UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF PREVENTION, PESTICIDES,
AND TOXIC SUBSTANCES

DATE: March 31, 2008

MEMORANDUM

SUBJECT: **Creosote**- Preliminary Risk Assessment for the Reregistration Eligibility Decision Document (RED). PC Codes 022003, 025003, and 025004.

FROM: Timothy F. McMahon, Ph.D.
Senior Toxicologist, Antimicrobials Division (AD) (7510P)

Norm Cook, Chief
A. Najm Shamim, Chemist
William Erickson, Ph.D., Biologist
Jonathan Chen, Ph.D., Toxicologist
Risk Assessment and Science Support Branch, AD (7510P)

Timothy Leighton, Environmental Scientist
Regulatory Management Branch II
Antimicrobials Division (7510P)

TO: Jacqueline Campbell- McFarlane, Chemical Review Manager
Regulatory Management Branch I
Antimicrobials Division (7510P)

Attached to this cover memorandum is the Preliminary Risk Assessment for the creosote RED document in support of the reregistration of creosote. The supporting disciplinary science chapters are included as attachments and are listed on the following page.

Updated Ecological Risk Assessment for Creosote. From William Erickson, Ph.D. Biologist to Timothy F. McMahon, Ph.D. Senior Toxicologist and Risk Assessor, March 7, 2008.

Creosote: Occupational and Residential Exposure and Risk Assessment for the Reregistration Eligibility Decision (RED). From Timothy Leighton, Environmental Scientist, to Timothy F. McMahon, Ph.D., Senior Toxicologist and Risk Assessor, March 25, 2008.

Epidemiology and Incident Reports Associated with Creosote. From Jonathan Chen, Ph.D. to Timothy F. McMahon, Ph.D., Senior Toxicologist and Risk Assessor, March 25, 2008.

Creosote- Toxicology Disciplinary Chapter for the Reregistration Eligibility Decision Document (RED). From Timothy F. McMahon, Ph.D., Senior Toxicologist and Risk Assessor, to Jacquie McFarlane, Chemical Review Manager, Marwch 25, 2008

Product Chemistry Science Chapter for Creosote (P1/P13 and P2 Fractions) Reregistration Eligibility Decision (RED) Process. From A. Najm Shamim, Ph.D., Chemist, to Tim McMahon, PhD, Senior Toxicologist, and Risk Assessor, February 14, 2008.

Residue/ Dietary Risk Assessment Science Chapter for Creosote (P1/P13 and P2 Fractions) Reregistration Eligibility Decision (RED) Process. From A. Najm Shamim, Ph.D., Chemist, to Tim McMahon, PhD, Senior Toxicologist, and Risk Assessor, February 14, 2008.

Environmental Fate and Transport Assessment of Creosote for the Reregistration Eligibility Decision (RED) Process. From A. Najm Shamim, Ph.D., Chemist, to Tim McMahon, PhD, Senior Toxicologist, and Risk Assessor, February 14, 2008.

TABLE OF CONTENTS

1.0 EXECUTIVE SUMMARY.....4

2.0 PHYSICAL AND CHEMICALPROPERTIES.....15

3.0 HAZARD PROFILE…….....17

3.1 ACUTE TOXICITY..... 17

3.2 DOSE_RESPONSE ASSESSMENT.....18

3.3 DERMAL ABSORPTION.....21

3.4 CARCINOGENICITY CLASSIFICATION.....22

3.5 ENDOCRINE DISRUPTION……...22

4.0 EXPOSURE ASSESSMENT AND CHARACTERIZATION.....23

4.1 SUMMARY OF REGISTERED USES.....23

4.2 OCCUPATIONAL EXPOSURE AND RISK.....23

.

5.0 AGGREGATE RISK ASSESSMENT AND CHARACTERIZATION.....28

6.0 CUMULATIVE EXPOSURE AND RISK.....29

.

7.0 ENVIRONMENTAL RISKS.....30

7.1 ENVIRONMENTAL FATE ASSESSMENT.....30

7.2 ENVIRONMENTAL EXPOSURE AND ECOLOGICAL RISK ASSESSMENT…...35

. 7.3 ENDANGERED SPECIES……..……49

8.0 INCIDENT REPORTS.....50

9.0 REFERENCES.....66

1.0 Executive Summary

Creosote is a fungicide, insecticide, and sporicide used as a wood preservative for above and below ground wood protection treatments as well as for treating wood in marine environments. All 16 Creosote products currently registered are Restricted Use Pesticides; 15 are End-Use Products and 1 is a Manufacturing-Use Product for formulating industrial end-use wood preservative products. Creosote wood preservatives are used primarily to pressure treat railroad ties/crossties (represents close to 70% of all Creosote use) and utility poles/crossarms (represents 15 - 20% of all Creosote use). Assorted Creosote-treated lumber products (e.g., timbers, poles, posts and groundline-support structures) represent the remaining uses for this wood preservative. The industry refers to different blends of creosote, based on the wood treatment standards set by the American Wood-Preservers' Association (AWPA), as P1/P13 and P2. Typically, railroad ties/crossties are treated with a P2 blend, which is more viscous than the P1/P13 blend used for treating utility poles.

A recent voluntary cancellation of all non pressure treatment uses resricts use of creosote to commercial and industrial settings. Creosote is applied 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.

The acute toxicity of both the P1/P13 and P2 blends of creosote is moderate by the oral, dermal, and inhalation routes of exposure in experimental animals (Toxicity Categories III and IV). Median lethal doses by the oral and dermal routes are above 2000 mg/kg, and median lethal doses by the inhalation route are above 4mg/L, which are considered limit doses in acute toxicity tests as set by Agency guidelines. The P1/P13 blend displays eye and skin irritation potential in experimental animals (eye irritation clearing in 8-21 days, skin irritation up to 14 days post-dosing). The P2 blend appears to show somewhat less potential for skin and eye irritation (eye irritation clearing within 7 days, skin irritation clearing after 72 hours) but data are incomplete. It is assumed that both blends are dermal sensitizers as there are no currently acceptable dermal sensitization studies for either blend.

Subchronic dermal testing with both the P1/P13 and P2 blends of creosote show a minimum of toxic effects in experimental animals (rats). Using the P2 blend, one mortality of questionable significance was observed at a dose of 400 mg/kg/day, while testing of the P1/P13 blend produced decreases in body weight gain. Effects on the skin in both studies were minimal to moderate.

Subchronic inhalation testing with creosote produced a wider spectrum of effects. At a dose of 0.049 mg/L, exposure to the P1/P13 blend produced myocardial pathology (degeneration, hemorrhage, cardiomyopathy) in males and females. Altered hematological parameters (decreased hemoglobin, hematocrit, erythrocytes; increased reticulocytes, polychromasia, poikilocytosis, anisocytosis) were also observed in males and females. Testing of the P2 blend

by the inhalation route also produced altered hematological parameters, and resulted in increased absolute and relative liver and thyroid weights. Follicular cell hypertrophy was observed. Lesions of the nasal cavity were also observed with the P2 blend.

Developmental and reproductive testing of creosote showed potential sensitivity of offspring to the P1/P13 blend. Decreased body weight gain and decreased food consumption were observed in maternal animals at a dose of 175 mg/kg/day, and at this same dose, increased post-implantation loss, increased mean resorptions, and decreased live fetuses per litter were also observed. An overall significant increase in incidence of developmental malformations was also observed at the 175 mg/kg/day dose. Testing of the P2 blend did not show any apparent susceptibility of developing offspring to creosote, and reproductive testing of P1/P13 creosote did not show any apparent susceptibility, but the reproductive toxicity study contained several deficiencies that compromised interpretation of the data, such as a low fertility and pregnancy index for F1 female parental rats. Thus, there is some uncertainty associated with concluding that creosote is devoid of any reproductive effects. In this light, the additional 10-fold safety factor mandated by FQPA was employed to account for this uncertainty as well as for the effects observed from developmental toxicity testing of P1/P13 creosote.

In consideration of the available evidence that creosote is a positive mutagen, the Agency waived the requirement for the standard mutagenicity battery, and instead required dominant lethal testing of both the P1/P13 and P2 blends. The results of testing of both the P1/P13 blend and P2 blend of creosote showed that, at doses toxic to the dosed animals (330.5 mg/kg for the P1/P13 blend, and 194 mg/kg for the P2 blend), there was no evidence of a dominant lethal effect of either creosote blend.

There are no reliable metabolism data on creosote, as the chemical is a complex mixture of several classes of polycyclic aromatic chemicals. Assays are in development to identify marker compounds to determine exposure to creosote.

A large body of experimental evidence exists which shows a positive relationship between exposure to creosote and development of tumors in experimental animals. In addition to its tumor-promoting potential, the ability of creosote to induce lung tumors after dermal application was examined. Dermally applied creosote (0.25ml undiluted, twice weekly for 8 months) induced 5.8 lung adenomas per mouse in mice housed in stainless steel cages, while untreated controls showed 0.5 lung adenomas/mouse (Roe et al, Cancer Res. 18: 1176-1178, 1958). Carcinogenicity of two high-temperature derived creosote oils was studied by Poel and Kammer (JNCI 18: 41-55, 1957). The light creosote fraction is composed mainly of benzene, toluene, xylene, and solvent naphtha, while the blended oil is composed of creosote oil, anthracene oil, and oil drained from recovery of naphthalene. Oils were applied by drops to the skin of mice at concentrations of 20%, 50%, or 80% three times a week for life. By weeks 21-26, both oils had induced skin tumors. Several mice exhibited metastases to the lungs or regional lymph nodes. In an oral carcinogenicity study conducted by Culp et al. (1996), the carcinogenicity of two coal

tar mixtures and pure benzo[a]pyrene (BaP) was studied. Coal tar mixture 1 (CTM-1) was a composite of coal tar samples from seven coal gasification plant waste sites and was dosed at 0.0, 0.01, 0.03, 0.1, 0.3, 0.6, and 1.0% of diet. Coal tar mixture 2 (CTM-2) was a composite of coal tars from three waste sites with high BaP content and was dosed at 0.0, 0.01, 0.03, 0.1, 0.3% of diet. An additional group was dosed with pure BaP at 0, 0.0005, 0.0025, and 0.01 % in diet. A control group received solvent control in diet. Each group consisted of 48 mice.
In mice exposed to coal tar, coal tar acted as a systemic carcinogen and induced a dose-related increase in hepatocellular adenomas and carcinomas, alveolar/bronchiolar adenomas and carcinomas, forestomach squamous epithelial papillomas and carcinomas, small intestine adenocarcinomas, histiocytic sarcomas, hemangiosarcomas in multiple organs, and sarcomas in several tissues.

The incidence of forestomach tumors increased sharply between the 0.1 and 0.3% doses. However, there was not a proportional increase in forestomach tumors above 0.3% because these mice died from adenocarcinomas of the small intestine. Tumors of the esophagus, observed in BaP treated mice, were not observed in the CTM1 or CTM-2 treated groups. Lung and hepatocellular tumors observed in the CTM1 and CTM2 groups were not observed in BaP treated animals.

Multiple neoplasms were noted in liver, lung, forestomach, and the small intestines of exposed mice. Liver neoplasms occurred in 1, 0, and 4 mice fed 0.03, 0.1 and 0.3% CTM-1, respectively. Lung neoplasms occurred in 21, 11 and 13 mice fed 0.3% , 0.6% , 1.0% CTM-1, respectively. Eight mice in the 0.3% CTM-2 group had multiple lung neoplasms. Forestomach carcinomas were found in 2 and 1 mice fed 0.6% or 1.0% CTM-1, respectively. Adenocarcinomas were found in the small intestines (jejunum) of 2 and 12 mice fed 0.6% and 1.0% CTM-1, respectively. Groups dosed with ≥0.3% CTM-1 or CTM-2 had significantly reduced survival.

In those mice exposed to benzo(a)pyrene, BaP appeared to act as a point-of-contact carcinogen, causing an increased incidence of papillomas and/or carcinomas of the forestomach, esophagus and tongue. The incidence of forestomach tumors increased steeply between 5 and 25 ppm, equivalent to 20.5 and 104 μg BaP/day. Tumors of the esophagus were not observed in the CTM1 or CTM-2 treated groups. Lung and hepatocellular tumors, observed in CTM treated mice, were not observed in mice fed BaP.

Multiple neoplasms of the forestomach occurred in 8 and 21 mice fed 25 and 100 ppm BaP, respectively. Multiple neoplasms of the esophagus and tongue were noted in 6 and 3 mice, respectively, fed 100 ppm BaP. It should be noted that all animals in 100 ppm group died before termination of the study.

In humans, evidence for carcinogenicity of creosote varies. Several studies have associated occupational exposure to creosote with development of skin cancer, with a latency period of 20-25 years. These studies are very old (1920's to 1940's), when occupational safety practices were much more lax than today. More recent reports (1980) show no increase in risk of skin, bladder, or lung cancer in wood treatment plant workers, or after treatment for 4 years with coal-tar medicinal therapy for treatment of dermatitis. These reports, however, were limited in scope. Those reports associated with therapeutic use of coal tar did not mention the fact that the composition of the coal tar used therapeutically is different than that used for wood treatment. In the report on wood treatment workers, the population studied was small, and the follow-up period was too short to allow a long enough latency for tumor development.

The Agency in 1988 acknowledged limitations on conducting a quantitative risk assessment from use of a single component of creosote (Guidance for the Reregistration of Pesticide Products Containing Coal Tar/Creosote As the Active Ingredient, USEPA, 1988), but it was also observed that creosote mixtures are "complex mixtures with known synergistic effects" on carcinogenicity.

In conjunction with the Pest Management Regulatory Agency, Health Canada, 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} $(\mu g/kg/day)^{-1}$ for the coal tar mixture 1 tested in this study was selected, on the basis of forestomach tumors observed.

Dose-Response Assessment

On April 1, 1999, the Health Effects Division's Hazard Identification Assessment Review Committee (HIARC) evaluated the toxicological endpoints selected for occupational and residential (dermal and inhalation) exposure risk assessments for Creosote. 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 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.

For creosote, there is no anticipated dietary exposure as there are no registered food uses for this substance and there is little concern for potential residues of creosote from trophic transfer (A. Najm Shamim, Ph.D., Residue/Dietary Risk Assessment.Chapter for Creosote). However, non-dietary exposures to creosote are expected via the dermal and inhalation routes of exposure. For these risk assessments, the following toxicity endpoints were selected: (1) for *short-term dermal risk assessment,* the NOAEL of 50 mg/kg/day was selected, based on the observation of decreased body weight gain at 175 mg/kg/day in a developmental toxicity study in rats (MRID # 43584201). (2) for *intermediate-term dermal risk assessment,* the NOAEL of 40 mg/kg/day was selected, based on the observation of decreased body weight gain at 400 mg/kg/day in a 90-day dermal toxicity study in rats (MRID # 43616201). (3) for *long-term dermal risk assessment,* the LOAEL of 25 mg/kg/day was selected based on the observation of decreased pre-mating body weight in parental animals from a 2-generation reproduction toxicity study (California EPA review, no MRID). (4) for *inhalation risk [short- and intermediate-term],* the NOAEL of 0.0047 mg/L was selected , based on decreased body weight gain, altered hematology and clinical chemistry, and increased absolute and relative weight of the liver and thyroid observed at 0.048 mg/L in a 90-day inhalation toxicity study (MRID # 43600901). 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. 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.

A Margin of Exposure of 100 is acceptable for short-term and intermediate-term dermal and inhalation occupational risk assessments. A Margin of Exposure of 300 is acceptable for long-term occupational dermal risk assessments. For long-term occupational inhalation risk assessment, a Margin of Exposure of 100 is acceptable. Separate Margins of Exposure should be calculated for dermal and inhalation routes since oral and dermal NOAELs were selected for dermal risk assessment and an inhalation NOAEL was selected for this route of exposure.

Occupational/ Residential Exposure and Risk

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.

Occupational Handler dermal risk estimates: 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).

Occupational Handler Inhalation risk estimates: 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 TLV of 52 mg/m^3.

Cancer risks from dermal and inhalation exposure : Based on the selected slope factor of 6.28×10^{-6} $(\mu g/kg/day)^{-1}$, 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}).

Occupational post-application exposure and risk: 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.

Although there are no creosote label registered uses of creosote for residential uses, EPA acknowledges that some creosote treated wood such as railroad ties are used outdoors in home landscaping. The potential dermal and incidental oral exposures to outdoor landscape timbers are expected to be episodic in nature.

Aggregate Exposure and Risk

In order for a pesticide registration to continue, it must be shown "that there is reasonable certainty that no harm will result from aggregate exposure to pesticide chemical residue, including all anticipated dietary exposures and other exposures for which there are reliable information." Aggregate exposure is the total exposure to a single chemical (or its residues) that may occur from dietary (i.e., food and drinking water), residential, and other non-occupational sources, and from all known or plausible exposure routes (oral, dermal, and inhalation).

Acute and Chronic Dietary Aggregate Risk

Under the current policy of the Office of Pesticide Programs, acute aggregate risk assessment determines the acute risk from combined dietary consumption of pesticide residues, separate from residential exposures (Health Effects Division, Standard Operating Procedure 97.2, April 1998). In the case of creosote, an acute aggregate (food + water) risk estimate was not performed for Creosote. Creosote is not registered for any food use, and it has also been determined that creosote is not likely to impact the diet or drinking water.

Short- and Intermediate-Term Aggregate Risks

Aggregate short and intermediate term risk assessments are designed to provide estimates of risk likely to result from exposures to the pesticide or pesticide residues in food, water, and from residential (or other non-occupational) pesticide uses. Due to the lack of exposure through food or water, short and intermediate term aggregate risks were not performed. Residential exposures to Creosote residues may occur, but data are not available to assess these risks.

Chronic (Non-Cancer) Aggregate Risk

Based on the lack of potential for chronic exposure to Creosote through food and water, a chronic (non-cancer) aggregate risk assessment was not performed.

Environmental Hazard and Risk

Process wastewater, dumpsite leachate, storage tank leaks, and spills are the major creosote sources to the environment (Merril and Wade, 1985). In addition, leachates from pressure treated wood can migrate out into soils, and water. The environmental fate and transport of creosote focuses primarily on the likely exposure of PAHs into three environmental compartments: 1) leaching of creosote mixture into surface and ground waters from the railroad ties and utility poles; 2) migration into soil/sediments from the railroad ties and utility poles, and 3) bioaccumulation into the aqueous and benthic organisms.

Recent studies conducted by Ken Brooke (Sooke Basin, 2001 and Railroad ties, 2004) illustrate some important trends: 1) Creosote treated wood does leach out its constituents; 2) leaching process is acute at the start of the and tapers off with time; 3) leachate in water soil consists of a mixture the PAHs ranging from low molecular weight to high molecular weight; 4) migration of PAHs from the pole or railroad ties is limited to a short distance: maximum up to 10 meters away from the line of origin and not more than 60 cm vertically down; 5) initial mixture ratio for low to high molecular weight PAHs is 1:1 while with the passage of time (385 days),in favor of high molecular weight PAHs.; 6) there is possibly an acute impact at short distances away from utility poles or railroad ties. 7) some high molecular weight PAH s like benzo (e)pyrene are persistent in soils.

Data on the migration into soils and water are contradictory, but the majority of data indicate that the migration process into these environmental compartments is not remarkable. Biodegradation processes of PAHs under aqueous aerobic conditions is rapid, and except for a few large sized PAHs like benz[a]pyrene, fluoranthene, benzo[k]fluoranthene, most PAHs show a tendency to biodegrade. Under anaerobic conditions, biodegradation is not commonly observed and the PAHs may become persistent, stable in soils/sediments and lose the migration capability.

Ecological Hazard and Risk

Because guideline toxicity studies are not available for the major component PAHs of creosote expected in the aquatic environment, the best available information from the open literature and from existing evaluations was used in this assessment. However, data gaps are extensive, especially for the complexes of PAHs likely to occur in the water column and in aquatic sediments. Therefore, much of the risk presumptions are based on a qualitative assessment of the available information.

