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OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

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MEMORANDUM

SUBJECT: Creosote: Occupational and Residential Exposure and Risk Assessment for the Reregistration Eligibility Decision (RED). PC Codes 022003, 025003, and 025004.

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EXECUTIVE SUMMARY

Creosote applications are limited to occupational handlers at pressure treatment facilities. Since it is a restricted-use pesticide that can only be applied by certified applicators or someone under their direct supervision, it is not available for sale to or use by homeowners. A recent voluntary cancellation of all non pressure treatment uses restricts creosote to commercial and industrial settings.

This chapter is a revision of the earlier draft Human Exposure RED Chapter for Creosote completed in 2003. Subsequent to the last release of the creosote assessment, additional data were made available to further refine the assessment. The previous version of the human risk assessment was based on relying on benzo(a)pyrene as an indicator of creosote risk because creosote-specific data were not available. Data are now available to relate dermal absorption and cancer risks to creosote. The previous draft risk assessment also included scenarios using data from the Pesticide Handlers Exposure Database (PHED) and the Chemical Manufactures Association (CMA). These scenarios have been deleted in this current assessment based on the voluntary cancellation of all non pressure treatment uses of creosote.

The results of the Creosote Council's worker exposure study (MRID 453234-01) at pressure treatment facilities indicate that the naphthalene inhalation exposures trigger EPA's non cancer risk level of concern for 16 of the 19 inhalation MOEs assessed. The non cancer inhalation MOEs for worker exposure to naphthalene range from 23 to 1,900 (i.e., target MOE of 300). However, none of the average naphthalene air concentrations for the various job functions exceeded the ACGIH TLV and OSHA PEL of 52 mg/m^3. Furthermore, the published literature for creosote exposure also indicates naphthalene air concentrations in the range of that monitored in MRID 453234-01, with some upper ends of the range slightly higher (but those concentrations are for "total vapor"). The results of the air concentrations reported in the literature support the results of the Creosote Council's worker exposure study indicating that exposure to creosote should be reduced.

For dermal worker risks, the results indicate the short-term (ST) non cancer dermal MOEs do not trigger a risk concern except for the treatment operator at site C where the dermal MOE is 68 and the target MOE is 100. The intermediate-term (IT) non cancer dermal MOEs trigger risk concerns for 8 of the 24 scenarios presented. IT MOEs range from 3 to 2700 and the target MOE is 100. The long-term (LT) non cancer dermal MOEs trigger risk concerns for 3 of the 24 scenarios. LT MOEs range from 34 to 34,000 and the target MOE is 300. IT risks being greater than the LT risks is an anomaly. However, in the case of creosote it is explainable because the IT toxicity endpoint is based on a dermal study while the LT endpoint is based on an oral study (i.e., there are differences in routes of exposure and dosing levels between the two studies).

All of the cancer risks exceed the Agency's level of concern of 1×10^{-6} but only 4 of the scenarios had risks exceeding 1×10^{-4} (i.e., risks range from 2.5×10^{-5} to 1.6×10^{-6}).

The registrants submitted a probabilistic worker risk assessment for creosote in February 2008. This probabilistic assessment has been included in the public docket. A thorough EPA review of the probabilistic assessment has not been conducted. The methodology and data inputs in this recent submission differ from that presented by EPA. EPA's assessment herein presents a deterministic risk assessment. In summary, the Creosote Council's probabilistic assessment includes cancer risk results with and without the probabilistic analysis of the cancer slope factor. The mean and 95th percentile cancer risks reported in Table 9 of the Creosote Council's probabilistic assessment range from 10^{-4} to 10^{-5}. These reported risks are within the range of the risks presented by EPA in Table 6 of Section 3.1.4 below. However, EPA's assessment reports one cancer risk lower than that reported in the probabilistic assessment (i.e., 1.6×10^{-3} for the treatment operators at a facility built in the 1940s).

1.0 Introduction

An occupational and/or residential exposure risk assessment is required for an active ingredient if (1) certain toxicological criteria are triggered and (2) there is potential exposure to handlers (mixers, loaders, applicators, etc.) during use or to persons entering treated sites after application is complete. For creosote, both criteria are met.

On April 1, 1999, the EPA/OPP Health Effects Division's Hazard Identification Assessment Review Committee (HIARC) evaluated the toxicology data base of creosote and selected toxicological endpoints for short-term, intermediate-term, and long-term occupational and residential exposure risk assessments. On September 3, 2003, the Antimicrobials Division Toxicity Endpoint Selection Committee (ADTC) met to verify the selected endpoints for dermal and inhalation risk assessment. On December 6, 2007, members of the Antimicrobials Division's Toxicity Endpoint Selection Committee and members of the Health Effects Division's Carcinogenicity Assessment Review Committee (CARC) met to discuss the quantitative carcinogenicity analysis performed for creosote by the Pest Management Regulatory Agency, Health Canada and to determine an appropriate potency factor for creosote.

2.0 Summary of Toxicity Data

Dermal Absorption: Submitted studies on the dermal absorption of creosote have been submitted and consist of an in vivo dermal absorption study in the rat as well as an in vitro dermal absorption study using both rat and human skin (MRIDs 47179501 and 47179502). The results of these studies support the conclusion that dermal absorption in human skin is approximately 8-fold lower than that of rat skin. The results of the submitted studies also support a value for dermal absorption of creosote in rat skin of approximately 34%. Thus, estimated dermal absorption of creosote in human skin is determined to be 5% (34% value divided by 8 and rounded to 5%). A lower dermal absorption value suggested by the registrant was not used because of the lack of data on solubility limit of the creosote mixture itself in the in vitro test system, and the continued absorption of creosote observed after 8 hours in the in vivo study suggesting the availability of creosote within the skin for absorption.

Short-Term Dermal (1 day - 1 month): An oral maternal NOAEL of 50 mg/kg/day and a LOAEL of 175 mg/kg/day, based on decreased body weight gain during the study, was chosen for this endpoint (USEPA, 2008). Although a 90-day dermal toxicity study was available, the developmental toxicity study was chosen because dermal toxicity studies (including the 2-week range-finding studies) did not measure developmental endpoints, which are present in both developmental toxicity studies. An uncertainty factor (MOE) of 100 is applied to this risk assessment (USEPA, 2008).

Intermediate-Term Dermal (1 month to 6 months): A dermal NOAEL of 40 mg/kg/day, based on decreased body weight gain in males at 400 mg/kg/day observed in the 90-day dermal toxicity study (MRID # 43616201). An uncertainty factor (MOE) of 100 is applied to this risk assessment (USEPA, 2008).

