DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Agency for Toxic Substances
and Disease Registry
Atlanta GA 30333

October 25, 2001

[redacted]

Mr. Craig Branchfield
Solutia, Incorporated
702 Clydesdale Avenue
Anniston, AL 36201-5328

Dear Mr. Branchfield:

Enclosed please find a copy of the health consultation for Solutia, Inc. [Aliases: Anniston PCB Site (Monsanto Company) and Monsanto Company], Anniston, Calhoun County, Alabama, dated October 22, 2001. The purpose of this exposure investigation was to assess the level of exposure to polychlorinated biphenyls for children and their families living near the facility who would most likely have been affected.

Please address correspondence to the Chief, Program Evaluation, Records, and Information Services Branch, Division of Health Assessment and Consultation, Agency for Toxic Substances and Disease Registry, ATTN: Solutia, Inc., 1600 Clifton Road, NE (E56), Atlanta, Georgia 30333.

If there are any questions, please direct them to Kenneth Orloff, the health assessor, at (404) 498-0506.

Sincerely yours,

for Max M. Howie, Jr.
Chief, Program Evaluation, Records,
and Information Services Branch
Division of Health Assessment
and Consultation

Enclosure

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DEFENDANT'S
EXHIBIT 1080

Health Consultation

EXPOSURE INVESTIGATION

SOLUTIA, INC.
[Aliases: ANNISTON PCB SITE (MONSANTO COMPANY)
AND MONSANTO COMPANY]

ANNISTON, CALHOUN COUNTY, ALABAMA

EPA FACILITY ID: ALD004019048

OCTOBER 22, 2001

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members.

This document has previously been released for a 30 day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The health consultation has now been reissued. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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HEALTH CONSULTATION

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[Aliases: ANNISTON PCB SITE (MONSANTO COMPANY)
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Prepared by:

Exposure Investigation and Consultation
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Preface

This document describes the exposure investigation (EI) conducted by the Agency for Toxic Substances and Disease Registry (ATSDR) in Anniston, Alabama. This EI is not meant to represent the community at large. The conclusions for this report are based on biological and environmental samples collected from 18 families living near the Solutia Inc. facility who volunteered to participate.

The purpose of the EI was to assess the level of exposure to polychlorinated biphenyls (PCBs) for children and their families living near the facility who would most likely have been affected. The EI is one of the series of public health activities underway in the Anniston area. Additional public health activities are planned to provide a clearer picture of exposure to PCBs within the greater Anniston community.

Background

The Monsanto Company produced polychlorinated biphenyls (PCBs) at a plant in Anniston, Alabama, from 1935 to 1971. Hazardous wastes, including PCB still bottoms, were disposed in two unlined landfill areas located adjacent to the production facility. Investigations conducted by Monsanto (now Solutia Inc.) under a Consent Order with the Alabama Department of Environmental Management (1996) documented the presence of PCB contamination in sediment samples from off-site drainage ditches and in soil samples from private residences east and north of the facility. These findings led to the remediation of off-site contaminated areas and property buyouts for some homeowners.

Recent investigations have detected elevated blood levels of PCBs in some residents of the community surrounding the Solutia facility and other neighborhoods in Anniston [1]. The source and exposure pathways by which residents have been exposed to PCBs have not been defined. Furthermore, it is uncertain whether significant exposures are still occurring. To address these issues, the Agency for Toxic Substances and Disease Registry (ATSDR) conducted this Exposure Investigation (EI).

This investigation was done to determine if a community health problem existed and to develop plans for its control. The results of this investigation are applicable only to the participants of this investigation and are not generalizable to other individuals or populations.

Methods

Target Population

Prior to conducting this investigation, staff from ATSDR and the Alabama Department of Public Health (ADPH) met with community representatives to explain the EI and solicit their input. In March 2000, ATSDR met with families who lived within a radius of approximately ½-mile of the site and invited them to participate in the EI. In order to be eligible for the study, at least one family member had to be a child between 1 and 7 years old. ATSDR staff and representatives of Community Against Pollution (CAP) went door-to-door in the designated neighborhoods to invite eligible families to participate. A total of 18 families fully participated in the EI. Environmental samples were collected from these 18 homes, and biological samples were collected from 78 residents of these homes. In addition, environmental samples, only, were obtained from one home; and biological samples, only, were obtained from two people who lived in the target area. The map in Figure 1 depicts the approximate locations of the homes that were tested; the locations of the homes on the map were slightly moved to protect the anonymity of the participants.

Biological Sampling

ATSDR staff made appointments with the residents to come to the Calhoun County Health Department (CCHD) where blood samples were collected. A few of the participants were unable to come to the CCHD. ATSDR staff traveled to the homes of these individuals to collect the blood samples.

A licensed phlebotomist collected a 7-ml blood sample from each participant using a Vacutainer® tube with no anticoagulant. After collection, the blood samples were allowed to clot for 2 hours at room temperature. The tubes were then placed on ice until they were delivered to the laboratory for analysis. Blood collection supplies and laboratory analyses were provided by the National Center for Environmental Health laboratory at the Centers for Disease Control in Atlanta, Georgia.

Blood serum samples were analyzed for 37 PCB congeners using gas chromatography/mass spectroscopy (GC/MS). Results were reported as concentrations of individual PCB congeners per unit volume of blood serum, and also as lipid-based concentrations. Individual congeners were added together to yield total PCB concentrations.

Environmental Samples

ATSDR staff used a metal trowel to collect a composite surface soil sample (0-3 inches in depth) from an area in the yard that the parent or child identified as a frequent play area. In addition, ATSDR staff used a Nilfisk® HEPA vacuum cleaner to collect an indoor house dust sample from a room frequented by family members; this was typically the living room of the house. The environmental samples were shipped by overnight mail to the Midwest Research Institute in Kansas City, Missouri, for analyses of PCB congeners and total PCBs. The house dust samples were strained through a wire mesh screen to remove food particles, fibers, and other debris prior to analyses. Soil and house dust samples were analyzed for PCB congeners using gas chromatography/mass spectroscopy (GC/MS).

Consent/Assent Form

Prior to testing, each adult and a parent or legal guardian of each minor participant was required to sign an informed consent/assent form. A separate informed consent form for environmental testing was also obtained for each house prior to testing.

Statistical Analyses

Descriptive statistics were used to characterize the blood serum and environmental media PCB data. To assess correlations between biological and environmental data, Spearman rank correlation coefficients were calculated. The correlation analyses were conducted using the lipid-based concentrations of blood serum PCBs.

Results

Biological Samples

The concentration of PCBs was determined in blood serum samples from 37 children (16 years old or less) and 43 adults. In adults, the blood PCB concentrations ranged from non-detected to 210 parts per billion (ppb). The mean concentration in adults was 14.2 ppb, and the median concentration was 2.2 ppb. In children, the blood PCB levels ranged from non-detected to 4.6 ppb. The mean concentration in children was 0.37 ppb, and the median concentration was non-detected.

Among the adults, five people had a blood PCB concentration in excess of 20 ppb; the PCB levels in these five people were: 22, 54, 93, 97, and 210 ppb. These five high values skewed the arithmetic mean of the adult population. If the five individuals with elevated PCB levels are not included in the mean, the average PCB concentration in the rest of the population of 38 adults was 3.5 ppb.