Based on the existing laboratory and field data and modeling of PAH aquatic concentrations from use of creosote-treated railroad ties and aquatic structures, the following conclusions for risks to freshwater and saltwater fish and invertebrates exposed in the water column and/or in aquatic

sediment are made:

Aquatic structures:

· the level of concern (LOC) is exceeded for acute risk to listed (i.e., endangered and threatened) freshwater and saltwater (estuarine/marine) fish and aquatic invertebrates exposed to PAHs in the water column

· the LOC is exceeded for acute risk to non-listed saltwater invertebrates exposed to PAHs in the water column

· chronic RQs can not be calculated due to lack of chronic toxicity data, but available evidence indicates that chronic risk (survival, growth, reproduction, immunotoxicity) is possible to aquatic organisms inhabiting the water column

· based on findings from the Sooke Basin field study, PAH accumulation in sediments is sufficiently high to pose a risk to benthic organisms in the immediate vicinity of creosote-treated pilings for at least a year

· although survival and spawning were not affected, short-term adverse affects on growth of mussels was reported immediately around pilings at Sooke Basin

· one year after pilings were installed in Sooke Basin, an assemblage of invertebrate organisms inhabited the creosote-treated pilings, suggesting minimal long-term impacts to some non-listed species

· laboratory and field investigation found a major detrimental impact on hatching and development of fish (herring) eggs attached to aquatic pilings, even pilings that were 40 years old, suggesting that some sensitive species may be adversely affected by creosote-treated pilings

· creosote had a significant and rapid adverse impact on total abundance, number of taxa, and population dynamics of zooplankton in microcosms

· impacts of creosote-treated aquatic pilings are likely to vary locally, depending on abiotic and biotic factors such as current speed, amount of structure per unit area, air and water temperature, salinity, and the aquatic species occurring in the immediate area of the structures; thus, a site evaluation is essential prior to installation of new structures

· potential impacts to aquatic organisms, especially less mobile species, may be exacerbated in the vicinity of treatment plants and loading docks in aquatic areas

Railroad structures:

· the level of concern (LOC) is exceeded for acute risk to listed freshwater and saltwater fish and aquatic invertebrates exposed to PAHs expected to reaching the water column; in wetter areas, PAHs may possibly remain at levels of concern to listed species for many weeks

· in wetter areas with more potential for movement of PAHs into the aquatic environment, the LOC also is exceeded for acute risk to non-listed fish (freshwater) and aquatic invertebrates exposed to PAHs in the water column

· chronic RQs can not be calculated due to lack of chronic toxicity data, but available evidence indicates that chronic risk is possible to aquatic organisms inhabiting the water column

· based on a mesocosm study, sediment PAH levels around railroad ties appear to be lower than those around aquatic pilings

Endangered Species

The Endangered Species Act (ESA) requires that federal agencies consult with the National Marine Fisheries Service and/or the United States Fish and Wildlife Services (FWS) for listed organisms if a proposed "action" may affect listed species or their designated habitat. Because the ecological risk assessment for creosote indicates a potential for exposure of listed fish and aquatic invertebrate species, a refined assessment is needed that includes delineation of the action area and species at risk and evaluation of direct, indirect, and habitat effects. A refined listed-species assessment has not been conducted for the current risk assessment but is deferred to Registration Review.

Incident Reports

Creosote and creosote-containing substances are widely used in industry and by certain subgroups of individuals, resulting in a large population of persons with potential exposure. According to California data, the majority of poisoning incident cases occurred as a result of handling creosote and applying it to wood without proper protection for the skin and eyes. The number of these cases has dropped quite markedly in the 1990s. Substantial contact with treated wood appears to be a risk factor for skin and eye burns, even years after the wood was treated. Symptoms experienced were burns and rashes on the exposed body areas, chemical conjunctivitis, headaches, nausea, and eye irritation.

While a number of human health studies are available that include creosote as a possible, or even likely, target exposure, few studies are available with enough information for a rigorous assessment of chronic health effects attributable to creosote specifically. By far, the most common limitation of studies aimed at evaluating effects of creosote exposure is the almost total absence of objective exposure measurements for the study participants. For most of the studies, assessment of exposure is based on information about past occupational activities provided by the participants or assigned by health studies professionals such as industrial hygienists with general knowledge of occupations and materials. In almost all cases, possible exposure to other materials, either separately or concomitantly, cannot be excluded. A second important limitation often seen in studies on effects of creosote is the lack of statistical significance calculated for many of the apparent associations between assigned creosote exposure and development of disease.

These limitations notwithstanding, among the epidemiological studies on effects of creosote exposure, increased risks for development of a number of diseases have been observed. Diseases typically found to be in excess include skin cancer and nonmalignant skin disorders, bladder cancer, lung cancer and nonmalignant respiratory diseases. Considering the information presently available, conclusions regarding chronic health effects from exposure to creosote alone should be considered tentative.

2.0 PHYSICAL/CHEMICAL PROPERTIES

Creosote ,as defined by the American Wood Preservers Association, is a distillate derived from coal tar, derived by the high temperature carbonization of bituminous coal. Creosote consists primarily of liquid and solid polyaromatic hydrocarbons (PAH's) and contains some tar acids and tar bases. The two major types of creosote in use are P1/P13 creosote (a straight creosote distillate used for ground contact, land, and fresh and marine water applications) and P2 creosote, used in treatment of railroad crossties. A detailed assessment of the physical/chemical properties of creosote can be found in the product chemistry chapter for the creosote RED (A. Najm Shamim, Ph.D.). A summary is shown here.

P1/P13 Fraction

Chemical Name:	Coal Tar Creosote
Molecular Wt.:	No Applicable
Color:	2.5Y2/2 to 2.5Y4/2 (Based on Munsell color scheme)
Odor:	Sharp, aromatic, wood-like
Solubility:	313 μg/ml
Vapor Pressure:	11.1 mm Hg at 24.4 ^{o}C
Log P:	3.247
Stability:	Short term(accelerated)stability was performed on four constituents of the mixture: naphthalene, phenanthrene pyrene and chrysene for a period of 30 days at 60 ^{o} C. At the end of thirty day period, naphthalene remaining was : 96.5%, phenanthrene: 87.2%, pyrene: 86.9% and chrysene: 92.4%
Viscosity;	14.60 mm/s
Storage Stability:	Not determined.

Notes:

1.The P1/P13 samples, provided by the Industry to Research Triangle Institute, were distilled, within 95% confidence limit, residues remaining were less than 1.1% as required by the AWPA Standard A1-91 (This test is similar to the EPA's Certified Limit Test required for other pesticides).

2.Insoluble mass in Xylenes: Duplicate determinations showed that this fraction contained between 0.21 to 0.23% insoluble materials.

3. Specific gravity of the fraction, for the industry sample (single determination) is 1.0934 (corrected to 38^{o}C)

4. Moisture (water) content for the industry sample (single determination) is 0.4%.

All these results were obtained by using the AWPA Method A1-91 Series.

P2 Fraction

Chemical Name:	Coal Tar Creosote
Color:	10YR2/1 to 2.5Y5/5 (Munsell color scheme)
Odor:	Strong aromatic, Petroleum-like
Solubility:	306 µg/ml
Vapor Pressure:	8.6 mm Hg at 24.4 to 24.5 ^{o}C
Log P	3.311
Stability:	
Viscosity:	15.5 mm/s at 25 ^{o}C

3.0 Hazard Characterization

3.1 Acute Toxicity

The acute toxicity profile for the P1/P13 blend is shown below.

Study Type	Animal	Results	Tox Cat	MRID No
81-1: Acute Oral	Rat	LD_{50} Male 2451 mg/kg Female 1893 mg/kg	III	43032101
81-2: Acute Dermal	Rabbit	LD_{50} Male > 2000 mg/kg Female > 2000 mg/kg	III	43032102
81-3: Acute Inhalation	Rat	LC_{50} > 5 mg/L	IV	43032103
81-4: Primary Eye Irritation	Rabbit	Irritation clearing in 8-21 days	II	43032104
81-5: Primary Dermal Irritation	Rabbit	erythema to day 14	III	43032105
81-6: Dermal Sensitization	Guinea Pig	study unacceptable	N/A	43032106

The acute toxicity profile for the P2 blend is shown below.

Study Type	Animal	Results	Tox Cat	MRID No
81-1: Acute Oral	Rat	LD_{50} Male 2524 mg/kg Female 1993 mg/kg	III	43032301
81-2: Acute Dermal	Rabbit	LD_{50} Male > 2000 mg/kg Female > 2000 mg/kg	III	43032302
81-3: Acute Inhalation	Rat	LC_{50} > 5.3 mg/L	IV	43032303
81-4: Primary Eye Irritation	Rabbit	Irritation clearing within 7 days	III	43032304
81-5: Primary Dermal Irritation	Rabbit	no irritation after 7 days	III	43032305
81-6: Dermal Sensitization	Guinea Pig	study unacceptable	N/A	43032306

3.2 Dose-Response Assessment

On April 1, 1999, the Health Effects Division's Hazard Identification Assessment Review Committee (HIARC) evaluated the toxicological endpoints selected for occupational and residential (dermal and inhalation) exposure risk assessments for Creosote. On September 3, 2003, the Antimicrobials Division Toxicity Endpoint Selection Committee (ADTC) met to verify the selected endpoints for long-term dermal risk assessments for creosote and inhalation risk assessment, and also discussed whether dermal and inhalation Margins of Exposure should be combined for creosote risk assessment. The toxicological endpoints selected for various exposure scenarios are summarized in the table below.

Toxicology Endpoints for Creosote

EXPOSURE SCENARIO	DOSE (mg/kg/day)	ENDPOINT	STUDY
Acute and Chronic Dietary	**These risk assessments not required.**		
Carcinogenicity (dermal)	Creosote has been shown to exert positive miutagenic 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 x 10^{-6} (µg 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 (1-30 days)	Oral NOAEL=50	decreased body weight gain at 175 mg/kg/day	Developmental Toxicity - Rat
	MOE = 100 (5% dermal absorption)		
Intermediate-term Dermal (1-6 months)	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[a] (>6 months)	Oral LOAEL = 25 mg/kg/day	decreased pre-mating body weight	2-generation reproduction study - Rat
	MOE = 300 (10x, 10x, 3x for use of a LOAEL)		
Inhalation-creosote[b] (any time period)	NOAEL = 0.0047mg/L MOE = 100	decreased body weight gain, altered hematology	90-day Inhalation Study in the Rat with P2 creosote (MRID **43600901**)
Inhalation –naphthalene (any time period)	LOAEL = 52 mg/m^3 MOE = 300	nasal effects: hyperplasia and metaplasia in respiratory and olfactory epithelium respectively	Two year inhalation toxicity study - mouse (USEPA, IRIS)
Dermal absorption[c]	5%, determined from the results of in vivo / in vitro testing in rats and in vitro testing using human skin.		

[a]after re-examination of the toxicology data, the ADTC concluded that the 2-generation reproduction toxicity study was appropriate for long-term dermal risk assessment for the following reasons**:** the duration of the 2-generation reproduction study is more representative of the time frame (i.e. long-term) than the 90-day dermal study, and is consistent with OPP policy regarding duration of the study vs. route of exposure; body weight gain decreases in the 2-generation reproduction toxicity study were observed in the F2 generation, supporting the time frame for the long-term endpoint (i.e. > 6 months). The 90-day dermal study effects are not as representative of the time frame for the long-term dermal risk assessment**.** However, the two studies can be considered co-critical studies for this endpoint. Correction of the LOAEL from the 2-generation reproduction toxicity study for dermal absorption (50%) and use of a LOAEL (3x extra UF) yields a MOE and endpoint (300 and 50 mg/kg/day) similar to the 90-day dermal toxicity study (40 mg/kg/day and MOE of 300 [extra 3x to extrapolate to long-term endpoint]).

[b]the ADTC re-examined the use of the inhalation toxicity study selected for inhalation risk assessment for creosote and concluded that a developmental toxicity study, as used for the oral and dermal risk assessments of creosote, is not appropriate for inhalation risk assessment because: (1) the inhalation toxicity study showed significant effects on body weight gain early in the study (one week) and is therefore relevant for short-term assessment (2) it is also a route-specific study; and (3) the inhalation NOAEL is more sensitive than the developmental NOAEL. Therefore, the inhalation study will remain as the study for the short-term inhalation endpoint.

[c]dermal absorption of creosote was determined from submitted in vivo and in vitro studies on creosote (MRIDs 47179501 and 47179502).

FQPA Considerations

As there are no existing food uses for creosote, an FQPA assessment is not necessary. Potential post-application exposures to residents, including children (e.g., from use of railroad ties by homeowners), could not be assessed due to lack of exposure data. 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.

3.3 Dermal Absorption

In 2003, the Toxicology disciplinary chapter for the reregistration eligibility decision (RED) document for creosote included toxicity endpoints of concern, among them an estimation of the magnitude of dermal absorption for creosote. As there were no data at that time specifically examining dermal absorption of creosote, a factor of 50% was estimated, based on comparison of the oral and dermal LOAELs from the developmental toxicity study in rats (MRID # 43584201) and the 90-day dermal toxicity study in rats (MRID # 43616101) using the P1/P13 blend. The oral LOAEL of 175 mg/kg/day observed in the developmental toxicity study, when compared to the dermal LOAEL of 400 mg/kg/day observed in the dermal toxicity study, yields an absorption factor of 44%, which was rounded up to 50% by the Committee. The rounding to 50% took into account the significant dermal irritation which occurs from dermal exposure to creosote.

Beginning in 2005, a series of meetings were held involving scientific and regulatory staff of the Antimicrobials Division and the Health Effects Division, Office of Pesticide Programs, U.S. Environmental Protection Agency, the Pest Management Regulatory Agency, Health Canada, and the Creosote Council III as the Creosote Couincil did not agree with the 50% dermal absorption factor determination. Over the next two years, a study protocol was discussed to examine dermal absorption of the creosote mixture. The study protocol was approved in 2007 and the dermal absorption study was submitted for review by the Office of Pesticide Programs in 2007. The studies submitted consisted 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 in vitro study was determined to be unacceptable for regulatory purposes from initial review, based on the lack of data demonstrating that the concentration of the creosote solution did not exceed solubility limits and therefore did not have an artefactual influence on the magnitude of absorption in the in vitro test system.

The Office of Pesticide Programs further determined that from the in vivo data, a value of 33.9 (34%) was appropriate for the magnitude of dermal absorption in the in vivo test system. This was concluded on the basis of data demonstrating continued absorption of the creosote mixture after 8 hours as evidenced by urinary and fecal excretion of radiolabel. The registrant presented an argument that the O-ring used in the initial study may have acted as a depot for creosote-derived readioactivity and should therefore be considered artefactual, based on a supplemental study conducted where the O-ring was not glued to the skin of the animal. However, significant radioactivity was recovered from the body wrap used in this supplemental study, compromising interpretation of the supplemental study results.

In January of 2008, the Creosote Council met again with scientific and regulatory staff of the Office of Pesticide Programs to discuss their interpretation of the dermal absorption data. The main point of their presentation was that the Office of Pesticide Programs should consider the ratio of absorption of creosote in rat skin vs. human skin based on the submitted data. These data show an approximate

8-fold difference in absorption through rat skin vs. human skin (34% in rat skin in vivo and in vitro vs. 4.2% in human skin in vitro).

Based on the available data, the 8-fold factor for rat skin vs. human skin with respect to absorption of creosote is supported by the submitted data. However, uncertainties still exist with these data:

1) The registrant submitted data on solubility limits of the 8 marker components used in the in vivo dermal absorption protocol as representative of creosote. The registrant did not submit solubility limits for the actual creosote mixture itself. While the solubility data submitted by the registrant indicated that the solubility limits were not exceeded for any of the 8 marker compounds used as radiolabelled tracers, there is uncertainty as to how the mixture behaves.

2) There is uncertainty regarding absorption after 8 hours exposure. The O-ring data are not conclusive of an effect of the O-ring acting as a depot, as absorption is still seen after 8 hours of exposure and washing of the skin surface contained by the O-ring.

Based on the uncertainties, 8-fold factor describing the difference in dermal absorption between rat and human skin is acceptable, but the dermal absorption value for creosote is concluded to be 5% (34% value from the rat study divided by 8 and rounded upward).

3.4 Classification of Carcinogenic Potential

In conjunction with the Pest Management Regulatory Agency, Health Canada, 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} $(\mu g/kg/day)^{-1}$ for the coal tar mixture 1 tested in this study was selected, on the basis of forestomach tumors observed

3.5 Endocrine Disruption

EPA is required under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act (FQPA), to develop a screening program to determine whether certain substances (including all pesticide active and other ingredients) "may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or other endocrine effects as the Administrator may designate." Following recommendations of its Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC), EPA determined that there was a scientific basis for including, as part of the program, the androgen and thyroid hormone systems, in addition to

the estrogen hormone system. EPA also adopted EDSTAC's recommendation that EPA include evaluations of potential effects in wildlife. For pesticides, EPA will use FIFRA and, to the extent that effects in wildlife may help determine whether a substance may have an effect in humans, FFDCA authority to require the wildlife evaluations. As the science develops and resources allow, screening of additional hormone systems may be added to the Endocrine Disruptor Screening Program (EDSP).

4.0 EXPOSURE ASSESSMENT AND CHARACTERIZATION

4.1 Summary of Registered Uses

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.

Dietary Exposure

Based upon its classification as a restricted use pesticide and restrictions on use sites since 1984, dietary exposure to creosote is not expected through food. In drinking water, the Agency has determined that the use pattern of Creosote is not expected to impact water resources through labeled uses. In light of this finding, EPA believes that Creosote's use will not impact ground or surface water and therefore is not expected to lead to exposure to humans through drinking water.

4.2 Occupational Exposure and Risk

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. The specific job functions and their descriptions for creosote workers are detailed in the occupational and residential chapter for the creosote RED.

The worker exposure study on pressure treatment applications submitted by the Creosote Council II provided chemical-specific handler dermal and inhalation exposure data in support of the re-registration of pressure treatments of creosote (Creosote Council II, 2001).
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.

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:

1MOE $_{[dermal]}$ = NOAEL or LOAEL / Potential and/or Absorbed Dermal Dose

2MOE $_{[inhalation]}$ = 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:

1Cancer Risk = absorbed LADD (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 : 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	Absorbed Dermal Dose	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	6756	270	3378
	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	4032	161	2016
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	1129	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	5524	221	2762
	C	5	1940s	0.203	0.010	4926	197	2463
	D	1	1970s: Automated	0.077	0.004	1295	518	6477
LLO(	D		1970s: Automated	0.244	0.012	4098	164	2049
LH	B	4	1983: Eng. Controls	0.023	0.001	4386	175	2193
	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	2132	853	1066
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)

Inhalation MOEs: 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 TLV 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

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: All of the cancer risks exceed the Agency's level of concern of 1 x 10^{-6} but only 4 of the risks exceed 1 x 10^{-4} (i.e., risks range from 2.5 x 10^{-5} to 1.6 x 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 $(mg/kg/day)^{-1}$

Occupational 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.

5.0 Aggregate risk assessment

Under the current policy of the Office of Pesticide Programs, acute aggregate risk assessment determines the acute risk from combined dietary consumption of pesticide residues, separate from residential exposures (Health Effects Division, Standard Operating Procedure 97.2, April 1998). In the case of creosote, an acute aggregate (food + water) risk estimate was not performed for Creosote. Creosote is not registered for any food use, and it has also been determined that creosote is not likely to impact the diet or drinking water.

Short- and Intermediate-Term Aggregate Risks

Aggregate short and intermediate term risk assessments are designed to provide estimates of risk likely to result from exposures to the pesticide or pesticide residues in food, water, and from residential (or other non-occupational) pesticide uses. Due to the lack of exposure through food or water, short and intermediate term aggregate risks were not performed. Residential exposures to Creosote residues may occur, but data are not available to assess these risks.

Chronic (Non-Cancer) Aggregate Risk

Based on the lack of potential for chronic exposure to Creosote through food and water, a chronic (non-cancer) aggregate risk assessment was not performed.

6.0 CUMULATIVE RISK

FQPA (1996) stipulates that when determining the safety of a pesticide chemical, EPA shall base its assessment of the risk posed by the chemical on, among other things, available information concerning the cumulative effects to human health that may result from dietary, residential, or other non-occupational exposure to other substances that have a common mechanism of toxicity. The reason for consideration of other substances is due to the possibility that low-level exposures to multiple chemical substances that cause a common toxic effect by a common mechanism could lead to the same adverse health effect as would a higher level of exposure to any of the other substances individually. A person exposed to a pesticide at a level that is considered safe may in fact experience harm if that person is also exposed to other substances that cause a common toxic effect by a mechanism common with that of the subject pesticide, even if the individual exposure levels to the other substances are also considered safe.