Long-Term Dermal (greater than 6 months): A parental oral LOAEL of 25 mg/kg/day, based on decreased pre-mating body weight, was selected for this endpoint. An extra uncertainty factor of 3x is applied for use of a LOAEL in this study for occupational risk assessments (USEPA, 2008). Based on the results of this study, the Parental Systemic NOAEL is < 25 mg/kg/day, and the Parental Systemic LOAEL is 25 mg/kg/day, based on decreased pre-mating body weight. The developmental NOAEL in this study is < 25 mg/kg/day, and the developmental LOAEL is 25 mg/kg/day, based on a dose-related decrease in pup body weight for the F0 pups from days 14-21. The reproductive NOAEL is < 25 mg/kg/day, and the reproductive LOAEL is 25 mg/kg/day, based on reduced pregnancy and fertility indices in F1 female parental rats (USEPA, 2008).

Inhalation (any time-period): An NOAEL of 0.0047 mg/L, based on decreased body weight gain, altered hematology and clinical chemistry, and increased absolute and relative weight of the liver and thyroid and increased incidence of lesions of the nasal cavity observed at 0.048 mg/L in P2 creosote CMT in rats (USEPA, 2008). In a 13-week inhalation toxicity study (MRID # 43600901), 20 Sprague-Dawley rats/sex/group were treated for 5 days/week, 6 hours/day with P2 Creosote CTM via whole body exposure at doses of 0, 4.7, 48 or 102 mg/m^3 (0, 0.005, 0.048 or 0.102 mg/L) in air measured gravimetrically. The aerosol size MMAD was between 2.4 and 2.9 microns with a geometric standard deviation between 1.85 and 1.91. For worker risk, naphthalene was selected as an indicator because 100 percent of the inhalation samples monitored at the pressure treatment facilities were detectable. For naphthalene, the Antimicrobials Division used the inhalation reference concentration (RfC) for naphthalene published in the EPA's IRIS database adjusted for the work week (i.e., EPA recognizes that the 24 hour/day 7 day/week adjustment to the RfC is not representative of a typical work day). The RfC was derived from a 2 year chronic inhalation study in the mouse in which exposure was for 6 hours/day, 5 days/week. The inhalation route-specific LOAEL is 52 mg/m^3 with a target MOE of 300 (10x intra species variability, 10x inter species extrapolation, and 3x for a lack of a NOAEL).

Carcinogenicity: In conjunction with Health Canada's Management Regulatory Agency (PMRA), a quantitative risk assessment on carcinogenicity of creosote has been performed using the data of Culp et al. (1998). A dermal carcinogenicity study by Bushmann et al. (1997) was also available, but was determined not suitable for quantitative assessment of carcinogenicity. Ulceration of the skin was significant finding of the study which potentially affected tumor response. In addition, systemic toxicity was not examined, and complete histopathology data were not available. Based upon the analysis of the Culp et

al. data, an oral cancer potency factor of 6.28×10^{-6} $(\mu g/kg/day)^{-1}$ or 6.28×10^{-3} $(mg/kg/day)^{-1}$ for the coal tar mixture 1 tested in this study was selected, on the basis of forestomach tumors observed.

2.1 Acute Toxicology Categories

Table 1 provides the acute toxicity categories for creosote. It also provides the results of the toxicity tests (USEPA 2008).

Table 1. Acute Toxicity Categories for Creosote

Test	Results	Toxicity Category
Acute Oral Toxicity	LD_{50} = 2,451 mg/kg (M); 1,893 mg/kg (F)	III
Acute Dermal Toxicity	LD_{50} > 2,000 mg/kg	III
Acute Inhalation Toxicity	LC_{50} > 5 mg/L	IV
Primary Eye Irritation	Irritation clearing in 8-12 days	II
Primary Dermal Irritation	Erythema to day 14	III
Dermal Sensitization	Study unacceptable	NA

NA - Not applicable, no toxicological endpoint.

2.2 Summary of Endpoints of Concern

Endpoints for assessing occupational and residential risks are presented in Table 2 (USEPA 2008).

Table 2. Summary of Toxicological Endpoints for Creosote.

EXPOSURE SCENARIO	DOSE (mg/kg/day)	ENDPOINT	STUDY

EXPOSURE SCENARIO	DOSE (mg/kg/day)	ENDPOINT	STUDY
Acute and Chronic Dietary	Acute and Chronic Dietary risk assessment not required		
Carcinogenicity (dermal)	Creosote has been shown to exert positive mutagenic effects in vitro, and has been shown to be positive for carcinogenicity in an initiation/promotion study. Creosote has been classified as a B1 carcinogen in IRIS. An oral cancer slope factor of 6.28×10^{-3} (mg CTM1/kg/day)$^{-1}$ was selected for creosote using the data of Culp et al (1998) for the coal tar mixture 1 (CTM1) on the basis of forestomach tumors.		
Short-Term (Dermal)	Oral NOAEL=50	decreased body weight gain at 175 mg/kg/day	Developmental Toxicity - Rat
	MOE = 100 (5% dermal absorption used to correct for use of oral endpoint)		
Intermediate-term (Dermal)	Dermal NOAEL = 40	Decreased body weight gain at 400 mg/kg/day	90-Day Dermal Toxicity Study in the Rat
	MOE = 100		
Long-Term (Dermal)	Oral LOAEL = 25 mg/kg/day	decreased pre-mating body weight	2-generation reproduction study - Rat
	MOE = 300 (10x interspecies, 10x intraspecies, 3x for use of a LOAEL)		
Inhalation (any time period)	Creosote NOAEL = 0.0047mg/m^3	MOE = 100 decreased body weight, body weight gain, altered hematology	90-day Inhalation Study in the Rat

EXPOSURE SCENARIO	DOSE (mg/kg/day)	ENDPOINT	STUDY
	Naphthalene LOAEL = 52 mg/m^3	nasal effects: hyperplasia and metaplasia in respiratory and olfactory epithelium respectively	Two year inhalation toxicity study - mouse (USEPA, IRIS)
Dermal absorption	5%, determined from the results of in vivo / in vitro testing in rats and in vitro testing using human skin.		

