UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
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OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

**MEMORANDUM**

SUBJECT: The Risk Assessment and Science Support Branch/Antimicrobials Division's Science Chapter for the Reregistration Eligibility Decision Document (RED) for Pentachlorophenol (PC Code: 063001, Reregistration Case Number 2505)

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The attached presents the science chapter for the Pentachlorophenol Reregistration Eligibility Decision Document (RED). The document addresses pentachlorophenol as the active ingredient, and does not address the microcontaminants, dioxins and furans. The microcontaminants are not addressed because initial discussions on resources, time constraints and harmonization efforts with the Office of Research and Development/EPA (the lead Office for evaluating dioxin exposures and risks) dictated that assessments would not be conducted on the microcontaminants. However, since that time it has been decided that the human and environmental risks posed by these microcontaminants will be assessed by the Antimicrobials Division in collaboration with ORD. Therefore, a separate document on the microcontaminants will be forthcoming.

**Pentachlorophenol Integrated RED Science Document**

# I. EXECUTIVE SUMMARY

## A. HUMAN RISKS

### 1. OCCUPATIONAL/RESIDENTIAL RISKS

#### a. Handler Risks

An occupational and/or residential exposure risk assessment is required for an active ingredient if (1) certain toxicological criteria are triggered, and (2) there is potential exposure to handlers (i.e., mixers, loaders, applicators, etc.) during use. For pentachlorophenol, both criteria are met. EPA has determined that there are potential exposures to mixers, loaders, applicators, and other handlers during typical use-patterns associated with pentachlorophenol and from use in commercial, industrial, and residential settings. Short and intermediate term exposures (1-7 days and 7 days to several months, respectively) occur through dermal contact and inhalation of crystalline technical grade product and the liquid formulation during mixing/loading and/or application. Acute, sub-chronic and chronic toxicity endpoints related to dermal exposures to pentachlorophenol have been identified. A MOE of greater than 100 for pentachlorophenol is considered to indicate no risk concern for short-term and intermediate-term exposures, and a MOE of greater than 300 for pentachlorophenol is considered to indicate no risk concern for chronic exposures. Based on the best available data and default assumptions used in this RED chapter, **the short- and intermediate-term non-cancer dermal risk estimates for occupational handlers exceed the risk level of concern after maximum risk mitigation measures have been applied** [(e.g., personal protective equipment (PPE), or engineering controls] **for the following scenarios:**

- **mixing/loading formulation at pressure treatment plants (closed mixing using the maximum estimate for the amount of PCP handled per day);**
- **applying liquid formulation at joinery mills - airless spraying; and,**
- **applying grease formulation for groundline remediation of utility poles - brushing.**

**The remainder of the scenarios have MOEs that do not exceed the risk level of concern.** Data gaps prevented the calculation of MOEs for the worker applying liquid formulation at pressure treatment plants - retort workers (helper/switchman).

The chronic non-cancer risk estimates range from $<1$ to 55. **The chronic non-cancer risk estimates for the occupational handler exceed the Agency's level of concern using maximum protective measures (e.g., PPE or engineering controls) for all scenarios.** Several scenarios were not assessed because the exposure was assumed to be less than 180 days/year (i.e., not considered to be a chronic exposure).

**Cancer risk estimates for thirteen (13) exposure scenarios exceed the Agency's level of concern. Cancer risk estimates for occupational handlers using typical application rates do not exceed the Agency's risk level of concern ($>10^{-4}$), using maximum protective measures (e.g., PPE or engineering controls) for only one scenario: mixing/loading crystalline technical grade product to make ready-to-use product.** Data gaps prevented the calculation of (dermal) cancer risks for the scenario, applying liquid formulation at pressure treatment plants - retort workers (helper/switchman).

### b. Postapplication Exposures/Risks

The Agency has determined that there are potential exposure concerns relating to post-application exposure to individuals following pentachlorophenol applications in commercial, industrial, and residential settings. **Short- and intermediate-term dermal occupational post-application risk estimates exceed the Agency's level of concern (i.e., MOE values below 100) for the pressure treatment facility QA/QC inspector exposure scenario.**

**Chronic dermal occupational post-application risk estimates exceed the level of concern for all scenarios.** Note that data gaps exist for certain scenarios such as, pressure treatment facility retort maintenance (e.g., inspecting or performing routine maintenance of the retort); other activities adjacent to a pressure treatment plant (e.g., operating equipment); pressure treatment facility storage yard worker/distributer (e.g., loading and unloading utility poles or treated wood); pressure treatment facility retort maintenance (e.g., inspecting or performing routine maintenance of the retort); other activities adjacent to a pressure treatment plant (e.g., operating equipment); and pressure treatment facility storage yard worker/distributer (e.g., loading and unloading utility poles or treated wood).

**The following scenarios indicate cancer risks which exceed the level of concern, based on dermal and inhalation exposures:**

- **pressure treatment facility yardman;**
- **pressure treatment facility QA/QC inspector;**
- **pressure treatment facility yard worker/ distributor; and**
- **pole installer.**

#### (1a) Residential (Homeowner) Post-application Exposure/Risks

An adult homeowner could potentially become exposed to PCP pressure-treated wood after dermal contact with treated decks, fence posts or utility poles. In addition, with the advent of FQPA, there is more emphasis on assessing residential exposure concerns related to infants, children and other sensitive sub-populations. In the case of a child or infant, PCP exposure may occur as a result of contact with PCP-contaminated soil near the base of a utility pole, or incidental ingestion of PCP residues from hand-to-mouth contact with treated wood in outdoor residential settings.

