ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Air Exposures to Wood Treatment Chemicals

KERR-McGEE CHEMICAL CORPORATION
COLUMBUS, MISSISSIPPI
EPA FACILITY ID: MSD990866329
OCTOBER 21, 2009

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was 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 public health assessment has now been reissued. This concludes the public health assessment 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.

Agency for Toxic Substances & Disease Registry ..... Julie L. Gerberding, M.D., M.P.H., Administrator
Howard Frumkin, M.D., Dr.P.H., Director

Division of Health Assessment and Consultation…. ..... William Cibulas, Jr., Ph.D., Director
Sharon Williams-Fleetwood, Ph.D., Deputy Director

Health Promotion and Community Involvement Branch……..Hilda Shepeard, Ph.D., M.B.A., Chief

Exposure Investigations Site Assessment Branch ..... Susan M. Moore, M.S., Chief

Site and Radiological Assessment Branch ..... Sandra G. Isaacs, B.S., Chief

Cooperative Agreement and Program Evaluation Branch ..... Richard E. Gillig, M.C.P., Chief

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PUBLIC HEALTH ASSESSMENT

Air Exposures to Wood Treatment Chemicals

KERR-McGEE CHEMICAL CORPORATION
COLUMBUS, MISSISSIPPI
EPA FACILITY ID: MSD990866329
OCTOBER 16, 2009

Prepared by:

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

Summary and Statement of Issues

This Public Health Assessment document focuses on air exposures only; other documents will focus on other pathways. This Public Health Assessment utilizes the new data learned about exposures from the air pathway. Because most of the new information is not available in the Agency for Toxic Substances and Disease Registry's (ATSDR's) Toxicological Profiles or Environmental Protection Agency guidance, we added the new technical data to this document.

Kerr-McGee Chemical Corporation, Forest Products Division (Kerr-McGee) owned the Columbus MS wood treating site. The facility manufactured pressure treated railroad products such as wooden crossties, switch ties, and timbers from approximately 1928-2003. Residential and industrial properties surround the site. In November 2004, approximately 1850 linear feet of the ditches near the Kerr-McGee facility were remediated [ERG 2005].

Kerr-McGee produced treated railroad products that used creosote and creosote coal tar solutions. The facility also used pentachlorophenol for wood treating from the 1950's until the mid 1970's [Dahlgren 2003]. Both of these processes exposed people to airborne contaminants: The pentachlorophenol process exposed people to pentachlorophenol and to a much lesser degree to dioxin. The creosote process exposed people to polycyclic aromatic hydrocarbons (PAHs).

Airborne levels of pentachlorophenol were likely to be lower than 60 $\mu g/m^3$ on average. Little dioxins are expected to have been released into the air.

Airborne levels of naphthalene were also likely to be lower than 60 $\mu g/m^3$ on average and probably below the highest level measured at the fence-line (13 $\mu g/m^3$). Little of the other PAHs are expected to have become airborne.

Studies on exposures to these chemicals indicate that little body burden occurs from the air pathway as compared with touching or ingestion.

ATDSR concludes:

Pentachlorophenol that was released into the air contributed to a low theoretical health risk (from 1950-1975), with no observable adverse health effects expected. During that time, most Americans had unacceptable pentachlorophenol exposures because of pesticide use. Any air releases would have contributed to the exposures.

- Naphthalene released into the air during the creosote treatment process posed a risk of respiratory irritation.
- Small amounts of naphthalene and associated chemicals are released when it rains because rainwater fills the pore spaces in the soil and pushes the vapors out. While unpleasant, these levels do not pose a health risk.

ATSDR Recommends:

- That residents remove any treated wood that may be in the home (regardless of the wood's origins).
- That the property owner remove or cover those soils that have strong odors.

Background and Statement of Issues

This document focuses on air exposures only; other documents related to this site will focus on other pathways. The petition to evaluate exposures near the Kerr-McGee facility came at a time when we just began to research the fate and transport of air emissions for wood treatment.

The Agency for Toxic Substances and Disease Registry (ATSDR) wanted to utilize the new data from the then ongoing research, but delays in those results impacted on the trust of the community. Therefore, ATSDR chose to complete the Health Assessment to address the soil and water pathways separately in order to be more timely. This Public Health Assessment utilizes the new information learned about exposures from the air pathway. Because most of the new information is not available in ATSDR's Toxicological Profiles or Environmental Protection Agency guidance, we added the new technical data to this document. The report is organized with an introductory paragraph for each section. The introductory paragraph summarizes the application of the new information. Elaboration of the new science then follows.

Site Description and History

Kerr-McGee Chemical Corporation, Forest Products Division (Kerr-McGee) owned and operated a wood preserving facility in Columbus, Lowndes County, Mississippi. The site occupies approximately 90 acres. Residential and industrial properties surround the site. The facility has been in operation since approximately 1928. The facility manufactured pressure treated railroad products such as wooden crossties, switch ties, and timbers. Kerr-McGee purchased the facility from Moss American Corporation in 1968. Kerr-McGee closed the facility in 2003. In November 2004, approximately 1850 linear feet of the ditches near the Kerr-McGee facility were remediated [ERG 2005].

Kerr-McGee produced treated railroad products that used creosote and creosote coal tar solutions. The facility also used pentachlorophenol for wood treating from the 1950's until the mid 1970's [Dahlgren 2003].The facility previously maintained an unlined surface impoundment, as part of the wastewater treatment system, to settle out solids and preservatives from the process wastewater prior to final discharge to the city publicly owned treatment works. Preservative was removed and recycled back to the production process in accordance with a closure plan approved by Mississippi Department of Environmental Quality (MDEQ); Kerr-McGee closed the impoundment in 1986.

In latter years of operation, the facility instituted several modifications designed to reduce the potential for impact on the local environment. This includes upgrading the process oil/water separators, installation of a concrete drip track that meets §40 CFR Part 264, Subpart W standards and the installation of concrete containment systems for the tank farms. In conjunction with the production changes, the facility has excavated visually impacted soils from the drip track, work tank and black tie storage areas.

Demographics and Land Use

Approximately 8,976 people live within 1 mile of the Kerr-McGee site. Approximately 1,030 persons living within one mile of the site are children under the age of six. Approximately 1,166 persons are age 65 or older [Census 2000].

Six public school sites are located within approximately one mile of the facility. The nearest school to the site is Hunt Intermediate School. Hunt Intermediate is located west of the site. Hunt Intermediate school has approximately 863 students in grades 5 through 6 [NCES 2003]. The other schools located within approximately one mile of the site are Stokes Beard Elementary School, S.D. Lee Intermediate School, Hughes Alternative School, Union Academy, and Joe Cook Elementary. Approximately 16 daycare facilities are located within a mile of the site [NCES 2003]. The closest hospital is Baptist Memorial Hospital - Golden Triangle, located 1.6 miles from the site. It is a 328 bed hospital and a Level 2 trauma facility [VRISK 2006].

Environmental Data Reviewed

The environmental data used for evaluating air exposures includes:

- Fence-line air data at Kerr-McGee for pentachlorophenol and polycyclic aromatic hydrocarbons (PAHs);
- Fence-line and community data from other operational facilities for pentachlorophenol and PAHs;
- TRI data from Kerr-McGee for PAHs;
- TRI data from other operational facilities for pentachlorophenol and PAHs;
- Blood-dioxin samples from community members;
- Soil and dust samples for PAHs;
- Worker exposure data for Kerr-McGee;
- Various exposure studies.

Summary of the Exposure Conclusions

Kerr-McGee had two processes that exposed people to airborne contaminants, the pentachlorophenol process and the creosote process.

The pentachlorophenol process exposed people to pentachlorophenol and to a much lesser degree to dioxins. The creosote process exposed people to polycyclic aromatic hydrocarbons (PAHs). We expect that exposures caused some respiratory irritation, but the exposures were lower than levels known to cause permanent health effects.

The Pentachlorophenol Process: The pentachlorophenol process can cause exposures to pentachlorophenol and dioxins. Air exposures to pentachlorophenol in the community were higher than those found in the general population, but not as high as those found in workers; and there is no evidence of significant airborne dioxin exposures.

In the 1970's, most of the US population was exposed to pesticides through their diet. Exposure to pentachlorophenol through the air pathway would have added to an already unacceptable body burden of pentachlorophenol. While no adverse health effects were proven to be directly linked to the presence of pentachlorophenol in food, pentachlorophenol was among the pesticides with associated effects [ATSDR 2001]. We expect that peak airborne exposures were likely to be below 1200 μg of pentachlorophenol per day and average airborne exposures were likely to be less than 200 μg/day. This exposure is in addition to the average dietary levels of 760 μg/day, which is a level that we expect the body can eliminate effectively. Individuals who may have contacted the pentachlorophenol in the ditches (addressed in ATSDR 2007) would have had additional exposures.

Although no health effects are expected, the past exposures from the site, from diet, and from other potential sources combined contribute to an unnecessarily high body burden of pentachlorophenol that posed a low theoretical health risk.

How Exposures were Estimated:

We estimated exposures by three different methods, namely:

1. By comparing ratios of exposure markers in people who lived near Kerr-McGee to exposed populations and unexposed populations, we determined that past exposures pentachlorophenol were elevated, but not likely to be at levels known to pose adverse health effects; we also determined that dioxin exposures are and were limited;
2. By comparing emissions data collected at Kerr-McGee to other sites, we estimated that peak air concentrations of pentachlorophenol were below 50 $\mu g/m^3$ (creating a maximum daily dose below 1000 μg) and average concentrations were below 10 $\mu g/m^3$ (creating an average daily dose below 200 μg);
3. By comparing modeled ratios of source strength at Kerr-McGee to the concentrations at the closest residence, we estimated that peak air concentrations of pentachlorophenol were below 60 $\mu g/m^3$ (creating a daily dose less than 1200 μg/day).

Method 1: Comparing Ratios of Biological Markers

Dioxin[1]

Four dioxin congeners are highly associated with high and moderate exposures to pentachlorophenol [Päpke 1992, Dahlgren 2003, Dahlgren 2006, Karouna-Reiner 2007]. Dioxin concentrations were as high as 100 ppm in the pentachlorophenol produced in the 1970's [Kerkvliet 1985]. Dioxin was an accidental by-product of the pentachlorophenol production process. Because of this, we expect to see 10,000 times less dioxin than pentachlorophenol in the soil after a spill (one reason that we express dioxin in units that are 1000 times smaller than those used for pentachlorophenol). Dioxin remains in the environment and inside people for a very long time. Dioxin remains in the blood 32-36 years after exposure [Päpke 1992; Shecter 1988]. Pentachlorophenol does not remain in the environment as long as dioxin, and it is removed from the body within days [ATSDR 2001]. Because pentachlorophenol degrades faster than dioxin, we expect the amount of dioxin to decrease at a slower rate than pentachlorophenol. The sediment near Kerr-McGee had both pentachlorophenol and dioxin at a ratio of 200 to 1 (ranging from 50-1 to 500-1) [Foster 2007].

Because of dioxin's persistence, blood dioxin could be an indicator of a possible pentachlorophenol exposure.[2] While there are many sources of dioxin, pentachlorophenol exposure is highly associated with four dioxin congeners (1,2,3,4,6,7,8-HpCDD, OCDD, 1,2,3,4,7,8-HxCDF, and 2,3,4,6,7,8-HxCDF). Because dioxin is much less volatile than pentachlorophenol, much less of it may enter the air, and therefore, it may not be a sensitive enough marker for air-only exposures. However, data suggests that it is a marker because of the persistence of dioxin. The ratio of dioxin to pentachlorophenol in the air is 2,000 to 1 ratio and the ratio in blood about a 200 to 1 ratio [Päpke 1989, ATSDR 2001, Gebefugi 1976, Oehme 1986].

Ten residents who had lived for more than 25 years near Kerr-McGee had TEQ's[3] that ranged from 19.6–37.1 ppt [Dahlgren 2003]. These levels can be compared with several other possible reference ranges, including other communities near wood treating facilities and the general population. In a study near another operational pentachlorophenol plant, Dahlgren found 29 adult residents (mean age of 51 years) with an average combined TEQ of 33 ppt [Dahlgren 2006]. Both of these groups compare to a study of non-exposed adults aged 45 through 59, whose TEQs, ranged from 0.8–55.4 ppt; a subsection of the group within Patterson's 2004 study

[1] Refers to a group of numerous chlorinated dioxins and furans, including: Octachlorodibenzo-p-dioxin (OCDD); 1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (1,2,3,46,7,8-HpCDD); 1,2,3,4,7,8 Hexaxchorodibenzofuran (1,2,3,4,7,8-HxCDF) ; and 2,3,4,6,7,8 Hexaxchorodibenzofuran (2,3,4,6,7,8-HxCDF).