2.3 FQPA Considerations

As there are no existing tolerances or other clearances for residues of creosote in food, an FQPA assessment is not necessary. The available evidence on developmental and reproductive effects of creosote was assessed by the Health Effects Division (HED) Hazard Identification Assessment Review Committee on April 1, 1999. The committee expressed concern for potential infants and children's susceptibility of creosote, based on the severity of offspring vs. maternal effects observed with testing of creosote in the P1/P13 blend developmental toxicity study in rats at the 175 mg/kg/day dose level as well as deficiencies observed in the 2-generation reproduction toxicity study in rats.

Although there are no current Agency guideline neurotoxicity studies available for creosote, the existing studies on creosote indicate no evidence of neurotoxicity for either the P1/P13 or P2 blends of creosote (ATSDR, 2002). Based on the above, and realizing that creosote is currently registered only for non -food use and is a restricted use pesticide, no additional neurotoxicity testing will be required at this time.

3.0 Occupational Exposures and Risks at Pressure Treatment Facilities

Creosote is used by occupational handlers only. Since it is a restricted-use pesticide that can only be applied by certified applicators or someone under their direct supervision, it is not available for sale to or use by homeowners. Furthermore, the non pressure treatments of creosote have been voluntarily cancelled by the registrants. Creosote applications are now restricted to pressure treatment cylinders.

EPA has determined that there are potential exposures to mixers, loaders, applicators, and other handlers during typical use-patterns associated with creosote

pressure treatment uses. Table 3 provides a summary of worker exposure scenarios at pressure treatment facilities. Although specific job functions have been defined within each exposure scenario, EPA acknowledges the occasional need for workers to cross over into other job functions. Table 3 also provides the numbers of monitoring events at each of four sites from MRID 453234-01.

Table 3: Job Descriptions of Workers Exposed at Pressure Treatment Facilities (Creosote Council Study – MRID 453234-01).

Job Function	Description of worker activities	Monitoring Events		
		Site	Dermal	Inhalation
Treatment Operator TO (engineer)	Operates and manages the treatment system; may open and close cylinder doors; cleans accumulated creosote from doors and latches; operates valves to transfer creosote solution between holding tanks and treatment cylinders; handles leads and bands.	 A B C D	total: 18 4, 1/day 4, 1/day 5, 1/day 5, 1/day	total: 14 0 4, 1/day 5, 1/day 5, 1/day
Treatment Assistant TA (helper)	Performs and assists with tasks of the TO; charge preparation, cylinder cleaning, maintenance, filter cleaning, mixing treatment solution; loader operation and movement of charges.	 B	total: 4 4, 1/day	total: 4 4, 1/day
Oil unloader OU	Operates creosote tank car unloading and transfer system; takes samples from tank cars; inserts siphons into tanks. (At site C, the tasks for this position were performed by the TO; position was not monitored at Site B)	 A D	total: 9 4, 1/day 5, 1/day	total: 5 0 5, 1/day
Loader Operator CLO (cylinder area) LLO (load out area)	Operates self-propelled vehicles for loading wood on and off trams, moving charges in and out of cylinders, and to and from load out areas. Out-of-cab tasks include tram placement, and handling chains and leads.	CLO A B C D	total: 18 4, 1/day 4, 1/day 5, 1/day 5, 1/day	total: 14 0 4, 1/day 5, 1/day 5, 1/day
		LLO B C	total: 19 4, 1/day 5, 1/day	total: 19 4, 1/day 5, 1/day

Table 3: Job Descriptions of Workers Exposed at Pressure Treatment Facilities (Creosote Council Study – MRID 453234-01).				
Job Function	**Description of worker activities**	**Monitoring Events**		
		Site	**Dermal**	**Inhalation**
		D	10, 2/day	10, 2/day
Loader helper CH; LH	Assists the LO in some tasks; works mainly on the drip pad and load out area, placing and removing charge leads, opening and closing cylinder doors, retrieving leads, adjusting track switches, and banding and unbanding charges.	 B C D	total: 14 4 LH, 1/d 5 CH, 1/d 5 CH, 1/d	total: 14 4 LH, 1/d 5 CH, 1/d 5 CH, 1/d
Checker CK	Performed tasks of the loader helper as well as inspecting treated lumber. Worker part time in the treatment area.	 C	total: 5 5 CH, 1/d	total: 5 5 CH, 1/d
Test Borer/QC Person TB	Takes core samples to test for creosote penetration; may test creosote solution concentration (site C); other QC laboratory duties. (These tasks performed by CLO at site B)	 A C	total: 9 4, 1/day 5, 1/day	total: 5 0 5, 1/day
Water Treatment System Operator WO	Operates chemical/biological water recovery equipment (At Site C, the tasks associated with this position were performed by the TB; position not monitored at Site D)	 A B	total: 8 4, 1/day 4, 1/day	total: 4 0 4, 1/day
Drip pad cleaner DP	Steam-cleans drip pad area; disposes of sludge and treated wood waste; other cleanup duties in treatment and drip pad area.	 C	total: 4 4, 1/day	total: 4 4, 1/day
Total			108	88

Site A is Florence, SC. Site B is Delson, Quebec. Site C is Denver, CO. Site D is Somerville, Tx.

The worker exposure study on pressure treatment applications submitted by the Creosote Council II to provide chemical-specific handler dermal and inhalation exposure data in support of the re-registration of pressure treatments of creosote (Creosote Council II, 2001, MRID 453234-01) is presented in Section 3.1. Other published studies for creosote are presented in Section 4.0.

Because of the overall variability in the composition of creosote (e.g., over 100 known chemicals are components of creosote), it is difficult to characterize its exact nature. Since neither the characterization of airborne creosote nor the development of inhalation sampling methods is specific for creosote, there exists a high variability in the creosote inhalation data presented in the literature. Most of the studies presented in the literature were conducted by industrial hygienists using methods approved by the National Institute for Occupational Safety and Health (NIOSH) and Occupational Safety and Health Administration (OSHA) for polycyclic aromatic hydrocarbons (PAHs), phenols/creosols, and the individual constituents of the PAHs (i.e., naphthalene, phenanthrene, anthracene, etc). The Creosote Council study is the most recent study presented on creosote exposure and presents both dermal and inhalation exposure. This study provides the best available data on worker exposure estimates and encompasses all of the worker activities contributing to exposure. Nonetheless, other studies available in the literature are also presented below in Section 4.0.