**Short- and intermediate-term non-cancer risk estimates do not exceed the level of concern for all residential post-application scenarios. The chronic dermal MOE estimate exceeds the level of concern for homeowner contact** (i.e., dermal) **with PCP-contaminated soil** [(e.g., soil contaminated by PCP-treated utility poles) **(child)**] at maximum soil concentrations. **All of the other residential post-application risk estimates do not exceed the level of concern.**

**Cancer risk estimates exceed the level of concern for the following scenario: homeowner contact (i.e., dermal and incidental ingestion) with PCP-contaminated soil [(e.g., soil contaminated by PCP- treated utility poles) (child)]. All the other scenarios were of a limited risk concern for cancer (i.e, do not exceed the level of concern).**

## 2. DIETARY RISK ESTIMATES FROM FOOD SOURCES

### a. Acute Dietary Risk Estimate

The percentage of the acute RfD occupied by acute dietary exposure of non-nursing infants (the highest exposed subpopulation) was 0.39%. This is a conservative estimate, based upon use of maximum detectable residues as reported by FDA. All other population subgroups occupied less than 0.5% of the acute RfD. **Therefore, the acute dietary risk is below the level of concern for any subpopulation.**

### b. Chronic Dietary Risk Estimate

Chronic dietary risk is estimated at 2.2% of the chronic RfD for the highest exposed subpopulation in this scenario (children ages 1-6). All other subpopulations are at 2.2% of the chronic RfD or less. **Thus, chronic dietary risk does not exceed the RfD for any subpopulation and therefore is below the level of concern.**

### c. Carcinogenic Risk Estimate

Carcinogenic risk estimates calculated from residential and dietary exposures to pentachlorophenol show that the estimated cancer risk for adults is less than the level that the Agency usually considers acceptable for negligible cancer risk estimates. **For children contacting soil contaminated with pentachlorophenol, however, the estimated cancer risk ($2.2 \times 10^{-4}$) is above the Agency's level of concern for negligible cancer risk.**

### d. Dietary Risk Estimates from Drinking Water Sources

Estimated Environmental Concentrations (EECs) for surface water (PRZM3-EXAMS have been calculated by the Antimicrobials Division, OPP. Drinking water levels of concern (DWLOCs) for acute and chronic dietary risk from drinking water were calculated. DWLOCs calculated for surface water for pentachlorophenol were 10,465 ppb for adult males and females and 2,990 ppb for children ages 1-6. Using the PRZM3-EXAMS model, available environmental fate data, and conservative assumptions, the estimated environmental concentrations calculated by AD for surface water were less than 1 ppb. EEC's for groundwater were not available for comparison against DWLOC values; however, based on pentachlorophenol's physical/chemical characteristics and available, but limited monitoring data, it is not expected to add significantly to this risk assessment.

## 3. AGGREGATE RISK ESTIMATES

### Acute (Dietary) Aggregate Risk:

The Antimicrobials Division concludes with reasonable certainty that use of pentachlorophenol **does not result in estimates of aggregate acute human health risk that exceed the Agency's level of concern.** The aggregate acute dietary risk estimate includes exposure to pentachlorophenol residues in food and water. Acute percentages of the RfD occupied (from food sources only) at the 95th percentile, were less than 0.5% for non-nursing infants, the highest exposed subpopulation for dietary exposure. Acute DWLOCs calculated for surface water for pentachlorophenol were 10,465 ppb for adult males and females and 2,990 ppb for children. EECs estimated from the PRZM3-EXAMS model were 0.176 ppb. This value is well below the Agency's level of concern. EEC's for groundwater were not available for comparison against DWLOC values; however, based on pentachlorophenol's physical/chemical characteristics and available, but limited monitoring data, pentachlorophenol levels in groundwater are not expected to add significantly to this risk assessment.

### Chronic (Dietary) Aggregate Risk:

**Chronic (dietary) aggregate risk estimates do not exceed the Agency's level of concern.** The chronic aggregate risk assessment for pentachlorophenol includes risk estimates associated with dietary exposure through food, water, and registered residential uses. Exposure to pentachlorophenol through food (based on FDA monitoring data) represents 2.4% of the chronic RfD for the most exposed subpopulation in the U.S. (Children ages 1-6). Exposure to all other groups represents less than 0.5% of the chronic RfD.

AD has calculated drinking water levels of concern (DWLOCs) for chronic exposure to pentachlorophenol for the following four subpopulations: non-nursing infants, $<1$ year old and children ages 1-6 (49 ppb), adult males (174 ppb), and adult females (149 ppb). These subpopulations were selected because they represent the most highly exposed subpopulations

representing males, females, and children and infants, respectively. A conservative estimate of average concentrations of pentachlorophenol in surface water is 0.014 ppb. The estimated average concentration of pentachlorophenol in surface water is less than the Agency's levels of concern for exposure to pentachlorophenol in drinking water as a contribution to chronic aggregate exposure. Estimated average concentrations of pentachlorophenol in ground water were not available for comparison against DWLOC values; however, based on pentachlorophenol's physical/chemical characteristics and available, but limited monitoring data, pentachlorophenol levels in groundwater are not expected to impact this risk assessment significantly.