AD did not perform a cumulative risk assessment as part of this RED for creosote. Creosote is a complex mixture of chemical substances that may act by various modes to produce toxicity. A review to determine if there are any other chemical substances that have a mechanism of toxicity common with that of creosote has not been initiated, and it is unlikely that there are other such mixtures that approach that of creosote in terms of complexity.

Notwithstanding, the registrant must submit, upon EPA's request and according to a schedule determined by the Agency, such information as the Agency directs to be submitted in order to evaluate issues related to whether creosote shares a common mechanism of toxicity with any other substance. If the Agency identifies other substances that share a common mechanism of toxicity with creosote, the Agency will perform aggregate exposure assessments on each chemical, and will begin to conduct a cumulative risk assessment.

The Health Effects Division, Office of Pesticide Programs, has recently developed a framework proposed for conducting cumulative risk assessments on substances that have a common mechanism of toxicity. This guidance was issued for public comment on January 16, 2002 (67 FR 2210-2214) and is available from the OPP Website at:
http://www.epa.gov/pesticides/trac/science/cumulative_guidance.pdf. In the guidance, it is

stated that a cumulative risk assessment of substances that cause a common toxic effect by a common mechanism will not be conducted until an aggregate exposure assessment of each substance has been completed.

Before undertaking a cumulative risk assessment, AD will follow procedures for identifying chemicals that have a common mechanism of toxicity as set forth in the *YMBOL 65 \f "WP TypographicSymbols" \s 12 Guidance for Identifying Pesticide Chemicals and Other Substances that Have a Common Mechanism of Toxicity* (64 FR 5795-5796, February 5, 1999).

7.0 ENVIRONMENTAL RISKS

7.1 ENVIRONMENTAL FATE ASSESSMENT

The major uses of creosote since 1988 have been railroad ties, crossbars, decks on marinas and utility poles. Polyaromatic hydrocarbons (PAHs) constitute the highest percent (85%) of coal tar creosote while the phenolic substances are about 10 percent, and N– and S-containing substances represent the remainder of the mixture. Most of the PAHs are non-volatile, therefore; creosote normally does not contaminate the air. The major route of exposure from creosote is through water and soil, and from these environmental compartments into the aquatic and benthic organisms (bioaccumulation).

Estimated half-lives of PAHs in water, soil and sediments are presented in Table 1. The half-lives of the PAHs in these environmental compartments tends to increase with increasing complexity of the molecules (Howard et al. 1991). K_{ow} values also increase with the complexity of the PAH. Half-lives in water (and air) are much lower than in soils and sediments.

Table 1. Estimated Half-lives of PAHs in Various Environmental Media

PAH	No. aromatic rings	Molecular wt	Est. half-life (weeks)			K_{ow}
			water	soil	sediment	
Naphthalene	2	128	1	8	32	3.37
Acenapthene	3	152	3	32	104	4.03
Fluorene	3	166	3	32	104	4.12
Phenanthrene	3	178	3	32	104	4.57
Anthracene	3	178	3	32	104	4.54
Fluoranthene	4	202	8	104	312	5.22
Pyrene	4	202	8	104	312	5.18
Chrysene	4	228	8	104	312	5.91

Half-lives in tissues of aquatic organisms are discussed in the Updated Creosote Environmental Fate Chapter. Aquatic organisms remove residues with half-lives of only a few days for the PAHs addressed in this assessment (Table 2). Biomagnification apparently is not a concern, because vertebrates metabolically transform PAHs and excrete them before they accumulate to higher levels (Brummelen et al. 1998 in Poston 2001).

Table 2. Half-lives of PAHs in Aquatic Organisms

Species	PAH	Half-life (days)
Vertebrates:		
Rainbow trout	fluoranthene	6
	fluorene	7
	anthracene	7
	phenanthrene	9
Invertebrates:		
Mussel	anthracene	1.9
	naphthalene	2
	phenanthrene	1.9-2.2
	fluoranthene	2.0-11.1
	pyrene	4.1-5.5
	chrysene	5.0-14.2
	fluoranthene	29.8

Species	PAH	Half-life (days)
Clam	napthalene	2.0
	chrysene	3.3
	fluoranthene	3.3
	pyrene	3.6-10.3
	phenanthrene	4.5-6.1
	fluoranthene	8.4
Oyster	phenanthrene	3.4
	pyrene	6.7
	fluoranthene	5.9
	chrysene	15.1
Shrimp	fluoranthene	0.8
	pyrene	0.8
	phenanthrene	0.9
Polychaete	chrysene	4.3
	phenanthrene	4.8
	pyrene	4.8
	fluoranthene	5.8

Abiotic Degradation

The PAHs are fused aromatic polycyclic rings which have no hydrolyzable hydrogens and the solubility of these compounds are very low in water. Environmentally, hydrolysis does not appear to be an important pathway for dissipation of the composite mixture of PAHs in water; however, some molecules like benzo[k]fluoranthene and benz[a]pyrene could persist in water.

Very few studies are reported in the open literature on field volatilities for PAHs present in creosote. Gevao et al. (1998) showed that acenaphthene, fluorene, phenanthrene, anthracene, and fluoranthene volatilized at a faster rate at 30°C than at 4°C. The study also showed that 85 percent of these components remained in the wood after seven weeks. The rate of volatilization was slow for acenaphthene (half-life, one year) and fluoranthene (half-life, one year). In most cases, the initial rates of dissipation are caused by partitioning between the wood and air and biodegradation in the presence of microbial populations. Therefore, exposure to air does not appear to be an important factor in fate assessment for most PAHs.

Photooxidation is a common phenomenon, and therefore an important degradation pathway for the creosote PAHs. The photolytic half-lives of the PAHs in aqueous medium are

dependent on the season, geographical location, surface water measurements, and complexities of the parent molecules (two fused rings vs. five fused rings). In most cases, half-lives under the conditions mentioned do not appear very long. Because of this, and the fact that most of the PAHs are not readily soluble (except for a few low molecular weight ones), the PAHs may not be a problem in surface and groundwater runoffs. However, it should be noted that the photooxidized products of PAHs are stable; therefore, may persist in air/water and soils and become an environmental concern as these photooxidized products are also bioaccumulative.

Mobility

Once introduced to an aquatic environment, creosote components are subjected to several fractionation processes. Many PAHs adsorb to sediments and may persist for long periods of time. Creosote contaminated sediments usually contain relatively higher levels of hydrophobic PAHs than whole creosote (Bieri et al., 1986). Eventually, sediment adsorbed PAHs may dissolve or become re-suspended in the water by tides, storms, bioturbation, shipping, or dredging. As a result, local biota may be exposed to low level PAHs over the long term (Fowler et al., 1993). Therefore, the adsorption/desorption processes in water involved with creosote-derived PAHs are a significant consideration in fate assessments of creosote contamination.

Additionally, colloidal matter present in a cresote-contaminated environment has been found to affect the desorption rates of specific PAHs. One study found that PAHs partitioned to course (of sizes >100 nm) colloid fractions and were linearly correlated with the PAH octanol-water partition coefficient, indicating the partitioning was hydrophobic (Villholth, 1999).

The PAHs from creosote-treated utility poles and/or railway ties tend to leach out initially in the first seven days and remain in the sediment surrounding the poles or railroad ties not migrating far from the wood. Most of the PAHs, however, tend to stay inside the wood (~85%). One study showed that background levels for PAHs leached from wood were attained within three months and may have been due to photolysis or biodegradation of the PAHs. A detailed study of 200 U.S. estuaries showed that PAHs that leached from the treated wood of decks and bulkheads, 175 had muddy sediments. Higher amounts of PAHs leached into such soil types. No systematic work has been carried out on all the PAHs and the representative soil types to show which one would have a higher tendency of retention for the PAHs.

Migration studies of PAHs into groundwater have shown that migration of some of the PAHs have been observed. Vertical or lateral migration of the PAHs from the utility poles indicated that at ground level the migration was not significant beyond 150 meters from the site of contamination (base of utility poles). The vertical or downward migration of the PAHs was even more limited and the existence of the PAHs were not found below a 12 meter depth.

Biodegradation

Most of the PAHs have a tendency to biodegrade under aerobic conditions. It has been reported that over eighty percent of biodegradation occurs in the first month after the wood

preservative application, with the exception of benz[a]pyrene and benzo[k]fluoranthene, which have shown resistance to biodegradation. A number of aerobic soil metabolism studies on PAHs conducted at various contaminated sites as well as in simulated microcosms reported that low molecular weight PAHs generally metabolized in aerobic conditions and the greater the oxic environment, the higher the biodegradation level.

Aerobic degradation of PAHs associated with soil and groundwater often leads to a rapid depletion of dissolved oxygen which eventually decreases the redox potential. This decrease in the redox potential can result in favorable environments for denitrifying, sulfate-reducing, or methanogenic microbes. Therefore, anaerobic transformations may be a significant factor in oxygen-depleted habitats (Karthikeyan and Bhandari, 2001). Under these conditions, anoxic or anaerobic degradation stimulated by denitrifying or sulfate-reducing bacteria can become an important pathway for the cleanup of contaminated sites.

Bioaccumulation in Fish

The major components of the PAHs in creosote have shown the ability to form neutral to oxidized transformation products under aerobic soil/aquatic conditions. For example, fluorene forms hydroxy fluorene and acenaphthene converts into diacetic acid acenaphthene. These oxidized species are stable and bioaccumulative. Numerous studies have shown that photooxidized transformation products of these PAHs are bioaccumulative and result in adverse effects on the aqueous biota as well as on the organisms in the soils and benthic sediments.

In aquatic habitats, fish, shellfish, and crustaceans readily bioaccumulate PAHs from the environment and store these at high levels in the tissues. Seven PAHs: naphthalene, anthracene, phenanthrene, pyrene, 9-methyl anthracene, benz[a]anthracene and perylene were shown to bioaccumulate in *Daphnia pulex*. PAHs like naphthalene, biphenyl/acenaphthylene, fluorene, phenanthrene/anthracene/chrysene, and benzopyrene were found to bioaccumulate in clams (*Rangia cuneate)*. The most dramatic increases were in cases such as benz[a]anthracene/chrysene which reported a bioaccumulation of 41 ppb (week zero) and increased to 190 ppb (week 4). Similarly, benzopyrene bioaccumulated from 8 ppb (week zero) to 600 ppb (week 4). Depuration was within two weeks. This study was conducted after the creosote spill into the Bayou Bonfuca at the American Creosote Works Plant Site at Slidell, Louisiana.

A study on benthic invertebrates showed a bioaccumulation concentration ranging from 0.10 to 11.00 ppm. A bioconcentration study on zebra mussels in the Great Lakes found that pre-spawning species (high lipid) bioaccumulated benzo[a] pyrene at a faster rate than the post-spawning (low lipid) species.

Bioaccumulation data on marine mammals are not readily available, and only one study on whales and seals has been reported. That study indicated a bioaccumulation of 0.10 and 1.21 ppm in the muscles of these mammals, respectively.

Some of the PAHs, particularly those that have a high molecular mass (higher number of the fused aromatic rings) have a higher tendency to adsorb to soil organic carbon. Such adsorption coefficients (K_{OC}) have been reported in literature. Some PAHs with a high K_{oc} can bind strongly with the organic carbon of the soils/sediments and may not be bioavailable to the aquatic organisms. However, if the octanol/ water coefficients (K_{ow}) is high, and if the PAHs are desorbed from the soils/sediments to which they are bound, some of these PAHs can become bioaccumulative to the benthic organisms.

It has been suggested that based on theoretical calculations and modeling, the half-lives of the PAHs obtained from coal tar creosote can be estimated in air, water, soils and sediments. From these calculations and modeling, one can arbitrarily divide the PAHs into 3 distinct groups: PAHs with two fused rings, PAHs with three fused rings and PAHs that have 4 to 5 fused rings. The half-lives in the environmental compartments (air, water, and soils) for PAHs are as follows: the half-lives of two fused rings PAHs < three fused rings < 4/5 fused rings. The K_{ow} values lie between 3 and 4 for PAHs with two fused rings, between 4 and 5 for PAHs with 3 fused rings, and at 6 or above for the third group of the PAHs. In general, the half-lives in air and water environmental compartments are much lower than in soils/sediments because the soil adsorption coefficients are higher. The longer the half-life, the greater the persistence of the PAHs in soils. Some of the 4/5 fused ring PAHs are more persistent in soils and sediments and since their K_{ow} values are higher, they can bioaccumulate because some of them adsorb onto soils and they may not be bioavailable for benthic organisms.

The third group of PAHs show a higher degree of bioaccumulation, persistence in soils and water, resistance to biodegradation and photooxidation. Additionally the components of these group have less of a tendency to leach from the wood structure and have high sorption constants to soils. On the other hand, these higher members of the PAHs (4/5 fused ring compounds) are not readily soluble in water and their percent on a mass basis in the creosote mixture is very low compared to the first group (2 fused rings) of the PAHs, and may not be available for biomagnification and migration into surface and ground waters.

7.2 ENVIRONMENTAL EXPOSURE AND ECOLOGICAL RISK ASSESSMENT

The toxicity endpoints typically used in OPP's assessments are obtained from guideline toxicity studies conducted for wildlife, aquatic organisms, and plants (40 CFR §158.2060). Guideline studies are required for all pesticides to provide acute and chronic measures of effect for one or more test species in several taxonomic groups. As noted in the 2003 preliminary ecological risk assessment, guideline toxicity studies are not available for creosote. The preliminary assessment relied on the whole creosote data available in the open literature, but insufficient data were obtained to assess chronic effects to freshwater invertebrates or to marine/estuarine aquatic organisms. For the updated assessment, available acute and chronic toxicity information for the

PAHs has been obtained from the open literature, including relevant laboratory, microcosm, and field studies obtained through ECOTOX searches and other sources, including EPA Sediment Quality Criteria documents for fluoranthene (EPA 1993a), phenanthrene (EPA 1993b), and acenaphthene (EPA 1993c).

Acute toxicity

Most PAHs for which data are available are highly to very highly toxic to freshwater and saltwater fish and invertebrates (Table 8), with anthracene and fluoranthene being the most toxic PAHs in the water column.

Table 8. Acute Toxicity of Creosote PAHs to Fish and Invertebrates Exposed in the Water Column

PAH/ media[a]	Species	Exposure duration (h)	LC50/EC50 (µg/L)	Source
Anthracene				
SW	Fish - no data			
	Mysid shrimp	48	3.6	Pelletier 1997
FW	Bluegill (*Lepomis macrochirus*)	96	1.27	McCloskey 1991
	Scud (*Hyalella azteca*)	240	5.6	Hatch 1999
Fluoranthene				
SW	Sheepshead minnow	96	0.8	EPA 1993b
	Mysid shrimp	96	0.58	Spehar 1999
FW	Fathead minnow	96	6.8	Diamond 1995
	Water flea	48	0.97	Spehar 1999
Acenaphthene				
SW	Sheepshead minnow	96	2200	Heitmuller 1981
	Mysid shrimp	96	160	EPA 1993c
FW	Brown trout (*Salmo trutta*)	96	580	Holcombe 1983
	Stone fly (*Tallaperla maria*)	96	240	Horn 1983
Fluorene				
SW	Fish - no data			
	Polychaete worm	96	1000	Rossi 1978
FW	Bluegill	96	760	Mayer 1986
	Water flea	48	420	
Naphthelene				
SW	Sheepshead minnow	24	2400	Anderson 1974
	Humpy shrimp (*Pandalus goniurus*)	96	971	Korn 1979
FW	Pink salmon (*Oncorhyncus gorbuscha*)	96	890	Rice 1989

PAH/ media[a]	Species	Exposure duration (h)	LC50/EC50 (µg/L)	Source
	Water flea (*Daphnia pulex*)	96	1000	Trucco 1983

Chrysene				
SW	Fish - no data			
	Polychaete worm (*Neanthes arenaceodentata*)	96	<1000	Rossi 1978
FW	Fish - no data			
	Water flea	20	1900	Kagan et al.1987
Pyrene				
SW	Fish - no data			
	Opossum shrimp (*Americamysis bahia*)	48	0.89	Pelletier et al. 1997
FW	Fathead minnow	3.2	25.6	Oris 1987
	Water flea	2	4	Kagan et al. 1987
Phenanthrene				
SW	Atlantic silverside (*Menidia menidia*)	96	108	EPA 1993a
	Mysid shrimp	96	17.7	
FW	Bluegill	96	234	EPA 1993a
	Hydra (*Hydra* sp.)	96	96	

[a] SW = saltwater; FW = freshwater

Chronic toxicity

No guideline chronic-toxicity studies are available to assess chronic risks of the PAHs addressed in this assessment. Some data are available in the EPA Sediment Quality Criteria documents for fluoranthene, acenaphthene, and phenanthrene (Table 9). Adverse affects on survival, growth, and reproduction have been reported at concentrations as low as 8 µg/L for phenanthrene and 18.8 µg/L for fluoranthene.

Table 9. Chronic Toxicity of Creosote PAHs to Fish and Invertebrates Exposed in the Water Column

PAH/ media[a]	Species	NOEC/LOEC (µg/L)	Effect	Source
Fluoranthene				
SW	Fish - no data			
	Mysid	11.1 / 18.8	survival, reproduction	Champlin and Poucher 1991
FW	Fathead minnow	10.4 / 21.7	survival, growth	Brooke 1991
	Daphnia magna	10.6 / 21.2	growth	Brooke 1992
Acenaphthene				
SW	Fathead minnow	332 / 495	growth	Cairns and Nebeker 1982

PAH/ media[a]	Species	NOEC/LOEC (µg/L)	Effect	Source
	Mysid (*M. bahia*)	44.6 / 91.8	reproduction	Thursby et al. 1989
FW	Sheepshead minnow	520 / 970	survival	Ward et al. 1981
	Midge (*Paratanytarsus* sp.)	295 / 575	egg hatching	NAS 1982
Phenanthrene				
SW	Fish - no data			
	Mysid	5.5 / 11.9	survival	Kuhn and Lussier 1987
FW	Rainbow trout	5 / 8	survival	Call et al. 1986
	Daphnia magna	57 / 163	reproduction, survival	

[a] SW = saltwater; FW = freshwater

Some additional but scanty information is available for some PAHs. Brooks (2000) reviewed the available data and concluded that "it appears that sustained water column concentrations of 30 to 50 ppb PAH can have subtle, but important, chronic impacts on populations of marine organisms." He cites Neff (1979) in reporting that copepod (*Eurytemora affinis*) exposed to 10 ppb of naphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, or 2,3,5-trimethylnaphthalene displayed significantly reduced lifespan and brood size. Moore et al. (1989 in Brooks 2000) reported reduced feeding rates of mollusks at PAH levels of 30 to 40 ppb in seawater.

Poston (2001) cites two developmental studies with salmon and herring exposed to PAHs from weathered crude oil (Heintz et al. 1999, Carls et al. 1999). He concluded that developmental affects might occur at about 0.4 to 1.0 µg/L total dissolved PAH in the water column, and that similar concentrations of PAHs leached from creosote-treated wood could be expected to exert the same impact on aquatic organisms.

Karrow et al. (1999) evaluated the effects of creosote on immunotoxicity to female rainbow trout in 12,000-L outdoor microcosms dosed with liquid creosote. The fish were exposed for 28 days to creosote concentrations ranging from 0 to 100 µl/L. Stimulatory and suppressive effects were observed; a concentration-response relationship was evident. The LOEC was 17 µl/L nominal creosote concentration, which represents 0.61 µg/L total PAHs in the water. The researchers concluded ". . . that environmental concentrations of PAHs can impair fish immune parameters, possibly to a degree where resistance to disease is compromised."

Sediments

Sediment toxicity studies are not available for most of the PAHs. However, the EPA Sediment Quality Criteria documents provide some sediment and pore water LC50s for several species of saltwater and freshwater organisms, mostly invertebrates, exposed to sediments spiked with fluoranthene, acenaphthene, or phenanthrene (Table 10).