3.1 Worker Exposure at Pressure Treatment Facilities

The 2001 Creosote Council II study was conducted to determine the dermal and inhalation exposure of workers exposed to creosote while performing routine tasks related to pressure treatment of lumber, utility poles, and railroad ties. The study was conducted at four typical commercial treatment facilities in the U.S. and Canada, per the requirements of the U.S. Environmental Protection Agency, Canada's Pesticide Management Regulatory Agency (PMRA), and the California Department of Pesticide Regulation. The four sites include older facilities from the 1940s as well as more modern facilities with additional engineering controls. Therefore, the exposure and risk estimates have been presented separately for each site. The job functions monitored in the study are presented in Table 3 above.

Previous drafts of the EPA's creosote assessment have defined the job functions at pressure treatment facilities as either handlers or postapplication. Since the job functions previously categorized as handlers (treatment operators and assistants) perform many functions, this creosote assessment does not highlight the job functions as being either handler or postapplication. Workers in the study performed typical tasks related to their job functions and were monitored during a full work cycle beginning at 7 AM and ending at 3 PM.

3.1.1 Pressure Treatment Process

Pressure treatment is often required because of the resistance of wood to deep penetration by preservatives. The pressure treatment process begins when untreated wood is loaded onto rail/tram cars that are pushed into the treating cylinder using locomotives,

forklifts, or similar equipment. The cylinder door is sealed via a pressure-tight door and the operation remains a closed system during the entire treatment process. Treating solutions are then pumped into the cylinder and the inside pressure is raised. At the end of the treatment process, the excess treating solution is pumped out of the treating cylinder and back to storage for reuse. The cylinder is opened, and the rail/tram cars holding the treated wood are pulled out of the cylinder using a locomotive, forklift, or similar equipment.

The amount of creosote handled in a given day among pressure treatment facilities depends on such factors as the size of the facility and the number of treatment cylinders on site. In a given facility, the amount of creosote handled per day varies depending on the wood conditioning techniques used for a given charge, on the type of wood being treated, and the type of product being produced (e.g., marine piling vs utility poles).

According to information provided by industry sources (Krygsman, 1994), wood pressure treatment of railroad ties in a retort may last anywhere from 4 to 24 hours. A typical retort cylinder has a diameter of about 8 feet and a length of about 120 feet. About 16 rail/tram cars can be placed in a retort at one time. The rail/tram cars usually are connected together and are pushed in and out of the retort on railroad tracks using a locomotive. Wood preservative is loaded into the wood pressure treatment retort facilities from rail tank cars using hoses and metered pumps. The wood preservative is stored in two or three holding tanks that may be as large as 60,000 gallons. During the wood treatment process the wood is sprayed under pressure in the enclosed retort. In the retort, a "charge" of liquid preservative is pumped into the trams and then later pumped out. After the wood preservative is pumped out, the wood is dried through a vacuum treatment and the tram cars containing wood (e.g. railroad ties) are then pulled out. Since the wood in the tram cars is pulled by mechanical means there is very little direct human contact with the exposed wood. Likely contact is through dermal contact with equipment that was previously in the retort, removing cables that separated layers of ties, dermal and inhalation contact to vapors inside the retort before and after pressure treatment, cleaning the retort, and inspecting wood pieces by coring the wood.

3.1.2 Dermal Exposure Monitoring

Since creosote is a complex mixture of over 100 chemicals including phenols, creosol, and aromatic hydrocarbons, it is analytically difficult and cost prohibitive to identify all of the chemicals in the mix. In addition, creosote cannot be measured directly because of its complex mixture. Dermal exposure to "total creosote" was estimated by measuring the levels of 10 individual polynuclear aromatic hydrocarbon (PNA) compounds. Each analyte was determined in each whole-body dosimeter (WBD) and glove sample as if it represented total creosote. The goal was to use these marker compounds to represent "total creosote".

The creosote dermal exposure to each worker was determined using a WBD, consisting of a 100% cotton thermal shirt and long pants. Each worker at Sites A, C, or D wore his WBD under a fresh work uniform consisting of a cotton long-sleeved work shirt and cotton work trousers (or one-piece cotton coverall) provided by the test site. The workers at Site B were not provided uniforms therefore; each worker wore a WBD under a fresh lightweight cotton/polyester sweat shirt and pants purchased locally by study personnel. The workers at all four sites wore a lightweight 100% cotton glove dosimeter on each hand under his chemical-resistant or work gloves, as appropriate. Each of these 10 analytes was determined for each WBD and glove sample as if it represented total creosote. The average of the analyte concentrations were used to estimate the level of total creosote present in/on the individual sample.

3.1.3 Inhalation Exposure Monitoring

Inhalation exposure for each worker was monitored by a personal air sampling train. Inhalation exposure was estimated for 11 individual PNA compounds as well as for benzene-soluble PNAs and related compounds collectively known as coal tar pitch volatiles (CTPVs). The Polytetrafluroethylene (PTFE) filter retained the CTPVs, while the PNAs were retained in the XAD-2 resin tubes. Each worker wore a sampling train consisting of a PTFE filter upstream from two in-line XAD-2 resin-filled air sampling tubes. However, there was no attempt by the study sponsors to relate inhalation levels found for PNAs and CTPVs to "total creosote" -- a significant weakness with the study. Moreover, there were analytical problems encountered with the CTPV samples and all samples were non detect. Therefore, EPA did not rely on the CTPV inhalation exposure monitoring results. Instead, naphthalene was used to indicate inhalation exposure concerns.

Inhalation exposure monitoring at Site A was unsuccessful because a single XAD-2 tube was used along with a non-solvent-resistant filter cassette. Therefore, the sampling methodology was changed to include the use of a second XAD-2 resin tube in the sampling train prior to sampling at Sites B, C, and D. Inhalation exposure monitoring was performed successfully at these sites. Each worker at Sites B, C, and D was equipped with an air sampling train consisting of a PTFE filter in an opaque, solvent-resistant plastic cassette connected upstream from two in-line XAD-2 resin-filled air sampling tubes. The intake orifice of the filter was placed in the worker's breathing zone, directed downward. Air was pulled through the sampling train by a portable air sampling pump attached to the worker's belt. The pump drew air through the sampling tube at approximately 1 L/minute while the worker performed his tasks. Pumps were calibrated immediately prior to and after each monitoring period using a mass flow meter or bubble calibrator. The pumps were turned on at the beginning of each work cycle and were left running during restroom, coffee, or other short breaks, but were turned off or set on "hold" during lunch breaks.