Therefore, based on the available information, the Agency concludes with reasonable certainty that residues of pentachlorophenol in drinking water (when considered along with exposure from food uses) **would not result in an unacceptable chronic aggregate human health risk estimate at this time.** AD bases this determination on a comparison of estimated concentrations of pentachlorophenol in surface water to back-calculated "levels of concern" for pentachlorophenol in drinking water. The estimate of pentachlorophenol in surface water is derived from a water quality model that uses conservative assumptions (health-protective) regarding the pesticide transport from the point of application to surface water. Because the Agency considers the aggregate risk resulting from multiple exposure pathways associated with a pesticide's uses, levels of concern in drinking water may vary as those uses change. However, no new uses are expected for pentachlorophenol based upon agreements reached as published in the Position Document 4 for Wood Preservatives.

**Cancer Aggregate Risk:**

Combined exposure and risk estimates for one residential exposure scenario plus dietary exposure to pentachlorophenol residues **results in cancer risk estimates that are greater than $10^{-6}$ for children's exposure.** Individual risks associated with dietary exposure and residential exposures must be reduced before the cancer aggregate risk is below the level of concern. Based upon the persistence of pentachlorophenol in treated wood, it is likely that residential uses will need to be removed in order to obtain level of carcinogenic risk that is not of concern to the Agency.

**Short-term Aggregate Risk:**

**Short-term aggregate risk estimates do not exceed the Agency's level of concern.** Aggregate risk estimates associated with short-term risk include exposures to average residues of pentachlorophenol in the diet (food and water) and oral, dermal, and inhalation exposure (1 to 7 days in duration) through the residential application use of pentachlorophenol. The default assumptions used in this aggregate risk estimate are that the homeowner's inhalation exposure to pentachlorophenol is equivalent to an oral exposure (100% absorption of the inhaled residues), while a dermal exposure is equivalent to 40% of an oral exposure, based on available dermal absorption data. Dietary exposures (average residues for food) for the

aggregate assessment were obtained from FDA regulatory monitoring data for the years 1985-1991 and as reported by the California Environmental Protection Agency. The uncertainty factor for the short-term risk assessments is 100. The aggregate risk assessment includes exposures to average concentrations of pentachlorophenol residues in the diet (food + water), and the high-end exposure scenario associated with residential uses assuming oral (child), dermal (child and adult), and inhalation (child and adult) exposures. The resulting aggregate Margins of Exposure (MOEs) for both children and adults are well above the Agency's level of concern. Therefore, AD concludes with reasonable certainty that residues of pentachlorophenol in drinking water (when considered along with exposure from food and residential uses) would not result in an unacceptable short-term aggregate human health risk estimate at this time.

**Intermediate-term Aggregate Risk:**

**Intermediate-term aggregate risk estimates do not exceed the Agency's level of concern.** Aggregate risk estimates associated with intermediate-term risk includes exposures to average residues of pentachlorophenol in the diet (food and water) and residential exposure (7 days to several months in duration) through the residential uses of pentachlorophenol. The default assumptions used in this aggregate risk estimate are that the homeowner's inhalation exposure to pentachlorophenol is equivalent to an oral exposure (100% absorption of the inhaled residues), while a dermal exposure is equivalent to 40% of an oral exposure, using available dermal absorption data. Dietary exposures (average residues for food) for the aggregate assessment were obtained from monitoring data through FDA. The uncertainty factor for the intermediate dietary and residential risk assessments is 100. The aggregate risk assessment includes exposures to average concentrations of pentachlorophenol residues in the diet and the high-end exposure estimates through residential uses, assuming oral (child), dermal (child and adult), and inhalation (child and adult) exposures. The resulting aggregate Margins of Exposure for both children and adults are well above the Agency's level of concern. Therefore, AD concludes with reasonable certainty that residues of pentachlorophenol in drinking water (when considered along with exposure from food and residential uses) would not result in an unacceptable intermediate-term aggregate human health risk estimate at this time.

**Chronic Aggregate Risk:**

**Chronic aggregate risk estimates do not exceed the Agency's level of concern for adults, but do exceed the Agency's level of concern for children.** Aggregate risk estimates associated with chronic risk includes exposures to average residues of pentachlorophenol in the diet (food and water) and residential exposure (greater than 6 months continuous exposure) through the residential uses of pentachlorophenol. The default assumptions used in this aggregate risk estimate are that the homeowner's inhalation exposure to pentachlorophenol is equivalent to an oral exposure (100% absorption of the inhaled residues), while a dermal exposure is equivalent to 40% of an oral exposure, using available dermal absorption data.

Dietary exposures (average residues for food) for the aggregate assessment were obtained from monitoring data through FDA. The uncertainty factor for the chronic dietary and residential risk assessments is 300. The aggregate risk assessment includes exposures to average concentrations of pentachlorophenol residues in the diet and the high-end exposure estimates through residential uses, assuming oral (child), dermal (child and adult), and inhalation (child and adult) exposures. The resulting aggregate Margins of Exposure for adults are well above AD's level of concern, but the resulting aggregate MOE's for children's exposure are unacceptable. Therefore, the Agency concludes with reasonable certainty that residues of pentachlorophenol in drinking water (when considered along with exposure from food and residential uses) would not result in an unacceptable chronic aggregate human health risk estimate for adults, but that residues of pentachlorophenol in drinking water (when considered along with exposure from food and residential uses) pose an unacceptable chronic risk to children.