[2] Elevated levels of Dioxin is a possible marker of pentachlorophenol exposure, but there are many sources of dioxin other than pentachlorophenol and therefore, elevated dioxin could indicate exposure to another source.

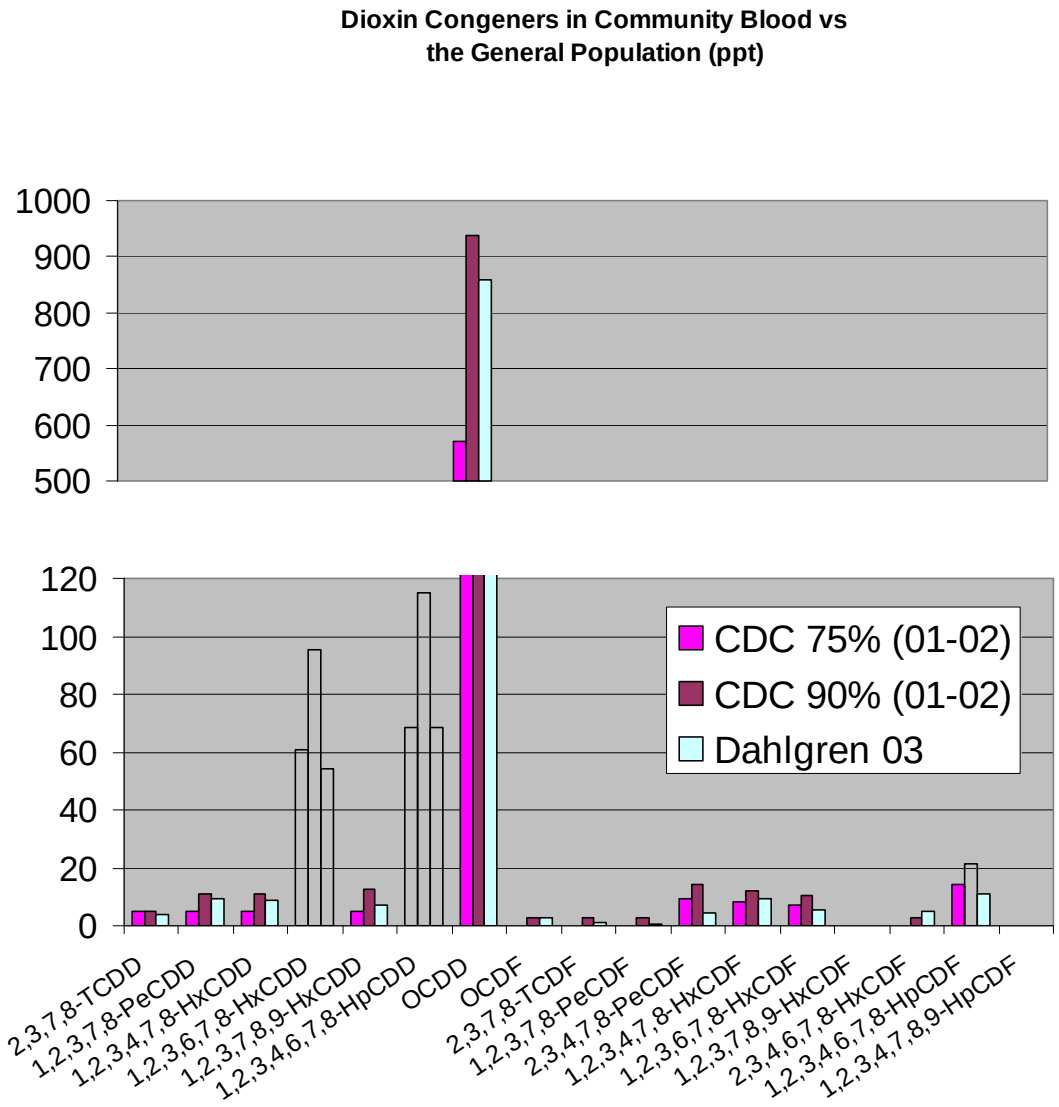
[3] The Word Health Organization provided a weighting of dioxin congeners to estimate toxicity in 1998 called Toxic Equivalents (TEQs). The weight factors associate each dioxin congener with the very toxic congener 2,3,7,8-Tetrachlorodibenzo-p-dioxin. This congener was not elevated in blood from the Kerr McGee residents.

had WHO 2002 TEQ's[1] which ranged from 11.8–36.1 ppt. All these values contrast with the results of a fifth study that included 9 workers and 37 residents living near a pentachlorophenol wood treatment plant in Florida where the TEQ's ranged from 8.4 to 711.1 ppt [Karouna-Reiner 2007]. The higher concentrations reflect the fact that there were some workers included in the study. The highest TEQ level was 19 times higher than the Kerr-McGee residents. Comparing the TEQ's of the study groups, we conclude that the Kerr-McGee residents do not have an elevated body burden of all dioxins.

While the TEQ results report the overall toxic burden, congener-specific analysis sometimes indicates if there are unusual exposures to a source of dioxins. Elevated 1,2,3,4,6,7,8-HpCDD, OCDD, 1,2,3,4,7,8-HxCDF, and 2,3,4,6,7,8-HxCDF would be expected from pentachlorophenol-treated wood exposures. Since pentachlorophenol workers were found to have the greatest levels of many congeners, with OCDD blood levels of 300,000 ppt, and because levels of many of these congeners remain in the blood for decades, it is likely that lower exposures would result in a detectable fingerprint years later [Päpke 1992]. However, a definitive fingerprint was not apparent in the blood of the residents. The average congener-specific blood dioxin values of the residents near Kerr-McGee are compared to the general population in Figure 1 [CDC/NCEH 2005].

[1] Data provided by Patterson used the WHO's newer 2002 TEQ assessment, which has some changes in the calculation of the relative toxicities of the congeners compared with the TEQs by WHO in 1998.

Figure 1: Blood Dioxin: The Kerr-McGee Neighbors and the General Population



CDC 75% (01-02) = the 75th percentile of the general population as represented by 1237 people in the years 2001 and 2002

CDC 90% (01-02) = the 90th percentile of the general population as represented by 1237 people in the years 2001 and 2002

Dahlgren 03 = the average of the 10 residents near Kerr-McGee [Dahlgren 2003]

The chart was split to compare the congener values on a single page

The Kerr-McGee neighbor's concentrations averaged below the 75th percentile of the general population and were below the 90th percentile for all except one congener, 2,3,4,6,7,8-HxCDF (at 10.1 ppt). This congener is associated with pentachlorophenol, therefore, it might indicate that there was some exposure. However, 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,7,8-HxCDF, and OCDD were not remarkably elevated. Therefore, if there were exposures they were much less than those of workers.

Karouna-Reiner et al. (2007) studied a community near another wood treatment plant in Florida. That community included workers and non-workers. In contrast with the Kerr-McGee population, the Karouna-Reiner et al. (2007) study's highest 2,3,4,6,7,8-HxCDF level was nearly 6 times higher (at 60 ppt). Furthermore, 1,2,3,4,7,8-HxCDF was 16.5 ppt, 1,2,3,4,6,7,8-HpCDD was 281 ppt and OCDD was 1622 ppt (also much higher). These levels are indicative of a worker and are much higher than levels found in the population near Kerr-McGee. Congener profiles of other highly exposed individuals can be found in Appendix A.

Because blood dioxin levels measured in the older residents near Kerr-McGee were within the normal range of TEQ's and the congener profiles were similar to the general population, we do not expect that there were harmful dioxin exposures to residents from the air pathway.

We do not expect that the residents were highly exposed to pentachlorophenol because elevated dioxin levels are generally found in people with elevated pentachlorophenol exposure.

Method 2: Comparing Emissions Data Collected at Kerr-McGee to Other Sites

It is possible that airborne pentachlorophenol levels near Kerr-McGee could have occasionally been as high as low-level worker exposure (air levels = 10-100 µg/m^3), but there is no evidence to suggest that it was as high as highly exposed workers (60-1000 µg/m^3) [Arsenault 1976; ACGIH 2001]. This estimate was derived by comparing operations at Kerr-McGee with the few available residential studies and contrasting them with the many worker studies.

There have been three recent investigations of communities near comparable pentachlorophenol wood treating facilities in Georgia, Mississippi, and Oregon. Each study is limited. Air was only sampled at two sites and biological markers of exposure (urine and blood) were only sampled at two sites. Pentachlorophenol was detected in the air in Georgia, but not detected in Oregon; and biological markers of exposure were detected in Georgia and Mississippi [Stone 2004, Zarus 2004, 2007, Dahlgren 2004, 2006].

While no air data are available for the operational facility in Mississippi (not Kerr-McGee), the blood results indicate that residents have low exposures to pentachlorophenol [Dahlgren 2004, 2006]. Urine data from the Georgia site also indicates low-level exposures from an operational plant where air data was also collected [Zarus 2007]. The pentachlorophenol levels in urine (in Georgia) and the blood (in the other Mississippi site) were much lower than those found in wood-workers; neither community had pentachlorophenol levels as high as people who lived in pentachlorophenol-treated log homes; and all were much lower than levels associated with health effects [CDC 1980, ATSDR 2001].

Using several emission and uncertainty factors, designed to provide a reasonable over estimate of emissions, we calculate that it is possible that the highest airborne concentration at Kerr-McGee could be 1.67 times higher than the highest level measured in other communities.

Using the highest maximum (30 µg/m^3) and highest average (<6 µg/m^3) at these sites, the following calculations are made:

max concentration 30 µg/m^3 x 1.67 = 50 µg/m^3

average concentration of: <6 µg/m^3 x 1.67 = <10 µg/m^3

This is within the range of less-exposed workers (air levels = 10-100 µg/m^3) and near the maximum (of 69 µg/m^3) measured at 21 of the 28 plants where worker air exposures were investigated [ACGIH 2001; Wyllie 1975; Arsenault 1976; Zober 1981]. Additional supporting data are provided in Appendix B and Appendix C.

Method 3: Comparing Modeled Ratios

Fugitive emissions pose large uncertainty for modeling. Uncertainty is larger when those emissions have a large seasonal variation (due to volatility and photo-degradation). A contractor for Kerr-McGee estimated their emissions using measurements, calculations from mass balance, and production data along with years of meteorological data [Hoffnagle 2001]. The average modeling results compared well with the average sampling results, which suggests that we can use the models to calculate long-term average exposures. However, the maximum and minimum at any one location did not compare well, indicating that the model cannot be used to predict an individual's peak exposures. The maximum 24-hr predicted value[1] for all sampling sites was 49 µg/m^3 and the maximum measured was 13 µg/m^3; however, the measured maximum 24-hr measured for each site varied by a factor of 10 to 27. Therefore, while the model appeared to over predict the values measured at the limited sampling locations, the data suggests that there is much more spacial variability than predicted.

Naphthalene can be used as a pentachlorophenol surrogate by the use of ratios. Then, these ratios can be used to determine potential pentachlorophenol emissions and the resultant downwind concentrations. Since the same production rates of creosote and pentachlorophenol occurred at the three other sites (Oregon, Georgia, and the other Mississippi site) as Kerr-McGee, the emission ratios are comparable. Studies indicating similar concentration levels of pentachlorophenol and naphthalene support the comparison, pentachlorophenol in the wood is at 5-6000 ppm [Päpke 1989; Oehme 1986] and naphthalene in creosote logs is at 650-13,000 ppm [ATSDR 2001].

Both pentachlorophenol and naphthalene are somewhat volatile, but very dependant on temperature. Pentachlorophenol, being nearly a solid, is less likely to volatilize. This is confirmed as studies find that naphthalene emission are higher at creosote facilities than pentachlorophenol emission at pentachlorophenol facilities, with ratios of pentachlorophenol to

[1] Naphthalene is used as a surrogate for pentachlorophenol in this case.

naphthalene in communities less than 1/1 and average near 1/1.8 [Elovaara 1995, Heikkilä 1997, Zarus 2004, ATSDR/ORDOH 2006, ATSDR 2001].