Table 10. Toxicity of PAHs to Aquatic Organisms Exposed to Spiked Sediments (from EPA 1993a,b,c)

PAH/ media[a]	Species	Exposure duration	TOC[b] (%)	Sediment LC50 (µg/g dry wt)	Pore-water LC50 (µg/L)	Source
Fluoranthene						
SW	Amphipod (*Rhepoxynius abronius*)	10 d	0.18	3.4	22.7	Swartz et al. 1990
			0.31	6.5	29.4	
			0.48	10.7	24.2	
	Amphipod (*R. abronius*)	10 d	0.34	15.0	14.1	De Witt et al. 1992
			0.40	12.6	19.2	
			0.31	8.65	9.38	
	Amphipod (*Eohaustorius estuaries*)	10 d	nm	5.1	nd	De Witt et al. 1989
FW	Fathead minnow	30 d	nm	0.43	nd	Gendusa 1990
		96 h		0.97		
	Channel catfish (*Ictalurus punctatus*)	96 h	0.70	3.68	nd	
	Amphipod (*Hyallela azteca*)	10 d	0.46	2.3	45.9	Suedel et al. 1993
			0.50	7.4	236.5	
			0.44	5.5	97.6	
	Midge (Chir*onomus tentans*)	10 d	0.46	7.3	91.2	
			0.50	8.7	251.0	
			0.44	3.0	75.7	
	Daphnia magna	nr	nm	4.2	nd	
Acenaphthene						
SW	Amphipod (*E. estuaries*)	10 d	1.23	44.4	800	Swartrz 1991
			2.49	47.8	609	
			4.21	68.4	542	
	Amphipod (*Leptocheirus lumulosus*)		1.62	>193	>1720	
			2.52	193	1410	
			3.66	382	1490	
Phenanthrene						
SW	Amphipod (*E. estuaries*)	10 d	1.02	39.2	138	Swartrz 1991
			2.47	97.2	139	
			3.33	122	146	
	Amphipod (*L. plumulosus*)		1.96	92.4	387	
			2.50	162	306	
			3.60	255	360	

[a] SW = saltwater; FW = freshwater

[b] OC = organic carbon; nm = not measured; nd = not determined

Some additional information is available for creosote or creosote components. Tagatz et al. (1983) evaluated the toxicity of creosote-contaminated sediment (sand) to field- and laboratory-colonized estuarine benthic communities exposed for 8 weeks to concentrations of 177, 844, or 4420 µg/g. At the two highest concentrations, the number of individuals and species were significantly reduced from the control numbers. Animal abundance also was reduced at 177 µg/g in the field. Echinoderm, annelid, and arthropod numbers were adversely affected at the lowest concentration. Mollusks numbers were significantly reduced at 844 µg/g. Changes in indices of species diversity and dominance occurred at the middle and high concentrations. Creosote sediment concentrations decreased by 30% in the laboratory and 42% in the field during the 8 weeks of exposure.

Padma et al. (1998) exposed mysid shrimp to creosote-contaminated sediment and to the water-soluble portion of the sediment. The 24-h LC50 was 700 µg/L for mysid exposed to the whole sediment. The 24-h LC50 was 180 µg/L for mysid exposed to the water-soluble fraction of the sediment, which contained higher concentrations of the low-molecular PAHs than did the whole sediment.

Other Effects Information

Zooplankton and phytoplankton communities

Sibley et al. (2001a,b) examined the structure and dynamics of zooplankton communities (86 spp.) and phytoplankton (200 spp.) for up to 83 days following single applications of liquid creosote directly to water in 12,000-L outdoor microcosms. Concentrations ranged from 0.06 to 109 mg/L. Results indicate that zooplankton communities may be highly sensitive to creosote exposure. Creosote had a significant and rapid adverse impact on total abundance, number of taxa, and population dynamics of individual taxa at all concentrations. Community-level EC50s were 44.6 µg/L (NOEC = 13.9 µg/L) at 5 days and 46.6 µg/L (NOEC = 5.6 µg/L) at days. Creosote had no direct adverse affect on the phytoplankton community based on total abundance and number of taxa.

Herring

Vines et al. (2000) investigated the effects of creosote-treated wood on embryonic development in Pacific herring (*Clupea pallasi*) in the laboratory and in the field. Herring spawn their eggs onto various substrates, including aquatic pilings, and creosote diffused from pilings had a detrimental affects on hatching rate and survival of embryos. In the laboratory, embryos were incubated in glass bowls of seawater containing either creosote-treated wood (from pilings at least 40 years old), untreated wood (Douglas fir), or only seawater. The LC50 for hatching success was 50 µg/L, with statistically significant reductions in hatching success at ~9 µg/L. None of the eggs adhering to treated wood hatched, and only 9% of free-floating eggs hatched in bowls containing treated wood. Hatching success was 89% for the control and 73% for untreated wood. The effects observed in the laboratory also were observed in the field, where herring eggs deposited onto aged creosote-treated pilings (Fort Baker, CA) did not hatch.

Other effects were observed in the laboratory study. For embryos not attached to wood but exposed to creosote in the water, 40-50 % exhibited delayed development. All creosote-exposed embryos that survived to hatch were abnormal as compared to 20% in the wood-only treatment and 8% in the control. Abnormalities included fluid retention in the visceral cavity (ascites), fluid accumulation around the heart (pericardial edema), and spinal curvature (scoliosis). Embryonic heart rates measured post-fertilization increased from 92 and 97 beats/min on Day 5 to 127 and 130 beats/min at Day 9 in the control and wood-only treatments, respectively, but decreased from about 50 beats/min to <10 beats/min in the creosote-exposed treatment. Other effects recorded only in the exposed treatment included severe arrhythmic heart contraction patterns, erratic and increased body movements within the chorion (egg wall), and embryo tremors and twitches.

Mussels

No adverse effects on the survival of mussels (*Mytilus edulis edulis*) suspended in cages from the Sooke Basin pilings were observed in a two year *in-situ* bioassay. However, mussels grown in close proximity (15 to 30 cm) to either the WP piling or BMP piling grew more slowly than did mussels grown farther away (Table 11).

Table 11. Survival and Growth of Mussels after Dolphin Installation in the Sooke Basin Field Study

Station (distance from pilings)	**% survival over time**			
	0 d	**14 d**	**185 d**	**384 d**
BMP treated dolphin (0.5 m)	100	100	97	79
BMP treated dolphin (2.0 m)	100	100	100	88
BMP treated dolphin (10.0 m)	100	99	98	81
WP piling (0.5 m)	100	100	99	88
Open control	100	100	99	80
Station and distance from pilings	**Growth (valve length, mm) over time**			
	0	**14**	**185**	**384**
BMP treated dolphin (0.5 m)	29	32.5	46.7	59.3
BMP treated dolphin (2.0 m)	29.5	32.7	51.6	67.2
BMP treated dolphin (10.0 m)	31.2	33.3	56.6	68.7
WP Piling (0.5 m)	29.1	31.4	49.1	64.2
Open Control	30.2	32.4	55.6	69.5

Prior to being placed in the water at Sooke Basin, mussel-tissue PAH concentrations were

relatively low (16.15± 2.19 ng/g). These levels increased an average of 328% on Day 14 of the study. The most significant increases were observed in close proximity (within 0.5 m) of either the BMP or WP dolphins. Following that initial increase, levels returned to normal by Day 185 and were slightly lower than the baseline levels by Day 384. Higher levels of PAHs were observed in gonadal tissue than were observed in whole body tissues as shown below (Table 12). Overall, tissue concentrations were low.

Table 12. Low and High Molecular Weight PAHs in Gonad and Whole-body Tissue of Mussels (*Mytilus edulis edulis*) Grown at Varying Distances from Creosote-treated Dolphins

PAH component	concentration (ng/g wet tissue) at each site[1]					
	OC[2]	BMP 0.5[3]	BMP 2.0[3]	BMP 10[3]	WP 0.5[4]	Avg.
LPAH in Gonad	14.8	15.1	18.2	10.0	16.4	15.0
HPAH in Gonad	22.0	29.2	32.4	15.6	32.4	26.3
LPAH in Whole Tissue	6.7	6.4	9.8	5.6	6.1	6.9
HPAH in Whole Tissue	15.0	15.4	26.0	11.5	16.7	16.9
Total PAH in Gonad	36.7	44.3	50.6	25.5	48.8	41.2
Total PAH in Whole Tissue	21.7	21.9	35.8	17.0	25.5	24.4

[1] tissues were analyzed at Day 185 when the mussels were ripe and ready to spawn
[2] OC = open control site located upstream of all structures
[3] BMP = creosote-treated piling produced using Best Management Practices at specifed distance (m)
[4] WP = 8-year-old weathered creosote treated piling

Aquatic Risk Assessment

Water column

Acute risks

When a new creosote-treated wood structure is installed in an aquatic environment, there is an immediate release of creosote components into the water column. During their study in the Sooke Basin, Goyette and Brooks (1998) report that creosote leaching from the portions of aquatic pilings above the water line initially forms a sheen on the water surface. They speculated that microdoplets from the surface sheen subsequently move down through the water column and into the sediment, with little of that creosote dissolving in the water column. However, they did not measure water-column concentrations until 6 months after pilings (dolphins) were installed in Sooke Basin. Ingram et al. (1982) and Bestari et al. (1998) measured PAH concentrations in the water column in the initial days and weeks and found levels that might be of concern for exposure of aquatic organisms.

Acute RQs for aquatic organisms exposed to the PAH component expected in the water column are presented in Table 13. The weighted acute toxicity values used to calculate RQs for the total PAH component are as follows:

Freshwater fish weighted LC50 = 405 µg/L
Freshwater invertebrate weighted EC50 = 267 µg/L
Saltwater fish weighted LC50 = 1150 µg/L
Saltwater invertebrate weighted EC50 or LC50 = 399 µg/L

Table 13. Acute RQs for Exposure of Aquatic Organisms to PAHs in the Water Column

Site	Time after initial exposure	Freshwater[a]		Estuarine/Marine[a]	
		Fish	Invert.	Fish	Invert.
300-gal tanks; seawater (Ingram et al.1982)	72 hr	n/a	n/a	**0.37***	**1.08****
	12 day	n/a	n/a	**0.13***	**0.39***
1200-L outdoor microcosms; freshwater (Bestari et al.1998)	7 day	**0.24***	**0.36***	n/a	n/a
	3 mo.	0.02	0.02	<0.01	0.02
Sooke Basin; marine (Goyette and Brooks 1998)	6 mo.	n/a	n/a	<0.01	<0.01
Railroad (wet scenario)	0 hr	**0.77****	**1.16****	**0.27***	**0.78****
	96 hr	**0.45***	**0.69****	**0.16***	**0.46***
	21-day avg.	**0.23***	**0.35***	**0.08***	**0.24***
	60-day avg.	**0.15***	**0.22***	**0.05***	**0.15***
	90-day avg.	**0.13***	**0.20***	0.04	**0.13***
Railroad (dry scenario)	0 hr	**0.12***	**0.18***	0.04	**0.12***
	96 hr	**0.07***	**0.11***	0.02	**0.07***
	21-day avg.	0.03	0.04	<0.01	0.03
	60-day avg.	0.01	0.02	<0.01	0.01
	90-day avg.	<0.01	0.01	<0.01	<0.01

[a] based on weighted toxicity values: FW fish = 405 ppb; FW invertebrate = 267 ppb; SW fish = 1150 ppb; SW invertebrate = 399 ppb

** exceeds the acute LOC for non-listed spp. (RQ ≥0.5) and listed spp. (RQ ≥0.05)

* exceeds the acute LOC for listed spp.

The RQs determined for Sooke Basin do not exceed the Agency's acute LOC; however, those concentrations were measured 6 months after pilings were installed and may simply represent background PAH concentrations. Based on the total PAH concentrations reported in seawater by Ingram et al. (1982) and in freshwater by Bestari et al. (1998) and the weighted toxicity values,

the acute LOC is exceeded for listed fish and aquatic invertebrates. The acute LOC also is exceeded for non-listed estuarine/marine invertebrates. Exceedance of an LOC indicates a potential for adverse effects on nontarget organisms and identifies a need for regulatory action to mitigate risk (Appendix B).

Based on the EECs modeled for railroad structures, the acute LOC for listed freshwater and saltwater species is exceeded in wet areas (MS scenario). Exposure levels of concern potentially exist for several months. Non-listed species also are at acute, short-term risk. In drier areas (CA scenario), the acute LOC is only exceeded for listed species and only in the short-term.

Zooplankton communities also may be at acute risk. Comparing the 5-day EC50 of 44.6 µg/L for community-level effects to the aquatic EECs (Tables 3 and 5) indicates that the LOC would be exceeded 1- to 10-fold for acute risks due to creosote leaching from aquatic structures and railroad structures.

Chronic risks

Insufficient data exist to calculate weighted toxicity values for the PAH component; therefore, chronic RQs are not calculated. However, comparing EECs to the available data (previously presented in the Toxicity Data section) indicate that adverse affects on survival, growth, and/or reproduction could be expected in some situations (Table 14). Chronic risk is presumed in OPP risk assessments when the chronic EEC (21-day-avg. for invertebrates and 60-day-avg. for fish) exceeds the NOEC. Chronic exposure in the water column potentially poses risks to fish and/or aquatic invertebrates around aquatic structures and, especially in wetter areas, where leachate from railroad structures may move into the aquatic environment.

Table 14. EECs and Adverse Effects from Chronic Exposure in the Water Column

Site	EEC (µg/L)	Reported effect concentrations (µg/L)
300-g SW tanks	156 (12 d)	0.4-1.0 (salmon, herring; development) 0.61 (rainbow trout; immunotoxicty) 9 (herring; hatching sig. reduced) 44.6 (zooplankton; community EC50) 30-50 (invertebrates; survival, brood size) 5-57 (phenanthrene NOECs[a]) 10-11 (fluoranthene NOECs[a]) 44-520 (acenapthene NOECs[a])
12,000-L FW microcosms	0.8-6.7 (84 d)	
Railroad (wet scenario)	94.4 (21-d-avg.) 59.3 (60-d-avg.)	
Railroad (dry scenario)	11.4 (21-d-avg.) 5.0 (60-d-avg.)	

[a] NOECs based on survival, growth, and reproduction

Sediments

Due to lack of sediment-toxicity data for most PAHs and the total PAH component, RQs cannot be calculated for acute and chronic risks to benthic organisms potentially exposed to the PAH matrix in sediments. However, the Sooke Basin study provides information on the potential

adverse affects of creosote-contaminated sediments. Goyette and Brooks (1998) compared sediment loads (see Table 4) to proposed or interim sediment quality criteria for Canada and Washington State. Based on those criteria, several PAHs pose potential risks to benthic organisms in sediments immediately around aquatic pilings (Table 15).

Table 15. PAHs Exceeding Canadian and/or Washington State Sediment Quality Criteria at Various Distanced from the BMP Dolphin in Sooke Basin After 384 Days (from Goyette and Brooks 1998)

PAH	Distance (m) from pilings				
	0.5 m	1 m	1.5 m	7.5 M	10 m
Fluoranthene	●	●			
Acenaphthene	●	●			
Phenanthrene	●	*	*	*	
Chrysene	●	*			
Fluorene	●				
Pyrene					
Anthracene					
Naphthalene					

● exceeds Canadian and WA State Sediment Quality Criteria

* exceeds Canadian Sediment Quality Criteria

Goyette and Brooks (2001**)** also conducted various tests to evaluate sediment toxicity in proximity to the creosote-treated pilings in Sooke Basin. Marine amphipods (*Rhepoxynius abronius* and *Eohaustorius washingtonianus*) were exposed to sediments from the BMP and control sites. Because these amphipods are not supported in the Sooke Basin, control reference sediments also were collected from elsewhere for comparison to the toxicity from exposure to BMP sediments. Sediments collected within 0.5 m of the BMP dolphin at Days 14 and 185 were toxic in comparison to the reference sediments. Fluoranthene and phenanthrene appeared to be the major contributors to this toxicity. At Days 1360 and 1540, toxicity was found in some, but not all, samples within 2 m of the BMP dolphin. However, by this time elevated hydrogen sulfide levels were elevated in the sediments (from buildup and decomposition of fouling organisms and their waste falling from the pilings) and were thought to have contributed to the observed toxicity. Pore-water toxicity testing with Microtox™ assays and a marine bioluminescent bacterium demonstrated toxic conditions in sediments within 2 m of the BMP dolphin and beneath the WP dolphin, but not at the open control site. Fertilization tests with sea urchins at Day 535 also indicated toxicity within 0.5 m and below the BMP, WP, and untreated dolphins.

Regarding the PAH sediment concentrations detected in mesocosm sediments, Brooks (2004) concluded that newly treated railway ties pose minimal environmental risk to the endangered

dragonfly inhabiting wetland areas crossed by a rail line. Using benchmark methodology proposed by Swartz (1999), Brooks (2004) also concludes that no adverse biological effects can be expected at the observed PAH levels. However, due to the lack of sediment-toxicity tests and actual field monitoring, the potential for adverse affects to aquatic organisms exposed to PAH in sediments from leaching from railroad structures remains uncertain.

One other study deserves mention regarding contaminated river sediments. Pastorok et al. (1994) investigated the effect of creosote-contaminated sediments on aquatic organisms nearby a creosote-treatment plant in the lower Willamette River in Oregon. Endpoints evaluated included amphipod (*Hyalella azteca*) mortality and Microtox (*Photobacterium phosphoreum*) bioluminescence. Toxicity occurred within approximately 300 feet of the shoreline, with a highly toxic area near a dock used for creosote off-loading. A low level of contamination of crayfish and lack of serious liver lesions in suckers collected near the site suggest that risk to mobile species from chronic contamination is low. There was no evidence of adverse effects throughout the rest of the main channel of the river.

Risk Characterization

Aquatic structures

Based on the available toxicity and exposure data, acute risk is presumed for listed freshwater and saltwater fish and aquatic invertebrates exposed to PAHs in the water column. Acute risk also is presumed for non-listed saltwater invertebrates. These risk presumptions are made from LOC exceedances determined from RQs calculated from water-column EECs generated in outdoor microcosms and large tanks and from weighted toxicity values for the PAH complex expected in the water column. Insufficient data are available to determine chronic toxicity of the PAH complex or even the toxicity of the individual PAHs addressed in this assessment. Therefore, chronic RQs cannot be determined. Limited data from the literature indicate that adverse chronic effects (survival, growth, reproduction, immunotoxicity) are possible from some PAHs or mixtures of PAHs at exposure levels expected in the water column. Guideline studies for acute and chronic toxicity testing with freshwater and saltwater fish and invertebrates exposed to the PAH complex would be needed to further characterize acute and chronic risks to aquatic animals inhabiting the water column. Guideline data also are not available to assess risks to aquatic plants. Uncertainties exist with the available data on creosote leaching from treated wood into the environment. Additional data would help to refine acute and chronic EECs. Field monitoring of aquatic concentrations also could be undertaken in areas of newly installed structures to further refine PAH concentrations over time.

Based on findings from the Sooke Basin field study in Canada, PAH accumulation in sediments is believed to be sufficiently high to pose a risk to benthic organisms in the immediate vicinity of creosote-treated pilings for at least a year. Risk could be better characterized if acute and chronic sediment-toxicity guideline studies were available. These guideline studies are outstanding for creosote. Moreover, movement into and retention of PAHs in sediments around creosote-treated aquatic pilings are likely to vary locally, depending on abiotic and biotic factors such as current speed, amount of treated structure per unit area, air and water temperature, salinity, and the

infaunal species occurring in the immediate area of the structures. Therefore, many uncertainties exist in extrapolating findings from the Sooke Basin to warmer locales in the southern U. S. Because of those uncertainties, prior to installation of any new aquatic structures, a site evaluation is essential to evaluate potential risks, particularly to listed (i. e., endangered or threatened) or other sensitive species or sensitive life stages. Risk could be further characterized with additional field or simulated field studies representing the environmental conditions and species assemblages occurring in the southern U. S.

Existing aquatic structures are likely to continue leaching creosote to the aquatic environment, but at lower rates than newly installed structures. In some situations, such as noted at Sooke Basin after about a year, leaching from submerged pilings might possibly be limited due to the mass of organisms attached to the pilings. Even in those situations, leaching may still occur in the pilings above the waterline, especially in hot weather. In other areas, such as where herring or other organisms attach their eggs directly to creosote-treated pilings, even pilings that have been submerged for many years, eggs may not hatch or surviving embryos may not develop normally. Developmental or other sublethal impacts also might occur where aquatic organisms are exposed to low PAH concentrations but for prolonged periods. Chronic guideline studies with freshwater and saltwater fish and invertebrates would provide additional information for assessing such risks.

