.11	0.00	0.09
6-7:	3.36	1.13	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.82 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0 1 2 3 4 5 6 7 8 9 10 11 12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.82
% Below: 99.18
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

AR309605

APPENDIX H, SECTION 9

HISTOGRAMS FOR SEEP NO. 6 SURFACE WATER IEUBK MODEL RUNS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER: alternate water selected by user as follows:
Flushed water: 4.00 ug Pb/L 100.0 %
First-Draw: 4.00 ug Pb/L 0.0 %
Fountain: 32.60 ug Pb/L 0.022% 0.022% estimated as follows:

WATER Consumption: DEFAULT $\left(\frac{0.001\ L/day\ SW_{IR}}{0.5625\ L/day\ Total\ DW_{IR}}\right) \times \left(\frac{45\ days/yr\ EF_{SW}}{365\ days/yr\ EF_{DW}}\right) \times 100\%$

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

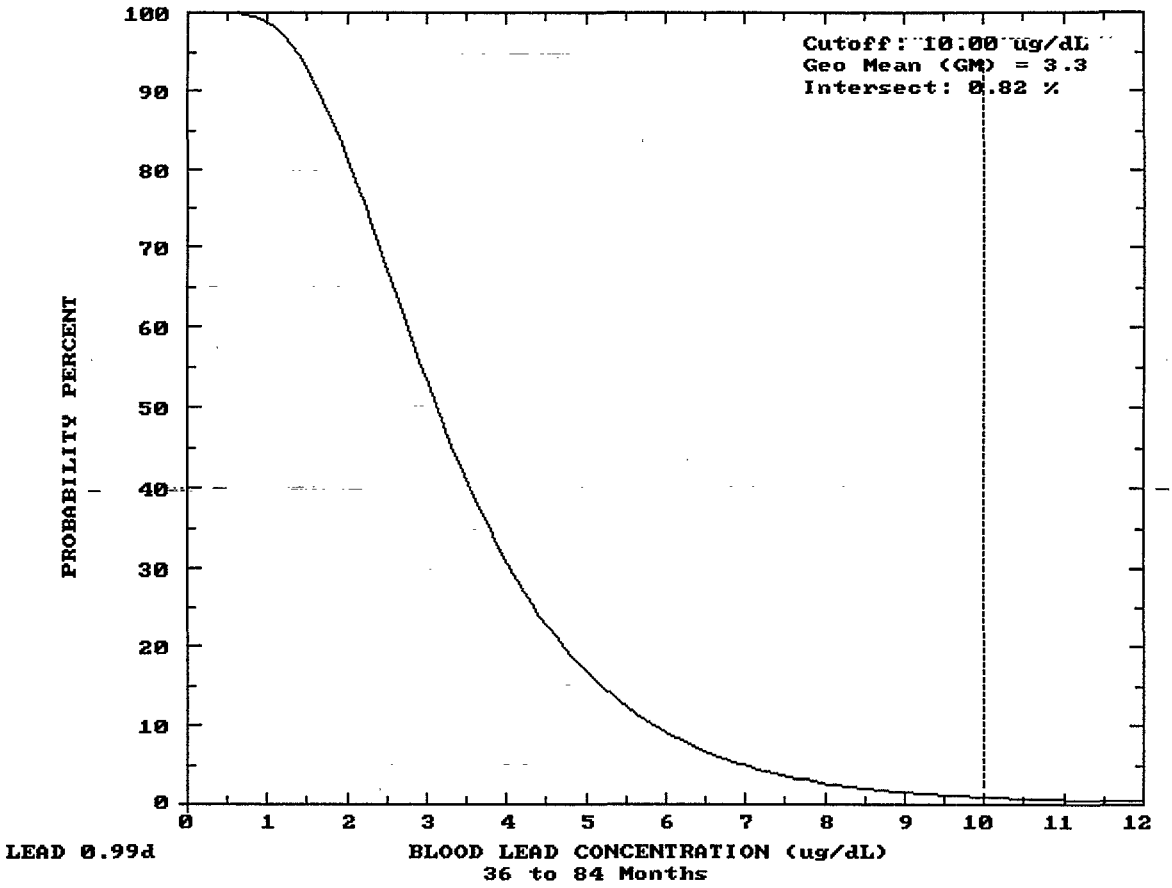
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