By using naphthalene measurements and model estimates from Hoffnegle (2001) and factoring to account for ratios of pentachlorophenol to naphthalene (1/1.8) at wood treatment sites, and scaling of the emission rates to account for the maximum production volume plausible at Kerr-McGee, we calculate a maximum concentration of 60 μg/m^3.[1]

Discussion of the Kerr-McGee Pentachlorophenol Exposures

All three estimation methods produced worst-case past air concentrations less than 60 μg/m^3 and average daily levels less than 10 μg/m^3. These levels would produce a peak dose of 1200 μg/day and average dose less than 200 μg/day,[2] which is close to the levels that US citizens used to get from eating foods that contained pesticides. Since the total dose from all pathways (past and present) are much less than low-level exposed workers, who have not been shown to suffer adverse effects, no pentachlorophenol toxicity effects are expected in the population near Kerr-McGee.

Before comparing doses, it is important to note a few issues with the metabolism of pentachlorophenol (discussed in more detail later):

- Urine is a better marker (than blood) of total recent exposures until a person is exposed to a point where they cease to excrete pentachlorophenol efficiently;
- Most people can effectively remove 1000 μg of pentachlorophenol in each liter (L) of urine excreted [Begley 1977].

Assuming a standard elimination rate of 2 liters/day, a person can effectively eliminate 2000 μg/day, but somewhere between 2000 μg/day and 3000 μg/day, they begin to accumulate pentachlorophenol in their blood. In some cases, blood and urine levels continue to rise after daily exposure [Begley 1977, Zober 1981, ACGIH 2001]. Therefore, exposures below 2000 μg/day (for adults) compare differently from levels above 2000-3000 μg/day. Based on several factors (discussed later), doses for children will be much lower, perhaps averaging 1000 μg/day [CDC 1980].

The U.S. EPA estimated that 6 μg of pentachlorophenol was inhaled by the general population each day [EPA 1980]; this is much lower than our estimate for inhalation near Kerr-McGee of 200 μg/day. However, an inhalation dose of 200 μg/day is much lower than the FDA's estimate of 760 μg/day of pentachlorophenol from food (for all of the population at that time). Many efforts were made during the 1980's to reduce pesticides in foods, which resulted in much lower levels within that decade. Since pentachlorophenol regulations, the total daily dose dropped to 16 μg, with root vegetables contributing most [Hattemen-Frey and Travin 1989]. We expect much less today as little or no pentachlorophenol is detected in the majority of the US population today.

[1] Assuming production rate of 385,000 six ft ties: 49 μg /m^3 x 2.20 (max logs) x 1/1.8 (conversion) = 60 μg /m^3.
[2] Air concentration x inhalation rate = 60 μg /m^3 x 20 m^3/day = 1200 μg /day.

Workers at wood treatment plants were estimated to have daily exposures ranging from 900-14,000 µg/day [ACGIH 2001]. This is because workers have multiple routes of exposure and their inhalation exposures continue daily. This contrasts with residential exposures because once in the air, wind will disperse pentachlorophenol and on some days will direct pentachlorophenol away from residents, reducing average exposures.

The urine levels and blood levels of pentachlorophenol measured in communities adjacent to operational wood treatment plants (similar to Kerr-McGee) are lower than the levels measured in the general population in 1980, and much lower than those who lived in log homes (summarized in Appendix D). We see little body burden in these communities (where most of the exposures are from the inhalation pathway). The opposite is observed in nearly all the worker studies. The worker's urine levels (and blood levels) are high, while the air levels are low. This suggests that a worker's exposure is mostly from pathways other than air, as there is more pentachlorophenol in the urine going out than is inhaled. If a worker excreted 2000 µg/day, he would have to inhale 20 m^3 of air (a days worth of breathing) with 100µg/m^3 of pentachlorophenol.

We would expect the Kerr-McGee neighbors to have exposures that resemble those of the other two resident communities. As in all cases, the access to the logs was limited. The inability to touch the logs and the changing wind patterns moderated the exposures near the treatment plants, giving the residents' bodies time to remove the pentachlorophenol –thus reducing their body burden.

As measured in other communities, exposures should vary daily and peak levels cannot be experienced more than 18% of the time due to wind variability alone (See Appendix E).

By contrast, the people living in treated log homes (with urine levels of 2-87µg/L and blood levels of 116-1084 µg/L) had internalized more pentachlorophenol than the residential communities near treatment plants. We expect that the higher levels in the log home residents were associated with the many exposure pathways; people living in log homes can touch the logs. Furthermore, living inside the log home gives no time to recover from inhalation exposure (all relevant studies are summarized in Appendix D).

Health Effects Associated with Pentachlorophenol Exposures

There are no studies that find an association of *low-level* worker pentachlorophenol (200-2000 µg/day) inhalation and cancer in humans, despite a long history of worker and residential exposures to pentachlorophenol [ATSDR 2001]. The only health effect observed in residents of treated log homes was a rash, which went away after exposure was reduced [CDC 1980]; however, the populations were too small to study appropriately.

There are *occupational* studies that suggest that *high-level* exposures to pentachlorophenol (2000-20,000 µg/day) treated wood are associated with non-Hodgkin's lymphoma[1] [Hertzman

[1] Non-Hodgkin's lymphoma includes a large group of cancers of the immune system.

1997, Hardel 1994]. There is a study indicating that higher exposed workers have higher rates of chloracne[1] [Hryhorczuk 1998]. However, those workers were also exposed by touching the pentachlorophenol and accidentally ingesting small amounts in addition to their high air levels.

One study determined that the ingestion of fish and water with high levels of pentachlorophenol was associated with non-Hodgkin's lymphoma [Lampi 1992]. There are several animal studies that associate ingestion of pentachlorophenol with cancer in animals [ATSDR 2001 –p65].

The ACGIH has calculated that a worker can absorb a total of 3000 µg/day pentachlorophenol and have little tissue uptake because of the body's ability to eliminate it [ACGIH 2001]. However, one worker study revealed that 1000 µg/day might be the mean level associated with a decrease in renal function [Begely 1977], as shown in an increase in urinary pentachlorophenol excretion and a decrease in creatinine excretion. That study showed that renal function was restored once exposure was reduced. Therefore, adult men can eliminate pentachlorophenol effectively until they are exposed to more than 2000 µg/day before effects are observed, but those effects go away after exposure stops. While it is possible that Kerr-McGee neighbors could have had peak exposures above 200 µg/day, it is unlikely that inhalation alone could produce levels as high as 2000 µg/day.

Additionally, residents in communities have time to eliminate pentachlorophenol after peak levels are inhaled. The fact that the log home dwellers had a larger body burden than levels near wood treatment facilities (despite higher air levels), suggests that people receive lower body burdens by peak inhalation exposures than they do from other routes of exposure.

The studies of the treated log home dwellers reported no serious health effects and no inhalation health effects, but did report rashes associated with touching the logs.

During the period when Kerr-McGee used pentachlorophenol, the general population had higher dietary exposures to pentachlorophenol. The National Health and Nutrition Examination Survey (NHANES II) conducted about this time (1976-1980), showed that most people at that time had pentachlorophenol in their urine –compared with very few having it today. In early samples of NHANES II, 79% of the people had pentachlorophenol in their urine [Kutz 1978]; by the end of the survey 71.6% had measurable levels [Kutz 1992]; for the 1988-1994 survey, the value dropped to 64% [Hill 1995]; and for the 1999-2002 (NHANES 99-02) survey, that value dropped to below 15.8% [NCEH 2005]. This is because earlier pesticide practices allowed pentachlorophenol to get into the food supply. The average pentachlorophenol urine level in the general population in the 1970's was 63 µg/L and the maximum was 1190 µg/L. While the peak doses estimated for air exposures from Kerr-McGee were not high, the emissions would have contributed to the total body burden of the community. An air dose of 200 µg/day would increase the 1970 mean level to over 163 µg/L.[2] This level is higher than the average of (111 µg/L) log home dwellers, but 1.6-6 times lower than worker levels (of 258-968 µg/L) [CDC 1980, Wyllie 1975, Zober 1981, Arsenault 1976].

[1] Chloracne is an acne-like condition linked to dioxin exposure.
[2] 200 µg/L ÷ 2 L (urine) + 63 µg/L = 163 µg/L.

ATSDR does not expect to see adverse health effects in the Kerr-McGee community as a result of pentachlorophenol inhalation exposures in the past because levels were expected to have been much lower than those associated with health effects. However, because the general population had dietary exposures to pentachlorophenol and because there was the possibility for contact exposures, the margin of safety to protect people from the risk of non-Hodgkin's lymphoma and developmental effects is unacceptable. While most of the pentachlorophenol exposure was due to pesticides that were in the food that all Americans ate at the time, the added exposures from the air pathway and through incidental contact with residues in the community added to a margin of safety that is not acceptable for prudent public health practice.

The following factors have contributed to an unacceptable past risk:

1) The general population had elevated levels of pentachlorophenol due to their diet during the time when Kerr-McGee was using pentachlorophenol;

   Americans ingested **760 µg/day** in food

2) It was possible to have skin contact with wood treatment residues and to accidentally ingest small amounts that adhere to hands;

   Skin absorption is high, intermittent, and not known.

   Ingestion was likely to be intermittent and less than **2 µg/day** (on the low measurements in ATSDR 2007)

3) Air concentrations near Kerr-McGee may have created intermittent exposures near those of *low-level* occupational exposures;

   Average exposures could have been as high as **200 µg/day**.

4) Occupational studies suggest that *high-level* exposures to pentachlorophenol treated wood are associated with non-Hodgkin's lymphoma;

   For doses of **2000-20,000 µg/day**.

5) Children may be more susceptible to pentachlorophenol exposures [Chapman 1965, 1970; McConnachie 1965]; (see Child Health Considerations)

   CDC found children to have twice the exposure of adults (lb for lb)

6) Pentachlorophenol may cross the placenta and enter into the developing fetus [Larsen 1975] and pentachlorophenol is a developmental toxicant at high doses [Schwetz 1978, Argus 1993, 1997; Bernard 2001, 2002; Beard 1999; Courtney 1976; Larsen 1975; Welsch 1987; Schwetz 1974]. (see Child Health Considerations)

Adding the total exposures **760 µg/day + 2 µg/day + 200 µg/day = 962 µg/day**

This value is about half of the level where there is potential for a low adverse effect level in men (of **2000 µg/day**). This leaves little margin of safety for those individuals who may also have skin contact with the logs.

The Creosote Process: Creosote is a complex mixture of polycyclic aromatic hydrocarbons (PAHs), phenols, sulfur, and nitrogen compounds. Creosote has a naphthalene odor, similar to mothballs. Much of the ingestion toxicity of creosote is linked to a small group of PAHs, benzo[a]pyrene and dibenzo[a,h]anthracene being among the most toxic. However, the sampling data from Kerr-McGee and other sites indicate that only naphthalene is elevated and that the levels are only expected to result in reversible nuisance irritation effects.

PAH's are semi-volatile chemicals that are formed when oils are cooked. Naphthalene is among the more volatile of the PAHs while benzo[a]pyrene and dibenzo[a,h]anthracene are less volatile. Therefore, we expect there to be more naphthalene in the air and more of the benzo[a]pyrene and dibenzo[a,h]anthracene to be in the soils near a creosote facility. The screening sampling at the fence-line of Kerr-McGee suggest this.

Table 1: Screening ERM Monitored Levels at Fenceline, Kerr-McGee Chemical Corporation (µg/m^3)

Chemical	*Low*	*High*
Benzo(a)pyrene	Non-Detect	0.0032
Dibenzofuran	Non-Detect	0.9939
Naphthalene	**Non-Detect**	**13**

Concentrations in micrograms per cubic meter (µg/m^3)
Analysis was by EPA method TO-13
Samples collected in 2000 [Hoffnagle 2001]

These levels are consistent with those found in the two known investigations in the air of communities near creosote facilities [Zarus 2004, Stone 2004]. Levels near both sites showed that naphthalene but no other chemicals were elevated. Furthermore, none of the hazardous PAH's (benzo [a]pyrene or dibenzo[a,h]anthracene) were found[1] as is normally found in the soils contaminated by creosote [ATSDR 1995, ATSDR 2001]. More data from these sites are provided in Appendix F.

How high could the Naphthalene Levels Get?

Airborne levels of naphthalene were also likely to be lower than 60 µg/m^3 on average and probably closer to or below 13 µg/m^3 when the facility operated and lower between batches. There are 10 samples collected at Kerr-McGee during 2000 that suggest a neighboring ambient air level. Every sample reported in Hoffnagle 2001 had a detectable value ranging from 0.048 -

[1] The Lowest Observed Adverse Effect Level for naphthalene is 10,000 ppb --rats and mice had lesions in the nose when exposed chronically.