The pumps and samplers were removed from the worker during the lunch break. At the conclusion of the lunch break, the pump and sampling train were reinstalled and the pump restarted. All start and stop times for breaks were recorded.
During each work cycle, start times and end times of each task performed by the worker were recorded. Pump parameters during use were also recorded. At the end of each work cycle, the pumps and sample trains were collected. Each filter cassette and sampling tube were capped, labeled, bagged, and placed on dry ice for shipment to USX Engineers and Consultants, Inc. (UEC) for extraction and analysis. After the collection of the air samples, the air sampling pump was re-calibrated.

3.1.4 Exposure and Risk Characterization

Estimated Dose: The short-term dermal endpoint is based on a maternal toxicological endpoint; therefore, a female body weight of 60 kg was used for the dose calculation. The median adult male/female body weight of 70 kg was used for the intermediate-term, long-term, and cancer endpoints. Short-term, long-term, and cancer endpoints are all based on oral administrations. Therefore, the 5 percent dermal absorption factor was used to estimate an absorbed dermal dose for comparison to an orally administered dose in the toxicity studies. The intermediate-term endpoint is based on a dermal toxicity study, and therefore, no absorption factor was necessary. A route specific inhalation assessment has been developed comparing the air concentrations monitored for workers directly to the human equivalent concentration (HEC) without the need for an extrapolated dose estimate.

The following equation was used to estimate the dermal dose. Because EPA traditionally uses an adult body weight of 70 kg and female body weight of 60 kg in its exposure assessments which is slightly different then the 71.8 kg body weight used in the Creosote Council II exposure assessment, the doses used in this assessment had to be normalized back to daily dermal exposures. The normalization was performed by multiplying the exposure dose times the 71.8 kg body weight. Subsequently, the dermal exposure was converted into an absorbed and/or potential dose using the 60 and 70 kg body weights.

- Absorbed Daily Dermal Dose (mg/kg/day) = Dermal Exposure (mg/day) x Dermal Absorption (%) x (1/Body Weight)

The estimated absorbed dermal lifetime average daily dose (LADD) is based on the following equation:

- $LADD_{[absorbed]}$ (mg/kg/day) = Absorbed dermal dose (mg/kg/day) x (250 days worked/365 days) x (35 years worked/70 year lifetime)

Estimated Non Cancer and Cancer Risks: The calculations of the daily dermal dose of creosote received by workers were used to calculate the non cancer MOEs for the short-term, intermediate-term, and long-term durations. The dermal MOEs were calculated using (1) a NOAEL of 50 mg/kg/day for short-term exposure with a target MOE of 100: (2) a NOAEL of 40 mg/kg/day for intermediate-term exposures with a target MOE of 100; and, (3) a LOAEL of 25 mg/kg/day for the long-term duration with a target MOE of 300. Note: The intermediate-term dermal endpoint was selected from a dermal toxicity study, and therefore, a dermal absorption factor was not necessary to calculate the potential dose. The dermal and inhalation non cancer MOE equations are as follows:

- $MOE_{[dermal]}$ = NOAEL or LOAEL / Potential and/or Absorbed Dermal Dose
- $MOE_{[inhalation]}$ = Human Equivalent Concentration (HEC) / Worker's air concentration

The cancer risk for creosote is based on the estimated absorbed dermal lifetime average daily dose (LADD) multiplied by the cancer slope factor for creosote dose as follows:

- Cancer Risk = $LADD_{[absorbed]}$ (mg/kg/day) x CSF of 6.28×10^{-3} $(mg/kg/day)^{-1}$

Using these equations, the worker exposure and risk estimates from the Creosote Council's exposure study are presented in Table 4 (dermal MOEs), Table 5 (inhalation MOEs), and Table 6 (dermal cancer risk).

Dermal MOEs (Table 4): The results indicate the short-term (ST) non cancer dermal MOEs do not trigger a risk concern except for the treatment operator at site C where the dermal MOE is 68 and the target MOE is 100. The intermediate-term (IT) non cancer dermal MOEs trigger risk concerns for 8 of the 24 scenarios presented. IT MOEs range from 3 to 2700 and the target MOE is 100. The long-term (LT) non cancer dermal MOEs trigger risk concerns for 3 of the 24 scenarios. LT MOEs range from 34 to 34,000 and the target MOE is 300. IT risks being greater than the LT risks is an anomaly. However, in the case of creosote it is explainable because the IT toxicity endpoint is based on a dermal study while the LT endpoint is based on an oral study (i.e., there are differences in routes of exposure and dosing levels between the two studies).

Table 4. Creosote Dermal MOEs.								
Job	Site	n=	Site Description	Potential dermal dose (mg/kg/day)	Absorbed Dermal Dose (mg/kg/day)	Dermal MOEs		
						ST	IT	LT
TO	A	4	1940s; manual	0.414	0.021	2415	97	1208
	B	4	1983; Eng. Controls	0.015	0.001	67568	2703	33784
	C	5	1940s	14.800	0.740	68	3	34
	D	5	1970s; Automated	0.132	0.007	7576	303	3788
TA	B	4	1983; Eng. Controls	0.025	0.001	40323	1613	20161
OU	A	4	1940s; manual	0.887	0.044	1127	45	564
	D	5	1970s; Automated	0.938	0.047	1066	43	533
CLO	A	4	1940s; manual	0.212	0.011	4717	189	2358
	B	4	1983; Eng. Controls	0.089	0.004	11299	452	5650
	C	5	1940s	2.120	0.106	472	19	236
	D	5	1970s; Automated	0.117	0.006	8547	342	4274
LLO	B	4	1983; Eng. Controls	0.018	0.001	55249	2210	27624
	C	5	1940s	0.203	0.010	4926	197	2463
	D	10	1970s; Automated	0.077	0.004	12953	518	6477
LLO(F)	D		1970s; Automated	0.244	0.012	4098	164	2049
LH	B	4	1983; Eng. Controls	0.023	0.001	43860	1754	21930
	C	5	1940s	1.810	0.091	552	22	276
	D	5	1970s; Automated	0.383	0.019	2611	104	1305
CK	C	5	1940s	0.822	0.041	1217	49	608
TB	A	4	1940s; manual	0.112	0.006	8929	357	4464
	C	5	1940s	1.060	0.053	943	38	472
WO	A	4	1940s; manual	0.204	0.010	4902	196	2451
	B	4	1983; Eng. Controls	0.047	0.002	21322	853	10661
DP	C	4	1940s	0.150	0.008	6667	267	3333

Site A,B,C,D indicate differences in site setup (e.g., eng controls).
Dermal exposures are not normalized to the various amount of wood treated.
Arithmetic mean of the dermal dose from Table 9 of the PMRA worker study review.
Abs Dermal Dose (mg/kg/day) = dermal dose (mg/kg/day) x 5% dermal absorption
Where ST NOAEL is 50 mg/kg/day (Target MOE = 100) and LT LOAEL is 25 mg/kg/day (Target MOE = 300).
Where IT NOAEL is 40 mg/kg/day (Target MOE = 100) from a dermal study.