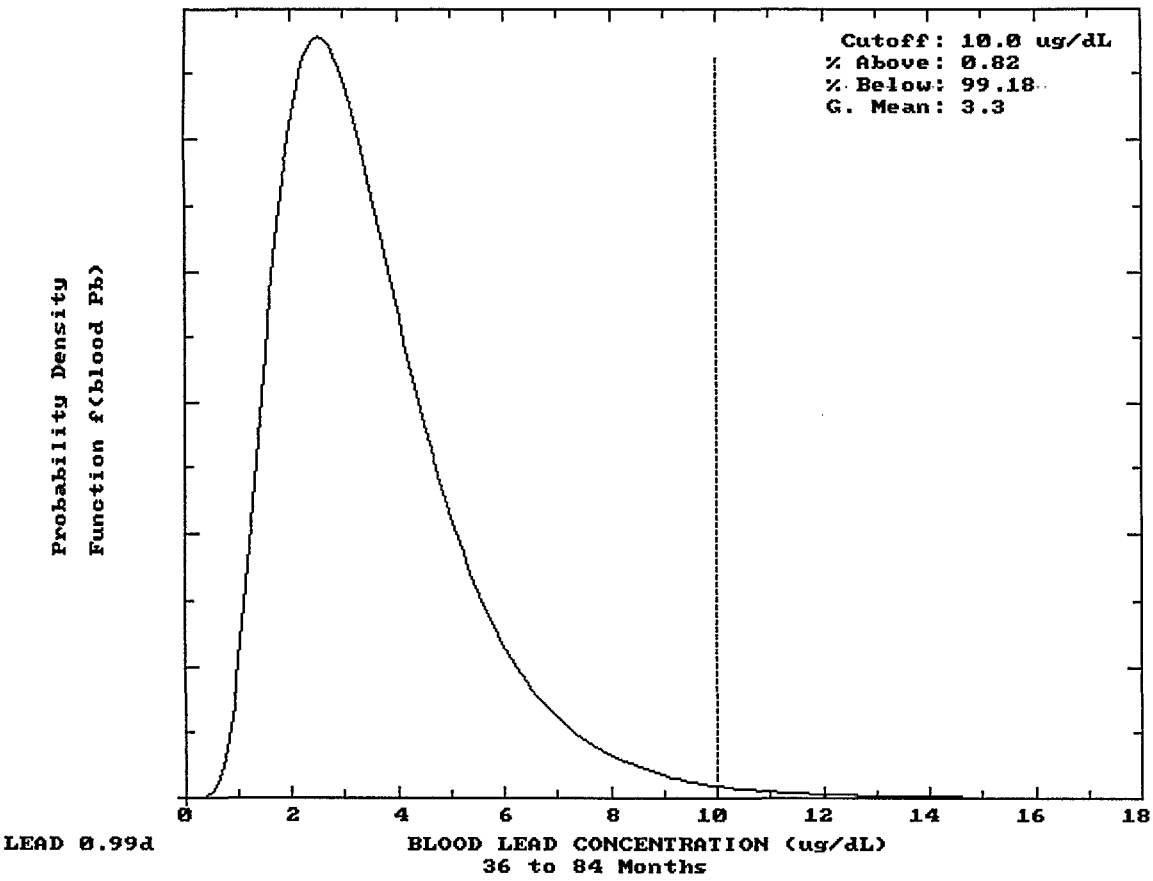
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.1	7.60	4.68
1-2:	4.5	10.93	7.36
2-3:	4.2	11.44	7.44
3-4:	4.0	11.49	7.53
4-5:	3.4	9.66	5.69
5-6:	3.0	9.39	5.16

6-7:	2.7	9.47	4.89	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.63	0.91	0.00	0.03
2-3:	2.98	0.96	0.00	0.06
3-4:	2.90	0.99	0.00	0.07
4-5:	2.85	1.05	0.00	0.07
5-6:	3.03	1.11	0.00	0.09
6-7:	3.36	1.13	0.00	0.09


Cutoff: 10.00 ug/dL
Geo Mean (GM) = 3.3
Intersect: 0.82 %
PROBABILITY PERCENT
100
90
80
70
60
50
40
30
20
10
0
0 1 2 3 4 5 6 7 8 9 10 11 12
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months


Cutoff: 10.0 ug/dL
% Above: 0.82
% Below: 99.18
G. Mean: 3.3
Probability Density
Function f(blood Pb)
0
2
4
6
8
10
12
14
16
18
LEAD 0.99d
BLOOD LEAD CONCENTRATION (ug/dL)
36 to 84 Months

APPENDIX I

COMPARISON OF ROUTINE AND LOW DETECTION LIMIT MERCURY DATA

TABLE I-1
COMPARISON OF SAMPLES ANALYZED BY ROUTINE AND LOW DETECTION LIMIT METHODS FOR MERURY
KEYSTONE SANITATION LANDFILL SITE, OU-2

SAMPLE IDENTIFIER	MASTER LOCATION	ROUND	Concentration UG/L or MG/KG	QUAL	M=NORMAL METHOD H=LOW D.L
SD-03	SD-03	SD940401	0.28	U	M
SD-03-0308	SD-03	SD950201	2.2		M
SD-03	SD-03	SD951101	**0.0742**	K	H
SD-03	SD-03	SD951101	0.1		M
SD-03	SD-04	MW940901	0.1		M
SD-04	SD-04	SD940401	0.3		M
SD-03	SD-04	SD950201	1.3	L	M
SD-03-DUP	SD-04	SD950201	0.25	L	M
SD-04	SD-04	SD951101	**0.209**		H
SD-04	SD-04	SD951101	0.1	U	M
SD-06	SD-06	SD940401	0.25	U	M
SD-06	SD-06	SD950201	0.26	U	M
SD-06-DUP	SD-06	SD950201	0.62		M
SD-06	SD-06	SD951101	**0.0545**	K	H
SD-06	SD-06	SD951101	0.1	U	M
SD-08	SD-08	SD940401	0.26	U	M
SD-08	SD-08	SD950201	0.13	U	M
SD-08	SD-08	SD951101	**0.0286**		H
SD-08	SD-08	SD951101	0.1	U	M
SD-13	SD-13	SD940401	0.36	U	M
SD-13-DUP	SD-13	SD940401	0.34	U	M
SD-13	SD-13	SD950201	1.3		M
SD-13	SD-13	SD951101	**0.057**		H
SD-13-DUP	SD-13	SD951101	**0.0543**		H
SD-13	SD-13	SD951101	0.1	U	M
SD-13-DUP	SD-13	SD951101	0.1	U	M
SD-18	SD-18	SD940401	0.27	U	M
SD-18	SD-18	SD950201	0.27	U	M
SD-18	SD-18	SD951101	**0.0516**		H
SD-18	SD-18	SD951101	0.1	U	M
SD-20	SD-20	SD940401	0.24	U	M
SD-20-DUP	SD-20	SD940401	0.27	U	M
SD-20	SD-20	SD950201	0.19	U	M
SD-20	SD-20	SD951101	**0.0231**		H
SD-20	SD-20	SD951101	0.1	U	M
SD-22	SD-22	SD940401	0.27	U	M
SD-22	SD-22	SD950201	0.72		M
SD-22	SD-22	SD951101	**0.0518**	K	H
SD-22	SD-22	SD951101	0.1	U	M
SD-29	SD-29	SD950201	0.7		M
SD-29	SD-29	SD951101	**0.0522**		H
SD-29	SD-29	SD951101	0.1	U	M
SD-34	SD-34	SD950201	1.2		M
SD-41	SD-34	SD951101	**0.025**		H
SD-41	SD-34	SD951101	0.1	U	M
SD-34	SD-41	SD951101	**0.0218**		H
SD-34	SD-41	SD951101	0.1	U	M
SD-42	SD-42	SD951101	**0.0599**		H
SD-42	SD-42	SD951101	0.1	U	M
SD-47	SD-47	SD951101	**0.0116**		H
SD-47	SD-47	SD951101	0.1	U	M
SD-48	SD-48	SD951101	**0.0362**		H
SD-48	SD-48	SD951101	0.1	U	M
SD-49	SD-49	SD951101	**0.055**	K	H
SD-49	SD-49	SD951101	0.1	U	M
SD-50	SD-50	SD951101	**0.0666**	K	H
SD-50	SD-50	SD951101	0.1	U	M
SD-51	SD-51	SD951101	**0.033**	K	H
SD-51	SD-51	SD951101	0.1	U	M