13 μg/m^3. These samples were collected in 2000, a year of relatively low production, only 1.6 million cubic feet compared with 2.9 million cubic feet in 1999. The maximum level is close to the average level of naphthalene near the two available community studies (of 12.6 μg/m^3 and 16.3 μg/m^3) [Zarus 2004, Stone 2006]. Elevated levels of 2-methylnaphthalene and 1-methylnapthylene were also found at other sites; those analytes were not reported for Kerr-McGee sampling. The multiple detections of these compounds are characteristic of a very odorous area.

The average levels measured in communities are about 20 times lower than the levels measured during an onsite evaluation of workers at Kerr-McGee of 210 to 330 μg/m^3 [Borak 2001]. Dispersion of these levels would reduce the levels measured at the fenceline (to as low as that measured, 13 μg/m^3), but short-term higher levels are also possible other years when production was higher. Off site concentrations elsewhere were shown to be 15 to 20 times lower (due to downwind dispersion). Modeling and sampling of other odorous chemicals indicated ratios of onsite to off site concentrations from 3-1 to 20-1 due to dispersion [Zarus 2002, Zarus 2004]. A 3-to-1 reduction would suggest peak levels of 110 μg/m^3. These concentrations are well below effect levels but at levels that warrant further evaluation.

Implication of the Exposures to PAH's (including naphthalene)

We expect very little exposure to most of the PAHs, but expect that naphthalene and other odorous chemicals may have posed respiratory irritation. Data collected from Kerr-McGee workers confirm low-level exposures to the less volatile PAH's, but provides no information about the internal dose from naphthalene exposures. While levels known to be harmful are about 100 times higher than the highest levels measured, the levels were likely to be above levels that are considered an odor nuisance[1] to the general public.

There are many sources of PAH's. PAH's are given off when fuels and other carbohydrates are burned [DeMarini 1997, ATSDR 1995]. Firefighers and coal tar workers have high exposures to PAH's [Kato 2003, ATSDR 1995]. Air monitoring data shows that fire fighters are exposed to 10,000 times more PAHs than the general population. After fire fighters are exposed, their biological levels are two to three times higher than the general population [Caux 1986, Pintos 1998, Stang 2003, Kato 2003, Plas 1995, Larson 1994, ATSDR 1995]. Creosote worker PAH exposure is much different from fire fighter exposure. Several studies find that creosote wood operations similar to Kerr-McGee, exposes workers to high levels of PAHs through the skin rather than through inhalation. Those studies show that wood treatment workers had among the highest PAH body burden of all occupations [Unwin 2006, Levin 1995, Elovaara 1995, Bentsen 1997].

Our bodies are efficient at eliminating PAH's. Most PAHs that enter the body leave within a few days, primarily in the feces and urine. Also, most people get high exposures to PAH's by

[1] The measured levels of naphthalene are below olfactory detection levels in single chemical studies, but at concentrations found at odorous wood treatment facilities.

ingesting them [ATSDR 1995]. This was confirmed in the Kerr-McGee-funded study of their own workers [Borak 2001]. Levels of PAHs in workers were very low (as compared with other occupational studies) and constituted a small portion of the workers overall body burden of PAH's shown by a small difference between the staff in contact with the logs and those not in contact. Because other studies indicate much higher levels are typical of creosote workers, Kerr-McGee workers must have had less direct skin contact with creosote than typical [Unwin 2006, Levin 1995, Elovaara 1995, Bentsen 1997].

Table 2: Summary of the Air Sampling Results near Kerr-McGee Workers

Chemical	*Maximum*	*Comparison Value*
Naphthalene	**330**	**3 MRL***
Fluorene	44 (e)	150*
Phenanthrene	52.5 (e)	>1100**
Anthracene	6.3 (e)	1100*

Concentrations in micrograms per cubic meter ($\mu g/m^3$)
(e) = estimated from a figure provided in Borak 2001 (no raw laboratory data provided)
MRL = ATSDR's Chronic Minimum Risk Level (this is numerically identical to the EPA RfD)
* EPA risk-based value (10^{-6}) assuming continuous exposure for 70 years (EPA 1998)
** Expected to be less toxic than anthracene (factored by Toxic Equivalency Factor) (10^{-6})
Concentrations above the comparison value are in **bold case**
These community-based values were used because the samples were collected outdoors.
Worker's exposures would normally be compared with much higher worker-related values.

Residential comparison values are not used to evaluate worker exposures, but they help to rule-out concern for PAH's other than naphthalene. Marker chemicals (1-hydroxypyrene, 1-OHP) found in urine indicates exposure to tar products despite the low levels of PAHs in the air data. 1-OHP is a break down product of PAHs; pyrene typically contributes the most to 1-OHP. The highest measurement of pyrene in the air at Kerr-McGee was 0.33 $\mu g/m^3$ and therefore little of the urinary 1–OHP came from air exposure. As mentioned previously, most of the PAH exposure to wood-workers is from skin absorption, maybe up to 50 times more [Elovaara 1995].

Results for 68 urine samples collected from 36 Kerr-McGee workers are provided below. The table separates the results of 1-OHP into concentration categories (in $\mu g/g$). No individual analytical results were available for the workers; so the collective groups were used as reported by Borak 2001.

Table 3: Distribution of Urinary 1-OHP in Kerr-McGee Workers

1-OHP Levels (μg/g)	*Number of Samples*
<1. 00	22
1.00-2.49	28
2.50-4.99	9
>4.99	9

(e) = estimated from a figure provided in Borak 2001 (no raw laboratory data provided)
MRL = ATSDR's Chronic Minimum Risk Level
* EPA risk-based value (10^{-6}) assuming continuous exposure for 70 years (EPA 1998)
** Expected to be less toxic than anthracene (factored by Toxic Equivalency Factor) (10^{-6})

Of the 68 urine samples tested for 1-OHP, 46 (67%) were over 1 μg/g and 9 (7.5%) were over 5 μg/g. The report indicated no difference between smokers and non-smokers. The urine sampling results were compared with air sampling results and showed no correlation between air levels and urine 1-OHP. Because other studies indicate much higher levels are typical of creosote workers, these workers must not have had direct skin contact with the creosote than is typical.

The biological exposure limit of 1-OHP for coke oven workers is 4.4 μg/g (g indicates gram of creatinine; 1 g of creatinine is normally found in 1 L of urine) [Jongeneleen 1992]. Levels in creosote workers in Sweden ranged 2-6 μg/g, ranged 2.8-120 μg/g the UK, and ranged 8-170 μg/g in Finnland [Levin 1995, Unwin 2006, Elovaara 1995]. Levels of non-exposed individuals range from 0.03 to 1.4 μg/g [Jongeneleen 1987, Levin 1995, Elovaara 1995, Caux 2002]. Smoking is a known confounder of 1-OHP in urine as average levels of 1-OHP in smokers are about twice that of nonsmokers (a mean level of about 1 μg/g) [Ichiba 2007, Kim 1999, ATSDR 1995].

These measures confirm that there was little overall exposures to the less volatile PAHs in addition to the limited amounts of PAH found in the air pathway (as is typical of creosote workers).

However, this does not provide any indication of inhalation exposure to naphthalene. Naphthalene levels were 100 times higher than pyrene, and is often found to be 1000 times higher at other sites, but naphthalene marker, 2-Napthol (2-NAP) in urine was not sampled [Elovaara 1995]. Since no 2-NAP samples were collected, we cannot fully evaluate the total

exposure to naphthalene. (It should be noted that the highest level measured near the workers was over 150 times lower than worker 8-hr recommended thresholds.)

Naphthalene, a respiratory irritant, has a unique toxicity when exposure is through the air pathway [ATSDR, 2004]. Naphthalene has a large range between levels that are definitely safe and levels that are definitely harmful. Because of the severity of the Lowest Observed Adverse Effect Level (LOAEL), ATSDR used a large safety factor (of 300) to calculate a Minimal Risk Level (MRL) of 3 $\mu g/m^3$, making the value very protective. Few human inhalation studies are available, and very few animal studies are available at low doses; so, we are left with animal studies at high doses to evaluate the inhalation pathway. Studies found repeated results at 52,400 $\mu g/m^3$. It is considered relevant to discuss these high exposures here because, there are very few studies at lower doses and the effects were found in many of the animals exposed. Inhalation of 52,400 $\mu g/m^3$ of naphthalene caused inflammation and deterioration of the epithelium inside the nose of rats and mice after 105 weeks [ATSDR 2004].[1] No heart, eye, GI, muscle, renal, endocrine, or body weight changes were observed in the rat study at 52,400 $\mu g/m^3$ and none were observed in the mice study at 157,200 $\mu g/m^3$. The rat study also found neuroblastomas at 52,400 $\mu g/m^3$. Another mouse study found a decrease in airway cell mass after exposure to 52,400 $\mu g/m^3$ for 4 hours. While the acute high levels appear to pose less serious irritation in the short term, they are linked to cancer after long exposures.

While ATSDR's Health Assessment process involves the evaluation of all health hazards from all chemicals found at a site, EPA's Health Assessment process evaluates the cancer hazards of a single chemical. As of November 26, 2008, EPA is undertaking an update of the Integrated Risk Information System (IRIS) Health Assessment for carcinogenicity resulting from the inhalation of naphthalene [EPA 2008]. EPA has initiated the peer review plan and proposed promulgation of the new assessment in 2010. EPA's work could result in a lower cancer slope factor and thus a higher theoretical risk of exposure. This report has incorporated the recent studies that have occurred since EPA's prior Health Assessment in 1998. Therefore, this Kerr McGee Health Assessment includes the new information about naphthalene toxicity to assess the hazards, qualitatively.

The naphthalene level measured at the Kerr-McGee fenceline (13 $\mu g/m^3$) and near the other creosote plants averaged 4000 times lower than the animal study concentrations (52,400 $\mu g/m^3$). The highest worker level (350 $\mu g/m^3$) was 150 times lower [Borak 2001]. Furthermore, the degeneration of the outermost layer in rats and mice were associated with continuous exposure to high levels. Because the exposures at Kerr-McGee were episodic and low, there was time to recover in between the low-dose exposure events. Therefore, we do not expect naphthalene-specific toxicity to occur in the community. This finding will remain, regardless of the conclusion of the EPA naphthalene health assessment. However, odor-associated health effects are an issue with the several compounds found near multi-process wood treatment facilities.

[1] The effects include a list of types of degeneration that occurred in the outermost tissue in the nose.

Odors

Some evidence suggests that people can experience symptoms as a result of exposure to odors [RosenKranz 2003]. Odor-related mechanisms that may result in symptoms include innate odor aversion, stress-induced illness, aversive conditioning phenomena, and aggravation of existing medical conditions, such as bronchial asthma. After exposure to noxious odors, these processes may occur in some individuals and not in others. Smokers and the elderly may be less susceptible to odors. In general, women are more sensitive to smells than men [Dempsey 2002, Hertz 2000] are. However, women's sensitivity is extremely variable over time. Current feeling of wellbeing and other emotional states also affect our odor acuity [Royet 2003].

Odor-related aversive conditioning may occur when a person experiences low-level odors after an initial traumatic exposure. In some cases, conditioning may cause an aversion for preferred odors [Dingfelder 2004]. A common response is the panic or hyperventilation cluster of symptoms such as fast heart rates, dizziness, nausea, sweating, and anxiety. Stress, which can result in health effects, may to some extent be related to the degree to which an individual believes an odor is causing risk. Many contaminants have odor thresholds that are lower than the levels thought to be hazardous, and may provoke odor-related symptoms described above.

Often, the health complaints expressed by a community defy classic toxicological explanation. This may occur when contaminants are detected below levels associated with known adverse health effects [Schusterman 1991]. In addition, a high amount of uncertainty exists concerning the significance of low-level, long-term exposures to contaminants compared with the known health effects of occupational or high dose exposure.

Communities near operational wood treatment facilities often complained of odors, including the times when the associated chemicals could not be detected by instruments. Agency investigators especially smelled the characteristic chemicals during rain events, even when the facility was not treating wood [Zarus 2004, 2007, Stone 2004].

Child Health Considerations

Toxicity from inhalation (only) exposures is discussed in this document. There is no evidence to suggest that children are or were inhaling dioxin near Kerr-McGee; however, because of the possibility of other pathway exposure to dioxin, we recommend referencing the Soil and Sediment Health Assessment for Kerr-McGee (2007). For dioxin fact sheets, see: www.atsdr@cdc.gov.