Long-term findings from the Sooke Basin field study deserve mention. The researchers reported that a substantial community of mussels, anemones, and other invertebrates inhabited the creosote-treated pilings after the first year. Sediment concentrations also decreased at that time, which they speculate may have been due to the physical barrier of organisms precluding additional movement of creosote into the water column and sediments. However, despite the apparent lack of long-term adverse affects on some organisms, sensitive species might be adversely affected by short-term exposure. Additional acute-toxicity data generated in guideline studies and refinement of initial EECs in the water column would help address that risk, especially for listed species.

Railroad ties

Based on modeled EECs using PRZM/EXAMS scenarios, and the weighted toxicity values for the PAH complex, acute risk is presumed for listed freshwater and saltwater fish. Based on the scenario for wetter areas, acute risk also is presumed for non-listed freshwater fish and aquatic invertebrates exposed to PAHs in the water column. Chronic risk can not be assessed due to the lack of chronic toxicity data, but some available evidence indicates that chronic risk is possible to aquatic organisms inhabiting the water column. As noted for aquatic structures, further characterization of acute and chronic risks would require acute and chronic toxicity testing with fish and aquatic invertebrates exposed to the PAH complex in the water column. Acute and chronic EECs could be refined from creosote leaching studies in the water column over time, or from field monitoring in areas where railroad ties are in proximity to surface waters. Data obtained from a mesocosm study indicate that sediment PAH levels leached from railroad ties appear to be lower than those around aquatic pilings, and the PAHs do not move any substantial distance from the railway ballast.

7.3 ENDANGERED SPECIES CONSIDERATIONS

Section 7 of the Endangered Species Act (ESA), 16 U.S.C. Section 1536(a)(2), requires that federal agencies consult with the National Marine Fisheries Service (NMFS) for marine and andronomus listed species, or with the United States Fish and Wildlife Services (FWS) for listed wildlife and freshwater organisms, if proposing an "action" that may affect listed species or their designated habitat. Each federal agency is required under the Act to insure that any action they authorize, fund, or carry out is not likely to jeopardize the continued existence of a listed species or result in the destruction or adverse modification of designated critical habitat. To jeopardize the continued existence of a listed species is to "to engage in an action that reasonably would be expected, directly or indirectly, to reduce appreciably the likelihood of both the survival and recovery of a listed species in the wild by reducing the reproduction, numbers, or distribution of the species." 50 C.F.R. §402.02.

To comply with subsection (a)(2) of the ESA, EPA's Office of Pesticide Programs has established procedures to evaluate whether a proposed registration action may directly or indirectly appreciably reduce the likelihood of both the survival and recovery of a listed species in the wild by reducing the reproduction, numbers, or distribution of any listed species (U.S. EPA 2004). If any of the Listed Species LOC Criteria are exceeded for either direct or indirect effects in the Agency's screening-level risk assessment, the Agency identifies any listed or candidate species that may occur spatially and temporally in the footprint of the proposed use. Further biological assessment is undertaken to refine the risk. The extent to which any species may be at risk determines the need to develop a more comprehensive consultation package as required by the ESA.

The ecological risk assessment for creosote indicates a potential for exposure of listed fish and aquatic invertebrate species that warrants a more refined assessment to include direct, indirect, and habitat effects. The refined assessment should involve clear delineation of the action area associated with proposed use of creoste and best available information on the temporal and spatial co-location of listed species with respect to the action area. This analysis has not been conducted for this assessment. An endangered species effect determination will not be made at this time.

8.0 INCIDENT REPORTS

The following databases have been consulted for the poisoning incident data on the active ingredient creosote (PC Code: 025002):

OPP Incident Data System (IDS) - The Incident Data System of The Office of Pesticide Programs (OPP) of the Environmental Protection Agency (EPA) contains reports of incidents from various sources, including registrants, other federal and state health and environmental agencies and individual consumers, submitted to OPP since 1992. Reports submitted to the Incident Data System represent anecdotal reports or allegations only, unless otherwise stated. Typically no conclusions can be drawn implicating the pesticide as a cause of any of the reported health effects. Nevertheless, sometimes with enough cases and/or enough documentation risk mitigation measures may be suggested.

Poison Control Centers - as the result of a data purchase by EPA, OPP received Poison Control Center data covering the years 1993 through 1996 for all pesticides. Most of the national Poison Control Centers (PCCs) participate in a national data collection system, the Toxic Exposure Surveillance System, which obtains data from about 65-70 centers at hospitals and universities. PCCs provide telephone consultation for individuals and health care providers on suspected poisonings, involving drugs, household products, pesticides, etc.

California Department of Pesticide Regulation - California has collected uniform data on suspected pesticide poisonings since 1982. Physicians are required, by statute, to report to their local health officer all occurrences of illness suspected of being related to exposure to pesticides. The majority of the incidents involve workers. Information on exposure (worker activity), type of illness (systemic, eye, skin, eye/skin and respiratory), likelihood of a causal relationship, and number of days off work and in the hospital are provided.

National Pesticide Telecommunications Network (NPTN) - NPTN is a toll-free information service supported by OPP. A ranking of the top 200 active ingredients for which telephone calls were received during calendar years 1984-1991, inclusive, has been prepared. The total number of calls was tabulated for the categories human incidents, animal incidents, calls for information, and others.

Published Incident Reports - Some incident reports associated with creosote related human health hazard are published in the scientific literature.

Incidents by database are summarized here:

1) OPP's Incident Data System (IDS)

The following cases from the IDS do not have documentation confirming exposure or health effects. Registrants are not required to report incidents involving exposure to previously treated wood, only direct exposure to creosote itself. Therefore, it is possible that serious adverse effects involving exposures to treated wood have been missed by this review. Legal claims of severe damage to eyes and skin including infections requiring amputation have been reported but only in a cursory way and without enough documentation to be included in this review.

Incident#2796-100

An incident was investigated in the United Kingdom in 1994 or 1995 (date of incident unknown) involving creosote. After a landlord treated a residence with creosote the male tenant complained of headache, stomach ache, and respiratory irritation. No further information is available on the disposition of this case.

Incident #2796-119

An incident was investigated in the United Kingdom in 1994. After creosoting work was done on the flat below theirs, a male and female reported tearing, burning throat, nausea, and vomiting. No further information is available on the disposition of this case.

Incident #8760-1

In 1997 a 38 year old railroad worker alleged inhalation and dermal exposure to creosote. The timing and duration of exposure are not reported. A legal claim has been filed alleging nodular malignant melanoma. No further information is available on the disposition of this case.

Incident #8760-3

A worker at a creosote plant was exposed in 1994 while testing boring treated wood. He reportedly developed skin rash on wrists and forearms and visited a dermatologist.

2) Poison Control Center

No data were reported in the Poison Control Center database covering the years 1993 through 1996**.**

3) California Data - 1982 through 1996

Detailed descriptions of 124 cases submitted to the California Pesticide Illness Surveillance Program (1982-1996) were reviewed. In 114 of these cases, creosote was used alone and was

judged to be responsible for the health effects. Only cases with a definite, probable or possible relationship were reviewed. Creosote ranked 88th as a cause of systemic poisoning in California (1982-1994). Most of the cases that could definitely be attributed to creosote (80% of the 50 cases categorized as definite) involved workers who handled creosote directly but did not have proper protection for eyes or skin. A significant number of cases have resulted when workers have been exposed to treated wood, usually by handling or sawing the wood. Most of these cases experienced chemical burns to the skin or eyes.

4) National Pesticide Telecommunications Network (NPTN)

On the list of the top 200 chemicals for which NPTN received calls from 1984-1991 inclusively, creosote was ranked 118th with 26 incidents in humans reported and no incidents in animals.

5) Open Scientific Literature

Dean et al. (1992) reported on a white ten week old female, who weighed 6 kilograms, and experienced cyanosis, irritability, metabolic acidosis, and a lethal methehemoglobin level of 71.4%. She was taken to the hospital and remained for three days. Three days earlier, the child's father replaced an aluminum stove pipe leading from the wood-burning stove to the chimney and installed a straight section of the stove pipe. Green slab pine wood was continuously burning in the stove. Pine tar fumes emitted from the stove were the suspected source of creosote oils. The girl's cradle was approximately five feet from the stove.

Bowman et al. (1984) reported on a seventy year old man who was found unconscious with a cup of creosote beside him. On admission to the hospital, the man's respiratory effort was weak and on auscultation, widespread crackles were heard. His face and clothes were stained with vomit and creosote. He was immediately administered endotracheal intubation and artificial ventilation. He experienced anuria and died. After his death, a liter of mostly creosote fluid was found in his stomach.

Thompson et al. (1994) reported that during 1989 to 1991, 250 children (124 boys and 126 girls) under 10 years old out of 6, 478 cases were taken to accident and emergency departments in the United Kingdom for suspected pesticide poisoning. Seven percent of these cases were due to creosote.

The following excerpts were taken directly for the Hazardous Substances Data Bank (HSDB). HSDB is a toxicology data file on the National Library of Medicine's Toxicology Data Network (TOXNET). Data are derived from "a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the bank's scope."

> Death from large doses of creosote appears to be due largely to cardiovascular collapse. Fatalities have occurred 14 to 36 hr after the ingestion of about 7 g by adults or 1 to 2 g

by children. The symptoms of systemic illness included salivation, vomiting, respiratory difficulties, thready pulse, vertigo, headache, loss of pupillary reflexes, hypothermia, cyanosis, and mild convulsions. The repeated absorption of therapeutic doses from the gastroenteric tract may induce signs of chronic intoxication, characterized by disturbances of vision and digestion (incr peristalsis & excretion of bloody feces). In isolated cases of "self-medication," hypertension & also general cardiovascular collapse have been described. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2603]

Contact of creosote with the skin or condensation of vapors of creosote on the skin or mucous membranes may induce an intense burning and itching with local erythema, grayish yellow to bronze pigmentation, papular & vesicular eruptions, and gangrene and in isolated instances cancer. ... Heinz bodies have been noted in the blood of a patient one yr after his exposure to creosote. ... Similar observations following percutaneous absorption of this preparation. Eye injuries can include keratitis, conjunctivitis, and abrasion of the cornea. ... Permanent corneal scars result in about one third of such cases. Photosensitization has been reported ... and severe systemic illness. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2603]

Contact of liquid creosote with the eye has caused painful protracted keratoconjunctivitis. This has involved loss of corneal epithelium, clouding of the cornea, miosis, and long lasting irritability and photophobia. In one report concerned with creosote, two patients have been described, one examined 2 wk and the other 2 months after working with this material, both complaining of haziness of vision, which was found to be associated with numerous gray spots of varied size in the corneas, plus a superficial keratitis. [Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. 283]

Injuries to the skin or eyes have occurred mainly among men engaged in dipping or in "pickling" and handling "sleepers," mine timbers, and woods for floors and other purposes. ... Calls attention to burns induced by fine particles of sawdust from creosote-treated lumber. ... The burns were reduced to a minimum on rainy days, probably because of the decreased dispersion of both the wood particles and creosote. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2601]

Epitheliomas can result from prolonged exposure to creosote. [Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V22 592 (1983)]

Vapor causes moderate irritation of nose and throat. Liquid may cause ... reddening and itching of skin. [U.S. Coast Guard, Department of Transportation. CHRIS - Hazardous Chemical Data. Volume II. Washington, D.C.: U.S. Government

Printing Office, 1984-5.]

Old creosote treated lumber ... retains a considerable portion of the oil for periods up to 25 or 30 years. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2604]

6) Epidemiology Studies Associated with Creosote Exposure

Case Series Involving Chronic Effects Associated with Health Effects of Creosote in Humans

Garrett (1975)

In a letter-to-the-editor, Garrett reported two patients diagnosed within eighteen months with multi-focal transitional cell carcinoma of the bladder with muscle invasion. Both men were determined to have had chronic exposure to cresol and creosote, but no details of the exposures were provided.

Reports of this kind may be useful when combined with other reports and studies. Considered alone, no conclusion regarding association of exposure to creosote with development of bladder cancer can be made.

6.1 Cross-Sectional Studies Associated with Health Effects of Creosote in Humans

Koppers (1979a)

The Koppers Company sponsored a cross-sectional study of workers at four wood preservative plants in Pennsylvania, South Carolina, West Virginia, and Kentucky where creosote and creosote/coal tar were the predominant treatments. The study was specifically aimed at identifying any health problems known to be related to exposure to these major process materials. An array of medical examinations were performed on 257 participants (73% of 351 total workers). The ratios of men to women participants were similar among all four plants. However, the ratios of black to white workers differed significantly among the plants, therefore the ratios of black to white participants differed also. The battery of examinations included a medical questionnaire, chemical exposure questionnaire, chest x-ray, pulmonary function test, clinical chemistry analysis, hematology analysis, urinalysis, sputum cytology exam, and urine cytology exam.

No exposure parameter was evaluated in the health assessment other than length of service. With the exception of a greater than expected number of pustular eruptions of the skin, all other tests revealed only infrequent and borderline abnormal findings. There was no evidence of cancer at

any site associated with work at these plants.

Due to the broad nature and limited depth of this study, only gross negative health effects could be observed. Since no exposure assessment for creosote was performed, no association between observed health conditions and creosote exposure was possible. Within these limitations, no evidence of detrimental health effects from working with creosote was seen.

Koppers (1979b,c and 1980a,b,c)

Cross-sectional studies were conducted at five coal tar processing plants to assess the health status of the work forces and thereby identify possible adverse health problems associated with exposure to coal tar and its derivatives. The studies were conducted by contracted researchers as part of a continuing health and safety program sponsored by the parent organization. The five plants studied were located in California, West Virginia, Alabama, Ohio, and Illinois; all five provided potential exposure to many industrial products, including creosote, resulting from distillation of coal tar. From a toxicological evaluation of coal tar products, an appropriate medical examination protocol was designed to measure a number of health parameters that should reveal toxic effects from the target coal tar products. Included among the procedures were collection of medical and work history, chest x-ray, pulmonary function test, clinical chemistry analysis, blood and urological analysis, and sputum cytology examination.

The study populations included men and women, white and black, but participation was voluntary resulting in an overall participation rate of 42%. Length of employment ranged between less than one year to 50 years, but a majority of the workers who participated in the study worked 10 years or less. No assessment of personal exposure to specific substances was performed. The sole exposure parameter, which was collected through the work history questionnaire, was the number of years of potential exposure to coal tar and its derivatives.

Among the results from the broad medical examination, a number of excesses and atypical findings were observed, although few could be directly associated with working at the coal tar plants. Restrictive respiratory deficits were found in the populations at all of the study sites and considerable excesses were seen at three sites. A few individuals at four of the five plants also were observed with obstructive respiratory deficits. Increases in gamma glutamyl transpeptidase (GGTP) and lactic dehydrogenase (LDH) levels were found in a few individuals at two plants. Results from hematological examinations showed atypical cells or abnormal cell counts in a few workers at all five plants. Of particular interest were the increased eosinophil counts observed in 13% of the workers at one plant. The only notable result from the urine analyses was the observation of excess RBCs (eight workers) and WBCs (11 workers) in 10% of the participants from one plant. The prevalence of folliculitis was greater than expected at three of the plants, with one of the plants having an incidence significantly increased (11 out of 105 workers examined). At one plant, no folliculitis was seen, but tar warts which are known to be associated with exposure to coal tar, were in excess. In general, few atypical cells were found during examinations of sputum. One exception was the increased C-reactive protein observed in five

workers at the same plant at which the excess blood cells in urine and the greatest excess in folliculitis occurred. No cancer at any site was discovered during the broad medical examination program.

This group of studies showed evidence of increased prevalence of folliculitis and tar warts consistent with prolonged exposure to coal tar products. The only chronic health effect observed was an excess of restrictive respiratory deficit. No excess cancer occurrence was reported. The usefulness of results of this study are weakened by the lack of specificity to creosote exposure, by only 42% participation of eligible workers, and the lack of individual exposure assessment to coal tar products.

NIOSH (1981)

Following a request from a carpenters' union, NIOSH conducted an evaluation of exposure among six dock builders engaged in driving creosote-preserved logs into a river bottom. Health surveys also were administered for five of the six dock builders.

Breathing zone and area air concentration measurements collected for the cyclohexane-extractable fraction of the coal tar pitch volatiles ranged from below the detectable limit to 0.06 mg/m^3. However, because of atypical weather conditions on the day of sampling and because the pile driver was in operation for less than one hour, the industrial hygiene results were not representative of normal working conditions.

A medical questionnaire was administered to five of the six workers. The questionnaire covered work conditions and work history, past exposures, current health problems, medical history, the use of personal protection and personal hygiene. Questions on health problems focused on skin, respiratory, gastrointestinal, and central nervous system problems. The five participating workers were also given skin examinations. The pile drivers were between 24 and 61 years of age (average age 44.6 years), and all had worked at the current site for at least five months. All of the participants had been employed as pile drivers for an average of 16.6 years of which an average of 8.3 years had involved pile-driving creosote-preserved piles. A number of health problems were reported by the workers, including eye irritation, nausea, lightheadedness, and swelling of the face, eyes, and hands. Skin problems reported by the workers included irritation, rashes, erythema, burning, dryness, desquamation, itching, and cracking. On hot days, symptoms were reported to be worse, and in addition, the workers experienced tearing and burning eyes, red eyes, swollen or puffy eyes, and photophobia. Four of the five workers responding to the questionnaire reported that their visual acuity had gradually worsened.

Skin examinations of the workers revealed erythema on the face, neck and hands, dry skin with desquamation in sun exposed areas, black comedones, plugged hair follicles on hands and forearms, and mild folliculitis on the forearms.

The symptoms reported by the dock building workers and the observations made during skin examinations were consistent with phototoxic skin reactions. The folliculitis was consistent with prolonged and direct contact with creosote. No chronic health effects, including cancers, were reported or observed, and because of the small number of workers examined, encountering these diseases would not be expected.

EPA (1981a)

A broad health evaluation was performed in 1981 on 59 workers (total of 79 workers eligible) at a wood preservative treatment plant in Ohio. The workers (51 males, eight females) were aged between 20 and 69 years, with only a slightly higher frequency of workers aged between 55 and 59 years. Creosote had been used at the plant since the 1920s, but had been discontinued in 1979. A large battery of tests including chest x-rays, pulmonary function tests, clinical chemistry analyses, hematology and urology analyses, and sputum and urine cytology were used to assess effects on organs and body systems known to be at risk from exposure to chemicals used in the plant. No industrial hygiene monitoring data were available, and no exposure assessments for individual participants were made.

Fifteen workers were observed with restrictive or obstructive respiratory deficits. One participant had elevated serum enzyme levels indicative of liver disease. Two workers had proteinuria and one other had evidence of urinary tract inflammation. Thirteen workers were found to have elevated serum triglycerides, but only one with levels above 400mg/100ml.

This study identified no occupationally related disease and showed little evidence of chronic effects from working for long periods in a wood preservative treatment plant. The small size of the study cohort and the lack of assessment of individual exposures, including the absence of data on number of years employed, seriously limited the possibility of observing negative health effects.

EPA (1986)

A cross-sectional study was conducted on 113 of the total 140 workers at a lumber preservative treatment plant. Thirty-nine of the participants worked less than one year, 40 had worked between one and 10 years, and 34 had worked between 11 and 35 years. The plant had used creosote, creosote/tar solution, Wolman salt (CCA), and pentachlorophenol (PCP) for many years since 1946 as wood preservatives. A fire retardant, NCX, also was used since 1978. The study focused on creosote and PCP since these were considered the chemicals of concern.

Health effects from working at the wood treatment plant were evaluated by a battery of tests including chest x-ray, pulmonary function test, clinical chemistry analysis, hematology and urology analyses, and sputum and urine cytology studies. Detailed medical and work history questionnaires were administered, however, no individual exposure assessment was conducted.

Air concentrations for coal-tar pitch volatiles were available from a single industrial hygiene survey conducted in 1978.

No evidence of skin cancer, bladder cancer, or lung cancer were seen in the study population. Pustular eruptions likely related to exposures at the plant were observed in a greater than expected number of workers. A number of workers had restrictive or obstructive pulmonary deficits, and two workers showed evidence of liver disease. There was no evidence of kidney disease or blood disease.

This study showed little evidence of chronic effects from working for long periods in a wood preservative treatment plant. The small size of the study cohort and the lack of assessment of individual exposures limited the possibility of observing negative health effects.