## B. ENVIRONMENTAL RISK

### 1. Environmental Fate

#### a. Abiotic degradation of Pentachlorophenol:

Pentachlorophenol does not hydrolyze in acidic, neutral or basic conditions and can therefore be a persistent molecule in abiotic aqueous conditions. It does, however, photodecompose under ultraviolet light in water quickly with a half-life of 3.5 hours at pH 7.3 and 100 hours at pH 3.3. Some of the identifiable degradates are: tetrachlorocathecol, tetrachlororesorcinol, tetrachlorohydroquinone, chloranil, hydroxyquinones, 2,3 dichloromaleic acid which slowly decompose to carbon dioxide, chloride ion and other organic fragments which are hard to identify. In the vapor phase, pentachlorophenol is moderately stable with a photodegradation half life of about 37 days under simulated sunlight. 2,3,5,6 tetrachlorphenol was identified as a major photoproduct. Pentachlorophenol showed no photolytic breakdown tendency on soil surface (sandy loam soil) under dark conditions. However, in the presence of light, it is moderately stable with an estimated half life of about 38 days.

#### b. Biotic degradation of Pentachlorophenol:

Pentachlorophenol metabolizes rapidly under aerobic aquatic conditions and has a half life of less than five days. Under anaerobic conditions, it metabolizes a little more slowly with a half life of about 34 days. (These results were obtained with blue sandy loam soil.) It is, therefore, not a persistent substance in natural waters. Under dark but aerobic conditions, soil (sandy loam) metabolizes pentachlorophenol slowly with a half life of 63 days. Tri and tetrachlorophenols were identified as degradates. Adsorption/desorption studies on four soils (Georgia sandy loam, Ohio Clay loam, California sandy loam and Nebraska Blue sandy loam) showed that pentachlorophenol binds moderately to strongly to the soils. $K_{OC}$ values revealed that pentachlorophenol to soil binding is tight with Georgia, Ohio and Nebraska sands and

moderately so with the California sand.

### c. Bioaccumulation

Bioaccumulation studies on bluegill sunfish revealed that when exposed to 2.5 $\mu$g/L pentachlorophenol for 28 days, the bioconcentration factors (BCF) were 190X, 740X and 490X for edible tissue, non-edible tissue and whole body tissue, respectively. Depuration for the whole body was one day and 98% is depurated within fourteen days. No metabolites were found in these studies.

### d. Leaching from treated wood

One study on three Southern pine poles soaked with pentachlorophenol was conducted and then exposed to different solutions: unbuffered water, buffered water at pH 5, 7 and 9, sea water, sea water in 0.10N HCl and 0.10N HCl. The average leach rate varied between $1.76x10^{4}$ to $6.33x10^{-3}$ mg pentachlorophenol/kg leachate/$in^2$ surface area/day. The leaching peaks in one day for most of the solutions except the solution at pH 9 for which the leaching peaked in 3 days.

Pentachlorophenol has a great tendency to attach to the organic contend of sediments (high $K_{OC}$) and is found to bind more strongly in acidic soils but is mobile in neutral to basic conditions. Pentachlorophenol can be transported to surface waters and potentially can be a hazard to drinking water.

## 2. Water Resources

### a. Modeling

The PRZM3/EXAMS model was used to predict estimated environmental concentrations (EECs) for pentachlorophenol leached out of the surface of utility poles. Pentachlorophenol concentrations were estimated for the water column, pore water, benthic sediment, and benthic organisms, with instantaneous, 96 hours, 21 days, 60 days, 90 days, and yearly values predicted for each environmental compartment. The PRZM3 model simulated the data for 36 years of the weather data. However, during the 36 years of simulation, Pentachlorophenol was applied only once when the treated pole was installed into the ground. The application rate was equal to the maximum amount of the leaching rate from the treated pole as described in the Environmental Fate section of the science chapter of this document. This assumption is very conservative because all of the chemical will not leach out of the pole at once and leaching will be gradual through the years. The data reported in the table are not the average for the duration of the simulation run, but from the first year of the simulation during which the model assumes the chemical was applied to the soil. The results for the EECs simulated by PRZM3-EXAMS models are shown in the Modeling Results Table in the Water Resources section of the science chapter of this document. Benthic sediment had the highest values, ranging from

7.368 $\mu$g/kg (instantaneous) to 1.497 $\mu$g/kg (yearly). Water column EECs ranged from an instantaneous value of 0.176 ppb to a yearly value of 0.014 ppb. The EECs were used to calculate risk quotients for human dietary risks from drinking water sources, as well as for acute and chronic risks to fish and aquatic invertebrates.

Pentachlorophenol is not mobile and has low persistence in the environment. It dissipates through photo-degradation. After leaching out of the utility pole surface and reaching the soil, pentachlorophenol is adsorbed to the soil particles. Pentachlorophenol has a very low solubility. Because of its affinity for soil particles, pentachlorophenol will not move downward into the ground water. Pentachlorophenol moves into surface waters adsorbed to the soil particles through run-off. Therefore, pentachlorophenol levels in ground and drinking water will be minimal.

### 3. Ecological Effects

#### a. Toxicity Summary

The available acute toxicity data on the TGAI indicate that pentachlorophenol is practically nontoxic to moderately toxic to birds ($LD_{50}$ = 380-627 mg/kg; $LC_{50}$ = 3400-5581 ppm), and moderately toxic to small mammals ($LD_{50}$ = 155 mg/kg, male rat). The data indicate that pentachlorophenol is highly toxic to very highly toxic to freshwater organisms ($LC_{50}$ = 0.015-0.600 ppm), and highly toxic to very highly toxic to estuarine/marine organisms ($LC_{50}$ = 0.048-0.240 ppm). Chronic toxicity studies establish the following maximum acceptable toxicant concentration (MATC) values: 0.014 ppm for freshwater fish; 0.240 ppm for freshwater invertebrates, and 0.064 ppm for estuarine/marine fish. Chronic toxicity studies were not available for birds and small mammals.