Children inhale creosotes, PAH's, and pentachlorophenol in the same way as adults. However, children pose a special health consideration. They should not be viewed simply as small adults. Their unique physiology as well as their behavior can have a profound influence on their exposure risk. While playing or at rest children breathe more rapidly and inhale more pollutants per pound of body weight than do adults. In addition, a child's airway passages are narrower than adults are and, thus, irritation results in proportionally greater airway obstruction. Children also metabolize chemicals differently and eliminate liquids at a different rate. The result of these

differences creates a different body burden when children are exposed to chemicals. In a study of families that lived inside treated log homes, children were found to have 80% more pentachlorophenol in their bodies due primarily to their inhalation rates and reduced elimination rates [Cline 1989].

Some chemicals uniquely affect children. There are some signs that pentachlorophenol effects women and children uniquely and some signs that naphthalene exposure affects some babies uniquely.

Pentachlorophenol-Specific Child Health Considerations:

Children exposure to the same air concentrations of pentachlorophenol in the environment is higher per pound of body weight than for adults [Cline 1989; CDC 1980]; children may be more susceptible to pentachlorophenol exposures [Chapman 1965; McConnachie 1965]; pentachlorophenol may cross the placenta and enter into the developing fetus [Larsen 1975]; and pentachlorophenol is a developmental toxicant at high doses [Schwetz 1978, Argus 1993b, Bernard 2001b, Argus1997, Bernard 2001c, Beard 1999b, Courtney 1976, Larsen 1975, Welsch 1987, Schwetz 1974].

Despite the number of health studies listed above, the degree to which children are uniquely susceptible to adverse outcome is not understood. In a study of upholstery workers, women with a mean blood level of 73 μg/L had an association with infertility and those with a mean of 42 μg/L had an association with menstrual dysfunction [Gerhard 1991]. The data from this and other studies on women are insufficient for appropriate statistical analysis; however, the results all suggest a unique toxicity for women and children. One such study found gynecological or endocrine disorders associated with elevated pentachlorophenol in blood [Gerhard 1998, Gerhard 1999]. We do know that exposures included contact and inhalation, and other chlorinated compounds were present in the women (PCBs, DDT). The many exposure pathways to these women were analogous to the exposures of the men who touched wood in the wood treatment process, and thus resulting in elevated HpCDD and OCDD in blood (as discussed above) as well as the pentachlorophenol. While there is an association between effects and pentachlorophenol in both blood and urine, the relative relationship depends on the nature of exposures (duration and peak levels). The implications of these studies suggest that there are unique effects of exposures that are not understood and the dose associated with those effects is unknown. Therefore, our evaluations of the current exposures need to be protective of children. These uncertainties justify the need for a larger margin of safety when protecting children from exposures.

Naphthalene-Specific Child Health Considerations:

While there are only a few studies available to assess the specific toxicity of naphthalene on children (no genotoxic data is available), those available showed little developmental toxicity. They did, however, suggest a unique sensitivity to inhaled naphthalene in infants and unborn fetus. Clinical reports suggest that prolonged exposure to naphthalene vapors can cause adverse health effects in all humans and, although the rate and extent of exposure is unavailable, children appear to be more sensitive [Harden 1978, Linick 1983, Valaes 1963].

There are cases of hemolysis (breaking open of red blood cells) when individual children have inhaled naphthalene (from mothballs) [Valaes 1963, Dhillon 2003, Kaplan 2004 and 2005, Anziulewicz 1959, Molloy 2004]. Valaes documented hemolysis in 21 infants, who inhaled mothball fumes from a diaper pail, with no data to determine dose. However, in Molloy's case, an African American pre-term child was born with severe hemolytic anemia a week after his mother inhaled naphthalene. The exposure was household air after mothballs (100% naphthalene) were spilled into a heating vent; due to the odors, the windows were left open. Kaplan suggests that the frequency (13%) of G-6-PD deficiency in male African-American babies, makes them predisposed to naphthalene-induced hemolysis [Kaplan 2005]. While these are serious acute affects, the children had no observed effects when discharged from the hospital. This unique predisposition is germane to the exposures near Kerr-McGee, because the neighbors bordering Kerr-McGee are mostly African-American and because an elementary school (which attracts mothers) borders the Kerr-McGee property.

In the Malloy case, the mother had urinary metabolites for naphthalene, but the 36-week preterm child did not. Since naphthalene metabolites have a half-life of approximately 24 hours and since some people do not have detectable levels after exposure, the acute dose must have been extremely high to be elevated a week later. Because the widespread use of naphthalene mothballs for several years and the low number of poisoning incidents, we anticipate that hemolytic anemia only occurs after acute exposure to very high levels.

The mother and child were both discharged in 16 days and mother and child were healthy 1-yr later.

While we do not know the exposure levels in the above cases, we know that they were much higher than possible at Kerr-McGee. Creosote typically contains 10% naphthalene, but is known to contain up to 40% naphthalene and is used outdoor compared with the above indoor exposures to 100% naphthalene. If acute exposures of 100% naphthalene to a predisposed preterm baby (as well as other babies) did not result in long-term effects, then lower acute exposures are not expected to cause long-term results.

Uncertainties and Limitations

There are limited data to evaluate airborne exposure to wood treatment operations. Some air samples were collected at the Kerr-McGee site while the facility used the creosote process. These compare well with the two available community exposure investigations. All data reveals slightly elevated levels of naphthalene, a respiratory irritant. The health exposure study data used to interpret the Kerr-McGee community exposures are 1) acute case studies to extremely high levels or 2) intermediate term exposures to high levels. Kerr-McGee exposures differ and were likely to be lower, episodic and over a longer period.

While the past pentachlorophenol dose calculations have some uncertainty due to the lack of site-specific data from Kerr-McGee's past, there is some exposure data collected in three communities near operational plants and there are many exposure studies of wood workers. These formed the basis of three estimation methods for this evaluation. None of the three estimation methods produced results considered hazardous.

Conclusions

- Pentachlorophenol that was released into the air during the pentachlorophenol process contributed to a low health risk (from 1950-1975). No adverse health effects are expected from the residents' airborne exposures. However, the air exposures occurred at a time when the US population was unnecessarily exposed to pentachlorophenol in their diet. While the total exposure dose is much lower than those known to cause health effects, there are study data that suggest that the margin of safety for children and women should be higher for pentachlorophenol.
- Naphthalene released into the air during the creosote treatment process posed a risk for respiratory irritation. While levels were several hundred times lower than those known to cause health effects, we cannot rule out the low risk due to the lack of studies conducted at lower levels and the absence of genotoxic studies. Furthermore, African American children appear to be uniquely susceptible to acute exposure effects.
- Measurements of PAHs (other than Naphthalene) indicate that air pathway exposures are much less than exposures from contact or ingestion. Measurements of PAH markers in Kerr-McGee workers indicate that inhalation exposures to PAHs are low.
- Small amounts of naphthalene are released when it rains because rainwater fills the pore spaces in the soil and pushes the vapors out. These levels are much lower than most instruments can detect and do not pose a health risk. However, the unpleasant odors themselves are known to be linked with a sense of low quality of life and contribute to lowered immune response.

Recommendations

- Remove any treated wood that may be in the home, regardless of the where it was manufactured.
- Remove or encapsulate those soils that have strong odors in order to improve the quality of life for the residents.

Authors, Technical Advisors

Principal Authors

Greg Zarus
Atmospheric Scientist
Site and Radiological Assessment Branch
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Teresa Foster
Environmental Health Scientist
Site and Radiological Assessment Branch
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Reviewed by:

James T. Durant, MSPH C.I.H.
Environmental Health Scientist
Exposure Investigations and Site Assessment Branch
Division of Health Assessment and Consultation

Tonia Burk, PhD
Environmental Health Scientist
Site and Radiological Assessment Branch
Division of Health Assessment and Consultation

Laura Frazier
Geologist
Site and Radiologic Assessment Branch
Division of Health Assessment and Consultation

Hatice Zahran, MD, PhD
Medical Epidemiologist
Exposure Investigations and Site Assessment Branch
Division of Health Assessment and Consultation

George Prince
Technical Information Specialist
Office of the Assistant Administrator
Agency for Toxic Substances and Disease Registry

Sandy Isaacs
Branch Chief
Site and Radiological Assessment Branch
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Daphne Moffett, PhD
Associate Director of Science
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

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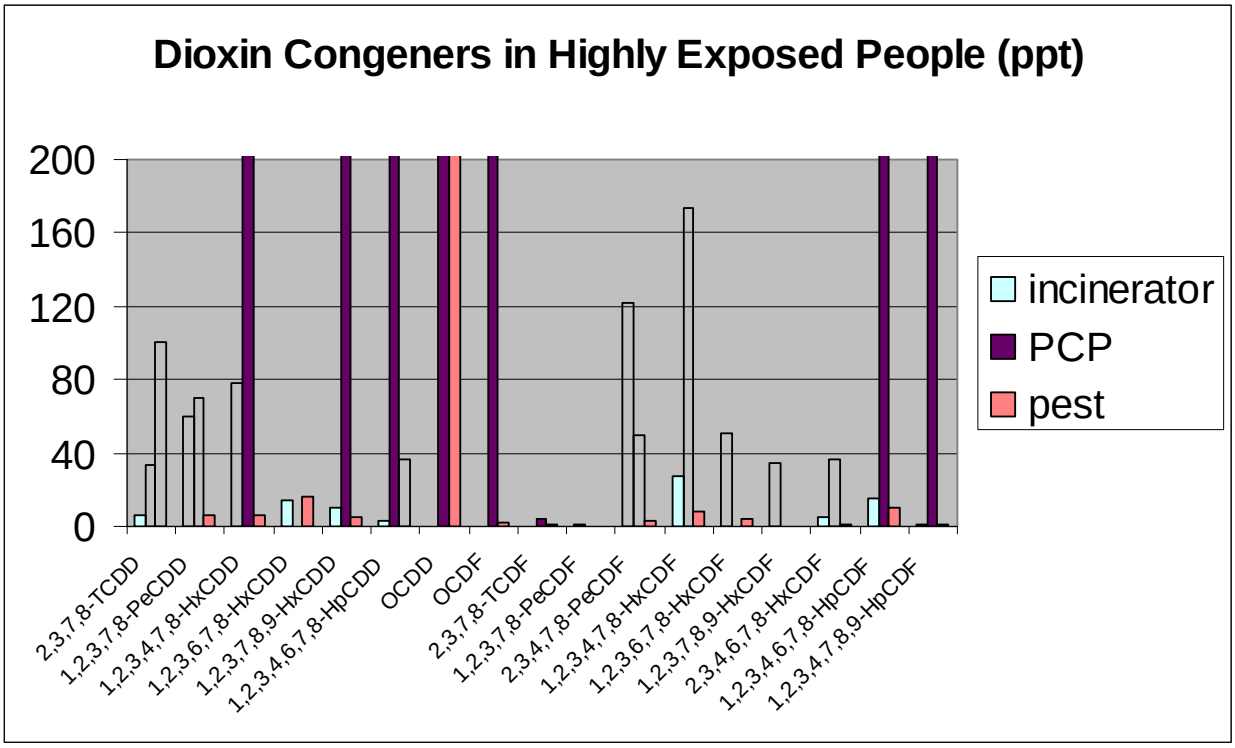
Appendix A

Dioxin Exposure Data

Blood dioxin congener profiles for highly exposed people are illustrated below.



incinerator = exposure to incinerator ash

PCP = pentachlorophenol exposures

pest = agent-orange pesticide application

source: Dahlgren et al. 2003

The figure was truncated in order to see the congener levels for people exposed to incinerator ash and agent-orange pesticide as compared with pentachlorophenol.

Appendix B

Emissions and Air Concentrations

Measured and Predicted

There is some data to compare the pentachlorophenol emissions at Kerr-McGee (prior to 1976) with other sites. The highest reported production rate at Kerr-McGee (while pentachlorophenol was used) was 2.8 million cubic feet per year, with pentachlorophenol accounting for approximately 1 million cubic feet per year [Hoffnagle 2001].[1] The Georgia producer treats 1.8 million cubic feet per year –with creosote accounting for approximately ½ or less than 1 million cubic feet. The Oregon site treats 3 million cubic feet per year with ¼ to ½ being pentachlorophenol –or about 1 million cubic feet. Like Kerr-McGee, the other pressure treatment operations were located near communities.

The table below illustrates the comparability of the available pentachlorophenol-related air exposure data for communities near operational facilities.