6.2 Cohort Studies Associated with Health Effects of Creosote in Humans

EPA, (1981b and 1982)

An in-depth study of mortality in 4048 males who worked at eight Koppers coal tar plants was conducted by Tabershaw Occupational Medicine Associates and reported by Koppers in 1981. The plants were located in Illinois, West Virginia, California, New Jersey (two plants), Texas, Alabama, and Ohio; and all plants except for one of the New Jersey plants distilled crude coal tar. Creosote was among the distillation by-products resulting from the plants' operations . The cohort was initially defined as all males who worked at least 10 days between 1946 and 1977. Persons who worked in strictly clerical or secretarial positions were excluded, as were women because of their small number.

The cohort consisted of 2,150 workers (53.1%) known to be white, 1,104 workers (27.3%) known to be black, and 794 workers (19.6%) whose race was unknown. Demographic information including date of hire, date of termination, and complete work history was collected from plant personnel files. Vital status follow-up information was collected by using plant records, SSA, motor vehicle bureaus, and finally local phone directories. The total cohort provided 64,600 person-years of observation with 9,917 person-years attributed to workers whose race was unknown. Of the total cohort, 703 (17.4%) were identified as deceased, and the vital status of 359 (8.9%) remained unknown.

During the analysis of the 1981 study, it was recognized that the lack of race information for almost 20% of the cohort presented a serious weakness in the study and imposed considerable difficulties with the interpretation and validity of results. This was further complicated by the fact that 163 of the workers classified as "race unknown" also had unknown vital status. Because of this weakness, a re-analysis of data for only those workers whose race was verified was performed in 1982, therefore, the results from the 1981 study are not presented here. The redefined cohort excluded the 794 workers with unknown race. The number of person-years of

follow-up was 36,635 for the white workers and 18,047 for the black workers. Within the cohort, 701 deaths had occurred by the close of the study (12/31/77), and death certificates were retrieved for 632 workers (359 white, 273 black).

The second analysis looked at cause-specific deaths for six subgroups of the total population of workers with known race. These groups were (1) all white workers, (2) white workers employed for less than six months, (2) white workers employed for six months or more, (4) all black workers, (5) black workers employed for less than six months, (6) black workers employed for six months or more.

For the entire population of white workers, the standard mortality r
atio (SMR) for all causes was 109. However, the SMR for deaths from all cancers was considerably elevated (SMR=126) largely due to the significant excess in cancers of the lung (SMR=160, p=0.05). Excesses also were observed for cancers of the stomach, large intestine, rectum, bladder, and kidney, however, none of the SMRs were statistically significant. When only white workers employed for less than six months were considered, a large excess in total mortality was observed (SMR=137, p=0.01), and the SMR for deaths from all cancers was 125, though not significant. The increases in overall mortality were due largely to significant excesses in deaths from cirrhosis of the liver (SMR=340, p=0.01), accidents (SMR=238, p=0.01), and cancer of the stomach (four observed, 0.74 expected, SMR=540, p=0.05). When only white workers employed for six months or more were considered, the only significant excesses observed were for cancer of the respiratory system (SMR=182, p=0.01), largely due to an excess of lung cancer (SMR=180, p=0.01). Deaths from all other cause-specific cancers were within expected numbers.

For the combined population of black workers, a number of statistically significant excesses (p=0.05) were found, including deaths from all causes (SMR=113), all cancers (SMR=138), cancer of the rectum (SMR=439), and lung cancer (SMR=173). The number of deaths from accidents, poisoning, and violence were also highly elevated (SMR=186, p=0.01). When only black workers employed for less than six months were considered, large excesses were seen for total mortality (SMR=154, p=0.01), for deaths from all cancers (SMR=171, p=0.05), and for accidents (SMR=241, p=0.01). The SMR for cancer of the respiratory system was significantly increased (226, p=0.05), influenced greatly by the SMR for lung cancer (SMR=243, p=0.01). The SMR for cancer of the esophagus was also greatly increased (326), though it was based on only three deaths with 0.92 expected. When only black workers employed for more than six months or more were considered, the SMR for all causes of death was 90, and the only significant excess observed was for bladder cancer (SMR=531, p=0.05) based on three deaths. Nonsignificant excesses also were observed for deaths from all cancers and several specific diseases, including cancers of the digestive system and skin, diseases of the hematopoietic system, and accidents. None of the excesses were statistically significant and were based on small numbers of deaths. Overall, mortality in the group of black workers employed six months were higher than in the black workers employed less than six months.

This study provided a large amount of mortality data on a reasonably large occupational cohort. Moderately convincing evidence is presented that employment at the eight coal tar distillation plants may result in increased risk of death from a range of malignancies. The study appeared to be well planned and executed, though the validity of the findings is limited by a number of shortcomings. These include the lack of race information on a large fraction of the cohort, the small number of deaths observed for many of the diseases reported in excess, and the very crude measure of exposure based only on employment at one or more of the plants.

Steineck et al. (1989)

Steineck, et al. employed a complex job-exposure matrix to estimate exposure for calculating relative risk for development of renal pelvic cancer (RPC) or bladder cancer (BC) in a Swedish population. The cohort was defined as all males born in Sweden, aged 20-64 in 1960, who reported themselves employed. Cases of renal RPC or BC occurring during the 19-year study period were identified through the National Swedish Cancer Registry.

The job-exposure matrix used to determine exposed and unexposed subpopulations was based on self-reported job-related information collected in 1960 for census purposes. Based on this information, subjects were classified into 292 occupational titles and 308 industrial categories, yielding 292 X 308 possible work tasks. Potential exposure to 50 single agents or groups of substances were assigned for each possible work task defined by the matrix. Among the potential exposures selected for evaluation were most of those cited in the literature as potential risk factors for the two cancers of interest, and creosote.

Relative risks were calculated after adjusting for age in 1960 (six categories). For some calculations, adjustments also were made for marital status, socioeconomic group, and urbanization of residence. Among the total study population of 1,905,660 persons, 556, 429 were judged to be exposed to at least one of the selected substances. During the 19 years of observations within the study, there were 714 cases of RPC with 542 cases occurring among the unexposed subjects. There were 10,123 cases of BC with 7,432 cases occurring within the unexposed group. For individuals categorized as exposed to creosote, the relative risk for BC was 1.4 (95% CI 0.7-2.6) compared to cohort members not assigned any exposure. It is notable that all of the BC cases categorized as exposed were leather tanners who were also assigned a number of other exposures. When adjustments for applied for age, marital status, socioeconomic group, and degree of urbanization, the relative risk for BC remained between 1.25 and 1.30.

This study provides very limited evidence of association between exposure to creosote and occurrence of bladder cancer. Weaknesses include exposure assessment based solely on self-reported occupational information from a single census observation, lack of control for multiple exposures, and no consideration for nonoccupational exposures.

Karlehagen et al. (1992)

Karlehagen, et al. studied cancer incidence among 922 men exposed to creosote at 13 wood impregnating plants in Sweden and Norway. Most participants worked as impregnators while 36 men repaired or maintained railroad cars used to transport creosote. Study participants were employed at least one year between 1950 and 1975, and follow-up was 1958-1985 for the workers in Sweden and 1953-1987 for the workers in Norway. Cancers were identified through national cancer registries in both countries. Cancer registration is compulsory in both countries, and quality and completeness of the registries was considered to be good.

No individual exposure measurements were available for participants, however, levels of naphthalene and benzo(a)pyrene (major constituents of creosote) at several of the plants had been determined to be 0.1-11 mg/m^3 and 0.03$\mu g/m^3$, respectively. Levels for both constituents were well below accepted exposure limits. Consequently, exposure assessment for study participants was based on minimum length of employment at plants known to use creosote regularly. Information on the type of work performed at each plant was collected through use of a questionnaire completed by plant personnel, but not by participants. No differences in exposure conditions among the 13 plants were observed.

The total incidence of cancer was lower than expected with 129 cases observed and 137 cases expected. Some differences were seen between the Swedish and Norway subgroups but the differences were small. Increased risks were observed for lip cancer (SIR=2.50, P=0.05), nonmelanoma skin cancer (SIR=2.37, P=0.02), and malignant lymphoma (SIR=1.9, P=0.06). When a latency period of 20 years since first exposure was applied, the SIRs for lip cancer, nonmelanoma skin cancer, and malignant melanoma were 3.7, 2.0, and 2.2 respectively. Only the SIR for lip cancer (five cases observed, 1.34 cases expected) was statistically significant. No increase in the incidence of lung cancer was observed in this population, with or without consideration for time since first exposure.

This study presents reasonable evidence that exposure to creosote, as measured by employment at creosote plants, is likely associated with development of nonmelanoma skin cancer. Increased risks of lip cancer and malignant melanoma (Norway subgroup only), and malignant lymphoma were also observed in the study population, but the risks were not statistically significant. Because the workers in the study worked outdoors part of the time, the validity of the associations observed, particularly for lip cancer, nonmelanoma skin cancer, and malignant melanoma, may be weakened.

6.3 Case Control Studies Associated with Health Effects of Creosote in Humans

Flodin et al. (1987)

Risk factors for development of multiple myeloma (MM) were investigated in a study of 131 cases and 431 controls in Sweden. The cases were identified from records at six hospitals in

southeast Sweden and were required to be less than 81 years of age, of Swedish ethnicity, resident in the catchment areas of the hospitals at the time of diagnosis, and capable of responding to a questionnaire. The 131 cases represented approximately one third of the total number of MM cases occurring in the area as reported to the cancer registry. The discrepancy between total number of cases and the number of cases identified from the six hospitals was attributed to simple administrative record keeping and was judged to not impose any bias on the study findings. Controls were randomly selected from population registries for the same catchment areas from which the cases were drawn. Differences in average age and distributions of gender were found between cases and controls. The average age for cases was 64 years and for controls, 58 years. Within the 131 cases, 57 percent were males; within 431 controls, 46 percent were males.

Assessment of exposure was through a nine-page questionnaire consisting of 17 major questions of which 10 related to occupational exposures. Some of the occupational questions also asked further questions regarding details of exposures. Reported exposures lasting less than one year and all reported exposures within five years prior to diagnosis were ignored in the analyses.

Crude rate ratios were significantly increased for occupational exposure to creosote (RR=6.0, 95% CI 2.00-18.2), fresh wood, engine exhaust, farming, and bricklaying. When the cases and controls were stratified into four age groups, the elevated risk ratios remained for creosote, fresh wood, and engine exhaust. The increased risks associated with creosote, engine exhaust, and fresh wood also remained significant when analyses controlled for confounding effects of other determinants.

This study provides moderate evidence that exposure to creosote, as measured by self-reporting via mailed questionnaire, may be linked to development of MM. The association is less convincing because the numbers of cases and controls reporting exposure to creosote were quite small. Also, the study suffers the same limits as other studies using similar assessment methods.

Persson et al. (1989)

A case-control study was conducted in Sweden by Persson, et al. to investigate associations between exposure to creosote and subsequent development of Hodgkin disease (HD) or non-Hodgkin's lymphoma (NHL). Cases were 160 patients (101 men, 59 women) with HD or NHL identified through the registry at Orebro Medical Centre Hospital and diagnosed between 1964 and 1986. The cases remained alive at least through the data collection period in 1986 and were required to be at least 20 years of age at diagnosis, born in Sweden, living in the area of the hospital at time of diagnosis, less than 80 years of age at time of data collection, and mentally capable of responding to the study questionnaire. The 275 controls (157 men, 118 women) were a subset of a larger set of controls, previously used in earlier studies, randomly drawn from general population registries in catchment areas of several hospitals. For the current study, only individuals in the catchment area from which the patients were drawn were used as controls. The

controls were required to meet the applicable inclusion criteria used for patients.

Information for assessment of exposures was collected through a nine page questionnaire mailed to each case and control. Of 17 main questions, 10 questions addressed occupational exposures with some of the occupational questions having additional subquestions asking for details. Questions also were asked about exposures during leisure activities. Exposures reported for periods of less than one year were not considered. A latency period between exposure and development of disease was imposed by considering only exposures within five to 45 years prior to diagnosis for the cases. For the controls, exposures were only considered if they occurred five to 45 years before the point in time of selection.

Age ranges for cases and controls were similar; 20-73 for HD, 22-79 for NHL, and 20-77 for controls. Crude odds ratios (ORs) for both HD and NHL were increased for exposure to wood preservatives and for exposure to creosote specifically (OR 10.5 for HD, OR 13.6 for NHL). Although the numbers of cases and controls exposed to creosote were small, logistic analyses were performed to control for age at time of case diagnosis, gender, and two exposure determinants, i.e., farming and exposure to fresh wood. For HD, the logistic OR for occupational exposure to creosote was still elevated (OR 10.7, CI 90% 1.1-103). For NHL, the logistic OR was 9.4 (CI 90% 1.2-69).

Assuming the instrument for exposure assessment and the methodology for administration was not biased, this study provides good evidence that exposure to creosote is a risk factor for development of both HD and HNL. The study is somewhat weakened by the small of number of persons reporting creosote exposure.

Feingold et al. (1992)

Feingold, et al. studied associations between parental exposures and cancers in children born subsequent to the exposures. The 252 incident cases, identified from a Colorado cancer registry, were in children 0-14 years of age, diagnosed between 1976 and1983. The cases were compared with 222 controls selected by random digit dialing in the same geographical area as the cases and matched on age (+/- three years), gender, and telephone exchange area.

Assessment of parental exposure was based on job history information (including job title, industry, and employment dates) collected by personal interview. A job-exposure matrix, derived from past industrial hygiene surveys and knowledge of industrial processes, was used to assign exposures to individuals on the basis of job title and industry of employment. All jobs held for six months or longer by mothers and fathers during the year prior to birth of the child were linked to all chemicals assigned to the job. Analyses were then performed to determine associations between cancer incidence and parental exposure to a large number of substances.

Creosote was not identified as an exposure for any of the mothers of cases or controls. An

adjusted odds ratio of 2.5 (CI = 0.8-8.1) was found for association of fathers' exposure to creosote during the year prior to birth of children with any type of cancer in the offspring. When associations between fathers' exposure to creosote and the incidence of specific cancers in children born subsequently were investigated, an odds ratio of 3.7 (CI = 0.8-16.6) was observed for childhood brain cancer. Fathers assigned exposure to creosote were chiefly in the construction industry or were farmers.

The major limitation of this study is the imprecision of the exposure assessment. Exposures to individuals with the same job titles and working in the same industries vary widely. Therefore, assignments of exposures to specific chemicals, such as creosote, based entirely on job titles and industries may be invalid for some individuals. Also, the credibility of occupation information collected from mothers for fathers is likely to be only 60-80%. However, exposure misclassification resulting from the lack of individual exposure data, or due to the necessary use of information from surrogates, is likely to be equal among parents of cases and controls and therefore, should be nondifferential.

Persson et al. (1993)

A case-control study was conducted in Sweden by Persson, et al. among 124 patients with HD or NHL to reexamine earlier findings of associations between exposure to creosote and HD and NHL. Cases diagnosed between 1975 and 1984 were identified through a regional cancer registry located at a university hospital serving a three county area. Only men were included in the study, and were required to be at least 20 year of age, born in Sweden, living in the area of the hospital at time of diagnosis, less than 80 years of age at time of data collection and mentally capable of responding to the study questionnaire. The 204 controls were randomly drawn from general population registries for the catchment area of the university hospital from which the patients were drawn. The controls were required to meet the applicable inclusion criteria used for patients.

Information for assessment of exposures was collected through a nine page questionnaire mailed to each case and control. Of 17 main questions, 10 addressed occupational exposures with some of the occupational questions having additional subquestions. Exposures of less than one year were not considered, and only exposures five to 45 years prior to diagnosis were considered pertinent for the cases. For the controls, the window of time during which exposures were considered had been determined based on the time of diagnosis of the patients in earlier studies.

None of the cases, and only four controls reported exposure to creosote. Assuming a null hypothesis for association of creosote with HD or NHL, the number of cases expected to report creosote exposure would be 2.4 based on the number of controls reporting creosote exposure and the ratio of cases to controls. This study shows no evidence of an association of creosote exposure with these diseases.

Tynes et al. (1994)

A nested case-control study was conducted to assess the presence of an association between exposure to electromagnetic fields existing at Norwegian railways and occurrence of brain tumors or leukemia in railway workers. Limited information on exposure to creosote was collected for analysis as a confounder.

The cohort from which the cases were selected included 13,030 male railroad workers employed in 1957 on either electric or non-electric railways and included line workers, outdoor station workers, and electrical workers. The cases identified from the Norway Cancer Registry to which all new cancer cases are reported included men diagnosed with brain tumors or leukemia during the follow-up period between 1958 and 1990. Four or five controls were selected for each case matched on year of birth. Controls were required to survive to the age at which the matching case was diagnosed. Information on whether the participants ever smoked was collected through telephone interviews.
Assessment of exposures to electromagnetic fields for the cases and controls was based on job titles, work histories, and job descriptions. Exposures to other potential hazards, including creosote, were estimated and analyzed as confounders. An exposure matrix was constructed using categories of exposure frequency (0=never, 1=monthly, 2=weekly, 3=daily) and years of employment as factors.

No association of brain tumors or leukemia with estimated exposure to creosote was observed in this study. As is true in many similar studies, assessment of exposures was based on qualitative information relevant to jobs and departments, and therefore is not precise, or accurate for any particular individual.

Schildt et al. (1999)

Associations between a number of occupational exposures including creosote with oral cancer was investigated in a case-control study in Sweden. The population-based study included 410 verified cases of squamous cell oral cancer reported to a four-county cancer registry during 1980-1989 and 410 controls drawn from a national population registry. Among the cases (175 alive, 235 deceased) were 134 women and 276 men. A control was matched to each case on age, gender, and county of residence. For deceased cases, deceased controls were selected from the the National Registry for Causes of Death. In addition to the other matching criteria, deceased controls also were matched on year of death.

Assessment of exposures was based on information collected through mailed questionnaires. For deceased participants, the questionnaire was sent to the next-of-kin in the order of spouse, child, parent, sibling, or other. The questionnaire included a lifetime work history and other questions concerning exposure factors of interest for oral cancer. Exposures associated with occupations

held for less than one year were ignored.

Analysis of association between exposure to creosote and oral cancer showed no increased risk (OR = 0.5, CI = 0.1-2.0). The reliability of this result is weakened by the method of exposure assessment and by the small numbers of individuals exposed (three cases and six controls).

9.0 REFERENCES

Product Chemistry References

None American Wood Preservers' Association Standards, 1998, pp 3-4.

None MacKay D. et al., 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals, Lewis Publishers.

None Mueller J.G. et al., 1989. Environ. Sci. Technol., Volume 23(10): 1197-1201.

None USEPA Document 540-RS-88-066. Guidance for the Registration of Pesticide Products containing Coal Tar/Creosote.

None Cited in reference 4 above.

None Cited in reference 4 above.

44141101 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analysis and Certification of Product Ingredients: Aristech Creosote P1/P13. Pesticide Assessment Guidelines Reference Series 62. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. July 23, 1992.

44141102 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. American Wood Preservers Association (AWPA) Standard Physical Characteristics: Aristech Creosote P1/P13. AWPA Standard Methods for Analyses of Creosote and Oil-Type Preservatives (A1-89). U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. August 29, 1992.

44141103 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analyses of Physical and Chemical Characteristics: Aristech Creosote P1/P13. Pesticide Assessment Guidelines Reference Series 63. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. September 15, 1992.

44141104 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analysis and Certification of Product Ingredients: Aristech Creosote P2. Pesticide Assessment Guidelines Reference Series 62. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. July 23, 1992.

44141105 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. American Wood Preservers Association (AWPA) Standard Physical Characteristics: Aristech Creosote P2. AWPA Standard Methods for Analyses of Creosote and Oil-Type Preservatives (A1-89). U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. August 29, 1992.

44141106 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analyses of Physical and Chemical Characteristics: Aristech Creosote P2. Pesticide Assessment Guidelines Reference Series 63. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. September 15, 1992.

45355601 Sparacino, Charles M. 2000. Product Chemistry for North American CTM Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355602 Sparacino, Charles M. 2000. Product Chemistry for North American CTM Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355701 Sparacino, Charles M. 2000. Product Chemistry for Western Tar Products Corp. Creosote P1 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355702 Sparacino, Charles M. 2000. Product Chemistry for Western Tar Products Corp. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355801 Sparacino, Charles M. 2000. Product Chemistry for KMG-Bernuth, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355802 Sparacino, Charles M. 2000. Product Chemistry for KMG-Bernuth, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355901 Sparacino, Charles M. 1998. Product Chemistry for Trenton Sales, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. October 15, 1998.