In Figure 2, blood PCB levels are plotted as a function of age. The ages of the participants ranged from 1 to 89 years old. In general, there was a tendency for blood PCB levels to increase with age, as has been reported for other populations [2,3].

In Table 1, the mean blood concentrations of individual PCB congeners are listed. The results are reported as μg/L and as ng/g serum lipid. Previous studies have shown that blood concentrations of PCBs can increase after the ingestion of large quantities of dietary fat [4]. To correct for this transient hyperlipidemia, blood serum concentrations of PCBs can be normalized by calculating them as a blood lipid concentration. As indicated, for the population of 80 people, the mean concentration of total PCB congeners was 7.80 μg/L or 1,397 ng/g lipid.

Environmental Samples

The concentration of PCBs detected in composite surface soil samples from 19 homes ranged from non-detected to 11.7 parts per million (ppm). The mean concentration of PCBs in soil was 1.4 ppm, and the median concentration was 0.60 ppm. The EPA has established a Recommended Soil Action Level - Analytical Starting Point of 1 ppm for PCBs in residential soil [5]. Soil samples from four homes contained PCB concentrations in excess of 1 ppm (11.7, 5.14, 1.31, and 1.20 ppm).

The concentration of PCBs detected in house dust samples from 18 homes ranged from non-detected to 10.3 ppm. The mean concentration of PCBs in house dust samples was 0.81 ppm, and the median concentration was 0.11 ppm. House dust samples from two homes contained PCB concentrations in excess of 1 ppm (10.3 and 1.71 ppm).

Indoor surface loading concentrations of PCBs in house dust ranged from non-detected to 31.9 $\mu g/m^2$. The mean surface loading concentration was 2.07 $\mu g/m^2$ of floor surface area, and the median concentration was 0.071 $\mu g/m^2$.

Discussion

Biological Samples

In the United States, no study of blood PCB levels in a statistically-based sample of the population has been conducted. Therefore, there is no national reference range that can be used as a comparison population for this EI. However, several studies have measured PCB levels in populations that had no known exposures to PCBs other than typical background levels. The results from these studies are listed in Tables 2 and 3.

As indicated in Table 2, mean background blood PCB levels in adults range from 3.7 to 6.8 ppb. The geometric mean and median PCB values calculated from these studies are somewhat lower, since these statistical measures of central tendency reduce the influence of individuals with unusually high PCB levels in the test population.

In comparing the results of these studies, several caveats must be considered. First, different analytical methodologies were used, which could account for some of the variability between studies. Earlier studies used packed column gas chromatography with electron capture detectors; more recent studies have used glass capillary columns with mass spectroscopy to detect individual PCB congeners. In addition, different populations were studied, and background PCB exposures of these populations could vary. In the United States, dietary intake of PCBs is thought to be the major source of exposure [6]. Therefore, regional variations in food consumption patterns could explain some of the variability in the studies. The age of the participants in these studies also varied. Since blood PCB levels increase with age, some of the variability between studies might be accounted for by the different age distributions of the study populations. Finally, these studies were conducted over the time period, 1982-1995. Since production of PCBs in the United States was halted in 1977, PCB releases to the environment have decreased. This could cause PCB blood concentrations to decrease over time, although this speculation has not been adequately documented.

In spite of these limitations and differences between studies, the results are fairly consistent. These studies suggest that mean PCB levels in blood serum from adults without unusual PCB exposures are 3 to 7 ppb, with lower levels being reported in more recent studies.

The upper end distribution of PCB levels in the general population has not been well characterized. In a review paper, Kreiss estimated that the 95th percentile PCB blood concentration in adults is 20 ppb [7]. Since this estimate was based on studies conducted in the late 1970s and early 1980s, it is likely that the 95th percentile level would be lower today. However, in the absence of a more recent estimate, ATSDR will assume that a blood PCB level in excess of 20 ppb is significantly elevated.

In this investigation, five adults had a blood PCB level (22, 54, 93, 97, and 210 ppb) that exceeded 20 ppb. It was noted that the blood serum sample from the individual with 22 ppb PCBs was hyperlipidemic (total lipid = 1,417 mg/dl; normal = 400-800 mg/dl). Following a heavy fat meal, blood PCB levels can be temporarily elevated by the transient lipidemia [4]. Therefore, the elevated blood PCB level in this individual may have been spuriously elevated by the presence of hyperlipidemia.

None of the other participants had a blood PCB level in excess of 20 ppb. If the five individuals with elevated PCB levels are excluded, the mean PCB level in the rest of the adult population is 3.5 ppb, which is within the normal background range. Therefore, these results indicate that blood PCB concentrations for most of the adult population were within the normal background range.

Relatively few studies have measured PCB levels in children. However, based on two published studies (Table 3), PCB levels in blood serum from children are about 2 to 4 ppb, which is less than for adults. In the EI population , the average blood PCB level in children was 0.37 ppb, and the median value was non detected. These levels are less than the values cited in the comparison studies.

Only 10 of 37 children tested in the EI had a detectable PCB level in their blood serum. The highest blood level detected in a child was 4.6 ppb, which was detected in an older child (a 13-year old girl). Reference ranges for PCB concentrations in teenagers are not available, but they would likely be between those for young children and adults. Nevertheless, all of the PCB concentrations detected in children from the EI were within the ranges detected in the comparison studies for children (Table 3). ATSDR concludes that none of the children tested in the EI had a significantly elevated blood PCB level.

Because PCBs are resistant to metabolism in the body, they bioaccumulate as a person ages. Therefore, blood PCB concentrations tend to increase with age. As indicated in Figure 2, all of the people with elevated blood PCB levels (> 20 ppb) were aged 45 years or older. Although blood PCB levels typically increase with age, PCB levels in excess of 20 ppb are unusual in unexposed populations, even among older adults. In a recent study, PCB levels were measured in a group of older adults (aged 50 to > 70 years old) [8, 9]. The mean blood PCB level in this population was 4.55 ppb, and the maximum level detected was 25.9 ppb.

There are 209 possible congeners of PCBs, which differ in the number and position of the chlorine atoms on the biphenyl ring structure. In this investigation, 37 PCB congeners were measured in the blood samples. As shown by the data in Table 1, the congeners that contributed most to the total blood concentration of PCBs in the participants were PCB congeners 153, 138/158, 180, 118, and 187 (IUPAC designation).

These congeners contain from five to seven chlorine atoms and are components of commercial PCB mixtures. Because these congeners contain chlorine atoms on the meta and/or para position of the biphenyl ring, they are resistant to metabolic degradation and bioaccumulate in humans [10, 11]. As a collective group, commercial mixtures of PCBs have an estimated half-life of 2 to 6 years [12]. However, the half-life of individual congeners may be even longer. For example, the half-life of PCB congener 153 was estimated to be 12.4 years [13].

In the adults with elevated blood PCB concentrations (>20 ppb), PCB congeners 153, 138/158, 180, 118, and 187 accounted for 59% of the total PCB concentrations. In the other adults, they accounted for 60% of the total PCBs. This indicates that the same PCB congeners composed the bulk of the PCBs in adults with either normal or elevated PCB levels. Therefore, variations in PCB exposure in adults appeared to be more quantitative than qualitative.