#### b. Risk Assessment:

**Birds:** Risk quotients cannot be calculated for birds from the use of pentachlorophenol as a wood preservative, because data are not sufficient to provide detailed terrestrial exposure values. However, risks to birds (acute and chronic) are not expected from the utility pole use, because data are sufficient to indicate low potential for exposure. Other uses of pentachlorophenol, such as the pier/piling use, could potentially result in exposure to certain bird species, such as waterfowl, that feed in aquatic habitats surrounding the treated wood; however, pentachlorophenol is unlikely to pose an acute risk to these species because of the low dietary toxicity of pentachlorophenol to birds. Chronic risk from this route of exposure cannot be determined due to lack of avian reproduction data.

**Mammals:** Risk quotients cannot be calculated for mammals from the use of pentachlorophenol as a wood preservative, because data are not sufficient to provide detailed terrestrial exposure values. However, risks to mammals (acute and chronic) are not expected because data are sufficient to indicate low potential for exposure.

**Nontarget Insects:** Risk quotients for nontarget insects were not calculated for pentachlorophenol due to the lack of data for use in estimating exposure. Wood treated with pentachlorophenol can be used in the construction of beehives. Pentachlorophenol used in this manner has been shown to cause adverse effects to bees, and has also been found in both honey and wax from the treated hives (Kalnins and Delroy, 1984).

**Aquatic Organisms:** Acute and chronic levels of concern (LOCs) are not exceeded for any aquatic organism, despite the very high acute and chronic toxicity of pentachlorophenol to the species tested.

**Plants:** Based on Tier II aquatic plant testing, acute LOCs are not exceeded for vascular and non-vascular aquatic plants from pentachlorophenol-treated utility poles.

## II. BACKGROUND

U.S. EPA's Office of Pesticide Programs (OPP), as required by the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA/1988) , and amended by the Food Quality Protection Act (FQPA) of 1996, is directed to implement procedures for the registration and reregistration of pesticides. OPP's Antimicrobials Division (AD) has responsibility for managing Agency regulations for antimicrobial chemicals, including wood preservatives, such as pentachlorophenol.

Note that as part of the reassessment of the wood preservative chemicals, North American Free Trade Agreement (NAFTA) harmonization efforts are underway between US EPA and Health Canada's Pest Management Regulatory Agency (PMRA). Science staff from AD and PMRA are collaborating on data sharing and in conducting joint data reviews in compliance with NAFTA.

The Antimicrobials Division has evaluated the database for pentachlorophenol and determined that the data are adequate to support reregistration. However, after evaluation of the existing database, data gaps have been identified. The following list captures the data deficiencies noted by discipline.

**Required Data:**

### 1. Chemistry Studies

a. Product Chemistry

***There are no outstanding data requirements for product chemistry in accordance with the proposed 40 CFR Part 158 Subpart W Data Requirements for Antimicrobial Chemicals.*** The Risk Assessment and Science Support Branch (RASSB) has no objections to the reregistration of pentachlorophenol with respect to product chemistry data requirements.

b. Residue Chemistry

***There are no pending data requirements for residue chemistry.*** As a result of the EPA Position Document 2/3 (1981), uses that were likely to contaminate food, feed or drinking water were canceled or denied registration. Even though there are no registered food uses for pentachlorophenol, PCP has been detected in milk, pears, and pork as indicated by the Food and Drug Administration's dietary monitoring data. The use of PCP on utility poles may result in surface water runoff from treated utility poles and may be a contributing source of PCP and/or its metabolites in drinking water. An estimation of this potential exposure was conducted using the PRZM3-EXAMS model. Based on the concentrations derived from the model, no residue data are required. RASSB has no objections to the reregistration of PCP with respect to residue chemistry data requirements.

## 2. Toxicology Studies

The toxicological database for pentachlorophenol is adequate and will support reregistration eligibility. ***However, an acute inhalation toxicity study (Guideline number 870.1300, formerly 81-3) is required for the technical test material to assess acute inhalation hazards.*** A waiver was previously granted for a 90-day inhalation toxicity study, but the issue of acute inhalation toxicity still needs to be addressed. ***The 2-generation reproduction study in rats is unacceptable and does not currently satisfy the guideline requirement OPPTS 870.3800, formerly 83-4.*** The study may be upgraded upon receipt and review of the range-finding toxicity study. Immunotoxicity data are required on the pure test chemical in in vitro human studies to provide insight regarding the potential for immunotoxicity of pentachlorophenol in humans.