TABLE I-1
COMPARISON OF SAMPLES ANALYZED BY ROUTINE AND LOW DETECTION LIMIT METHODS FOR MERURY
KEYSTONE SANITATION LANDFILL SITE, OU-2

SAMPLE IDENTIFIER	MASTER LOCATION	ROUND	Concentration UG/L or MG/KG	QUAL	M=NORMAL METHOD H=LOW D.L
SD-52	SD-52	SD951101	**0.039**	K	H
SD-52	SD-52	SD951101	0.1	U	M
SD-53	SD-53	SD951101	**0.026**	K	H
SD-53	SD-53	SD951101	0.1	U	M
SD-54	SD-54	SD951101	**0.038**	K	H
SD-54-DUP	SD-54	SD951101	**0.0356**	K	H
SD-54	SD-54	SD951101	0.1	U	M
SD-54-DUP	SD-54	SD951101	0.1	U	M
SD-56	SD-56	SD951101	**0.0304**	K	H
SD-56	SD-56	SD951101	0.1	U	M
SD-57	SD-57	SD951101	**0.0778**	K	H
SD-57	SD-57	SD951101	0.1	U	M
SD-58	SD-58	SD951101	**0.019**		H
SD-58-DUP	SD-58	SD951101	**0.0193**		H
SD-58	SD-58	SD951101	0.1	U	M
SD-60	SD-60	SD951101	**0.0375**		H
SD-60	SD-60	SD951101	0.1	U	M
SD-61	SD-61	SD951101	**0.0317**		H
SD-61	SD-61	SD951101	0.1	U	M
SD-62	SD-62	SD951101	**0.0402**		H
SD-62-DUP	SD-62	SD951101	**0.0316**		H
SD-62	SD-62	SD951101	0.1	U	M
SD-63	SD-63	SD951101	**0.058**		H
SD-63	SD-63	SD951101	0.1	U	M
SD-64	SD-64	SD951101	**0.0554**		H
SD-64	SD-64	SD951101	0.1	U	M
SD-65	SD-65	SD951101	**0.0971**		H
SD-65	SD-65	SD951101	0.1		M
SD-66	SD-66	SD951101	**0.0403**		H
SD-66	SD-66	SD951101	0.1		M
SD-67	SD-67	SD951101	**0.0282**		H
SD-67	SD-67	SD951101	0.1	U	M
SD-68	SD-68	SD951101	**0.0502**	K	H
SD-68	SD-68	SD951101	0.1	U	M
SD-71	SD-71	SD960701	**0.042**		H
SD-71	SD-71	SD960701	0.22	U	M
SD-72	SD-72	SD960701	**0.0362**		H
SD-72-DUP	SD-72	SD960701	**0.0577**		H
SD-72-DUP	SD-72	SD960701	0.17	U	M
SD-72	SD-72	SD960701	0.17	U	M
SD-73	SD-73	SD960701	**0.102**		H
SD-73	SD-73	SD960701	0.3	U	M
SD-74	SD-74	SD960701	**0.015**		H
SD-74	SD-74	SD960701	0.17	U	M
SD-75	SD-75	SD960701	**0.0928**		H
SD-75	SD-75	SD960701	0.15	U	M
SD-76	SD-76	SD960701	**0.0302**		H
SD-76	SD-76	SD960701	0.19	U	M
SD-92	SD-92	SD960701	**0.0318**		H
SD-92	SD-92	SD960701	0.14	U	M
SD-94	SD-94	SD960701	**0.0753**		H
SD-94	SD-94	SD960701	0.2	U	M
SD-95	SD-95	SD960701	**0.0454**		H
SD-95	SD-95	SD960701	0.16	U	M
SD-96	SD-96	SD960701	**0.0275**		H
SD-96	SD-96	SD960701	0.16	U	M
SD-02-0307	SD-BKG-01	SD950201	0.12	U	M
SD-BKG-01	SD-BKG-01	SD951101	**0.0135**		H
SD-BKG-01	SD-BKG-01	SD951101	0.1	U	M

TABLE I-1
COMPARISON OF SAMPLES ANALYZED BY ROUTINE AND LOW DETECTION LIMIT METHODS FOR MERURY
KEYSTONE SANITATION LANDFILL SITE, OU-2