45356001 Sparacino, Charles M. 2000. Product Chemistry for Coopers Creek Chemical Corp. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356002 Sparacino, Charles M. 2000. Product Chemistry for Coopers Creek Chemical Corp. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356101 Sparacino, Charles M. 2000. Product Chemistry for Koppers Industries, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356102 Sparacino, Charles M. 1999. Product Chemistry for Koppers Industries, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. October 26, 1999.

45356201 Sparacino, Charles M. 2000. Product Chemistry for Reilly Industries, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356202 Sparacino, Charles M. 2000. Product Chemistry for Reilly Industries, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45363901 Sparacino, Charles M. 2000. Product Chemistry for Allied Signal Incorporated Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45363902 Sparacino, Charles M. 2000. Product Chemistry for Allied Signal Incorporated Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

Toxicology References

BIBLIOGRAPHY

MRID 43032101: North American P1/P13 Creosote: Acute Oral Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032102: North American P1/P13 Creosote: Acute Dermal Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032103: North American P1/P13 Creosote: Acute Inhalation Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032104: North American P1/P13 Creosote: Eye Irritation Study in Rabbits. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032105: North American P1/P13 Creosote: Primary Dermal Irritation Test in Rabbits. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032106: North American P1/P13 Creosote: Dermal Sensitization Study (Buehler) in the Albino Guinea Pig. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032301: North American P2 Creosote: Acute Oral Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032302: North American P2 Creosote: Acute Dermal Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished.

MRID 43032303: North American P2 Creosote: Acute Inhalation Toxicity Evaluation in Rats.Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032304: North American P2 Creosote:Eye Irritation Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032305: North American P2 Creosote: Primary Dermal Irritation Test in Rabbits.Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43032306: North American P2 Creosote: Dermal Sensitization Study (Buehler) in the Albino Guinea Pig. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Unpublished. HED document no. 011033.

MRID 43601001: North American P1/P13 Creosote: Thirteen Week Subchronic Inhalation Toxicity Study on North American P1/P13 Creosote CTM in Rats: Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-016 (1995). Unpublished.

MRID 43600901: North American P2 Creosote: Thirteen Week Subchronic Inhalation Toxicity Study on North American P2 Creosote CTM in Rats: Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-018 (1995). Unpublished.

MRID 43616101: North American P1/P13 Creosote: 90-Day Subchronic Dermal Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-013 (1995). Unpublished.

MRID 43616201: North American P2 Creosote: 90 Day Subchronic Dermal Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-014 (1993). Unpublished.

MRID 43584201: North American P1/P13 Creosote: Developmental Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-020 (1995). Unpublished.

MRID 43584202: North American P2 Creosote: Developmental Toxicity Study in Rats. Study conducted by IRDC, Mattawan, MI for the Creosote Council II. Report No. 671-022 (1995). Unpublished.

York, Raymond G. (1994): Developmental Toxicity Study in New Zealand White Rabbits. Study conducted by IRDC, Mattawan, MI for Koppers Inductries, Pittsburgh, PA. Report no. 672-002. Unpublished.

York, Raymond G. (1995): Two Generation Reproduction/Fertility Study in Rats. Study conducted by IRDC, Mattawan, MI for Koppers Inductries, Pittsburgh, PA.Report no. 672-006. Unpublished.

MRID 44844401: A 6-Month Dermal Oncogenicity Study of Creosote in Mice. Study conducted by WIL Research Laboratories, Ashland, OH for Koppers Industries, Pittsburgh, PA. Report no. WIL-100005. Unpublished.

McMahon, Timothy F.: Creosote: Report of the Hazard Identification Assessment Review Committee. May, 1999. HED document no. 013375. Unpublished.

USEPA, 1984: Wood Preservative Pesticides: Creosote, Pentachlorophenol, Inorganic Arsenicals. Position Document 4

Agency for Toxic Substances and Disease Registry (1990): Toxicological Profile for Creosote. U.S. Department of Health and Human Services: Public Health Service.

Culp, S.J., Gaylor, D.W., Sheldon, W.G., Goldstein, L.S., and Beland, F.A. (1996): DNA adduct measurements in relation to small intestine and forestomach tumor incidence during the chronic feeding of coal tar or benzo[a]pyrene to mice. *Polycyclic Aromatic Compounds* 11, 161-168.

Culp, S.J., Gaylor, D.W., Sheldon, W.G., Goldstein, L.S., and Beland, F.A. (1998): A comparison of the tumors induced by coal tar and benzo[a]pyrene in a 2-year bioassay. *Carcinogenesis* 19(1): 117-124.

Gaylor, D.W., Culp, S.J., Goldstein, L.S., and Beland, F.A. (2000): Cancer Risk Estimation for Mixtures of Coal Tars and Benzo[a]pyrene. *Risk Analysis* 20(1): 81-85.

Schneider, K., Roller, M., Kalberlah, F., and Schumacher-Wolz, U. (2002): Cancer Risk Assessment for Oral Exposure to PAH Mixtures. *J. Appl. Toxicol.* 22: 73-83.

Exposure References

Agency for Toxic Substances and Disease Registry (ATSDR, 1990). Toxicological Profile for Creosote. U.S. Public Health Service. February 16, 1990.

Agency for Toxic Substances and Disease Registry (ATSDR, 1995). Toxicological Profile for Creosote. U.S. Public Health Service.

Butala J. H. February, 1999. Creosote Council II memorandum with attachment to Nader Elkassabany, U.S. Environmental Protection Agency, Antimicrobial Division.

Creosote Council II, 1998. "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure- Treatment of Wood with Creosote." Submitted by John H. Buttala, Creosote Council II. Field work by George M. Singer, Ph.D, American Agricultural Services Inc. Analytical work by David A. Winkler, EN-CAS Laboratories.

Creosote Council II, 2001. "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure Treatment of Wood with Creosote." Submitted by John H. Butala, Creosote Council II. Field work by Mark G. Bookbinder, Ph.D, c/o American Agricultural Services Inc. Analytical work by Bert Clayton, EN-CAS Laboratories and Stephanie Guilyard, USX Engineers and Consultants. MRID 453234-01.

Flickinger and Lawrence, 1982. Occupational Health Experience in the Wood Preserving Industry. Koppers Company, Inc. MRID 447595-02.

Heikkila PR, Hameila M, Pyy L, et al. 1987. Exposure to Creosote in the Impregnation and Handling of Impregnated Wood. Scand J Work Environ Health 13:431-437.

Krygsman, Adrian 1994. "Wood Preservation. An Overview of Biocide Use and Application."
NIOSH (1980a). Industrial Hygiene Report, Comprehensive Survey of Wood Preservative Treatment Facility at Santa Fe Centralized Tie Plant, Somerville, Texas. National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, Cincinnati, OH. (by Todd AS & Timbie CY). NTIS PB83-133892.
Markel, H.L.; Ligo, R.N.; Lucas, J.B. 1977. Health Hazard Evaluation/Toxicity Determination Report 75-117-372. Koppers Company, Inc. North Little Rock, Arkansas.

NIOSH (1980). Industrial Hygiene Report, Comprehensive Survey of Wood Preservative Treatment Facility at Santa Fe Centralized Tie Plant, Sommerville, Texas. National Institute of Occupational Safety and Health, Division of Surveillance Hazard Evaluations and Field Studies, Cincinnati, OH. (by Todd AS and Timbie CY). NTIS PB83-133892.

NIOSH (1981a). Industrial Hygiene Report, Comprehensive Survey of Wood Preservative Treatment Facility at Cascade Pole Company, McFarland Cascade, Tacoma, Washington. National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, Cincinnati, OH. (by Todd AS and Timbie CY). NTIS PB82-174160.

NIOSH (1981b). Health Hazard Evaluation: New York Port Authority, Brooklyn, New York. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Cincinnati, OH. (by Baker D & Fannick N). HHE 80-238-931.

Personal Communication, 1998. Personal Communication with industry experts and members of the Creosote Council II and Pentachlorophenol Task Force on April, 1998.
Pentachlorophenol Task Force

a) Mr. John Wilkinson, Vulcan Chemicals, Phone (202) 293-0635.
b) Mr. Gene Meyer, Vulcan Chemicals, Phone (205) 877-3543.
c) Mr. Martin Rollins, H.M. Rollins Co. Inc., Phone (601) 832-1738.
d) Mr. Herbert Estreicher, Consultant on Task Force, Phone (202) 662-6000.

Creosote Council II.

a) Dr. John Butala, Consultant for the Creosote Council II, Phone (724) 443-0097.
b) Dr. Mark Bookbinder, Consultant for the Creosote Council II, Phone (301) 540-5622.

Personal Communication, 1999. Personal Communication with Dr. Timothy McMahon, Senior Toxicologist EPA/OPP/AD/RASSB. April 19, 1999.

Pesticide Handler Exposure Database (PHED) Surrogate Exposure Guide, 1997. Estimates of Worker Exposure from the Pesticide Handler Exposure Database (PHED) Version 1.1. May, 1997.

Rotard W, Mailahn W (1987). Gas Chromatographic-Mass Spectrometric Analysis of Creosotes Extracted from Wooden Sleepers Installed in Playgrounds. Anal. Chem. 59:65-69.

SRI. 1993. Directory of chemical producers: United States of America. SRI International, Menlo Park, CA.

The Environmental Degradation of Creosote, 1998. http://inweh1.uwaterloo.ca/biol447new/assignments/creo2.html.

United States Environmental Protection Agency (USEPA), 1984. "Wood Preservative Pesticides: Creosote, Pentachlorophenol, Inorganic Arsenicals. Position Document 4." Registration Division. Office of Pesticides and Toxic Substances. Washington, DC. July, 1994.

United States Environmental Protection Agency (USEPA), 1997a. Standard Operating Procedures for Residential Exposure Assessments. December, 1997. Residential Exposure Assessment Work Group, U.S. Environmental Protection Agency.

United States Environmental Protection Agency (USEPA), 1997b. Exposure Factors Handbook. Volume 1. Office of Research and Development. Washington, DC. EPA/600/P-95/002Fa.

United States Environmental Protection Agency (USEPA), 1998a. Route to Route Extrapolations. Memorandum from John E. Whalan EPA/OPP/HED to Margaret Stasikowski, Director EPA/OPP/HED.

United States Environmental Protection Agency (USEPA), 1998b. Creosote LUIS Database. USEPA Report Run Date 3/09/98.

United States Environmental Protection Agency (USEPA), 1998c. International Harmonization Position Paper. Antimicrobial Exposure Databases. An Overview of the NAFTA Member Agencies Regulatory Authority Over Antimicrobial Pesticides And An Evaluation of Existing Databases Used in Antimicrobial Exposure Assessments. Prepared by Antimicrobial Division. Office of Pesticide Programs.

United States Environmental Protection Agency (USEPA), 1998d. Inhalation Risk

Characterizations and the Aggregate Risk Index (ARI). Memorandum from John E. Whalan, EPA/OPP/HED, to Margaret Stasikowski, Director, EPA/OPP/HED.

United States Environmental Protection Agency (USEPA), 1998e. Integrated Risk Information System (IRIS) database.

United States Environmental Protection Agency (USEPA), 1999. Creosote - Report of the Hazard Identification Review Committee, April 1, 1999.

United States Environmental Protection Agency (USEPA), 2001. Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote. Office of Pesticides and Toxic Substances, January 30, 2001.

Product Chemistry References

MRID

None American Wood Preservers' Association Standards, 1998, pp 3-4.

None MacKay D. et al., 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals, Lewis Publishers.

None Mueller J.G. et al., 1989. Environ. Sci. Technol., Volume 23(10): 1197-1201.

None USEPA Document 540-RS-88-066. Guidance for the Registration of Pesticide Products containing Coal Tar/Creosote.

None Cited in reference 4 above.

None Cited in reference 4 above.

44141101 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analysis and Certification of Product Ingredients: Aristech Creosote P1/P13. Pesticide Assessment Guidelines Reference Series 62. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. July 23, 1992.

44141102 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. American Wood Preservers Association (AWPA) Standard

Physical Characteristics: Aristech Creosote P1/P13. AWPA Standard Methods for Analyses of Creosote and Oil-Type Preservatives (A1-89). U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. August 29, 1992.

44141103 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analyses of Physical and Chemical Characteristics: Aristech Creosote P1/P13. Pesticide Assessment Guidelines Reference Series 63. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. September 15, 1992.

44141104 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analysis and Certification of Product Ingredients: Aristech Creosote P2. Pesticide Assessment Guidelines Reference Series 62. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. July 23, 1992.

44141105 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. American Wood Preservers Association (AWPA) Standard Physical Characteristics: Aristech Creosote P2. AWPA Standard Methods for Analyses of Creosote and Oil-Type Preservatives (A1-89). U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. August 29, 1992.

44141106 Wade, Terry L. 1992. Product Chemistry Methods Development and Validation for Creosote. Analyses of Physical and Chemical Characteristics: Aristech Creosote P2. Pesticide Assessment Guidelines Reference Series 63. U.S.E.P.A. Pesticide Assessment Guidelines Subdivision D: Product Chemistry. Unpublished study prepared by Geochemical and Environmental Research Group, Texas A&M University, for Aristech Chemical Corporation and John H. Butala, Technical Advisor, Creosote Council II. September 15, 1992.

45355601 Sparacino, Charles M. 2000. Product Chemistry for North American CTM Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355602 Sparacino, Charles M. 2000. Product Chemistry for North American CTM Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355701 Sparacino, Charles M. 2000. Product Chemistry for Western Tar Products Corp. Creosote P1 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355702 Sparacino, Charles M. 2000. Product Chemistry for Western Tar Products Corp. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355801 Sparacino, Charles M. 2000. Product Chemistry for KMG-Bernuth, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355802 Sparacino, Charles M. 2000. Product Chemistry for KMG-Bernuth, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45355901 Sparacino, Charles M. 1998. Product Chemistry for Trenton Sales, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. October 15, 1998.

45356001 Sparacino, Charles M. 2000. Product Chemistry for Coopers Creek Chemical Corp. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356002 Sparacino, Charles M. 2000. Product Chemistry for Coopers Creek Chemical Corp. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356101 Sparacino, Charles M. 2000. Product Chemistry for Koppers Industries, Inc.

Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356102 Sparacino, Charles M. 1999. Product Chemistry for Koppers Industries, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. October 26, 1999.

45356201 Sparacino, Charles M. 2000. Product Chemistry for Reilly Industries, Inc. Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45356202 Sparacino, Charles M. 2000. Product Chemistry for Reilly Industries, Inc. Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45363901 Sparacino, Charles M. 2000. Product Chemistry for Allied Signal Incorporated Creosote P1/P13 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

45363902 Sparacino, Charles M. 2000. Product Chemistry for Allied Signal Incorporated Creosote P2 Storage Stability and Corrosion Characteristics. Unpublished study prepared by Research Triangle Institute. March 31, 2000.

Environmental Fate References

Al-Bashir, B. et al. 1990. Appl. Microbiol. Biotech. 34:414-419.

Bauer, J.E. et al. 1985. Appl. Environ. Microbiol. 81-90.

Bauer, J.E. et al. 1988. Appl. Environ. Microbiol. 1649-1655.

Behymer, T.D. and R.A. Hites. 1985. Environ. Sci. Technol. 19(10):1004-1006.

Bestari, K.T. Jim et al. 1998a. Distribution and composition of polycyclic aromatic hydrocarbons within experimental microcosms treated with liquid creosote. Environ. Toxicol. Chem. 17(12):2359-2368.

Bestari, K.T. Jim et al. 1998b. Distribution and composition of polycyclic aromatic hydrocarbons within experimental microcosms treated with creosote-impregnated douglas

fir pilings. Environ. Toxicol. Chem. 17(12):2369-2377.

Bieri, R.H. et al. 1986. Polycyclic aromatic hydrocarbons in surface sediments from the Elizabeth River subestuary. Int. J. Environ. Anal. Chem. 26:97-113.

Bouwer, E.J. and P.L. McCarty. 1983. Appl. Environ. Microbiol. 45:1295-1299.

Bouwer, E.J. et al. 1996. Annals of New York Academy of Sciences. pp. 103-115.

Bruner, K.A. et al. 1994. J. Great Lakes Research. 20:725-734.

Callahan, M.A. et al. 1979. Water Related Environmental Fate of 129 Priority Pollutants, EPA-440-4-79-029a,b.

Chapman, P.J. et al. 1995. Fossil Fuel Biodegradation: Laboratory Studies. Environ. Health Perspec. 103, Supplemental 5:80-83.

Elder, J.F. and P. Dresler. 1988. Accumulation and bioconcentration of polycyclic aromatic hydrocarbons in a nearshore estuarine environment near a Pensacola (Florida) creosote contamination site. Environmental Pollution. 49:117-132.

Electric Power Research Institute (EPRI). 1992. Document EPRI TR-01000870.

Ehrlich, G.G. et al. 1982. Groundwater. 20(4):703-710.

Flyvberg, J.E. et al. 1993. J. Contamin. Hydrology. 12:133-150.

Fowler, M.G. et al. 1993. Preliminary results from a field experiment investigating the fate of some creosote components in a natural aquifer. Org. Geochem. 22:641-649.

Fukuda, K. et al. 1988. Chemosphere. 17(4):651-659.

Gevao, B. and K.C. Jones. 1998. Kinetics and potential significance of polycyclic aromatic hydrocarbon desorption from creosote-treated wood. Environ. Sci. Tech. 32:640-646.

Gile, J.D. et al. 1982. J. Agric. Food Chem. 30:295-301.

Godsy, E.M. et al. 1992. Ground Water. 30(2):232-242.

Goerlitz, D.F. et al. 1985. Environ. Sci. Tech. 19(10):955-961.

Grbic-Galic, D. et al. 1991. Anaerobic degradation of aromatic hydrocarbons and aerobic degradation of trichloroethylene by subsurface microorganisms. In: Organic Substances

and Sediments in Water, R.A. Baker, Ed., Lewis Publishers, Michigan:239-266.

Harris, J.C. 1982. Handbook of Chemical Property Estimation Methods. W.J. Lyman, W.F. Reehl, D.H. Rosenblatt, Eds. McGraw-Hill Book Company, New York: Chapters 7-8.

Hellou, J.G. et al. 1990. Polycyclic aromatic hydrocarbons in muscle tissue of marine mammals from the Norhtwest Atlantic. Marine Pollution Bulletin. 21(10):469-473.

Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, E.M. Michalenco, Eds. 1991. Handbook of Environmental Degradation Rates, Lewis Publishers, Inc. Chelsea, Michigan, USA.

Hurst, C.J. et al. 1996. Polycyclic aromatic hydrocarbon biodegradation as a function of oxygen tension in contaminated soil. J. Haz. Materials. 51:193-208.

Karthikeyan, R. and A. Bhandari. 2001. Anaerobic biotransformation of aromatic and polycyclic aromatic hydrocarbons in soil microcosms: a review. Journal of Hazardous Substance Research. 3:3-19.

Kirso, U. et al. 1991. Photochemical oxidation of PAH and heteroaromatic analogues in different model conditions. Polycyclic Aromatic Hydrocarbons: Proceedings of the Thirteenth International Symposium on Polynuclear Aromatic Hydrocarbons.

Kuhn, E.P. et al. 1988. Appl. Environ. Microbiol. 54:490:496.

Lindhardt, B. and T.H. Christensen. 1996. Volatilisation of aromatic hydrocarbons from soil: part II, fluxes from coal tar contaminated soils residing below the soil surface. Water Air Soil Pollution. 92:375-389.

Long, E.R. et al. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environ. Management. 19:81-97.

Mabey, W. et al. 1982. Aquatic Fate Process for Organic Priority Pollutants. EPA Report No. 440/4-81-14.

Mattraw, H.C. and B.J. Frank. 1986. Movement and fate of creosote waste in ground water, Pensacola, Florida; U.S. Geological Survey toxic waste -- ground-water contamination program. U.S. Geological Survey Water Supply Paper No.: 2285.

McGroddy, S.E. and J.W. Farrington. Sediment porewater partitioning of polycyclic aromatic hydrocarbons in three cores from Boston Harbor, Massachusetts. Environ. Sci. Technol. 29(6):1542-1550.