## 3. Occupational/Residential Exposure Studies

There are data gaps for the human exposure database for pentachlorophenol. In 1991 the Agency issued a Data Call-In which requested applicator exposure monitoring data to estimate dermal and inhalation exposures at outdoor sites (formerly guideline numbers 231 and 232) and estimation of dermal and inhalation exposures at indoor sites (formerly guideline numbers 233 and 234). In subsequent correspondence, the Agency granted the registrant's request to waive the data required for guideline numbers 233 and 234. The data requirements to address dermal and inhalation exposures at outdoor sites are still outstanding. Specifically, there are ***no acceptable chemical specific or surrogate data to estimate worker exposures while applying the liquid formulation at pressure treatment plants - retort workers (helper/switchman) (Guideline numbers 875.2400; 875.2500).***

In addition, to estimate potential exposures to pentachlorophenol after the wood has been treated, postapplication dermal and inhalation exposure **data are required to support guideline numbers 875.2300, 875.2400 and 875.2500.** Specifically, there are no data available to estimate human exposures for the following post-application exposure scenarios:

- ***pressure treatment facility retort maintenance (e.g., inspecting or performing routine maintenance of the retort)***
- ***other activities adjacent to a pressure treatment plant (e.g., operating equipment)***
- ***pressure treatment facility storage yard worker/distributer (e.g., loading and unloading utility poles or treated wood)***

## 4. Ecological Effects Studies

Important data gaps for pentachlorophenol are in the areas of chronic testing with aquatic invertebrates and sediment acute toxicity testing. ***The actual requirements which have not been fulfilled are the estuarine/marine invertebrate life cycle test and the whole sediment acute tests for freshwater and estuarine/marine invertebrates (Guideline numbers 73-1/850.1740; 72-4/850.1300).***

Data from sediment toxicity testing is important to this assessment because of the nature of pentachlorophenol. Fate characteristics indicate that this chemical may persist in the environment and will have a tendency to bind to sediment. Because of these factors, testing which is confined to the water column does not provide a complete picture of the potential risk to aquatic organisms. Data from sediment toxicity testing would reduce the uncertainty in the aquatic invertebrate risk assessment.

In addition to the above, it should be noted that there are a number of requirements in Part 158 that have been designated as reserved. In other words, studies may be required in addition to those discussed above. The 5 aquatic acute studies using the typical pentachlorophenol end use product (as determined by the Agency in 1995) are reserved, pending receipt of additional information on the degradates of pentachlorophenol.

Listed below are the data requirements which are reserved:

- 72-1(a)/850.1075 Acute Fish Toxicity, Rainbow Trout – Reserved[*1]
- 72-1(a)/850.1075 Acute Fish Toxicity, Bluegill – Reserved[*1]
- 72-3/850.1025; 850.1035; 850.1045; 850.1055; 850.1075 Acute Estuarine/Marine Organisms Toxicity – Reserved [*1]
- 72-6/850.1710; 850.1730; 85.1850 Aquatic Organism Bioavailability/Biomagnification/Toxicity Tests – Reserved [*2]
- 73-1/850.1735 Whole Sediment, Acute Freshwater Invertebrates – Reserved[*2]
- 73-2/850.1740 Whole Sediment, Acute Marine Invertebrates – Reserved[*2]
- 73-3 Acute Pore Water, Fish and Invertebrates – Reserved[*2]
- 74-1 Whole Sediment, Chronic Invertebrates – Reserved[*2]

[*1] Deferred pending review of special leaching study.
[*2] These studies might be required under the new guidelines established by the Agency in 1998.

However, because they were not required prior to the review of the PCP RED, the Agency has placed a reserve on these data until further notice.

## 5. Environmental Fate Studies

The environmental fate data requirements to support the reregistration of pentachlorophenol have been satisfied as typically required for agricultural pesticides. However, since the use pattern of PCP as a wood preservative is different from most agricultural chemicals which are more likely to come in contact with or be incorporated into the soil, there are questions that were not addressed by the normal data requirements.

The main unanswered question is to what extent PCP and its microcontaminants are depleted from treated wood (utility poles are the main use) and the levels of exposure to the soil, water and air in the vicinity of the treated poles. Studies were not conducted to measure the levels of PCP and its microcontaminants in treated utility poles at specified time intervals including when they were placed in service. ***To address this concern a special study is required – aqueous availability of the preservative from wood.***

## 6. Product Labeling Requirements

To be completed after risk mitigation discussions with the registrants (Pentachlorophenol Task Force).

## III. USE CHARACTERIZATION/PROFILE

### Registered Use Sites

Pentachlorophenol is registered for use as a wood preservative in the United States. Prior to the 1987 Federal Register Notice (Vol.52, No. 13) which canceled and restricted certain nonwood uses of PCP, pentachlorophenol was registered for use as a herbicide, defoliant, mossicide, and as a disinfectant. Pentachlorophenol is currently registered for wood protection treatments to existing buildings or parts of buildings, unseasoned forest products, seasoned forest products, wooden containers for growing plants, aquatic structures and items, non-food/feed wooden containers, forest products by pressure, and finished wood products. Examples of registered use sites include, utility poles, crossarms, crossties, timber, lumber, fencing, porches, posts, shingles, groundline building components, steps, walkways, laminated beams, pilings, piers, docks, bridges, and trusses.

Indoor applications of pentachlorophenol are prohibited in accordance with the restrictions indicated in the U.S. EPA Position Document 4 for Wood Preservative Pesticides: Creosote, Pentachlorophenol and Inorganic Arsenicals (1984, amended 1986). PCP may not be applied in interiors. The use of PCP to treat wood intended for use in interiors is prohibited, except for a few low exposure uses (i.e., except those support structures which are in contact with the soil in barns, stables and similar sites and which are subject to decay or insect infestation). PCP is a restricted use pesticide for sale to and use by certified applicators only.

The primary registrants are IBC Manufacturing Co., Vulcan Materials, Co., KMG-Bernuth, Inc., Wood Protection Products, Inc. and Osmose, Inc.