SAMPLE IDENTIFIER	MASTER LOCATION	ROUND	Concentration UG/L or MG/KG	QUAL	M=NORMAL METHOD H=LOW D.L
SD-BKG-02	SD-BKG-02	SD951101	**0.0178**		**H**
SD-BKG-02	SD-BKG-02	SD951101	0.1	U	M
SD-BKG-03	SD-BKG-03	SD951101	**0.0325**		**H**
SD-BKG-03	SD-BKG-03	SD951101	0.1	U	M
SD-BKG-04	SD-BKG-04	SD951101	**0.0251**		**H**
SD-BKG-04	SD-BKG-04	SD951101	0.1	U	M
SD-SEEP2	SD-SEEP2	SD940401	0.33	U	M
PD-02	SD-SEEP2	SD950201	0.15	U	M
PD-02	SD-SEEP2	SD951101	**0.0345**		**H**
PD-02	SD-SEEP2	SD951101	0.1	U	M
PD-05	SD-SEEP5	SD950201	0.75	U	M
PD-05	SD-SEEP5	SD951101	**0.0583**		**H**
PD-05	SD-SEEP5	SD951101	0.1	U	M
SD-SEEP9	SD-SEEP9	SD951101	**0.0543**	**K**	**H**
SD-SEEP9	SD-SEEP9	SD951101	0.1	U	M
SD-SEEP9	SD-SEEP9	SD960701	**0.043**		**H**
SD-SEEP9	SD-SEEP9	SD960701	0.19	U	M
SP-04	SP-02	MW940901	0.1	UL	M
SP-04	SP-02	MW950201	0.23	J	M
SW-02	SP-02	SW951101	**0.22**		**H**
SW-02	SP-02	SW951101	3.8		M
SS-49	SS-49	SD960701	**0.049**		**H**
SS-49	SS-49	SD960701	0.12	U	M
SP-03	SW-04	MW940901	0.15		M
SP-03	SW-04	MW950201	0.2	UJ	M
SP-03-DUP	SW-04	MW950201	0.2	UJ	M
SW-04	SW-04	SW940401	0.23		M
SW-04-F	SW-04	SW940401	4.2		M
SW-04	SW-04	SW951101	**0.0784**		**H**
SW-04	SW-04	SW951101	0.2	U	M
SW-06	SW-06	SW940401	0.2	U	M
SW-06-F	SW-06	SW940401	5.6		M
SW-06	SW-06	SW950201	0.2	U	M
SW-06-DUP	SW-06	SW950201	0.2	U	M
SW-06	SW-06	SW951101	**0.00116**	**B**	**H**
SW-06	SW-06	SW951101	0.2	U	M
SW-13	SW-13	SW940401	0.2	U	M
SW-13-F	SW-13	SW940401	1	B	M
SW-13-DUP	SW-13	SW940401	0.2	U	M
SW-13-DUP-F	SW-13	SW940401	0.47	B	M
SW-13	SW-13	SW950201	0.2	U	M
SW-13	SW-13	SW951101	**0.00175**	**B**	**H**
SW-13-DUP	SW-13	SW951101	**0.00151**	**B**	**H**
SW-13	SW-13	SW951101	0.2	U	M
SW-13-DUP	SW-13	SW951101	0.2	U	M
SW-18	SW-18	SW940401	0.2	U	M
SW-18-F	SW-18	SW940401	0.78	B	M
SW-18	SW-18	SW950201	0.2	UL	M
SW-18	SW-18	SW951101	**0.00124**	**B**	**H**
SW-18	SW-18	SW951101	0.2	U	M
SW-20-F	SW-20	SW940401	0.82	B	M
SW-20	SW-20	SW940401	0.2	U	M
SW-20-DUP	SW-20	SW940401	0.2	U	M
SW-20-DUP-F	SW-20	SW940401	0.84	B	M
SW-20	SW-20	SW950201	0.38		M
SW-20	SW-20	SW951101	**0.00182**	**B**	**H**
SW-20	SW-20	SW951101	0.2	U	M
SW-22	SW-22	SW940401	0.2	U	M
SW-22-F	SW-22	SW940401	0.62	B	M

TABLE I-1
COMPARISON OF SAMPLES ANALYZED BY ROUTINE AND LOW DETECTION LIMIT METHODS FOR MERURY
KEYSTONE SANITATION LANDFILL SITE, OU-2