In children, the same six PCB congeners accounted for 67% of the total blood PCB concentrations. The total PCB concentrations in children were generally lower than for adults. As a result, the children often lacked detectable quantities of the minor PCB congeners that were detected at low concentrations in adults. The absence of detectable quantities of these minor PCB congeners in children increased the relative proportion of the major congeners.

Correlation Analysis

To assess the relationships between blood PCB concentrations and age and length of residency at current address, Spearman rank coefficients were calculated. Age was strongly correlated with the blood PCB level ($r_s = 0.729$, $p < 0.001$). Length of residency was also correlated with the blood PCB level ($r_s = 0.645$, $p < 0.001$). By calculating the Spearman partial correlation coefficient, it was determined that if age were controlled for, there was still a significant correlation between the blood PCB level and length of residency ($r_s = 0.310$, $p = 0.0054$). In these calculations, a participant's length of residency was equated to how long he had lived at his current address. However, many of the participants indicated that they had lived at more than one house in the area over the course of their lives. Therefore, the length of residency value may have underestimated how long some people actually lived in the vicinity of the facility. If this information had been incorporated into the calculations, the correlation coefficient between blood PCB concentrations and length of residency may have been even higher.

Nevertheless, these statistical analyses suggest that people were exposed to PCBs while living near the facility, although it is not known when the exposures occurred or what the source of PCB exposure was.

Environmental Samples

Environmental sampling at the homes indicated the presence of PCB contamination in soil and house dust samples from many of the houses. This contamination poses a potential source of exposure, since children may intentionally (pica behavior) or inadvertently ingest soil and house dust [14, 15]. Soil and house dust ingestion by adults has not been well studied but is likely less than for children [16].

To examine the correlations between the concentrations of PCBs in blood and environmental samples, Spearman rank correlation coefficients were calculated. This statistical analysis demonstrated that there was a significant correlation between the concentrations of PCBs in soil and house dust samples from individual homes ($r_s = 0.628$, $p < 0.0052$). This correlation is expected since soil typically constitutes about 50 percent of the mass of house dust [17].

Further statistical analyses failed to demonstrate a significant correlation between blood PCB levels and the concentration of PCBs in either soil ($r_s = -0.128$, $p = 0.26$); house dust ($r_s = 0.078$, $p = 0.51$); or house dust loading ($\mu g/m^2$) ($r_s = 0.046$, $p = 0.70$). Additional analyses in which the test population was divided into adults and children also failed to demonstrate any significant correlations between blood and environmental PCB levels. The absence of a correlation between blood PCB concentrations and soil or house dust PCB concentrations suggests that other sources of PCBs, besides those in soil and house dust, have contributed to the body burdens of PCBs.

Health Implications

The blood levels of PCBs that cause adverse health effects have not been well characterized. In occupational studies, exposures to PCBs have been associated with chloracne and subtle evidence of liver damage, although it is possible that polychlorinated dibenzofuran impurities in the PCBs might have contributed to the toxicity [18, 19]. Slight elevations in serum enzymes of hepatic origin, such as γ-glutamyl transferase, have been reported in workers with blood serum PCB levels of several hundreds of parts per billion [18]. However, in occupational studies, it is difficult to unequivocally attribute the observed health effects to PCBs because of the possible confounding effect of concurrent exposure to other chemicals. Nevertheless, based on one such study, a blood PCB level of 200 ppb was suggested as a no effect level [18].

Based on this criterion, only one of the EI participants had a blood PCB level that exceeded this level of concern. However, it should be recognized that occupational studies have examined only a limited number of health endpoints and have limited statistical power to detect an effect. Therefore, no firm conclusions can be drawn regarding a safe blood level for PCBs in adults.

Several studies have reported that low level PCB exposure during fetal or neonatal development can effect the infant's neurobehavioral development [6, 20, 21]. Although several limitations of these studies have been noted [22, 23], they provide evidence that the developing fetus or infants may be the most sensitive human population for PCB toxicity. However, the reported health effects occurred in populations with background exposures to PCBs, so a threshold effect level has not been defined.

Therefore, it is not possible to predict the health impact of the PCB exposures in the EI participants. Nevertheless, to minimize any potential health risks, it is prudent public health policy to reduce exposure to environmental PCB contamination.

Characterization of PCB Exposures

In the general population, the major background source of PCB exposure is from food [6]. Among foodstuffs, the major contributors to the body burden of PCBs are fish, meat, and poultry. It is likely that trace levels of PCBs in comercially-available foods have contributed to the body burden of PCBs in the EI participants. However, for those EI participants with elevated PCB levels, additional sources of PCB exposure are likely.

The EI participants with elevated blood PCB levels share several characteristics: (1) They are older adults, aged 45 and above. (2) They reported no known occupational exposure to PCBs. (3) They grew up in neighborhoods near the facility and have lived there most of their lives. (4) They ate locally-grown fruits and vegetables and locally-raised chickens and eggs. In addition, three of the five individuals with elevated blood PCB levels reported that in the past they ate clay from the neighborhood. Geophagia, including clay eating, has been reported to be a cultural or social tradition in some Southern societies [24].

Once PCBs get inside the body, they are stored in adipose tissue and are resistant to metabolic degradation. The major PCB congeners detected in the participants of this investigation were the ones with long biological half-lives. Therefore, exposures to PCBs that occurred years ago could

have contributed to the high body burdens of PCBs seen in some long-term residents of the area. This hypothesis is supported by the absence of elevated blood PCB levels in younger residents of the community (Figure2), as well as by the strong correlations between blood PCB levels and age and length of residency in the area. Furthermore, analyses of environmental data (soil and house dust) failed to show a correlation between current environmental levels of PCB contamination and blood PCB levels.

In recent years, Solutia Inc. has purchased, remediated, and restricted access to some off-site, PCB-contaminated properties. However, it is likely that prior to remediation, long-term residents of the area had exposure to environmental contamination by direct contact with contaminated soil, sediment, surface water, and air, and by indirect contact from eating locally-raised animal and plant foodstuffs. Therefore, past exposure to environmental PCBs may have exceeded current ones, and could be responsible for the elevated blood PCB levels seen in some older, long-term residents of the community.

Nevertheless, this study and others conducted by the EPA have documented that elevated levels of PCBs remain in off-site soils and sediments. Since the future use of these areas cannot be predicted, they should be remediated to prevent further human exposure to environmental contamination.

Conclusions

(1) Five of 43 adults had an elevated blood PCB level (> 20 ppb).

(2) Blood PCB levels in 37 children were not elevated.

(3) Blood PCB levels were correlated with age and length of residency near the Solutia facility.

(4) Blood PCB levels were not correlated with soil or house dust PCB levels.

(5) Available evidence suggests that PCB exposures in the past may have exceeded more recent exposures.

Recommendations

(1) Homes with PCB contamination in soil in excess of EPA action levels should be evaluated for possible remediation.

(2) Residents should minimize further exposure to areas of known PCB contamination.

Report Prepared by:

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References

(1) R. Canady, Agency for Toxic Substances and Disease Registry, Draft Health Consultation; Evaluation of Soil, Blood, and Air Data from Anniston, Alabama; February 14, 2000.