Operational Pentachlorophenol Data from Wood Treatment Plants

Facility	**Kerr-McGee**	**Other Mississippi**	**Georgia**	**Oregon**
Production	Crossties	Crossties	Poles	Crossties
Amount treated	1 MM CF	1 MM CF	<1 MM CF	<3 MM CF
Highest air	?	?	30 µg/m^3	ND
Highest blood	?	26 µg/L	?	?
Highest Urine	?	?	6.7 µg/L	?

MM CF = Million Cubic Feet (per year)

? = Not measured

ND = Not detected when measured

It was not determined why pentachlorophenol was detected in the air near the Georgia producer but not near the Oregon producer. Unlike the Georgia producer, Kerr-McGee made crossties, as the Oregon producer did. However, all facilities changed from creosote to pentachlorophenol operations, making it difficult for samples to be collected at optimal times. While it is possible that pentachlorophenol levels may have been like the Oregon producer, to determine a worst-case, we assumed that they were more like that of the Georgia producer.

[1] Kerr McGee was permitted to treat up to 5 million cubic feet per year. The maximum reported between the 1970-1989 was 2.84 million cubic feet and it was expected that approximately one third was pentachlorophenol. The maximum total production of creosote of 3.292 million cubic feet occurred in 1998.

The area used by Kerr-McGee to dry logs is (and was) about twice the Georgia producer's. (Satellite photographs are provided below). The additional area is consistent with the differences in the total maximum production rates (of all logs) of 3.3 to 1.8.

Since railroad ties were rectangular, they could be stacked higher at Kerr-McGee (as documented) than at the Georgia producer. Furthermore, because the Kerr-McGee railroad ties have much more surface area than the Georgia producer telephone poles, we expect that emission rates from the drying of ties to be higher.

Crosstie Area = 2 (L W + L D + D W) Crosstie Volume = L W D

Length, L = 8.46' ; Width, W = 8.37"; Depth, D = 6.5"

Pole Area = 2π r h + 2 (π r^2) = π D (h + r) Pole Volume = π r^2 h

Radius, r = 6" ; Diameter, D = 12; height, h = 30'

If for a given volume we equate: π r^2 h = L W D

Then , we find that we have 7.48 times as many crossties as poles and the total area of the crossties is 1.67 times more than the area of the poles.

Being smaller, rectangular, and having 6 times as many ends, the ties have about 67% more surface area to emit from than the telephone poles. This could result in 67% higher emission per pound of pole. With pentachlorophenol production rates about the same, the average emission rate could have been 67% higher at Kerr McGee. The highest air level measured near the Georgia producer was 30 µg/m^3 (with a daily average of 5.8 µg/m^3).[1] The additional potential for emissions would result in peak air concentrations of 50.1 µg/m^3 (and maximum 3-day average of 9.7 µg/m^3).

[1] All measurements near residences were below 8 µg/m^3. Pentachlorophenol was not detected in many samples; however, the detection levels were as high as 4.5 µg/m^3 at times. The range of detected levels near homes was 1.3-8.1 µg/m^3. The highest average string of measurements taken in within 3 days at one location was 5.8 µg/m^3.

Supporting Data for Method 2: Comparing Emissions Data

Satellite Photograph of Kerr-McGee

The vertical line segment indicates 500 ft

Satellite Photograph of the Georgia producer (in Georgia)

The vertical line segment indicates 500 ft

Appendix C

Exposure Dose Calculations for Pentachlorophenol

Supporting Information to Provide Perspective of Pentachlorophenol Emissions

While the peak measured level near the Georgia facility (30 µg/m^3) and the maximum calculated near Kerr-McGee (60 µg/m^3) are similar to levels measured within work zones, workers are surrounded by treatment products and residents are down wind. Wind should disperse pentachlorophenol in the air and will flow in alternate directions, reducing exposures. In addition, the total body burden is expected to be higher for workers because wood workers have contact exposures. Below is a photograph of log-workers loading lumber. During dipping or pressure operations, workers have been known to get splashed with pentachlorophenol.

There are three studies of worker exposures (at 28 plants) where pentachlorophenol was measured in both the air and the worker's urine [Wyllie 1975; Arsenault 1976; Zober 1981]. The air levels averaged 10 times higher than those measured near the facility in Georgia and the urine levels averaged 400 times higher. The difference in these values indicates a large difference between the nature of exposures of workers and residents. Only one group of workers had air and urine ratios similar to the residents of the Georgia facility, and they were workers at one application plant and not at either the pressure, dipping, or spraying plants [Zober 1981], which indicates less touching. However, there urine levels averaged 4 times higher than the resident's near the Georgia plant. (Appendix D contains average and maximum worker exposure data.)

We saw very little pentachlorophenol in the urine of residents near the Georgia plant (urine levels ND-6.7 µg/L) following the elevated air levels. People living in treated log homes (with urine levels of 32-160 µg/L) had internalized more pentachlorophenol than the community near the Georgia plant, although their air levels averaged 10 times lower [CDC 1980]. The neighbors of the Georgia plant and log-home dwellers were much lower than the levels associated with chloracne in workers (117 µg/L) and much lower than the average of the workers without chloracne (90.6 µg/L) [CDC 1980, Wyllie 1975, Zober 1981, Arnsenault 1976, Zarus 2007].

The ATSDR investigator had urinary pentachlorophenol levels 25% higher while sampling air in the community near the Georgia plant than months after being away from the community [Zarus

2007]. This suggests that 25% of his total body burden came from inhalation exposures while in the community 8 hrs/day. Because most people get their pentachlorophenol body burden from diet, 75% of his body burden was likely to be from his diet. The investigator's levels were close to the mean of the community, implying that the majority of the community's body burden was also from their diet.

The community near the (other) plant in western Mississippi were recently found to have blood pentachlorophenol averaging 3 µg/L with a maximum of 26 µg/L [Dahlgren 2007]. This is much lower than the average collected from: 18 Vat dippers (of 3780 µg/L), 23 Pressure treaters (1720 µg/L), and 210 farmers (of 250 µg/L) [Klemmer 1980]. Also, the dioxin levels in these same individuals (reported in Dahlgren 2006) was much lower than wood treatment workers; and although, low, were higher than the dioxin levels of the ten Kerr-McGee residents (reported in Dahlgren 2003).

In another study of 32 individuals near an operational pentachlorophenol wood treatment plant in Georgia (known to have elevated air levels), about 40% had detectable levels of pentachlorophenol (with a maximum of 6.7 µg/L) [Zarus 2007]. Of 5022 people in the general population, 44 had more than 6.7 µg/L of pentachlorophenol in urine and 84% did not have any pentachlorophenol detected [CDC/NCEH 2005. As with the Dahlgren 2004 (and 2006) study, Zarus (2007) suggests that neighboring treatment operations are contributing to the community's pentachlorophenol, but the total body burden is not very high. Furthermore, these biological levels are well below any known to cause health effects [ATSDR 2001].

People living in treated log homes (with blood levels averaging 330 µg/L [116-1084 µg/L]) had internalized more pentachlorophenol than the community near that western Mississippi plant [CDC 1980]. Furthermore, the 11 unexposed (controls) residents in the log home study (with blood levels averaging 48 µg/L [15-55 µg/L]) had internalized more pentachlorophenol than the community near that western Mississippi plant [CDC 1980]. This comparison points to the fact that the body burden of pentachlorophenol from food was high prior to the late 1980's.

We expect that indoor lifestyles and changing wind patterns moderated the exposures near the Georgia Plant, giving the residents' bodies time to remove the pentachlorophenol –thus reducing their body burden. There also is the issue that the Georgia community could not touch the logs where as the workers and the log-home dwellers could.

By using EPA and FDA estimates of body burden of pentachlorophenol in the general population, we find that air contributes to 0.8% of the total body burden [EPA 1980, ATSDR 2001].

Empirical Evidence of Pentachlorophenol Concentrations in Air and Urine

The following photograph was taken after a period when fence-line air concentrations near the Georgia producer were 29 ug/m^3 and community concentrations ranged 1.3-7.9 µg/m^3. Peak air levels were measured during periods that included treatment and early drying.

During another sampling period, the logs were stacked much higher and the resultant concentration was 30 µg/m^3. This indicates that our sampling location was so close in both cases that there was little time for any dilution and it indicated a potential maximum concentration due to the emissions from drying logs.

The photograph reveals that treatment and drying of this amount of logs created community levels of 1.3-7.9 µg/m^3. The sampling scenario with wind direction blowing across the entire property was observed twice –and produced similar sampling results (3.9-8.1 µg/m^3). Wind blowing at slight angles resulted in levels roughly 1/3 as high as the maximum days. The Georgia producer followed a similar (low) stacking routine when they treated using creosote. (Of course, no pentachlorophenol was detected during those operations) [Zarus 2004; Zarus 2007].

Appendix D

Summary of Pentachlorophenol Exposure Studies

Of the available exposure studies:

- There are six studies on workers where pentachlorophenol exposure was measured.
- Pentachlorophenol in air, urine, and blood were measured at two of those worker studies [Wyllie 1975; Zober 1981].
- There are three studies on residential pentachlorophenol exposures; one study includes air, blood, and urine [CDC 1980].

The airborne pentachlorophenol concentrations can be used to estimate a daily dose for men by multiplying by 20 m^3 (of air breathed per day); The urine (and blood) pentachlorophenol concentrations can be used to estimate internal dose (or body burden) for men by multiplying by 2 L (the amount of urine excreted in a day and the amount of blood volume in a man).

If the estimated inhalation dose is smaller than the internal dose, then the people did not receive their body burden from inhaling the pentachlorophenol. But, if the estimated inhalation dose is larger than the internal dose, then it is possible the people did receive their body burden from inhaling the pentachlorophenol.

The ranges of concentrations from the available studies are reported in the table below along with estimates for the community near Kerr-McGee. The table also reports the possible exposure dose from air compared with the estimated dose from all sources (Dose ratio, in µg/day).

Summary of Pentachlorophenol Exposure Studies

Population	**Air** (µg/m^3)	**Blood** (µg/L)	**Urine** (µg/L)	**Dose Ratio** (µg/D)	**Reference**
KM neighbors in 1970's	*<60*	?	?	***<1***	*(Estimated)*
32 Residents	ND-30	?	ND-6.7	44	Zarus 2007
29 Residents	?	ND-26	?	?	Dahgren 2006
32 Log home residents	ND-0.38	116-1084	2-87	**0.044**	CDC 1980
11 Unexposed residents	?	15 – 55	1-7	**?**	CDC 1980
18 Production W	2-50	20-1500 (P)	10-2110 (Cr)	<**0.47***	Zober 1981
23 Application W	0.3-8.0	200-2400 (P)	6-410 (Cr)	<**0.008**	Zober 1981
18 Dip, spray, brush W	?	430-14000	90-3300	?	Begley 1977
23 Pressure W	?	20-7700	10-2400	?	Begley 1977
210 Farmers	?	10-8400	10-400	?	Begley 1977
32 Unexposed W	?	20-7200	10-1000	?	Begley 1977
7 Dipping plants	3-63	?	120-9680	**<0.13***	Arsenault 1976
11 Spray plants	3-69	?	130-2580	**<0.53***	Arsenault 1976
7 Pressure plants	4-1000	?	170-5570	<3.5*	Arsenault 1976
6 Pressure W	5-15.3	350-3550	40-760	<**0.20**	Wyllie 1975
(#UK) Vat Dip W	?	?	(2600)	?	Casarett 1969
130 W	?	3-35,000	?	?	Bevenue 1967
121 Oc. Exposed W	?	3-38,600	?	?	Bevenue 1967
290 non exposed W	?	3-1840	?	?	Bevenue 1967

W= Workers **#UK = Number of samples unknown** **P = Plasma, not whole blood**

Oc. = Occupationally **Cr = Creatinine corrected** **? = Not measured (µg/g)**

ND = Not detected when measured **(2600) = mean value only** **D = Day**

Dose = ratio of the air dose over the total body dose

Estimated Air dose = 20 m^3***/day x air concentration (assuming 24 hrs of inhalation exposure)***

Total body dose = urine concentration x 2L/day, when urine data is available (normal excretion rate)