SAMPLE IDENTIFIER	MASTER LOCATION	ROUND	Concentration UG/L or MG/KG	QUAL	M = NORMAL METHOD H = LOW D.L
SW-22	SW-22	SW950201	0.2	U	M
SW-22	SW-22	SW951101	0.00224		H
SW-22	SW-22	SW951101	0.2	U	M
SW-29	SW-29	SW950201	0.2	U	M
SW-29	SW-29	SW951101	0.00083	B	H
SW-29	SW-29	SW951101	0.2	U	M
SW-34	SW-34	SW950201	0.2	U	M
SW-41	SW-34	SW951101	0.0017	B	H
SW-41	SW-34	SW951101	0.2	U	M
SW-34	SW-41	SW951101	0.0014	B	H
SW-34	SW-41	SW951101	0.2	U	M
SW-42	SW-42	SW951101	0.00117	B	H
SW-42	SW-42	SW951101	0.2	U	M
SW-47	SW-47	SW951101	0.00099	B	H
SW-47	SW-47	SW951101	0.2	U	M
SW-48	SW-48	SW951101	0.0012	B	H
SW-48	SW-48	SW951101	0.2	U	M
SW-51	SW-51	SW951101	0.00099	B	H
SW-51	SW-51	SW951101	0.2	U	M
SW-52	SW-52	SW951101	0.00081	B	H
SW-52	SW-52	SW951101	0.2	U	M
SW-53	SW-53	SW951101	0.00096	B	H
SW-53	SW-53	SW951101	0.2	U	M
SW-54	SW-54	SW951101	0.00082	B	H
SW-54-DUP	SW-54	SW951101	0.00094	B	H
SW-54	SW-54	SW951101	0.2	U	M
SW-54-DUP	SW-54	SW951101	0.2	U	M
SW-56	SW-56	SW951101	0.00185	B	H
SW-56	SW-56	SW951101	0.2	U	M
SW-57	SW-57	SW951101	0.00688		H
SW-57	SW-57	SW951101	0.2	U	M
SW-58-DUP	SW-58	SW951101	0.00233	B	H
SW-58	SW-58	SW951101	0.00231	B	H
SW-58	SW-58	SW951101	0.2	U	M
SW-60	SW-60	SW951101	0.00242	B	H
SW-60	SW-60	SW951101	0.2	U	M
SW-62	SW-62	SW951101	0.001	B	H
SW-62-DUP	SW-62	SW951101	0.00111	B	H
SW-62	SW-62	SW951101	0.2	U	M
SW-63	SW-63	SW951101	0.0111		H
SW-63	SW-63	SW951101	0.2	U	M
SW-64	SW-64	SW951101	0.0012	B	H
SW-64	SW-64	SW951101	0.2	U	M
SW-65	SW-65	SW951101	0.00132	B	H
SW-65	SW-65	SW951101	0.2	U	M
SW-66	SW-66	SW951101	0.00084	B	H
SW-66	SW-66	SW951101	0.2	U	M
SW-67	SW-67	SW951101	0.00123	B	H
SW-67	SW-67	SW951101	0.2	U	M
SW-68	SW-68	SW951101	0.00161	B	H
SW-68	SW-68	SW951101	0.2	U	M
SW-71	SW-71	SW960701	0.0018	B	H
SW-71	SW-71	SW960701	0.2	U	M
SW-72	SW-72	SW960701	0.00098	B	H
SW-72-DUP	SW-72	SW960701	0.00124	B	H
SW-72	SW-72	SW960701	0.2	U	M
SW-72-DUP	SW-72	SW960701	0.2	U	M
SW-91	SW-91	SW960701	0.00897		H
SW-91-F	SW-91	SW960701	0.00605		H

TABLE I-1
COMPARISON OF SAMPLES ANALYZED BY ROUTINE AND LOW DETECTION LIMIT METHODS FOR MERURY
KEYSTONE SANITATION LANDFILL SITE, OU-2

SAMPLE IDENTIFIER	MASTER LOCATION	ROUND	Concentration UG/L or MG/KG	QUAL	M=NORMAL METHOD H=LOW D.L
SW-91	SW-91	SW960701	0.2	U	M
SW-91-F	SW-91	SW960701	0.2	U	M
SW-92	SW-92	SW960701	0.0087		H
SW-92-F	SW-92	SW960701	0.00534		H
SW-92	SW-92	SW960701	0.2	U	M
SW-92-F	SW-92	SW960701	0.2	U	M
SW-93	SW-93	SW960701	0.011		H
SW-93-F	SW-93	SW960701	0.00665		H
SW-93	SW-93	SW960701	0.2	U	M
SW-93-F	SW-93	SW960701	0.2	U	M
SW-94	SW-94	SW960701	0.0134		H
SW-94-F	SW-94	SW960701	0.00919		H
SW-94	SW-94	SW960701	0.2	U	M
SW-94-F	SW-94	SW960701	0.2	U	M
SW-95	SW-95	SW960701	0.00914		H
SW-95-F	SW-95	SW960701	0.00556		H
SW-95	SW-95	SW960701	0.2	U	M
SW-95-F	SW-95	SW960701	0.2	U	M
SW-96	SW-96	SW960701	0.00852		H
SW-96-F	SW-96	SW960701	0.00532		H
SW-96	SW-96	SW960701	0.2	U	M
SW-96-F	SW-96	SW960701	0.2	U	M
SW-02	SW-BKG-01	SW950201	0.2	U	M
SW-BKG-01	SW-BKG-01	SW951101	0.00053	B	H
SW-BKG-01	SW-BKG-01	SW951101	0.2	U	M
SW-BKG-02	SW-BKG-02	SW951101	0.00062	B	H
SW-BKG-02	SW-BKG-02	SW951101	0.2	U	M
SW-BKG-03	SW-BKG-03	SW951101	0.00043	B	H
SW-BKG-03	SW-BKG-03	SW951101	0.2	U	M
SW-BKG-04	SW-BKG-04	SW951101	0.00117	B	H
SW-BKG-04	SW-BKG-04	SW951101	0.2	U	M
SW-SEEP9	SW-SEEP9	SW960701	0.00603		H
SW-SEEP9-F	SW-SEEP9	SW960701	0.0007	B	H
SW-SEEP9-F	SW-SEEP9	SW960701	0.2	U	M
SW-SEEP9	SW-SEEP9	SW960701	0.2	U	M

APPENDIX J

SUMMARY OF ANALYTES DETECTED DURING OU-1 INVESTIGATION
REMEDIAL INVESTIGATION REPORT
KEYSTONE SANITATION LANDFILL SITE
BY WILLIAMS-RUSSELL AND JOHNSON, INC.
JULY 1990