(2) SL Gerstenberger et al; Concentrations of blood and hair mercury and serum PCBs in an Ojibwa population that consumes Great Lakes region fish; Clinical Toxicol. 35 377-386 (1997).

(3) DT Miller et al; Human exposure to polychlorinated biphenyls in greater New Bedford, Massachusetts: A prevalence study; Arch. Environ. Contam. Toxicol. 20 410-416 (1991).

(4) DL Phillips et al; Chlorinated hydrocarbon levels in human serum: effects of fasting and feeding; Arch. Environ. Contam. Toxicol. 18 495-500 (1989).

(5) U.S. Environmental Protection Agency, Solid Waste and Emergency Response; A Guide on Remedial Actions at Superfund Sites With PCB Contamination; Directive 9355.4-01 FS; August 1990.

(6) Agency for Toxic Substances and Disease Registry; Toxicological Profile for Polychlorinated Biphenyls (update); November 2000.

(7) Kathleen Kreiss; Studies on populations exposed to polychlorinated biphenyls; Environ. Health Perspec. 60 193-199 (1985).

(8) H. Humphrey et al.; PCB congener profile in the serum of humans consuming Great Lakes fish; Environ. Health Perspect. 108(2) 167-172 (2000).

(9) S. Schantz et al.; Impairments of memory and learning in older adults exposed to poychlorinated biphenyls via consumption of Great Lakes fish; Environ. Health Perspect. 109(6) 605-611 (2001).

(10) Larry G. Hansen; Stepping backward to improve assessment of PCB congener toxicities; Env. Health Perspec. 106 171-189 (1998).

(11) JF Brown; Determination of PCB metabolic, excretion, and accumulation rates for use as indicators of biological response and relative risk; Environ. Sci. Technol. 28 2295-2305 (1994).

(12) JH Shirai and JC Kissel; Uncertainty in estimated half-lives of PCBs in humans: impact on exposure assessment; Science of the Total Environment 187 199-210 (1996).

(13) JF Brown et al.; PCB metabolism, persistence, and health effects after occupational exposure: Implications for risk assessment; Chemosphere 29 2287-2294 (1994).

(14) EJ Calabrese et al.; How much soil do young children ingest: An epidemiologic study; Regulatory Toxicology and Pharmacology; 10 123-137 (1989).

(15) EJ Stanek and EJ Calabrese; Daily estimates of soil ingestion in children; Environ. Health Perspect. 103(3) 276-285 (1995).

(16) EJ Calabrese et al.; Preliminary adult soil ingestion estimates: Results of a pilot study; Regulatory Toxicology and Pharmacology 12 88-95 (1990).

(17) DJ Paustenbach, BL Finley, and TF Long; The critical role of house dust in understanding the hazards posed by contaminated soils; Int J Toxicol; 16 339-362 (1997).

(18) H Ouw et al; Use and health effects of Aroclor 1242, a polychlorinated biphenyl, in an electrical industry; Archives Environ. Health 31 189-194 (1976).

(19) A. Fischbein et al; Dermatological findings in capacitor manufacturing workers exposed to dielectric fluids containing polychlorinated biphenyls (PCBs); 37 69-74 (1982).

(20) JL Jacobson et al; Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive function in young children; J. Pediatrics 116 38-45 (1990).

(21) WJ Rogan, BC Gladen, JD McKinney et al.; Neonatal effects of transplacental exposure to PCBs and DDE; J Pediatrics 109 335-341 (1986).

(22) SL Schantz; Developmental neurotoxicity of PCBs in humans: What do we know and where do we go from here? Neurotox. Teratology 18 217-227 (1996).

(23) RF Segal; Epidemiological and laboratory evidence of PCB-induced neurotoxicity; 26 709-737 (1996).

(24) MD Feldman; Pica: Current perspectives; Psychomatics 27(7) 519-523 (1986).

(25) JD Sahl et al; Polychlorinated biphenyls in the blood of personnel from an electric utility; J Occup Med 27 639-643 (1985).

(26) MS Wolf et al; Blood levels of organochlorine residues and risk of breast cancer; J Nat Cancer Inst 85 648-652 (1993).

(27) ME Hovinga et al; Historical changes in serum PCB and DDT levels in an environmentally -exposed cohort; Arch Environ Contam Toxicol 22 362-366 (1992).

(28) DJ Hunter et al; Plasma organochlorine levels and the risk of breast cancer; New Eng J Med; 337 1253-1258 (1997).

(29) HEB Humphrey et al; PCB congener profile in the serum of humans consuming great lakes fish; Environ Health Perspec 108 167-172 (2000).

(30) LP Hanrahan et al; Serum PCB and DDE levels of frequent Great Lakes sport fish consumers – A first look; Environ Research Section A 80 S26-S37 (1999).

(31) JL Jacobson et al; Effects of exposure to PCBs and related compounds on growth and activity in children; Neurotoxicology and Teratology 12 319-326 (1990).

(32) A Schecter et al; Polychlorinated biphenyl levels in the tissues of exposed and nonexposed humans; Environ Health Perspect 102 Suppl 1: 149-158 (1994).

Table 2: Blood Serum PCB concentrations (ppb) in adults

Mean	Median	Number tested	Date of testing	Location	Notes	Reference
5	4	738	1982-1984	California	pre-employment screening	Sahl et al [25]
5.8	3.9	840	1985-1986	Massachusetts	random sample - New Bedford, MA	Miller et al [3]
6.7	–	171	1985-1991	New York City	breast cancer controls	Wolff et al [26]
6.8	–	90	1989	Michigan	controls for fish eaters	Hovinga et al [27]
5.16	4.68	230	1989-1990	US	women - median age of 59	Hunter et al [28]
4.56	--	78	1993-1995	Michigan	≥ 50 years old	Humphrey et al [29]
--	1.5 (geometric mean -males)	57	1994-1995	Great Lakes	infrequent fish eaters	Hanrahan et al [30]
--	0.9 (geometric mean - females)	42				
3.7	--	66	1997	Great Lakes	Ojibwa Indians	Gerstenberger et al [2]

Table 1: Average blood serum PCB congener concentrations in Anniston residents (n = 80)

Congener number	average concentration (μg/L)	average concentration (ng/g lipid)
28	0.0111	1.90
52	0.00971	1.60
49	0.00564	0.914
44	0.00799	1.32
74	0.170	30.3
66	0.0409	7.60
101	0.0315	5.76
99	0.358	65.8
87	0.0281	5.25
110	0.0138	2.57
118	0.621	112
105	0.144	26.3
151	0.0288	5.20
149	0.0126	2.63
146	0.261	46.4
153	1.58	283
138/158	1.15	205
128	0.0408	8.08
167	0.0654	11.5
156	0.144	24.8
157	0.0393	6.82
178	0.104	18.7
187	0.441	79.7
183	0.125	22.9
177	0.127	22.2
172	0.0721	12.4
180	0.829	148
170	0.327	57.5
189	0.0118	2.05
201	0.268	47.8
196/203	0.219	39.1
195	0.039	7.14
194	0.253	44.7
206	0.141	25.3
209	0.0830	14.9
Total	7.80	1397

Table 3: Blood Serum PCB concentrations (ppb) in children

Mean (arithmetic)	Range	Number tested	Age (years)	Date of testing	Location	Reference
2.1	0 - 23.3	285	4-5	1984-1985	Michigan	Jacobson et al [31]
4.3	–	11	–	1984	US	Schecter et al [32]