**** total daily dose was set less to the total amount in urine as the elimination rate > 1000*** µg/L

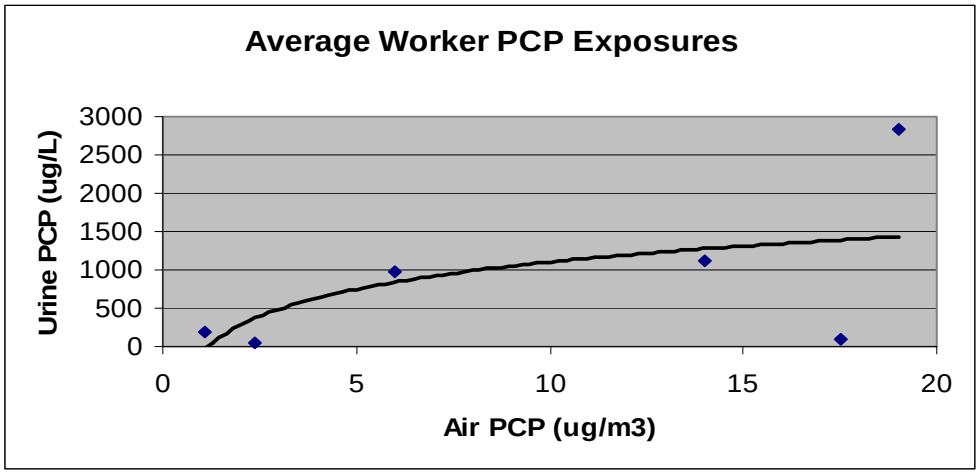
***** Total dose = blood concentration x 2L (total blood volume); no urine data was available for the dose calculation***

Bolded values indicate that air concentrations can not account for the body burden (or dose) of pentachlorophenol

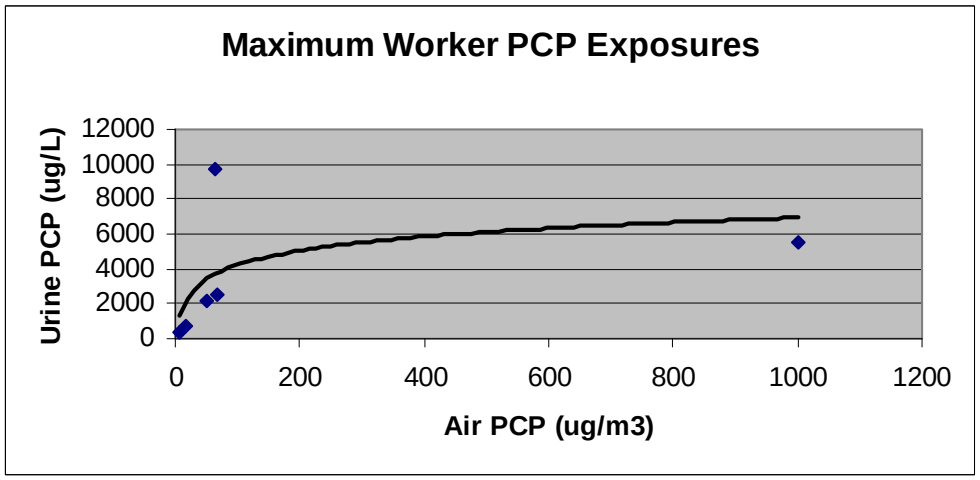
The dose ratio calculation (being much less than 1) suggests that most of the people's body burden is from exposures other than the air pathway. In most studies, more pentachlorophenol is excreted than is inhaled.

Worker Exposure Data

The following graphs represent data from three worker exposure studies (at 28 plants) where pentachlorophenol was measured in both the air and the worker's urine [Wyllie 1975; Arsenault 1976; Zober 1981]. Other studies are available for workers, but no others were found that provide mean and maximums of air and urine. One study is available for residents who lived in log homes. Pentachlorophenol ranged 0.2-0.38 µg/m^3 in air and 2-87 µg/L in urine [CDC 1980]. Because of contact dermatitis, we expect that some of these individuals had touched the logs in addition to inhaling the air levels. It also seems unlikely that 0.38 µg/m^3 of pentachlorophenol in air could result in 87µg/L in urine unless someone was very dehydrated. The dose from inhaling the contaminated air would be less than 7.6 µg/day (concentration in air times air inhaled = 0.38 µg/m^3 x 20 m^3/day) and the average excretion rate would be about 174 µg/day (concentration in urine times typical daily excretion = 87µg/L x 2 L/day). All urine levels measured in the community near the Georgia producer were below 7 µg/L [Zarus 2007].



PCP = pethachlorophenol ug/m3 = micrograms per cubic meter ug/L = micrograms per liter



PCP = pethachlorophenol ug/m3 = micrograms per cubic meter ug/L = micrograms per liter

Summary of study data from highly exposed workers not discussed in the main report:

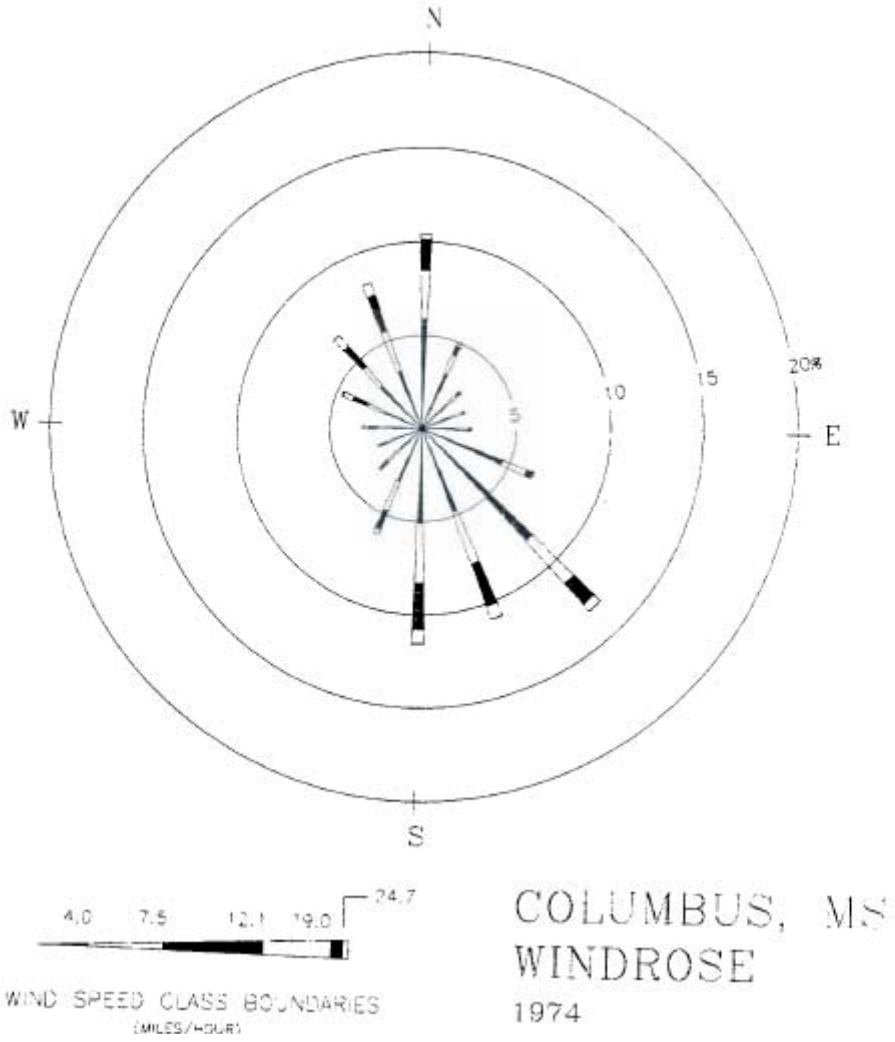
A treatment-operator-sponsored study on worker exposure found 17.8% of the workers to have exposed to the point where they developed chloracne –a scaring skin condition caused by high exposures to chlorinated compounds (dioxins in particular) [Hryhorczuk 1998]. These workers had higher elimination rates of pentachlorophenol (indicating higher exposures by all pathways). The average level of the workers with chloracne was 117 µg/L and workers without chloracne averaged 90.6 µg/L. The worker urine levels ranged 1.2-968 µg/L in several studies [CDC 1980, Wyllie 1975, Zober 1981, Arnsenault 1976].

Appendix E

Wind Variability Modifying Exposures

The wind roses from 1959-2000 showed that no wind direction was maintained more that 18% of the time [Hoffnagle 2001]. Wind direction for the years when pentachlorophenol were more variable. A wind rose for the year 1974 is provided in Figure 4.

Figure 4. Windrose for 1974: Showing the Frequency of Wind Direction



Source: Hoffnagle 2001

This figure indicates wind did not blow from any one direction more than 15% of the time.

Appendix F

PAH levels near Operational Creosote Facilities

The table below provides the results of the sampling for those chemicals detected above the background concentrations. The values for pentachlorophenol are also provided for a basis of evaluating the relative significance of the PAH exposure.

Data collected at other operational sites

Summary of Sampling Results at other Sites

	Oregon Site			Georgia Site		
Chemical	*Frequency of Detects*	*Maximum*		*Frequency of Detects*	*Maximum*	*Comparison Value*
Pentachlorophenol	0%	ND		**36%**	**30**	**0.052***
Naphthalene	**81%**	**24.5**		**85%**	**57.6**	**3 MRL***
Acenaphthene	50%	4.6		23%	1.2	220*
Fluorene	31%	2.3		23%	0.52	150*
Phenanthrene	22%	1.6		75%	1.24	>1100**
Anthracene	NR	NR		25%	1.34	1100*
a or b pinene	NR	NR		100%	ID only	NA
Terpene	NR	NR		33%	ID only	NA

Concentrations in micrograms per cubic meter ($\mu g/m^3$)
ND = Not Detected
NR = Not Reported
ID = Identified
MRL = ATSDR's Chronic Minimum Risk Level (the ATSDR MRL for naphthalene is numerically identical to EPA's RfD)
* EPA risk-based value (10^{-6}) assuming continuous exposure for 70 years (EPA 1998)
** Expected to be less toxic than anthracene (factored by Toxic Equivalency Factor); EPA risked-based factor (10^{-6})
Concentrations above the comparison value are in **bold case**

Pinene and terpene are characteristic chemicals within pine logs and they are included to serve as a marker that the samples were collected downwind. The results indicate that naphthalene is the only PAH that is significantly elevated. Comparison values provide a means to compare the significance of the naphthalene levels to the pentachlorophenol levels (discussed earlier). The results indicate that naphthalene is very frequently detected. Peak levels of naphthalene in both communities exceeded the comparison values. No other PAH comes near a screening comparison value, which indicates they are clearly at safe levels.

Related levels of Napthalene: The highest levels of naphthalene measured inside homes were 32-1600 $\mu g/m^3$ due to mothballs [Chuang 1991, Hung 1992, Wilson 1989]. While average naphthalene levels inside homes were 5.19 $\mu g/m^3$ [EPA 1988].

Appendix G

Response To Comments

During the public comment period, ATSDR received written comments on behalf of Tronox LLC (formerly Kerr McGee Chemical LLC); no comments were received from the general public. Tronox LLC, submitted comments in a November 21, 2008 document entitled "Public Comments to Public Health Assessment for Air Exposures to Wood Treatment Chemicals". This section provides ATSDR's response to their comments on our document. Additionally, some changes were made to this Public Health Assessment based on those comments. Tronox LLC provided a *General Comments* section to address the categorical concerns then followed with a *Specific Comments* section that often supported the general comments. Our responses to their comments follow this same format and correspond numerically with their comments. When Tronox LLC's comments offered clearer explanation of the community's exposure, those statements or comments were incorporated within the general text of the Public Health Assessment.

Responses to General Comments

1) ***Tronox LLC pointed out that the PHA failed to provide other sources of similar emissions from other industrial facilities in the region.*** We agree that other industry contributed to air pollution. We also expect that PAH exposure occurs from many sources, including living on an asphalt street. However, the focus was to address a few air pollutants associated with Kerr McGee. Naphthalene, and PAHs measured by Kerr McGee on site and at the fence line are unmistakably associated with Kerr McGee and those samples were collected during a relatively low production year.

2) ***Tronox LLC wanted to identify what the new information was that helped in the assessment and wanted an opportunity to review it.*** The new information refers to recent sampling conducted in communities near wood treatment facilities. Previously, scientists only had information about worker exposures. Therefore, health scientists had to extrapolate from those studies to determine the potential uptake and metabolism by residents. Those references are cited and available for review. The new information regarding exposures include:

   a. Stone 2004, Zarus 2004, and Zarus 2007 provided residential measurements of pentachlorophenol in air;

   b. ATSDR 2001, ATSDR/ORDOH 2006, and Zarus 2004 provided ratios of pentachlorophenol to other PAH concentrations in air;

   c. and Dahlgren 2006, Karouna-Reiner 2007, and Zarus 2007 provided new knowledge about residential uptake and elimination of chemicals associated with the pentachlorophenol process.