Inhalation MOEs (Table 5): The non cancer inhalation MOEs for worker exposure to naphthalene range from 23 to 1900 with a target MOE of 300. Sixteen of the 19 inhalation MOEs presented exceed the target MOE of 300, and therefore, are of concern. None of the average air concentrations for the various job functions exceeded the ACGIH TLV and OSHA PEL of 52 mg/m^3.

Table 5. Inhalation MOEs for Naphthalene.

Job	Site	n=	Site Description	Average Naphth (ug/m^3)	Average Naphth (mg/m^3)	% of TLV	MOE (Target 300)
TO	A	4	1940s; manual	NA	NA	NA	NA
	B	4	1983; Eng. Controls	221	0.221	0.4	235
	C	5	1940s	1320	1.32	2.5	39
	D	5	1970s; Automated	802	0.802	1.5	65
TA	B	4	1983; Eng. Controls	406	0.406	0.8	128
OU	A	4	1940s; manual	NA	NA	NA	NA
	D	5	1970s; Automated	925	0.925	1.8	56
CLO	A	4	1940s; manual	NA	NA	NA	NA
	B	4	1983; Eng. Controls	227	0.227	0.4	229
	C	5	1940s	2033	2.033	3.9	26
	D	5	1970s; Automated	574	0.574	1.1	91
LLO	B	4	1983; Eng. Controls	27	0.027	0.1	1926
	C	5	1940s	694	0.694	1.3	75
	D	10	1970s; Automated	195	0.195	0.4	267
LLO(F)	D		1970s; Automated	679	0.679	1.3	77
LH	B	4	1983; Eng. Controls	43	0.043	0.1	1209
	C	5	1940s	1870	1.87	3.6	28
	D	5	1970s; Automated	2251	2.251	4.3	23
CK	C	5	1940s	117	0.117	0.2	444
TB	A	4	1940s; manual	NA	NA	NA	NA
	C	5	1940s	853	0.853	1.6	61
WO	A	4	1940s; manual	NA	NA	NA	NA
	B	4	1983; Eng. Controls	917	0.917	1.8	57
DP	C	4	1940s	347	0.347	0.7	150

Site A,B,C,D indicate differences in site setup (e.g., eng controls)
TLV = 10 ppm (52 mg/m^3) STEL 15 ppm (79 mg/m^3)
mg/m^3 = ug/m3 / 1000
% of TLV = (mg/m^3 / 52) x 100
MOE = HEC / air conc; Where HEC = 52 mg/m^3.

Cancer Risks (Table 6): All of the cancer risks exceed the Agency's level of concern of 1×10^{-6} but only 4 of the risks exceed 1×10^{-4} (i.e., risks range from 2.5×10^{-5} to 1.6×10^{-6}).

Table 6. Creosote Dermal Cancer Risks.

Job	Site	n=	Site Description	Potential dermal dose (mg/kg/day)	Abs Dermal Dose (mg/kg/day)	Abs LADD (mg/kg/day)	Creosote Risk
TO	A	4	1940s; manual	0.414	0.0207	0.0071	4.5E-05
	B	4	1983; Eng. Controls	0.0148	0.0007	0.0003	1.6E-06
	C	5	1940s	14.8	0.7400	0.2534	1.6E-03
	D	5	1970s; Automated	0.132	0.0066	0.0023	1.4E-05
TA	B	4	1983; Eng. Controls	0.0248	0.0012	0.0004	2.7E-06
OU	A	4	1940s; manual	0.887	0.0444	0.0152	9.5E-05
	D	5	1970s; Automated	0.938	0.0469	0.0161	1.0E-04
CLO	A	4	1940s; manual	0.212	0.0106	0.0036	2.3E-05
	B	4	1983; Eng. Controls	0.0885	0.0044	0.0015	9.5E-06
	C	5	1940s	2.12	0.1060	0.0363	2.3E-04
	D	5	1970s; Automated	0.117	0.0059	0.0020	1.3E-05
LLO	B	4	1983; Eng. Controls	0.0181	0.0009	0.0003	1.9E-06
	C	5	1940s	0.203	0.0102	0.0035	2.2E-05
	D	10	1970s; Automated	0.0772	0.0039	0.0013	8.3E-06
LLO(F)	D		1970s; Automated	0.244	0.0122	0.0042	2.6E-05
LH	B	4	1983; Eng. Controls	0.0228	0.0011	0.0004	2.5E-06
	C	5	1940s	1.81	0.0905	0.0310	1.9E-04
	D	5	1970s; Automated	0.383	0.0192	0.0066	4.1E-05
CK	C	5	1940s	0.822	0.0411	0.0141	8.8E-05
TB	A	4	1940s; manual	0.112	0.0056	0.0019	1.2E-05
	C	5	1940s	1.06	0.0530	0.0182	1.1E-04
WO	A	4	1940s; manual	0.204	0.0102	0.0035	2.2E-05
	B	4	1983; Eng. Controls	0.0469	0.0023	0.0008	5.0E-06
DP	C	4	1940s	0.15	0.0075	0.0026	1.6E-05

Site A,B,C,D indicate differences in site setup (e.g., eng controls)
Dermal exposure not normalized to various amounts of wood treated per site
Arithmetic mean from Table 9 of the PMRA review.
Abs Dermal Dose (mg/kg/day) = dermal dose (mg/kg/day) x 5% dermal abs
Creosote Risk = LADD (mg/kg/day) x creosote oral CSF of 6.28E-3 $(mg/kg/day)^{-1}$

3.2 Post-application Exposures and Risks

There is the potential for post-application exposures to creosote. Potential post-application exposure may occur as a result of creosote treated wood in commercial, industrial, and residential settings. There is the potential for contact with creosote treated wood for occupational workers who install railroad ties and poles. Railroad workers may become exposed during the mechanical and manual installation of pressure treated railroad crossties as well as during inspection procedures (ATSDR, 1990). Pole installers may also contact creosote treated wood while attaching fittings on telephone poles, installing new telephone poles, conducting ground line treatment of telephone poles, and maintaining and repairing existing telephone poles (ATSDR, 1990). No dermal exposure data were available for these scenarios. Mechanical installation and/or the use of appropriate PPE are recommended to reduce exposure/contact with creosote treated wood.