Figure 2: Blood serum concentrations (ppb) in EI participants vs. age (years)

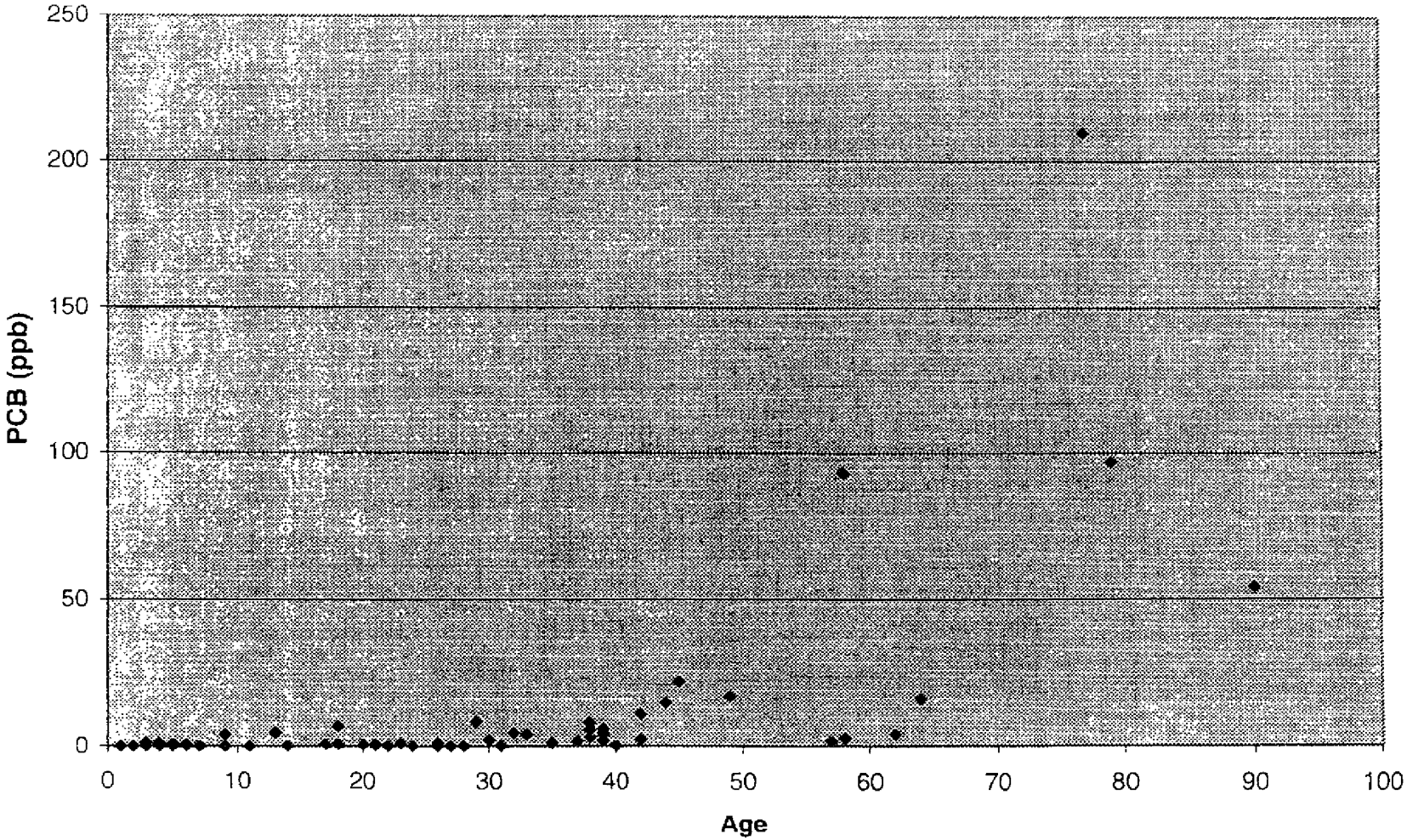
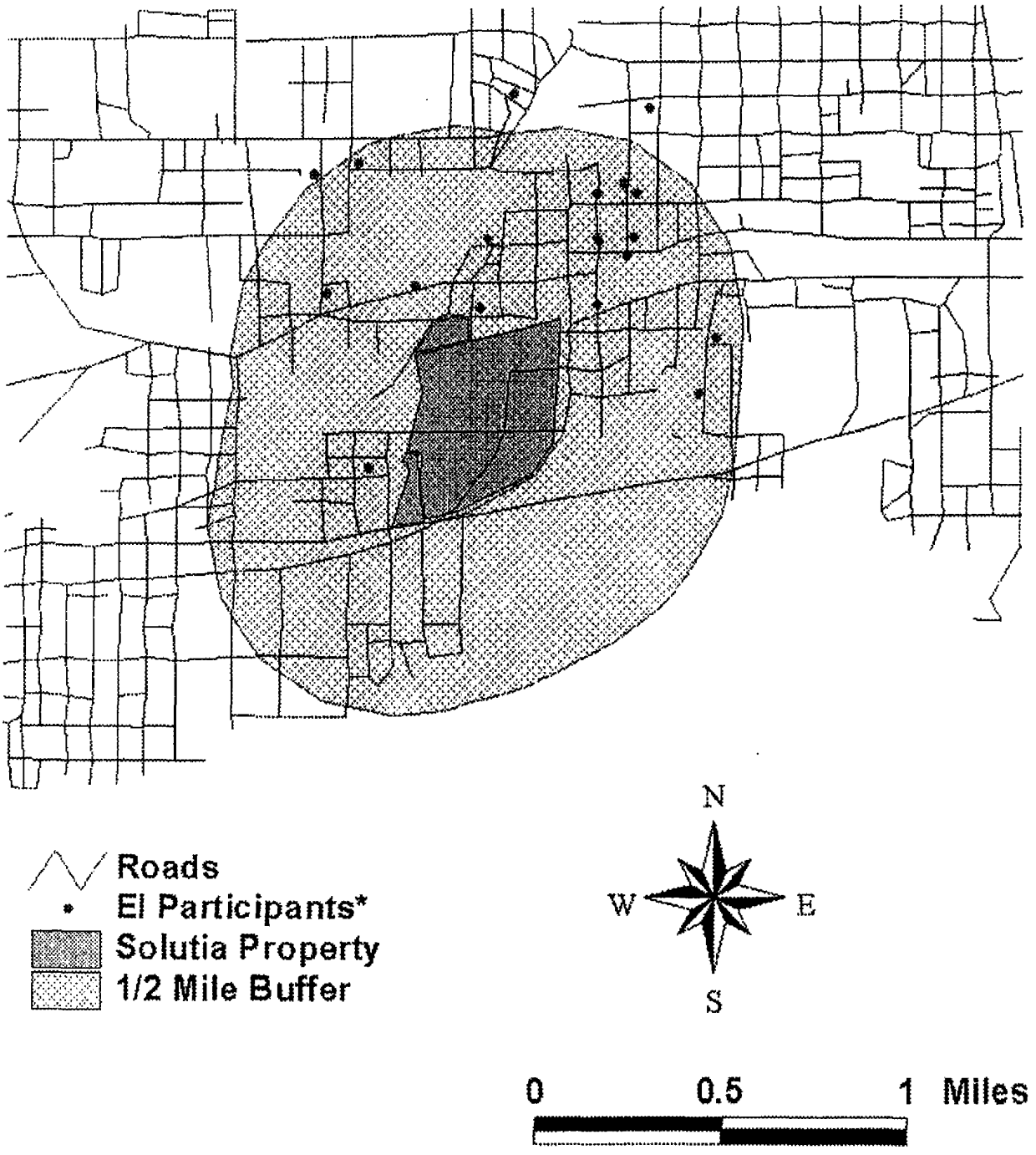