TABLE 5 - 1

COMPOUNDS/ANALYTES DETECTED IN ON-SITE SOIL SAMPLES

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUND/ANALYTE	RANGE OF CONCENTRATION (ug/kg)	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
VOLATILES (VOCs)			
1,1 - Dichloroethane	2J	2/9	3,4
1,2 - Dichloroethene(total)	6J	1/9	3
1,1,1 - Trichloroethane	2J - 9	2/9	3,4
Tetrachloroethene	43	1/9	3
BNAs/PESTICIDES/PCBs			
Naphthalene	19J	1/9	2
Acenaphthene	33J	1/9	7
Phenanthrene	19J - 160J	4/9	1,2,3,5
Anthracene	14J - 120J	2/9	1,3
Fluoranthene	14J - 200J	7/9	1,2,3,4,5,6,7
Benzo (a) anthracene	36J - 190J	2/9	3,5
Chrysene	20J - 89J	5/9	1,2,3,5,6
Benzo (b) fluoranthene	23J - 200J	5/9	1,2,3,5,6
Benzo (k) fluoranthene	13J - 160J	5/9	1,2,3,5,6
Benzo (a) pyrene	21J - 180J	5/9	1,2,3,5,6
Indeno (1,2,3-cd) pyrene	110 J	1/9	3
Dibenzo (a,h) anthracene	160 J	1/9	3
Benzo (g,h,i) perylene	100 J	1/9	3
Pentachlorophenol	73J	1/9	7
Benzoic Acid	28J - 240 J	5/9	1,2,5,6,8
Dimethylphthalate	68J - 88 J	2/9	2,3
Diethylphthalate	15J - 160J	4/9	1,3,5,6
Di-n-octylphthalate	10J - 140J	7/9	1,3,4,5,6,7,8
N-nitrosodiphenylamine	120J	1/9	3
Dieldrin	6.8J	1/9	5
4,4'-DDE	12J-14J	2/9	1,5
4-chloro-3-methyl phenol	96J	1/9	7

KEY: J - Reported value is estimated.

[ ] - Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.

* - Lists the number of detections per nine on-site sample locations.

** - Background locations are: 19,20,21.

TABLE 5 - 1 (Continued)

COMPOUNDS/ANALYTES DETECTED IN ON-SITE SOIL SAMPLES

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUND/ANALYTE	RANGE OF CONCENTRATION (ug/kg)	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
INORGANICS			
Antimony	[6.0] - [6.3]L	2/9	2,8
Arsenic	[0.6] - 4.8L	9/9	1,2,3,4,5,6,7,8,11
Beryllium	[0.5] - 1.5	8/9	1,2,3,4,5,6,7,8,11
Chromium	13.4 - 22.6	9/9	1,2,3,4,5,6,7,8,11
Copper	11.2J - 43.3	9/9	1,2,3,4,5,6,7,8,11
Lead	9.2 - 80	9/9	1,2,3,4,5,6,7,8,11
Mercury	0.11 - 1.2	4/9	3,4,5,6
Nickel	[6.1] - 29.1	9/9	1,2,3,4,5,6,7,8,11
Selenium	[0.81]J	1/9	5
Zinc	32.5 - 106	9/9	1,2,3,4,5,6,7,8,11

KEY: J - Reported value is estimated.

L - Analyte present but may be biased low. Actual value is expected to be higher.

[ ] - Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.

* - Lists the number of detections per nine on-site sample locations.

** - Background locations are: 19,20,21.

TABLE 5-2

COMPOUNDS/ANALYTES DETECTED IN OFFSITE SOIL SAMPLES

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUND/ANALYTE	RANGE OF CONCENTRATION ug/kg	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
BNAs/PESTICIDES/PCBs			
Phenanthrene	17J-36J	2/15	21,23
Fluoranthene	14J-63J	7/15	15,16,18,20,21,22,23
Chrysene	19J-35J	4/15	15,20,21,23
Benzo (b) fluoranthene	22J-31J	3/15	15,21,23
Benzo (a) pyrene	19J	1/15	15
Benzoic Acid	23J-130J	3/15	13,14,23
Dimethylphthalate	26J	1/15	21
Butylbenzylphthalate	35J	1/15	13
Di-n-octylphthalate	5J-28J	6/15	10,14,18,19,20,24
Heptachlor epoxide	2.3J-4.1J	1/15	23
Dieldrin	2.2J-12J	4/15	14,15,16,23
4,4'-DDT	76	1/15	23
INORGANICS			
Antimony	8.7L	1/15	10
Arsenic	[1.4]J - 86L	15/15	All Locations
Beryllium	[0.54] - 1.6	15/15	All Locations
Chromium	10.7 - 32.2	15/15	All Locations

Key:

J - Reported value is estimated.
L - Analyte present but may be biased low. Actual value is expected to be higher.
[ ] - Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.
* - Lists the number of detections per 15 offsite sample locations.
** - Background locations are: 19,20,21.

TABLE 5-2 (Continued)

COMPOUNDS/ANALYTES DETECTED IN OFFSITE SOIL SAMPLES

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUND/ANALYTE	RANGE OF CONCENTRATION ug/kg	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
Copper	8.3J - 38.2J	15/15	All Locations
Lead	5.1J - 102J	15/15	All Locations
Mercury	0.10 - 0.13	2/15	9, 13
Nickel	[6.2] - 29.3	15/15	All Locations
Selenium	[0.47]J	1/15	10
Silver	4.2	1/15	All Locations
Zinc	23.7 - 149	15/15	All Locations

J - Reported value is estimated.
L - Analyte present but may be biased low. Actual value is expected to be higher.
[ ] - Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.
* - Lists the number of detections per 15 offsite sample locations.
** - Background locations are: 19,20,21.