There is no creosote product registered for residential uses; however, EPA recognizes that some creosote-treated wood such as railroad ties are used outdoors in home landscaping. Based on the label directions of creosote products, EPA considers such uses of creosote-treated wood to be illegal under FIFRA 12(a) (2) (G). For creosote-treated wood that is misused in residential landscaping, the potential dermal and incidental oral exposures to outdoor landscape timbers are expected to be episodic in nature. During the public comment period on this risk assessment, EPA received comments recommending wipe studies to assess dermal and incidental oral exposure to children contacting creosote treated landscape ties. EPA has considered the need for surface residue data on recycled, creosote-treated railroad ties once they are removed from service. A similar type of assessment was conducted for CCA-treated lumber using the SHEDS model. The CCA SHEDS assessment was developed for arsenic exposure to treated dimensional lumber. The CCA SHEDS model assesses children that are exposed to play sets and decks specifically built for contact by children. Compared to play sets EPA expects there would be considerably less contact and less frequent contact by children with landscape ties and on wood not used for specific children's play structures. Based on this type of comparison, the fact that creosote used in residential settings is a misuse of the product, and creosote is less potent of a carcinogen then arsenic, EPA does not believe a SHEDS-type of an assessment for creosote treated ties used as landscape timbers is warranted at this time.

4.0 Summary of Literature Exposure Studies

Additional creosote exposure studies in the literature are summarized below and presented in Table 7. Some of the air concentrations in Table 7 from these published studies exceed the ACGIH TLV and PEL of 0.2 mg/m^3 for CTPV. The results of the air concentrations reported in the literature support the results of the Creosote Council's worker exposure study indicating that exposure to creosote should be reduced.

Todd and Timbie **(NIOSH 1980)** estimated occupational exposures of workers to creosote in a railroad tie treatment plant in Somerville, Texas. Petroleum oil/creosote solutions of 70/30 and 50/50 were used respectively to treat the cross ties and bridge timbers in the plant. The concentrations of creosote (i.e., coal-tar pitch volatiles; CTPV) in personal air samples over a two-day monitoring period ranged from 0.002 to 1.211 mg/m^3. Another NIOSH study **(NIOSH 1981a)** of occupational exposure to creosote at a wood-treatment facility in Tacoma, Washington reported CTPV concentrations in personal air samples ranging from less than 0.0004 to 0.112 mg/m^3 with the highest concentration found at the end of the treatment process when the cylinder was opened. NIOSH also reported creosote exposures of dock builders ranging from zero to 0.059 mg/m^3 based on cyclohexane extractable fraction of CTPV **(NIOSH; 1981b)**.

Studies conducted by **Markel et al. (1977) and SRI (1993)** indicated that particulate polycyclic organic materials (PPOM) was within 0.1 mg/m^3, the NIOSH permissible level for CTPV, when estimating occupational exposure to creosote in wood treatment plants. The concentrations of naphthalene, methylnaphthalene, and acenaphthene (the only components in the vapor-phase fractions that could be reliably measured) ranged from 0.54 to 2.0 mg/m^3. Benzene-soluble particulates (PPOM) ranged from 0.02 to 0.10 mg/m^3.

Hiekkila et al. (1987) conducted an occupational study in Finland estimating workers' exposure to creosote in the creosote impregnation plants and when they were handling the impregnated wood. The average vapor concentrations (naphthalene being the major component) ranged from 0.5 to 71 mg/m^3 in the impregnation plants; while the vapor concentrations ranged from 0.1 to 11 mg/m^3 in the handling of impregnated wood. Most of the airborne contaminants in workers' breathing zones were in the vapor phase; the proportion of particulate polycyclic aromatic hydrocarbons (PAHs) to total concentration of vapors was less than 0.5 to 3.7 percent.

Rotard and Mailahn (1987) reported high levels of carcinogenic PAHs, such as benzo[a]pyrene, benzo[b]-fluoranthene, and benzo[j]fluoranthene, and cocarcinogenic PAHs in samples of wooden sleepers (railroad cross ties) installed in playgrounds.

Borack et. al. 2002 conducted air sampling and biological monitoring of 36 workers at a wood treatment plant where railroad ties were treated with creosote. There were 18 low exposure workers who worked as secretaries or clerical staff, 13 moderate exposure workers who transported cured ties to the shipment yard and 3 high exposure workers who worked in the retort building and handled ties immediately after creosote application. Air sampling was conducted with a filters and adsorbent tubes both of which were analyzed for benzene soluble PAHs. Six filter samples had detectable levels of particulate PAHs and the highest level was 0.33 ug/m^3 for pyrene. Thirty two of the tube samples had

detectable levels of vapor phase PAHs but levels were generally low. The highest levels were for pyrene and naphthalene measured in the high exposure group and ranged from 1.5 to 2.5 ug/m^3 for pyrene and 210 ug/m^3 to 330 ug/m^3 for naphthalene. Biomonitoring was performed using urinary 1-hydroxypropene and the results suggested that more than 90% of the measured 1-hydroxypropene could be attributed to dermal exposure.

Elovarra et.al. 1995 conducted air sampling and biological monitoring of six workers (1 impregnator, 2 assistant operators, 1 lorry driver and 2 tie platers) at an impregnation plant in Russia where railroad ties were treated with creosote. The air sampling was conducted for five consecutive days using filters and adsorbent tubes. The filters were analyzed for nine PAHS other than naphthalene and the tubes were analyzed for naphthalene. The results for the filter samples ranged from 1.23 to 13.74 ug/m^3 with a GM of 4.77 ug/m^3 and an AM of 5.7 ug/m^3. The naphthalene results of the tube samples ranged from 370 to 4200 ug/m^3 with a GM of 1536 ug/m^3 and an AM of 1254 ug/m^3. Biomonitoring was performed using urinary 1-hydroxypropene and indicated that dermal uptake was much greater than inhalation uptake.