Figure 1
Location of EI Participant Homes



* the locations of EI homes have been slightly moved in random directions to protect the anonymity of participants

Appendix A

Responses to Comments Received During the Public Comment Period

Commentor 1 (The comments provided are reproduced *ver batim*):

(1) **Comment:** 1. Background
Not explicit enough

Response: Additional background information for the site is presented in numerous reports previously prepared by ATSDR and the Alabama Department of Public Health. Please refer to ATSDR's Public Comment Release Health Consultation (February 14, 2000) and the Alabama Department of Public Health's Public Comment Release Health Assessment (December 3, 1999).

(2) **Comment:** Target Population
A. The ½ mile radius is questionable due to denial of access by number of residents upon advice of their attorneys.
B. Would like to see a map of household locations, as displayed in other health consultation reports.

Response: A map indicating the approximate location of the houses tested in the EI is included in the final report.

(3) **Comment:** C. Number of children tested between the ages of 1-7 and the results chart not clear.

Response: ATSDR tested 31 children between the ages of 1 and 7 years old.

(4) **Comment:** D. What % would 18 households be of total number of households in targeted area?
E. Document number of universal households and number of children within radii from center of solutia plant.

Response: This cannot be estimated until the Year 2000 census data become avialable.

(5) **Comment:** 3. Biological Sampling
A. How many participants did the health department test?
B. How many were tested by ATSDR
C. What were the protocols for both agencies EI testing? Please submit and indicate as reference.

Response: ATSDR conducted all of the blood tests. The Calhoun County Health Department provided the facility where residents came to have blood samples drawn. The protocol for this EI is described in the Methods section of the report. A total of 80 people were tested in the EI.

(6) **Comment:** D. When did the samples arrive at CDC for testing and were holding times complied with?

Response: Sample handling procedures are described in the Methods section of the report. The samples were delivered to the laboratory within 24-48 hours of collection.

(7) **Comment:** E. May we have a copy of CDC'S actual analyzing procedure? Please submit raw data.

Response: The National Center for Environmental Health provided the following description of their analytical methodology.

Polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), non-ortho substituted or coplanar polychorinated biphenyls (cPCBs), other polychlorinated biphenyls (PCBs), persistent chlorinated pesticides and selected pesticide metabolites are measured in serum by high-resolution gas chromatography/ isotope-dilution high-resolution mass spectrometry (HRGS/ID-HRMS). Serum samples are spiked with $^{13}C_{12}$-labeled internal standards and the analytes of interest are isolated using a C_{18} solid phase extraction (SPE) procedure followed by a multi-column automated cleanup and enrichment procedure. The analytes are separated by HRCG using a DB-5 ms capillary column and quantified by ID-HRMS using selected ion monitoring (SIM) at 10,000 resolving power. The concentration of each analyte is calculated from an individual standard linear calibration. Each analytical run is conducted blinded and consists of three unknown serum samples, a method blank, and a quality control sample. After all data are reviewed using comprehensive quality assurance and quality control (QA/QC) procedures, the analytical results are reported on both a whole-weight and lipid-adjusted basis. Serum total lipids are calculated using an enzymatic 'summation' method. International toxicity equivalents (I-TEQs) are also reported for PCDDs, PCDFs, cPCBs and other 'dioxin-like' PCBs, based on the WHO-TEQ system. Detection limits, on a whole-weight and lipid-adjusted basis, are reported for each sample, corrected for sample weight and analyte recovery. The references for these analytical techniques and QA/QC procedures are listed below.

ATSDR does not release individual test results as stated in the Informed Consent Agreement with the participants.

(8) **Comment:** 3. Environmental Samples:

A. This appears to be a general assessment no specifics.
B. Please submit a copy of Midwest Research Institute procedure for testing soil and dust samples?
C. Indicate what EPA Protocol was adhered to.
D. Is Midwest Research Institute an approved EPA lab?

Response: The Midwest Research Institute provided the following information:

The procedure for analyzing PCB congeners in soils is not a standard procedure but a combination of standard procedures. The analysis follows Method 680. Basically, this is a low resolution mass spectrometry method that performs quantitation using internal standard analysis.

For preparation, the extraction procedure is soxhlet extration SW-846 Method 3540, the acid partitioning is a procedure within SW846 Method 8290 and 1613B, the Florisil procedure is SW-846 Method 3620.

EPA does not officially certify laboratories for PCB analysis. MRI has served as a subcontractor to the USEPA Office of Pollution Prevention and Toxics (OPPT) for PCB method development and analysis for the last 25 years.

(9) **Comment:** 4. Consent/Assent Form
A. No problem

Response: no response needed

(10) **Comment:** Statistical Analyses:
A. Please explain in laymen's

Response: The PCA statistical analysis was deleted. Instead, a descriptive analysis of the major PCB congeners detected in the blood samples was presented.

(11) **Comment:** Results.
1. Biological Samples:
A. Addressed concerns in target population with numbers and results.
B. If more samples were taken would have been reflective of the total community the mean and median concentrations would have been different.

Response: ATSDR offered testing to all families who lived near the Solutia Inc. facility and had a child aged 7 years old or less. The people who volunteered to participate in the EI were self-selected residents who lived within 0.6 miles of the facility. ATSDR has no way of estimating what the blood PCB levels might be in those individuals who chose not to participate in the EI.

(12) **Comment:** C. Why use blood lipid concentrations if there are few/no studies to compare with?

Response: Reporting the blood PCB concentration as a function of the blood lipid concentration reduces the variability caused by fluctuations in blood lipid levels. ATSDR believes that in future studies, more emphasis will be placed on reporting blood PCB levels as a function of blood lipid concentrations.

(13) **Comment:** D. Insert table of results within text.

Response: The final report includes a table of PCB congener concentrations in blood samples from the EI participants.

(14) **Comment:** E. Were results compared to results from community data used with ATSDR PCB Health Consultation (Feb. 2000)?

Response: This report evaluated only the data generated by ATSDR in its exposure investigation. The previous draft health consultation included an evaluation of data that was provided by an attorney who is representing residents of the area. Information provided to ATSDR during the public comment period for this health consultation suggests that some of the

information in the attorney's data base is incorrect. The final health consultation will be revised in accordance with the new information received.

(15) **Comment:** 2. Environmental Samples:
A. Were results considered a health risk/hazard or not?

Response: The purpose of this exposure investigation was to characterize PCB exposure in the community. For an assessment of the risk posed by environmental contamination, the reader is referred to ATSDR's Public Comment Release Health Consultation (February 14, 2000).