### Formulation types and percent active ingredient

There are 19 registered end-use products with active ingredient percentages ranging from 4.8 percent to 48 percent. There are 3 registered technical grade products and 3 registered manufacturing use products. Below is a list of the 25 products registered with the U.S. EPA (for wood preservation uses) which contain pentachlorophenol as the active ingredient in the formulations:

**Technical Grade Active Ingredient (TGAI):**

| | |
|---|---|
| Crystalline (Solid) | 96.00% |

**Manufacturing-Use Product (MUP):**

| | |
|---|---|
| (with additional use as an EUP) | |
| Soluble Concentrate (Liquid) | 37.05 to 39.40% |
| Emulsifiable Concentrate (Liquid) | 40.03% |

**End-Use Product (EUP):**

| | |
|---|---|
| Soluble Concentrate (Liquid) | 24.58 to 48.00% |
| Emulsifiable Concentrate (Liquid) | 24.03% |
| Ready-to-Use Compound (Grease) | 10.21 to 14.40% |
| Ready-to-Use Compound (Impregnated Material/Grease) | 10.23% |
| Ready-to-Use Solution (Liquid) | 4.80 to 10.00% |

**Type of Target Pests**

INVERTEBRATES

(Insects, Miscellaneous Invertebrates, and Related Organisms.)

- Insects
- Wood Destroying Insects
- Termites
- Subterranean Termites
- Drywood Termites
- Powderpost Beetles
- Lyctus Powderpost Beetles
- Carpenter Ants

PLANT PATHOGENIC ORGANISMS

(Bacteria, Fungi, and Other Fouling Organisms.)

- Blue Stain (Ceratocystis)
- Wood Mold
- Wood Stain Fungi
- Wood Rot/decay Fungi
- Wood Rot/decay Organisms

**Types and Rates of Applications**

The following covers only those application rates which are specified on the product labeling:

**Terrestrial Non-Food Crop**

Wood Pressure Treatment to Forest Products:

Nonsoil Contact Nonfumigation - maximum of 4 lb active ingredient in 10.3 gal of solvent and 9.5 lb active ingredient in 10 gal of solvent.

Soil Contact Nonfumigation - maximum of 4.75 lb active ingredient per 1000 sq ft and 9.5 lb active ingredient in 10 gal of solvent.

Vacuum Pressure System Wood Protection Treatment - maximum of 4.123 lb active ingredient in 10 gal of solvent.

Wood Protection Treatment to Buildings/Finished Wood Products Outdoor:

Soaking Treatment (Bath/Dip Tank) - maximum of 3.648 lb active ingredient in 10 gal of solvent.

Wood Surface Treatment (Brush, Spray, Swab) - maximum of 3.648 lb active ingredient in 10 gal of solvent.

Wood Protection Treatment to Forest Products (Seasoned):

Brush-on Treatment - maximum of 2.062 lb active ingredient per 1000 sq ft and 6.2208 lb active ingredient per pole.

Low Pressure Injection Treatment - maximum of 3.825 lb active ingredient per pole.

Nonsoil Contact Nonfumigation - maximum of 4.75 lb active ingredient per 1000 sq ft and 9.5 lb active ingredient in 10 gal of solvent.

Soil Contact Nonfumigation - maximum of 4.75 lb active ingredient per 1000 sq ft and 9.5 lb active ingredient in 10 gal of solvent.

Bandage Soak Treatment - maximum of 3.825 lb active ingredient per pole.

Soaking Treatment (Bath/Dip Tank) - maximum of 2.062 lb active ingredient per 1000 sq ft and 3.648 lb active ingredient in 10 gal of solvent.

Sponge-on/Swab Treatment - maximum of 2.062 lb of active ingredient per 1000 sq ft.

Spray Treatment - maximum of 6.2208 lb active ingredient per pole.

Wood Surface Treatment (Bandage) - maximum of 2.625 lb active ingredient per pole.

Wood Surface Treatment (Brush, Sprayer) - maximum of 3.648 lb active ingredient in 10 gal of solvent and 3.825 lb active ingredient per pole.

Wood Surface Treatment (Swab) - maximum of 3.648 lb active ingredient in 10 gal of solvent.

Wood Protection Treatment to Forest Products (Unseasoned): Not Specified

Dip, Nonsoil Contact Nonfumigation, Soak, and Wood Surface Treatment Application Rates are unspecified on the product labeling.

**Aquatic Non-Food Outdoor**

Wood Protection Treatment to Aquatic Structures/Items:

Brush-on Treatment - maximum of 2.062 lb active ingredient per 1000 sq ft and 6.2208 lb active ingredient per pole.

Soaking Treatment (Bath/Dip Tank) - maximum of 2.062 lb active ingredient per 1000 sq ft..

Soil Contact Nonfumigation - maximum of 4.75 lb active ingredient per 1000 sq ft.

Sponge-on/Swab Treatment - maximum of 2.062 lb of active ingredient per 1000 sq ft.

Spray Treatment - maximum of 6.2208 lb active ingredient per pole.

**Outdoor Residential**

Wood Protection Treatment to Buildings/Finished Wood Products Outdoor:

Brush-on Treatment - maximum of 2.062 lb active ingredient per 1000 sq ft and 6.2208 lb active ingredient per pole.

Low Pressure Injection Treatment - maximum of 2.625 lb active ingredient per pole.

Nonsoil Contact Nonfumigation - maximum of 4.75 lb active ingredient per 1000 sq ft.

Soaking Treatment (Bath/Dip Tank) - maximum of 2.062 lb active ingredient per 1000 sq ft..