TABLE 5-3

COMPOUNDS/ANALYTES DETECTED IN SURFACE WATER

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN PENNSYLVANIA
REM V

COMPOUND/ANALYTE	FRESH WATER CHRONIC WATER QUALITY CRITERIA	RANGE OF CONCENTRATION (ug/L)	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
VOLATILES (VOCs)				
1,1,1-Trichloroethane	N/A	2J	2/18	4,18
Dichloroflouremethane	N/A	0.4	1/18	18
1,1-Dichloroethane		0.3	1/18	18
INORGANICS				
Arsenic	48[+]	[2.2] - [5.0]	4/18	4,9,10,18
Chromium	11[++]	13.4	1/18	16
Copper	12[a]	[12.0]	1/18	16
Cyanide	5.2	10.0 - 18.0	13/18	1,3,4,5,6,8,10,13,14, 15,16,17,18
Lead	3.2[a]	[1.1] - 18.9K	9/18	6,9,10,11,12,15,16,17,18
Mercury	0.012	0.30 -7.8	5/18	6,7,11,13,15
Selenium	35	[1.3]	1/18	9
Zinc	47	[9.4] - 67.7	17/18	1,2,4,5,6,7,8,9,10,11, 12,13,14,15,16,17,18

Key:
- * - Lists the number of detections per 18 sample locations.
- ** - Background locations are: 1 and 3.
- a - Hardness dependent criterion (100 ug/l used).
- + - Insufficient data to develop criterian. The lowest observed effect value reported is applicable to pentavalent arsenic only, criterion for trivalent arsenics is 190 ug/l
- ++ - Applicable to hexavalent chromium only, hardness dependent criterion for trivalent chromium is 210 ug/l (100 mg/l used)
- N/A - Criterion Not Available
- J - Reported value is estimated.
- K -
- L - Analyte present may be biased low. Actual value is expected to be higher.
- [ ] - Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.

TABLE 5-4

COMPOUNDS/ANALYTES DETECTED IN SEDIMENT

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN PENNSYLVANIA
REM V

COMPOUND/ANALYTE	RANGE OF CONCENTRATION (ug/kg)	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)**
BNAs			
Benzoic acid	110J	1/10	3
Bis(2-ethylhexyl) phthalate	52J-130J	3/10	1,3,4
INORGANICS			
Arsenic	[2.1] - 13.3	10/10	All Locations
Beryllium	[0.46]- 2.1	8/10	1,4,5,6,8,10,11,14
Cadmium	2.7J - 11.1J	10/10	All Locations
Chromium	12.7J - 48.6J	10/10	All Locations
Copper	9.5 - 31.7	10/10	All Locations
Lead	16.6J - 200J	10/10	All Locations
Nickel	12.7J - 53.6J	10/10	All Locations
Selenium	[0.25]- [0.28]K	4/10	1,5,10,11
Zinc	62.7J - 206J	10/10	All Locations

Key:

** -	Background locations are 1 and 3
* -	Lists the number of detections per 10 sample locations.
[ ] -	Analyte present but near the instrument detection limit (IDL). As values approach the IDL the quantitation may not be accurate.
J -	Reported value is estimated.
k -	Analyte prevent but reported value may be biased high. Actual value is expected to be lower
N/A -	criterion not available
+ -	Applicable for pentavalent arsenic, hardness dependent criterion for trivalent arsenic 190 ug/l (100 mg/l hardness used)
++ -	Insufficent data to develop criterion. The lowest observed effect concentration reported is applicable to hexavalent chromium, criterion for trivalent chromium 210 ug/l

TABLE 5-5

COMPOUNDS/ANALYTES DETECTED IN ONSITE MONITOR WELLS

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUNDS/ANALYTES	FEDERAL MCL (ug/L)	RANGE OF CONCENTRATION (ug/L)	FREQUENCY OF DETECTION*	SAMPLE LOCATION(S)
VOLATILES VOCs				
Vinyl Chloride	2	4J - 40	2/9	K1,HS
Chloroethane	N/A	3J - 18J	4/9	K1,K5,HS,HD
1,1-Dichloroethene	7	5 - 16	2/9	K1, HD
1,1-Dichloroethane	N/A	4J - 71	5/9	K1,K5,K6,HS,HD
1,2-Dichloroethene (Total)	N/A	6 - 140	4/9	K1,K6,HS,HD
1,1,1-Trichloroethane	200	3J - 52	7/9	K1,K4,K5,K6,K7,HS,HD
Trichloroethene	5	3J - 57	5/9	K1,K5,K6,HS,HD
Benzene	5	7J - 14	2/9	K1,K3
Tetrachloroethene	N/A	8 - 48	6/9	K1,K4,K5,K6,HS,HD
Dichlorodifluoromethane	N/A	4J - 48	7/9	K1,K4,K5,K6, K7,HS,HD
BNAs				
Benzoic Acid	N/A	3L	1/9	K3
Diethylphthalate	N/A	2J	1/9	K3
Bis(2-ethylhexyl) phthalate	N/A	3J	1/9	K3
INORGANICS				
Arsenic	50	[1.2] - 20.2L	3/9	K5,HS,HD
Chromium	50	[6.4] - 658L	4/9	K5,K8,HS,HD
Copper	N/A	[23.4] - 1300J	2/9	K5,K8,HS
Lead	50	[2.0] - 13.7	4/9	K1,K4,K5,K8
Mercury	2	0.2J - 0.4J	3/9	K1,K6,K8
Nickel	N/A	[24.6]L - 1040L	4/9	K3,K5,K8,HS
Zinc	N/A	26.0J - 98J	8/9	K1,K3,K4,K5,K6,K7,K8,HS

* - Lists the number of detections per 9 sample locations.
N/A - Compound not listed or no value reported (40 CFR 141).
J - Reported value is estimated.
L - Analyte present but may be biased low. Actual value is expected to be higher.