(16) **Comment:** B. Please define/explain indoor surface loading concentrations

Response: Indoor house dust samples were collected using a HEPA vacuum cleaner. The concentration of PCBs in the house dust sample was expressed as a mass concentration (milligrams of PCBs per kilogram of dust), and indoor surface loading contamination was expressed as nanograms of PCBs per square meter of floor surface area.

Indoor dust samples were tested to determine if there was a correlation between the concentration of PCBs in house dust and concentration of PCBs in blood samples. No correlation was detected in this investigation.

(17) **Comment**: Discussion
1. Biological Samples
A. Are there really populations that have not been exposed to PCBs?

Response: Low level exposures to PCBs are ubiquitous throughout the world.

(18) **Comment:** B. Is this the total universe of studies and how were they selected?

Response: ATSDR assumes that the commentor is referring to the studies in Table 1. These studies were selected using the following criteria: (1) published in a peer-reviewed scientific journal within the past 15 years, (2) procedures and analytical methodology were adequately described, (3) test subjects were residents of the United States, (4) test population had no unusual PCB exposures, (5) study provided the mean, median, or other statistical measure of central tendency in the reported blood PCB concentrations.

(19) **Comment:** C. The major source of exposure in Anniston AL. has not been identified

Response: Possible sources of PCB exposure in the Anniston community are discussed in the Characterization of PCB Exposures section.

(20) **Comment**: D. PCB level increase with age? Is this with ongoing exposure?

Response: PCBs ingested in food and from other sources bioaccumulate in the body and cause the PCB level in the blood to increase with age.

(21) **Comment**: E. ATSDR is going to assume that blood PCB level in excess of 20 ppb is significantly elevated based on a REVIEW PAPER
F. In the previous sentence you stated that today levels would be lower.

Response: As discussed in the report, this reference provides the best available documented estimate of the upper end distribution of PCBs in the general population. In an upcoming National Health and Nutritional Examination Survey (NHANES), the CDC will characterize the distribution of PCBs in the general population. However, these data are not currently available.

(22) **Comment:** G. Were the adults with elevated levels samples taken after a fatty meal intake?

Response: As stated in the report, the person with a blood PCB level of 22 ppb had an elevated blood lipid level.

(23) **Comment:** H. Please submit supporting data.
I. How can a report be accurate if certain data is omitted from report?

Response: The final report has been expanded to include a map indicating the approximate locations of the houses sampled during the investigation and a table that lists the blood congener concentrations of EI participants.

(24) **Comment:** J. Third paragraph page 6, last sentence, would this indicate that further studies and evaluations are needed.

Response: ATSDR is working with the U.S. Environmental Protection Agency, the Alabama Department of Environmental Protection, and the Alabama Department of Public Health to identify and characterize sources of environmental contamination.

(25) **Comment:** PCB Congener Analyses
A. Congener Analyses indicates ongoing exposure.

Response: There is widespread exposure to PCBs through low level contamination of foodstuffs with PCBs.

(26) **Comment**: B. As you referenced breast milk studies, fatty tissue studies, from other human studies should EI not be expanded to include these types of studies along with umbilical and placental studies also here.

Response: Measuring PCB concentrations in blood samples from a population is the most practical way of characterizing exposure in a population. It was beyond the scope of this investigation to measure PCB levels in breast milk, fatty tissue, umbilical cord, or placental tissue. Moreover, the availability of these types of tissue samples is much more limited than blood samples.

(27) **Comment:** Health Implications
A. None of participants had any occupational exposure.

Response: No response needed

(28) **Comment**: B. Who is responsible for reducing exposure to environmental PCB contamination?

Response: ATSDR is working with the U.S. Environmental Protection Agency, the Alabama Department of Environmental Protection, and the Alabama Department of Public Health to identify environmental health hazards and implement actions to remediate them.

(29) **Comment:** C. ATSDR just where do you suppose that these participants were/got exposed to/from PCBs if not from AIR, DUST, and SOIL?

Response: Possible sources of PCB exposure are discussed in the Characterization of PCB Exposures section.

(30) **Comment:** D. Please explain studies used in health implications and their significance.

Response: ATSDR has provided fact sheets to the public on the health effects of PCBs. The EI report provides references for readers who want additional information on the studies cited.

(31) **Comment:** E. How and who is responsible for reducing exposure to environmental PCB contamination?

Response: See response to comment 28.

(32) **Comment**: Environmental Samples
A. Please explain statistical analyses exposure in layman's terms.

Response: See response to comment 10.

(33) **Comment:** Characterization of PCB exposure
A. The exposure here is site specific and not just from food.

Response: This issue was discussed in the Characterization of PCB Exposures section.

(34) **Comment**: B. PCB's were banned in 1977 by the U.S. government WONDER WHY?
C. There are no more locally grown fruits and vegetables and folks don't raise chickens anymore.

Response: No response needed

(35) **Comment:** D. Did you find any fresh eggs?

Response: None of the EI participants reported that they were currently eating eggs from home-raised chickens, although some reported that they had done so in the past.

(36) **Comment**: E. If the half-life of PCB's is 2-6 yrs, explain why the adults continue to have elevated levels if there is no ongoing exposure. FDA has established safe levels for food etc.

Response: As an illustrative example of a biological half-life, assume that a person has a blood level of a chemical of 100 ppb with a half-life of 10 years. Assuming that this person has no further exposure to the chemical, the blood level of the chemical will decrease as indicated in the following table:

0 years - 100 ppb
10 years - 50 ppb
20 years - 25 ppb
30 years - 12.5 ppb

Therefore, even in the absence of any further exposures to the chemical, a person would still have a detectable level of the chemical in their blood, even though several half-lives have transpired.

All residents of the United States have continuing, low-level exposure to PCBs in food. The major sources of PCBs in the diet are fish, meat, and poultry. These dietary exposures will contribute to the body burden of PCBs derived from other sources.

The biological half-life of commercial PCB mixtures was estimated to be 2-6 years. However, this is an average half-life for the mixture of congeners. Some individual congeners have a half-life of only a few months; others have estimated half-lives of 10 years or more. The predominant PCB congeners found in human blood samples are those with long half-lives.

(37) **Comment**: F. Why is there no reference to EPA's current release of exposure into air, soil sediment, and water?

Response: These issues were not the subject of this EI. Environmental contamination data collected by the EPA was discussed in ATSDR's Public Comment Release Health Consultation (February 14, 2000), and additional health consultations will evaluate new data as they become available.

(38) **Comment:** G. No reference to EPA's current 2yr investigation with RICA and Superfund program.

Response: See response to comment 37.

(39) **Comment:** Conclusions

Your conclusions were once again, based on outdated, inadequate, not sufficient, not site specific, and irrelevant references that produced a lot of suggestive and assumptive conclusions that are not going to benefit anyone especially this community.

Response: The data and information presented in the report support the conclusions.

(40) **Comment:** Recommendations
ARE YOU SEROUS? You would have been better off to have suggested that you do nothing. With your recommendations there is no specifics as to WHO, WHAT, WHEN, and HOW anything is going to be or get done.

Response: ATSDR provided the EPA with the data gathered during the EI and requested that they consider implementing remedial actions at those properties that had environmental contamination that exceeded their clean-up criteria.