Flickinger and Lawrence, 1982 data was cited in Wong and Harris, 2005 which is an epidemiological study of creosote workers at 11 plants in the United States. This data indicates that 95 percent of the workers at the woodtreating plants were exposed to no more than 0.14 mg/m^3 CTPV-BSF. In terms of specific jobs, the typical time-weighted average of treating operators at the participating plants ranged from 0.04 to 0.11 mg/m^3 CTPV-BSF with most measurements centered on 0.05 or 0.06 mg/m^3 CTPV. Because of the limited nature of the data, however, the epidemiology study was based on job/exposure categories rather than the data.

Heikkila et. al. 1997 conducted air sampling and biological monitoring of six workers at an impregnation plant where railroad ties were treated with creosote. The air sampling was conducted for one workweek and samples were analyzed for ten PAHs and naphthalene. The mean exposures were 1.5 mg/m^3 for naphthalene vapor, 5.9 ug/m^3 for particulate PAHs and 1.4 ug/m^3 for PAHs with 4-6 aromatic rings. Biomonitoring was conducted in conjunction with the air sampling and indicated that airborne naphthalene correlated fairly well with urinary 1-napthol ($r = 0.745$). It was also determined, however, that urinary 1-naphthol alone is not a suitable marker for inhalatory or cutaneous exposure to PAH originating from creosote.

Baker and Fannick, 1980 surveyed worker exposures to coal tar pitch volatiles during a NIOSH health hazard evaluation on October 14, 1980 at the New York Port Authority. The evaluation was requested by a union representative on behalf of six workers engaged in pile driving creosote preserved wood logs for a dock underpinning. Personal and area air samples were collected. Breathing zone CPTV concentrations ranged up to 0.06 mg/m^3 and area CPTV concentrations ranged up to 0.02 mg/m^3. These concentrations

were below the NIOSH recommended limit of 0.1 mg/m^3, however weather conditions on the sampling day probably caused substantial reductions in exposure. The authors concluded that on typical days workers may be exposed to significant amounts of CPTV.

Unwin et. al. 2006 conducted air sampling and biological monitoring of eleven workers at a timber impregnation plant as part of a larger study of PAH occupational exposure in the U.K that was funded by the British Health Executive. The workers included 2 pole fabricators, 2 pole loaders, 1 timber loader, 2 creosote plant operators, 1 unchainer, 2 labourers and 1 QC inspector. The air sampling was conducted for one day using an IOM fitted with glass fiber filters and followed by an XAD-2 adsorbent tube. The air samples were analyzed for 17 PAHs including napthalene. The results expressed as total PAH ranged from 29.9 to 1913 ug/m^3 with a mean of 835 ug/m^3. The PAH profile was dominated by naphthalene and the results for total 3-6 ring PAH and BaP were much lower with mean values of 0.05 ug/m^3 for 3-6 ring PAH and 0.01 ug/m^3 for BaP. Biomonitoring was performed using urinary 1-hydroxypropene and indicated that the creosote workers had the highest exposure among the 25 workplaces surveyed.

Table 7. Summary of Occupational Inhalation Exposure Studies of Creosote

Study	**Setting/Subjects**	**Components Reported (Analyzed)**	**Concentration** (mg/m^3)
NIOSH; 1980 (Todd & Timbie)	railroad tie treatment plant	coal-tar pitch volatiles (CTPV)	0.002-1.211
NIOSH; 1981a (Todd & Timbie)	wood treatment facility	CTPV	0.0004-0.112
NIOSH; 1981b (Baker & Fannick)	dock builder	CTPV (cyclohexane extractables)	0-0.059
Markel et al. (1977) and SRI (1993)	wood treatment facility	polycyclic organic materials (PPOM)	<0.1
Hiekkila et al. (1987)	creosote impregnation plant;	average total vapor (naphthalene being the major component)	0.5-71

Study	Setting/Subjects	Components Reported (Analyzed)	Concentration (mg/m^3)
Hiekkila et al. (1987)	handling impregnated wood	average total vapor (naphthalene being the major component)	0.1-11
Elovaara et. al., 1995	Railroad Tie impregnation plant in Russia/six workers, 5 days	10 PAHs include Naphthalene	0.40 to 4.2 Naphthalene
Borak, et.al., 2002	Railroad Tie impregnation plant /34 workers, all jobs including office/1 days	Benzene Soluble Fraction/16 PAHS	0.21 – 0.33 Naphthalene 0.015 to 0.0025 pyrene
Flicker and Lawrence, 1982 Cited in Wong and Harris, 2005	Wood Preserving Industry/Treating Operators	CTPV-BSF	0.04 – 0.11
Heikkila et. al. 1997	Railroad tie impregnation plant/six workers/one week	Naphthalene and 10 PAHs	1.5 Naphthalene (mean) 0.0059 PAH (mean)
Baker and Fannick, 1980	Pile Driving Creosote Dock/New York Port Authority/six workers/one day	CPTV	Up to 0.06 (n=6)
Unwin et. al. 2006	*Timber impregnation/UK*	*Total PAH (Primarily naphthalene)*	*0.030 – 1.9 (n=11)*

5.0 Uncertainties and Limitations

This section summarizes the uncertainties and data limitations in the creosote assessment.

- The amount of product applied and the amount of active ingredient handled by each worker was not calculated because the creosote was applied in a closed system which recovered and retained excess treatment solution from the wood and

treatment vessel while sealed. The amount of wood treated at the 4 sites is believed to be representative of the industry.

- The number of field fortification samples collected at the sites was less than the required number to satisfy Series 875 guidelines. According to the guidelines, there should be at least one fortification sample per worker per monitoring period (8 hour shift) per fortification level (three levels) for each matrix and at least one field blank per worker per monitoring period for each matrix. There were more workers monitored than there were field fortifications and field blank samples collected.

- The overall inhalation field fortification percent recoveries for the coal tar pitch volatiles (CTPVS) were poor. The overall recovery for Site B was 57%. The overall recoveries for Sites C and D were 51% and 57%, respectively. The analytical method for quantifying CTPVs in the creosote study was inadequate and only one filter had a detectable level. Therefore, inhalation exposure to CTPVs was not determined. Instead, risk concerns are indicated using the results of the naphthalene inhalation samples.

- There were some dermal fortification levels with extremely high recoveries for WBD's and some with unacceptable low recoveries for gloves. As an example, for a 60 µg/sample "total creosote" fortification for Site B, the recoveries for the WBD's were as high as 150% and recoveries for the gloves as low as 52.3%. There were measurable amounts of total creosote found in each of the control samples prepared at each facility.

- The study sponsors made no attempt to relate inhalation levels found for PNAs and CTPVs to "total creosote" -- a significant weakness with the study.

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