3) ***Tronox LLC stated that conclusions based on ratios of chemicals are borderline speculative.*** ATSDR believes that ratios of similar chemicals provide one useful model. Such models are commonly used to estimate concentrations. Like all models, the method is not always

accurate. For that reason, ATSDR used three methods to determine likely concentrations. Each of these methods produced similar values. ATSDR used the weight of evidence from all of these methods to conclude on exposures.

4) ***Tronox LLC is requesting that the two reports be combined into one.*** ATSDR addressed the air pathway separately from other pathways in the interest of addressing the community's concerns quickly and accurately. Furthermore, this separate assessment allowed a more comprehensive and detailed analysis of the air pathway. The public comment period provides an opportunity to address any statements that may appear to be contradictory or any other issues arising from the assessment.

Responses to Specific Comments

1. ***Tronox LLC stated that the PAH exposures described in the report were potential and not actual. They therefore recommended adding the word "potentially" before the word exposed.*** The Public Health Assessment process uses a weight of evidence approach to conclude on community exposures. The definitive statements on exposure are made because, if pollutants are at a facility and measured in the air near the fence line and a resident lives near the fence line, that person must breathe the air with its pollutants and is therefore exposed. Therefore, we do not concur with the language that people were "potentially exposed" or "may have" been exposed.
2. ***Tronox LLC agreed with ATSDR health conclusion regarding pentachlorophenol, but disagrees that naphthalene exposures were confirmed. Specifically, Tronox LLC stressed that the measured levels of naphthalene were lower than the published values for olfactory detection and therefore ATSDR can not say that the chemical is present when the characteristic odor is smelled.*** ATSDR agrees that the measurements of naphthalene were lower than the reported olfactory detection levels for single chemical controlled studies. We also expect that the rain-induced release of semi volatile and volatile chemicals will be much lower than the values measured during operation. Therefore, the statement regarding naphthalene's odors was revised to address naphthalene in creosote. The statement is necessary because the community (and other communities near creosote facilities) complain of the characteristic creosote odors during rains. Naphthalene is more volatile than the other creosote chemicals and does off-gas.
3. ***Tronox LLC believes that ATSDR's health recommendation to remove treated wood from the homes is likely to be construed as referring to Kerr McGee's treated wood products.*** ATSDR agrees that the first statement is general one and not specific to Kerr McGee. It was revised to ensure that it is understood that we mean all treated wood, regardless of producer. Most treated wood contains some hazards. Unlike the other facility emissions that are not relevant to community exposures to chemicals from Kerr McGee, treated wood in the home regardless of maker is relevant. The first statement is an important and relevant health protective recommendation, especially since Dahlgren (2006) identified that two people in the community had elevated levels of pentachlorophenol in their blood, indicating exposure.

The second statement also addresses a community concern. As mentioned above (#2), the community was concerned with soil odors that do smell like creosote. The statement addresses that concern.

4. ***Tronox LLC believes that ATSDR's risk conclusion on pentachlorophenol is speculative. Tronox LLC believes that there is no evidence of an unacceptable body burden of pentachlorophenol and that the potential added contribution from Kerr McGee operations of the past is speculative. Tronox LLC also wants ATSDR to remove the sentence referring to exposure to PCP in the ditch.*** The CDC, EPA, and FDA determined that the amount of pentachlorophenol exposure in the nation in the 1970s-1980s presented an unacceptable risk. Since the nation and this community was already at a level that is deemed to be an unacceptable risk, any additional exposures will contribute to that risk, whether using a linear or threshold based risk assessment paradigm. The sentence that discusses other pentachlorophenol exposure from ditches is germane in this report because it adds to the body burden of some individuals. No dose calculation is provided in this report because such doses are much more dependent on an individual's access to the ditches, which differs greatly from inhalation doses. Nevertheless, the community remembers children having contact, so it is germane to the issue of exposure.

5. ***Tronox LLC found an error within Figure 1 that also resulted in miss-alignment of data and a misstatement about the figure.*** Figure 1 incorrectly listed one of the congeners twice and was revised. The Dahlgren 2003 data was checked and verified, but the abscissa's labels were changed to improve readability. the identification of the duplication of one congener, lead to the revision of one figure in the appendix along with associated text. The result is that only one dioxin congener associated with pentachlorophenol appears to be elevated. This strengthens the support for the conclusion about very little dioxin exposure.

6. ***Tronox LLC identified a typo on page 13.*** The date was corrected from 1998 to1988.

7. ***Tronox LLC purports that if concentrations are below levels known to result known to result in a measurable adverse effect, then the exposures could not be "unacceptable".*** There are uncertainties when applying epidemiologic studies to determine hazards to other populations. Epidemiologic studies involve small populations and look at diseases that have very low prevalence. Even if chemical exposures result in a high relative risk of a disease, if the prevalence of that disease is low, the total number of people who will get that disease will be low. Because of low prevalence of the diseases related with wood-workers, we do not expect to see adverse health effects in a community who get lower exposures. However, because of the elevated relative risk of workers, acceptable risk models result in theoretical risk that we consider elevated. While ATSDR's multi-factorial weight of evidence approach may appear too qualitative, it is often supported by EPA's theoretical risk formula. For pentachlorophenol, EPA's 1998 theoretical risk assessment places an unacceptable risk within the range of 0.052-5.2 $\mu g/m^3$.

8. ***Tronox LLC disagrees that irritation is associated with the concentrations measured and calculated.*** The issue of odors and irritation was previously addressed in responses #2 and #3. Naphthalene measured at the Kerr McGee fence line was 13 $\mu g/m^3$ which is above the agency's Minimum Risk Level (MRL) of 3 $\mu g/m^3$, which necessitated a closer evaluation of the data.

9. ***Tronox LLC recommended that the date of sampling be placed in Table 1.*** The year of sampling was placed in Table 1.

10. ***Tronox LLC agrees with most of the naphthalene conclusions, but stresses there was "no evidence" of concentrations higher than13 µg/m^3.*** We do recognize that the number of samples available for Kerr McGee are few. However, that data is consistent with data from other sites and as previously stated in our response to comment #8, the value at the fence line for Kerr McGee was much higher than the MRL and it occurred on a year of low production. We also agree that long-term average concentrations could be lower than 13 µg/m^3, therefore, the text was changed to indicate the range of values measured during that low production year. It should be noted that levels as high as 350 µg/m^3 were measured on site.

11. ***Tronox LLC offers references of naphthalene-specific odor studies and sites the specific information about naphthalene-only odors .*** Tronox correctly recognizes that the olfactory detection studies of single chemicals conclude that chemicals are odorous at much higher concentrations than measured at mixed chemical sites. However, ATSDR has visited several wood treatment facilities and reviewed their associated sampling data. The text was revised to clarify the basis of the conclusion on odors and irritation. Additionally, revisions were made to reflect naphthalene and associated odors (Also see information in the other responses).

12. ***Tronox LLC identified a misspelling; identified that a cancer risk based value was used to screen a non-carcinogen; requested more details about the screening values used; and suggested the use of worker based values for some of the measurements reported .*** Fluorine was corrected to Fluorene as recommended. Tronox correctly identifies that the measured values were on site and not at the residences that are farther than 200 ft away. They are appropriately compared to occupational values in the referenced [Borak 2001] document. As requested by Tronox, we noted that these were worker-related samples in Table 2.

    However, because of the limited fenceline data ATSDR needed to use the onsite outdoor samples that were collected in an area that could migrate to the community in low wind conditions. ATSDR recognized that these contaminants would be diluted in the air as they traveled 200 ft toward the community. Therefore, ATSDR only used the concentrations as an upper bound level. Then ATSDR screens out those chemicals of minimal concern. ATSDR uses cancer and non-cancer comparison values when screening data to determine which chemicals deserve consideration. Such screening is important when many chemicals have a common health endpoint. Cancer screening helps prevent scientists from inappropriately screening out those chemicals that pose a low risk when they are present with other chemicals that pose a higher risk of the same health endpoint. Even though the measurements were collected outdoors, they were collected close to residences. The comparison values used were noted as being redundantly protective and they were not exceeded. Therefore, these chemicals were not considered any further. We recognize that the MRL is numerically identical to the RfD and that note was added to the table. (Also see response to #23.)

13. ***Tronox LLC identified that footnotes were not on the same page that they were referenced.*** The footnotes associated with the table were checked in the final document.

14. ***Tronox LLC requested that the last sentence on page 18 be rewritten to state that the levels measured at Kerr McGee were low for workers .*** The last sentence was changed as recommended to clarify that we believe the Kerr McGee workers had lower exposures than at other similar facilities.

15. ***Tronox LLC requested that we clarify the constituent being discussed on page 19; that Tronox does not agree with the odors causing irritation (citing a reference); that the 13 µg/m^{3}was a singular maximum; and that other sources are all or partially responsible for the soils and sediment contamination.*** The name of the specific constituent, Naphthalene, was added as suggested. A reference was also added as suggested. Sentences on naphthalene were modified to reflect the range of measurements rather than the maximum, when they were measured, and the issue of odors associated with asphalt being known to occur even at times when naphthalene cannot be detected using conventional analytical methods. (More discussion on the odors is provided above.)

    There is no change to the sediment and soil statements –contaminants associated with wood treatment were found in the soil and sediment and those contaminants were available for occasional exposure to those who accessed those areas.

16. ***Tronox LLC recommends removing the odor section on page 19.*** Odors were a concern by the community near Kerr McGee and are a concern in every other wood treatment facility that ATSDR has investigated. Our scientists visited these communities and witnessed these strong odors. It would be negligent not to address this major concern that affects the wellness of the community.

17. ***Tronox LLC recommends removing the odor section on page 20.*** See our previous responses to comments on odors.

18. ***Tronox LLC suggests that the relevance of the PCP effects on women is unclear when discussing children and they suggest deleting the discussion of these studies.*** The discussion of pentachlorophenol exposures to women and children are critical to support the limitations in applying only male worker studies to residents that include women and children. Since some children are female, it is especially critical to discuss the women studies along with the children. Animal studies show males and females metabolize and respond differently following pentachlorophenol exposure. Unfortunately, few epidemiologic studies are available for people other than male workers. The epidemiologic studies presented in this section lack sufficient data to determine a dose response relationship; however, the relative risks they present suggest unique toxicity to females.

19. ***Tronox LLC suggests modifying statements regarding irritation and naphthalene exposure on page 22.*** The issues of odors have been modified, but not as recommended. See previous discussion.

20. ***Tronox LLC suggests modifying the conclusions about exposures contributing to an already unacceptable overall risk in the past, calling it speculative. Furthermore, they conclude that the highest value of naphthalene reported by ATSDR was below the olfactory detection level and therefore, naphthalene odors are not possible.*** The issue of exposure and risk are addressed above in responses #8 and #10. The issue of odors are addressed in #1 and #2 and other responses above.

21. ***Tronox LLC recommends deleting the sentence about removing treated wood from the home and deleting the sentence about removing or encapsulating soils with bad odors.*** Neither sentence was removed, but the first sentence has been changed so that it does not appear to refer to Kerr McGee (only) wood. Also, see our response to comment #3. Our response to odors is addressed in our response to comments #1 and #2 and the statements referring to odors and naphthalene elsewhere in the report were modified.

22. ***Tronox LLC noted an apparent contradiction between the table and text on page 37.*** The table was renamed to include the word Pentachlorophenol and the text was changed to reduce the apparent contradiction (detected verses measured).

23. ***Tronox LLC wanted a reference for the risk-based value of* 0.052 µg/m^3*; also, they did not want screening values for carcinogens and non-carcinogen used in the same table; and they pointed to another misspelling of fluorene.*** The 0.052 µg/m^3 value for pentachlorophenol risk is found in EPA's 1998, Region 3, Risk Based Concentration Tables. Furthermore, the EPA's 1998, Region 9, Preliminary Remediation Goal for pentachlorophenol is 0.056 µg/m^3 and the California EPA 2003 inhalation unit risk is 0.0000046 (µg/m^3)$^{-1}$. The factored comparison values used for the less toxic PAHs were noted as being protective and were not exceeded. Therefore, these chemicals were not considered any further. The fact that the MRLs are dose based should not preclude their use as a screening value. However, we recognize that the MRL is numerically identical to the RfD and have added that note in the associated table. Fluorene's spelling has been corrected. (Also see response to #23.)