Merril, E.G. and T.L. Wade. 1985. Carbonized coal products as a source of aromatic hydrocarbons to sediments from a highly industrialized estuary. Environ. Sci. Technol. 19:597-603.

Middaugh, D.P. et al. 1991. Archives of Environ. Contamin. pp. 244-254.

Mihelcic, J.R. et al. 1988. Appl. Environ. Microbiol. 54:1182-1187.

Mill, T. et al. 1981. Chemosphere. 10:1283-1293.

Miller, M.M. et al. 1985. Environ. Sci. Technol. 19:522-529.

Mississippi State University. 1981. Cooperative Agreement Number 12-156.

Mueller, J.G. et al. 1991. Biodegradation of creosote in contaminated groundwater: chemical and biological assessment. Applied and Environmental Microbiology. 57(5):1277-1285.

Mueller, J.G. et al. 1993. Strategy using bioreactors and specially selected microorganisms for bioremediation of groundwater contaminated with creosote and pentachlorophenol. Environ. Sci. Technol. 27(4):691-698.

National Oceanic and Atmospheric Administration (NOAA). 1988. A Selected Summary of Data of Chemical Contaminants in Sediments Collected During 1984-1987, NTIS.

Neff, J.M. et al. 1976. Marine Biology. Volume 38:279-289.

Padma, T.V. et al. 1999. Toxicity of creosote water-soluble fractions generated from contaminated sediments t the bay mysid. Ecotoxicology and Environmental Safety. 42:171-176.

Politzer, I.R. et al. 1985. Impact on Human Health of Petroleum in the Marine Environment, American Petroleum Institute (API), Washington, DC.

Priddle, M.W. and K.T.B. MacQuarrie. 1994. Dissolution of creosote in groundwater: an experimental and modeling investigation. Journal of Contaminant Hydrology. 15:27-56.

Rutherford, P.M., M.R. Gray, and M.J. Dudas. 1997. Desorption of [^{14}C]naphthalene from bioremediated soils contaminated with creosote compounds. 31:2515-2519.

Sharak Genther, B.R. et al. 1977. Arch. Environ. Contamin. Toxicol. 32:99-105.

Shocken, M.J. et al. 1984. Bacterial oxidation of the polycyclic aromatic hydrocarbons acenaphthene and acenaphthylene. Appl. Environ. Microbiol. 48(1):10-16.

Smith, J.H. et al. 1978. Environmental Pathways of Selected Chemicals in Fresh Water Systems: Part II. Laboratory Studies: 304. EPA-600/7-78-074, USEPA, Athens, Georgia.

Southworth, R.G. 1977. Aquatic Toxicology, ASTM ATP 667. L.L. Marking, R.A. Kimerle, Eds., American Society for Testing and Materials: 359-380, Philadelphia.

Southworth, G.R., J.J. Beauchamp, and P.K. Schmieder, 1978. Water Research. 12:973-977.

Spacie, A. et al. 1983. Uptake, depuration, and biotransformation of anthracene and benzo[a]pyrene in bluegill sunfish. Ecotoxicology and Environmental Safety. 7:330-341.

Stegman, J.J. and J.M. Teal. 1973. Marine Biology. 22:37-44.

Tay K.L. et al. 1992. Sediment bioassessment in Halifax Harbour. Environmental Toxicology and Chemistry. 11:1567-1581.

Verasani, U. et al. 1978. Toxicol. Appl. Pharmacol. 44:277-289.

Villholth, K.G. 1999. Colloid characterization and colloidal phase partitioning of polycyclic aromatic hydrocarbons in two creosote-contaminated aquifers in Denmark. Environ. Sci. Technol. 33:691-699.

Wan, M.T. 1994. Utility right-of-way contaminants: polycyclic aromatic hydrocarbons. J. Environ. Quality. 23:1297-1304.

Webb, D.A. No date available. Creosote, its use as a wood preservative in the railroad transportation industry with environmental considerations. American Wood Preserver's Association.

Wendt, P.H. et al. 1996. Wood preservatives leachates from docks in an estuarine environment. Arch. Environ. Contamin. Toxicol. 31:24-37.

Zepp, R.G. et al. 1980. Assessing the photochemistry of organic pollutants in aquatic environments. In, Dynamics, Exposure and Hazard Assessment of Toxic Chemicals. R. Haque, Ed. Ann Arbor Sci. Publ. Inc., Ann Arbor, Michigan: 69-110.

Ecological Toxicity References

Brooks, K.M. 2004. Polycyclic aromatic hydrocarbon migration from creosote-treated railway ties into ballast and adjacent wetlands. USDA. Forrest Service. Research paper FPL-RP-617

EPA, 2006. "PE5 User's Manual for PRZM EXAMS Modeling Shell, Version 5.0." Environmental Fate and Effects Division. Office of Pesticide Programs, U.S. Environmental Protection Agency. November 15 2006.

Parker, Ronald. 2007. Office of Pesticide Programs. U.S. Environmental Protection Agency. Personal communication with ICF. November.

Anderson, J.W., J.M. Neff, B.A.Cox, H.E.Tatem, and G.M. Hightower. 1974. The effects of oil on estuarine animals: Toxicity, uptake and depuration, respiration. In: F.J. Vernberg and W.B.Vernberg (Eds.), Polution and Pysiology of Mar. Organisms, Academic Press, NY :285-310.

Bestari, K.T. Jim, R.D. Robinson, K.R. Solomon, T.S. Steele, K.E. Day and P.K. Sibley. 1998. Distribution and composition of polycyclic aromatic hydrocarbons within experimental microcosms treated with creosote-impregnated Douglas fir pilings. Environ. Toxicol. Chem. 17(12):2369-2377.

Brooke, L.T. 1991. Results of freshwater exposures with the chemicals atrazine, biphenyl, butachlor, carbaryl, carbazole, dibenzofuran, 3,3'-dichlorobenzidine, dichlorvos, 1,2-epoxyethylbenzene (Styene Oxide), isophorone, isopropalin (truncated). Ctr. For Lake Superior Environ. Stud., U. Of WI-Superior, Superior, WI: 110 p.

Brooke, L.T. 1993. Conducting toxicity tests with freshwater organisms exposed to dieldrin, fluroanthene, and phenanthrene. US EPA contract no 68-C1-0034, work assignment No 5 to R.L. Spehar, Duluth, MN.

Brooks, K.M. 2000. Assessment of the environmental effects associated with wooden bridges preserved with creosote, pentachlorophenol, or chromated copper arsenate. Res. Paper FPL-RP-587. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest products Laboratory. 100 p.

Brooks, K.M. 2004. Polycyclic aromatic hydrocarbon migration from creosote-treated railway ties into ballast and adjacent wetlands. Res. Paper FPL-RP-617. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest products Laboratory. 53 p.

Cairns, M.A., and A.V. Nebeker. 1982. Toxicity of acenaphthene and isophorone to early stages of fathead minnows. Arch. Environ. Contam. Toxicol. 11(6):703-707.

Call, D.J., L.T. Brooke, S.L. Harting, S.H. Poirier, and D.J. McCauley. 1986. Toxicity of phenanthrene to several freshwater species. Final report to Battelle Research Institute,

Columbus, OH. Center for Lake Superior Environmental Studies, University of Wisconsin-Superior. 18 pp.

Canadian Institute of Treated Wood - Western Wood Preservers Institute. 1997. Best Management Practices for the Use of Treated Wood in Aquatic Environments. Developed For Use In Specifying Materials For Use In Aquatic Projects in Canada and the Western United States.

Carls, M.G., S.D. Rice and J.E. Hose. 1999. Sensitivity of fish embryos to weathered crude oil: Part I. Low-level exposure during incubation causes malformations, genetic damage, and mortality in larval Pacific herring (*Clupea pallasi*). Environ. Toxicol. Chem. 18:481-493.

Champlin, D.M. and S.L. Poucher. 1991. Chronic toxicity of fluoranthene to the mysid, *Mysidopsis bahia*. Memorandum to D.J. Hansen, USEPA.

DeWitt, T.H., M.S. Redmnod, J.E. Sewall, and R.C. Swarz. 1992. Development of a chronic sediment toxicity test for marine benthic amphipods. US EPA-Erlin, Newport, OR

De Witt, T.H., R.C. Swartz, and J.O. Lamberson. 1989. Measuring the acute toxicity of estuarine sediments. Environ. Toxicol. Chem. 8:1035-1048.

Diamond, S.A., J.T. Oris, and S.I. Guttman. 1995. Adaptation to fluoranthene exposure in a laboratory population of fathead minnows. Environ. Toxicol. Chem. 14(8):1393-1400.

EPA. 1993a. Sediment Quality Criteria for the Protection of Benthic Organisms: Fluoranthene. USEPA Office of Water and Office of Research and Development, Washington, DC, EPA-822-R-93-012, September, 1993. 102 pp.

EPA. 1993b. Sediment Quality Criteria for the Protection of Benthic Organisms: Phenanthrene. USEPA Office of Water and Office of Research and Development, Washington, DC, EPA-822-R-93-014, September, 1993. 86 pp.

EPA. 1993c. Sediment Quality Criteria for the Protection of Benthic Organisms: Acenaptrene. USEPA Office of Water and Office of Research and Development, Washington, DC, EPA-822-R-93-013, September, 1993. 94 pp.

Gendusa, A.C. 1990. Toxicity of chromium and fluroanthene from aqueous and sediment sources to selected freshwater fish. Ph.D. Thesis, Univ. of North Texas: 138 p.

Goyette, D. and K.M. Brooks. 2001. Addendum Report: Continuation of the Sooke Basin Creosote Evaluation Study (Goyette and Brooks, 1998); Year four – Day 1360 & Day 1540. Prepared for: Creosote Evaluation Steering Committee Regional Program, Report PR00-03, May 12, 2001.

Goyette, D. and K.M. Brooks. 1998. Creosote Evaluation: Phase II Sooke Basin Study - Baseline to 535 Days Post Construction 1995-1996. Prepared for: Creosote Evaluation Steering Committee Regional Program, Report PR98-04, December, 1998.

Hatch, A.C. Jr. 1999. Photo-induced toxicity on PAHs to *Hyalella azteca* and *Chironomus tentans*: Effects of mixture and behavior. Environ.Pollut. 106(2):157-167.

Heintz, R. A., J. W. Short, and S. D. Rice. 1999. Sensitivity of fish embryos to weathered crude oil: Part II. Increased mortality of pink salmon (*Oncorhynchus gorbuscha*) embryos incubating downstream from weathered Exxon Valdez crude oil. Environ. Toxicol. Chem. 18(3): 494-503.

Heitmuller, P.T., T.A. Hollister, and P.R. Parrish. 1981. Acute toxicity of 54 industrial chemicals to sheepshead minnows. Bull. Environ. Contam. Toxicol. 27(5):596-604

Holcombe, G.W., G. Phipps, and J.T. Fiandt. 1983. Toxicity of selected priority pollutants to various aquatic organisms. Ecotoxicol. Environ. Saf. 7(4):400-409.

Horne, J.D., M.A. Swirsky, T.A. Hollister, B.R. Oblad, and J.H. Kennedy. 1983. Aquatic toxicity studies of five priority pollutants. Report No. 4398, EPA Contract No. 68-01-6201, NUS Corp., Houston, TX. 93 pp.

Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, E.M. Michalenco, Eds. 1991. Handbook of Environmental Degradation Rates, Lewis Publishers, Inc. Chelsea, Michigan, USA.

Hutton, K.E. and S.C. Samis. 2000. Guidelines to protect fish habitat from treated wood used in aquatic environments in the Pacific region. Canadian Tech. Rep. Fisheries and Aquatic Sci. 2314. Habitat and Enforcement Branch, Fisheries and Ocenas Canada, Vancouver, BC.

Ingram Jr., L.L, G.D. McGinnis, L.R. Gjovik and G. Roberson. 1982. Migration of creosote and its components from treated piling sections in a marine environment. Proc. Annual Meeting Amer. Wood-Preserver's Asso. 78:120-128.

Kagan, J., E.D. Kagan, I.A. Kagan, and P.A. Kagan. 1987. Do Polycyclic Aromatic Hydrocarbons, Acting as Photosensitizers, Participate in the Toxic Effects of Acid Rain? In: W.J.Cooper and R.G.Zika (Eds.), Photochemistry of Environmental Aquatic Systems, Chapter 14, m.Chem.Soc.Symp.Ser.No.327, Washington, D.C.:191-204

Karrow, N.A., H.J. Boermans, D.G. Dixon, A. Hontella, K.R. Solomon, J.J. Whyte, and N.C. Bols. 1999. Characterizing the immunotoxicity of creosote to rainbow trout (*Oncorhynchus mykiss*): a microcosm study. Aquatic Toxicol. 45:223-239.

Korn, S., D.A. Moles, and S.D. Rice. 1979. Effects of temperature on the median tolerance limit of pink salmon and shrimp exposed to toluene, naphthalene, and Cook Inlet crude oil. Bull.Environ.Contam.Toxicol. 21(4/5):521-525.

Kuhn, A. and S. Lussier. 1987. Phenanthrene results of acute and chronic tests (flow-thru) with *Mysidopsis bahia*. Memorandum to D.J. Hansen, August 3, 1987. 3 pp.

Mayer, F.L., Jr., and M.R. Ellersieck. 1986. Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemicals and 66 Species of Freshwater Animals. Resour. Publ. No. 160, U.S. Dept. of the Interieo, US FWS, Wash., DC:505 p.

McCloskey, J.T., and J.T. Oris. 1991. Effect of water temperature and dissolved oxygen concentration on the photo-induced toxicity of anthracene to juvenile bluegill sunfish (*Lepomis macrochirus*). Aquat.Toxiol. 21:145-156.

Moore, M.N., D.R. Livingston, and J. Widdows. 1989. Hydrocarbons in marine mollusks: biological effects and ecological consequences. In: Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. U. Varanasi, ed., Boca Raton, FL, CRC Press, Inc. 321 pp.

NAS. 1982. Round robin testing of the midge (Tanytarsus): acute and chronic toxicity tests of 2,4,6-trichlorophenol and acenaphthene. Northwestern Aquatic Sciences, Inc., Newport, OR report to USEPA, ERL-Duluth, MN. 66 pp.

Neff, J.M. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment; sources, fates and biological effects. London: Applied Science Publishers LTD. ISPN 0-85334-832-4.

Oris, J. T. and J. P. Giesy. 1987. The photo-induced toxicity of polycyclic aromatic hydrocarbons to larvae of the fathead minnow (*Pimephales promelas*). Chemosphere 16:1395-1404.

Padma, T.V., R.C. Hale, and M.H. Roberts, Jr. 1998. Toxicity of water-soluble fractions derived from whole creosote and creosote-contaminated sediments. Environ. Toxicol. Chem. 17:1606-1610.

Pastorok, R.A., D.C. Peek, J.R. Sampson, and M.A. Jacobson. 1994. Ecological risk assessment for river sediments contaminated by creosote. Environ. Toxicol. Chem. 13:1929-1941.

Pelletier, M.C., R.M. Burgess, K.T. Ho, A. Kuhn, R.A. McKinney, and S.A. Ryba. 1997. Phototoxicity of individual polycyclic aromatic hydrocarbons and petroleum to marine invertebrate larvae and juveniles. Environ. Toxicol. Chem. 16:2190-2199.

Poston, T. 2001. Treated Wood Issues Associated with Overwater Structures in Marine and Freshwater Environments. White Paper submitted by Battelle to the Washington

Department of Fish and Wildlife, Washington Department of Ecology, and Washington Department of Transportation. 96 pp.

Rice, S.D. and R.E. Thomas. 1989. Effedct of pre-treatment exposures of toluene or naphthalene on the tolerance of pink salmon (*Oncorhynchus gorbuscha*) and kelp shrimp (*Eualis suckleyi*). Comp.Biochem.Physiol.C 94:289-293.

Rossi, S. and J.M. Neff. 1978. Toxicity of polynuclear aromatic hydrocarbons to the polychaete *Neanthes arenaceodentata*. Mar. Pollut. Bull. 9:220-223.

SCI. 2005. Creosote-treated wood in aquatic environments: technical review and use recommendations. Prepared for NOAA Fisheries, Santa Rosa, CA by Stratus Consulting Inc., Boulder, CO with Duke University, Durham, NC.

Sibley, P.K., M.L. Harris, K.T. Bestari, T.A. Steele, R.D. Robinson, R.W. Gensemer, K.E. Day, and K.R. Solomon. 2001a. Response of zooplankton communities to liquid creosote in freshwater microcosms. Environ. Toxicol. Chem. 20:394-405.

Sibley, P.K., M.L. Harris, K.T. Bestari, T.A. Steele, R.D. Robinson, R.W. Gensemer, K.E. Day, and K.R. Solomon. 2001b. Response of phytoplankton communities to liquid creosote in freshwater microcosms. Environ. Toxicol. Chem. 20:2785-2793.

Spehar, R.L., S. Poucher, L.T. Brooke, D.J. Hansen, D. Champlin, and D.A. Cox. 1999. Comparative toxicity of fluoranthene to freshwater and saltwater species undr fluorescent and ultraviolet light. Arch. Environ. Contam. Toxicol. 37(4):496-502.

Suedel, B.C., J.H. Rodgers, Jr., and P.A. Clifford. 1993. Bioavailability of fluoranthene in freshwater sediment toxicity tests. Environ. Toxicol. Chem. 12(1):155-165.

Swartz, R.C., D.W. Schuts, T.H. DeWitt, G.R. Ditsworth, and J.O. Lamberson. 1990. Toxicity of fluoranthene in sediment to mrine amphipods: A test of the equilibrium partitioning approach to sediment quality criteria. Environ. Toxicol. Chem. 9(8):1071-1080.

Swartz, R.C. 1999. Consensus sediment quality guidelines for polycyclic aromatic hydrocarbon mixtures. Environ. Toxicol. Chem. 18:780-787.

Swartz, R.C. 1991. Acenaphthene and phenanthrene files. Memorandum to D. J. Hansen, June 26, 1991. 160 pp. [in EPA 1993c]

Tagatz, M.E., G.R. Plaia, C.H. Deans, and E.M. Lores. 1983. Toxicity of creosote-contaminated sediment to field- and laboratory-colonized estuarine benthic communities. Environ. Toxicol. Chem. 2:441-450.

Thursby, G.B., W.J. Berry, and D. Champlin. 1989. Flow-through acute and chronic tests with

acenaphthene using *Mysidopsis bahia*. Memorandum to D.J. Hansen, September 19, 1989. 5 pp. [in EPA 1993c]

Trucco, R.G., F.R. Engelhardt, and B. Stacey. 1983. Toxicity, accumulation, and clearance of aromatic hydrocarbonbs in *Daphnia pulex*. Environ. Pollut. Ser. A 31(3):191-202.

Valle, S., M.A. Panero and L. Shor. 2007. Pollution Prevention and Management Strategies for Polycyclic Aromatic Hydrocarbons in the New York/New Jersey Harbor. New York Academy of Science, New York. 171 pp.

van Brummelen, T.C., B. van Hattum, T. Crommentuijn, and D. F. Kalf. 1998. Bioavailability and Ecotoxicty of PAH. In: Neilson, A. H. (ed.). PAH and Related Compounds – Biology. (Vol. 3-J, The Handbook of Environmental Chemistry). Springer-Verlag, Berlin Heidenberg. pp. 203-263.

Vines, C.A., T. Robbins, F.J. Griffin, and G.N. Cherr. 2000. The effects of diffusible creosote-derived compounds on development in Pacific herring (*Clupea pallasi*). Aquatic Toxicol. 51:225–239.

Vlosky RP. 2006. Statistical Overview of the U.S. Wood Preserving Industry: 2004. Louisiana State University Agricultural Center.

Ward, G.S., P.R. Parrish, and R.A. Rigby. 1981. Early life-stage toxicity tests with a saltwater fish: Effects of eight chemicals on survival, growth, and development of sheepshead minnows. J,. Toxicol. Environ. Health 8(1-2):225-240.

WHO. 2004. Coal Tar Creosote. Concise International Chemical Assessment Document (CICAD) 62, World Health Organization, Geneva, Switzerland. http://www.inchem.org/documents/cicads/cicads/cicad62.htm

Xiao, Y., J. Simonsen, and J.J. Morrell. 2002. Effects of water flow rate and temperature on leaching from creosote-treated wood. Res. Note FPL-RN-0286. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest Products Laboratory. 6 p.