ATSDR is working with the U.S. Environmental Protection Agency, the Alabama Department of Environmental Protection, and the Alabama Department of Public Health to identify environmental health hazards and implement actions to remediate them.

Commentor 2 (The comments provided are reproduced *ver batim*):

(41) **Comment**: Thanks for the information you sent concerning testing being done in Anniston, Alabama. Monsanto Chemical plant recently changed name to Solutia facility. You stated in your report that testing has been done on residents blood, urine, soil, water, and air. I would like to know what other tests can be done?

Response: Biomonitoring for PCBs is usually done by analyzing a blood sample. In the past, when analytical methods were not as sensitive, PCBs were sometimes measured in a fat sample, which contains a higher PCB level than that found in blood. However, there is no need to analyze fat samples, since modern analytical techniques can detect PCBs in blood at a concentration of less than 1 part per billion, which is below normal background levels.

(42) **Comment**: We the victims of this horrendous disastrous afflictions caused by PCBs and other toxins that are a by-product of these chemicals? I have suffered with painful joint diseases for as long as I can remember, with something new being discovered each time I see the doctor. These chemicals stay in your adipose tissue and come out from time to time thereby entering our blood stream where it works havoc for a time then stores itself back in our adipose tissue. It (PCBs) will never leave our bodies. We will die with them, live with them, and suffer with them.

Sirs, I ask you, who do we blame for all our suffering, pain, and heartache? I'm trying to learn all I can about PCBs in order to help my children and grand children (which I only have one grand child) so far. How come all of the testing done on people and some animals for long term effects of PCBs are found to be inconclusive? Why was testing stopped in most cases? I would love to get answers to these and more questions. Can PCBs be destroyed? If so how?

P.S. Please take blood samples when persons have an out-break of skin eruptions. The PCB level will be much higher. I guarantee it.

Response: In laboratory studies, PCBs can be administered to animals in precisely measured doses and over a wide range of doses. This allows scientists to determine the relationship between exposure to a toxic chemical and an adverse health effect in the animal. In humans, it is seldom possible to determine how much PCB a person has been exposed to. Furthermore, in

occupational studies, a person is often exposed to more than one chemical, so it is not certain which chemical, if any, is responsible for an adverse health effect. In addition, many adverse health outcomes, such as cancer, can be caused by or influenced by factors other than chemical exposures. Therefore, it is much more difficult to prove a causal association between exposure to PCBs and disease in humans.

PCBs can be safely destroyed by burning them in a hazardous waste incinerator. Other techniques, such as chemical dechlorination and bioremediation, show promise but are still in the experimental stage. PCBs can also be safely disposed of in a hazardous waste landfill.

Commentor 3 (The comments provided are reproduced *ver batim*):

(43) **Comment: TITLE**

The use of the idiomatic "a/k/a" is inappropriate in a document of this type. In addition, the description of the site in the title is inaccurate and misidentifies the area to which the consultation is addressed. The name and current owner of the former PCB-manufacturing in Anniston is Solutia Inc. (The company name does not include a comma between the two words of the company name.) However, the area addressed by the Health Consultation is not the facility but rather is off-site areas in the greater Anniston area. Solutia suggests that the title of the document be changed to the following:

Health Consultation
Exposure Investigation Report
Near-site and Off-site Areas
Solutia Inc.
(formerly the chemical businesses of Monsanto Company)
Anniston, Calhoun County, Alabama
CERLIS No.ALD004019048

Response: ATSDR recognizes your concern. However, ATSDR is required to identify the site by its name in EPA's CERCLIS database, which is the Monsanto Company. Solutia Inc. has been listed as an alias.

(44) **Comment:** The current document appears to be an exact duplicate of a document dated August 18, 2000, and apparently released by ATSDR on or about that date. That version was titled as an Exposure Investigation Report, but it is not designated as a **"Public Comment Release"**. Solutia believes the existence of two essentially identical documents with different release dates, the earlier of which was not designated for comment, may lead to substantial confusion among persons with an interest in this site. ATSDR should acknowledge the earlier document in the final version of this Health Consultation and provide some explanation for existence of the earlier document.

Response: The report was released a second time in response to community's request for a public comment period so that they could comment on the report.

(45) **Comment: Preface**

In the third sentence, "residence" should read "residences" and the comma between "Solutia" and "Inc." should be deleted, as should the comma following "Inc."

Response: The preface was revised accordingly.

(46) **Comment: Background**

Page 1, first sentence: "Company" should be capitalized; the manufacturing of PCBs ceased in 1971, which is more precise that the generic "1970s".

Third sentence: The comma should be removed from the company name in the parentheses. The Consent Order under which Monsanto undertook sampling of residential properties in areas east and north of the facility was signed in 1996, not 1985 as denoted in the parentheses.

Response: The text was revised accordingly.

(47) **Comment: Results, Biological Samples:**

Page 3, third paragraph of this section: ATSDR correctly notes that blood concentrations of PCBs can be influenced by the intake of dietary fat. It is for this reason that most protocols for taking blood samples for PCB analyses specify that the subject should be in a fasting state, i.e., the subject should not have eaten on the day of the sampling prior to the blood being drawn. Were the blood samples in this exposure investigation taken from subjects in the fasting state?

Response: In investigations of this type, it is not practical to obtain fasting blood samples from the participants, who include young children. However, variations in blood PCB levels due to blood lipid levels would be expected to be comparable to those reported in other studies. It should be noted that the comparison studies cited in Table 1 and 2 were not conducted with blood samples from fasting individuals.

(48) **Comment: Discussion , Biological Samples:**

Page 4, second paragraph of this section and Table 1: Table 1 provides useful information about some of the many studies which have measured PCB levels in the blood of nominally unexposed populations. The table would be much more useful as a reference if ranges of PCBs levels in the subjects were included as a column in the table. Such information on the distribution of PCB levels in populations would provide additional context in which to consider the results presented in the Health Consultation.

Page 5, last full paragraph and Table 2: It is noted that Table 2 does include a column for the range of blood levels reported in the two studies noted. Such a column would be a useful addition to Table 1, as noted above.

Response: The amount of information presented in the Tables was limited by available space. The information referred to can be obtained from the original references.

(49) **Comment: PCB Congener Analysis**

Page 6, last paragraph and Page 7, top: Although the discussion implies that the two clusters identified in the cluster analysis of the PCB congener specific analytical data are largely, if not exclusively, a function of PCB concentration, ATSDR should make this point more clearly. Cluster A appears to be primarily the low-concentration samples, whether for adults or children, in which the lower concentration congeners were at concentrations less than the detection limits for those congeners. In Cluster B, the total concentrations of PCBs were high enough that those relatively less-concentrated congeners were present at levels above their respective detection limits.

Response: The PCA statistical analysis was deleted. Instead, a descriptive analysis and discussion of the major PCB congeners detected in the blood samples from adults and children was presented.

(50) **Comment: Characterization of PCB Exposures**

Page 9, second paragraph, first sentence: The phrase "the potentially responsible party" implies that Solutia is the only PRP associated with this site. While Solutia has undertaken extensive sampling and remedial activities in the near-site and off-site areas, there has been no determinaton that other PRPs may not have contributed to PCB levels in soils and sediments. Solutia suggests replacement of the phrase with "Solutia Inc."

Response: The text was revised accordingly.