TABLE 5-6

COMPOUNDS/ANALYTES DETECTED IN OFFSITE MONITOR WELLS

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN PENNSYLVANIA
REM V

COMPOUND	FEDERAL MCL (ug/l)	RANGE OF CONCENTRATION (ug/l)	FREQUENCY OF DETECTION*	LOCATION**
VOLATILES (VOCs)				
Carbon Disulfide	N/A	7-8	3/26	BD,GS,GD
1,1,1-Trichloroethane	200	20J	1/26	BS
Tetrachloroethene	N/A	11-15J	2/26	BS,MD2
Total - 1,2-dichloroethene	N/A	3J	1/26	MD2
BNAs/PESTICIDES/PCBs				
Benzyl alcohol	N/A	3J	1/26	FD
Benzoic Acid	N/A	3J-4J	2/26	AD,FD
N-Nitrosodiphenylamine	N/A	4J	1/26	FD
Bis(2-ethylthexyl)phthalate	N/A	3J-7J	5/26	AI,CD,GI,MD1,MD8
Indeno (1,2,3-cd)pyrene	N/A	7J	1/26	EI
Dibenzo (a,h) anthracene	N/A	4J	1/26	EI
benzo (g,h,i) perylene				
Aldrin	N/A	0.021J - 0.16	2/26	AD,GD
4,4'-DDT	N/A	0.04J - 0.35	6/26	AS,BS,BD,CS,FS,GI
Gamma-Chlordane	N/A	0.012J	1/26	FD

Key:

* - Lists the number of detections per 26 offsite well locations.
** - Well clusters MW-F and MW-I are background locations
N/A - Analyte not listed or no valve reported (40 CFR 141).
J - Analyte present. Reported value may not be accurate or precise.
L - Analyte present. Reported value may be biased low. Actual value is expected to be higher.
[ ] - Analyte present. As values approach the IDL the quantitation may not be accurate.

TABLE 5 - 6 (continued)
COMPOUNDS/ANALYTES DETECTED IN OFFSITE MONITOR WELLS

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

ANALYTE	FEDERAL MCL (ug/l)	RANGE OF CONCENTRATION (ug/l)	FREQUENCY OF DETECTION*	LOCATION
INORGANICS				
Arsenic	50	[1.3] - [8.1]	15/26	AD, CI, CD, DI, EI, FS, FD, GS, GD, IS, II, ID, MD4, MD5, MD6,
Cadmium	10	[2.8]	1/26	BS
Chromium	50	[4.0] - 117	14/26	AS, AI, AD, BS, BD, CS, CI, CD, DI, EI, FS, FD, GD, II
Copper	N/A	[24.3] - 156	23/26	AI, AD, IS
Lead	50	[3.0] - 25.1J	5/26	AI, AD, MD10
Nickel	N/A	[17.3] - 89.7	5/26	AS, AI, AD, CS, CI
Selenium	10	[1.0]L - [4.7]	2/26	MD1, MD5
Zinc	N/A	[26.6] - 77.6J	10/26	AI, AD, DI, FS, GS, IS, MD2, MD5, MD9,

Key:

* - Lists the number of detections per 26 offsite well locations.
** - Well clusters MW-F and MW-I are background locations
N/A - Analyte not listed or no valve reported (40 CFR 141).
J - Analyte present. Reported value may not be accurate or precise.
L - Analyte present. Reported value may be biased low. Actual value is expected to be higher.
[] - Analyte present. As values approach the IDL the quantitation may not be accurate.

TABLE 5-7
COMPOUNDS/ANALYTES DETECTED IN RESIDENTIAL WELL, RW-1

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN, PENNSYLVANIA
REM V

COMPOUND/ANALYTE	FEDERAL MCL (ug/L)	CONCENTRATION (ug/L)
VOCs		
1,1-Dichloroethane	N/A	19
1,2-Dichloroethene (total)	N/A	18
1,1,1-Trichloroethane	200	17
Tetrachloroethene	N/A	27
Dichlorofluoromethane	N/A	16
BNAs		
Di-n-butylphthalate	N/A	0.5J
INORGANICS		
Chromium	50	3
Copper	N/A	26
Zinc	N/A	3300

Key: J - Reported value is estimated.
Note that background locations are RW-13 and -15.
N/A - Chemical not listed or no value reported (40 CFR 141).

TABLE 5-8

COMPOUNDS/ANALYTES DETECTED IN OFFSITE RESIDENTIAL WELLS

KEYSTONE SANITATION COMPANY SITE
LITTLESTOWN PENNSYLVANIA
REM V

COMPOUND/ANALYTE	FEDERAL MCLs (ug/L)	RANGE OF CONCENTRATION (ug/L)	FREQUENCY OF DETECTION*	SAMPLES LOCATION(S)**
BNAs				
Dimethylphthalate	N/A	0.4J	1/14	7
Di-n-butylphthalate	N/A	0.6J-1J	10/14	2,4,7,8 9,11,12,13,14,15
Butylbenzylphthalate	N/A	0.1J	1/14	13
PESTICIDES				
Alpha-chlordane	N/A	0.043J	1/14	11
Gamma-chlordane	N/A	0.068J	1/14	11
INORGANICS				
Cadmium	10	2	1/14	2
Copper	N/A	2.11-514	14/14	All locations
Lead	50	6-26	7/14	2,4,5,9,6,11,12
Mercury	2	0.59	1/14	11
Nickel	N/A	5	1/14	4
Zinc	N/A	4-3300	14/14	All locations

KEY:
* - Background locations are: 13 and 15.
** - Lists the number of detections per 14 sample locations.
J - Reported value is estimated.

N/A - Chemical not listed or no value reported (40 CFR 141).