Sponge-on/Swab Treatment - maximum of 2.062 lb of active ingredient

per 1000 sq ft.

Spray Treatment - maximum of 6.2208 lb active ingredient per pole.

Wood Surface Treatment (Bandage, Brush, Sprayer) - maximum of 2.625 lb active ingredient per pole.

Wood Protection Treatment to Forest Products (Seasoned): Not Specified

Dip, Soak, and Wood Surface Treatment Application Rates are unspecified on the product labeling.

**Types of Treatment**

Wood Surface Treatment (Brush-on Treatment, Sponge-on/Swabbing, Spraying); Soil Contact Nonfumigation (Wrapping & Bandaging); Nonsoil Contact Nonfumigation; Low Pressure Injection; Pressure Treatment; Thermal Treatment (Hot & Cold Bath); Vacuum Treatment; Dip Treatment; Cold Soaking; Extended Soaking

**Equipment**

Bandage; Pad; Dip Tank; Tank; Bath; Pressure Treating Vessel; Cylinder; Low Pressure Injection Equipment; Caulk Gun; Brush; Sprayer; Swab

**Timing**

Prior to, or after end use of wood, but prior to significant pest damage (not specifically stated on label); when needed.

# IV. PRODUCT CHEMISTRY/ENVIRONMENTAL FATE/MODELING

## A. Physical and Chemical Properties Assessment

### 1. Identification of Active Ingredient

| | |
|---|---|
| **Chemical Name:** | Pentachlorophenol |
| **Chemical Family:** | Chlorophenols |
| **CAS Registry Number:** | 87-86-5 |
| **OPP Chemical Code:** | 63001 |
| **Empirical Formula:** | $C_6 H Cl_5 O$ |
| **Manufacturer:** | Vulcan Chemicals, Inc. |

### 2. Manufacturing Process

A Batch Process is used to manufacture pentachlorophenol in the USA. This process consists of multistage chlorination. Three hundred and fifty gallons of phenol are pumped into a reactor that already contains 850 gallons of partially chlorinated phenol. Chlorine gas is then injected into the reactor. This stage produces di- and trichlorophenols. As the process is highly exothermic, temperature of the reactor is kept between 30-50 ° C and only slowly increased to 65-95 ° C. This reaction mixture is transferred to another reactor and before additional chlorine is injected a Friedal Crafts catalyst ( $AlCl_3$) is added. Starting temperature of this reaction mixture is 90-100 ° C and under controlled conditions is increased to 130 ° C and again slowly brought down to 82 ° C. Technical grade Pentachlorophenol produced by this method is molded into large ( one ton) blocks.

The impurities or microcontaminants are present in the TGAI and consist of dioxins and furans. and these are structurally and chemically a variety of polycyclic compounds. Commonly found microcontaminants in the pentachlorophenol-treated wood are listed in the Product Chemistry Assessment Section.

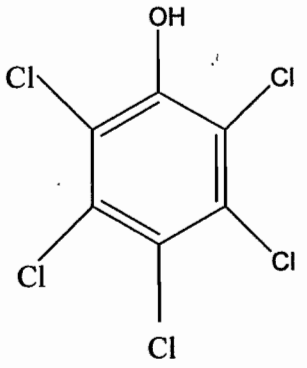
### 3. Physical and Chemical Properties

**Technical Grade Active Ingredient (TGAI):**

| | |
|---|---|
| **Chemical Name:** | Pentachlorophenol |
| **Molecular Weight:** | 266.34 g $mol^{-1}$ |
| **Color:** | Light brown to tan (Pure pentachlorophenol, however, is white needle-like crystals) |
| **Empirical Formula:** | $C_6 H Cl_5 O$ |
| **Vapor pressure:** | 0.00415 Pa (1.1E-4 Torr at 25 ° C) |
| **$K_{OW}$:** | A variety of values are reported: 3.81, 5.86, 5.01 |

| | |
|---|---|
| **Log $K_{ow}$** | 5.05 (at pH 5.1) |
| **$K_{oc}$** | 2430 ( Georgia, sandy loam) |
| | 3420 (Ohio, clay loam) |
| | 706 ( California, sandy loam) |
| | 1410 (Nebraska, blue sandy loam) |
| **Solubility** | At 20 ° C, |
| | Water: 14 ppm, |
| | Methanol: 1.7 g/g |
| | Benzene: 0.014 g/g |
| **Odor:** | Phenolic |
| **Physical State:** | Solid |
| **Melting Point:** | 190-191 ° C |
| **Boiling Point:** | 309 ° C ( decomposes) |
| **Density:** | 1.978 g/ml |
| **Dissociation Constant $K_a$:** | $1.6 \times 10^{-14}$ |
| **pH:** | 4.99 |
| **Stability:** | Sublimes at 54 $\pm$ 2 ° C |

## 4. Structural Formula




## 5. Microcontaminants

The Science Advisory Board of the US EPA's Environmental Health Advisory Committee put together a Report of the AD HOC Study Group on Pentachlorophenol Contaminants (Dec. 29, 1978) and it states that: ---" although 2,3,7,8 -TCDD has been known

to be extremely toxic, TCDD has not been identified as a contaminant of PCP manufactured in the US. The same document reports that for a TGAI pentachlorophenol analysis of Dowicide EC-7 (88.4% pentachlorophenol), the total $H_xCDD$ was 4 ppm in a 1978 sample while it was 9 ppm in a 1975 sample.

The National Toxicology Program (NTP) in 1987 conducted a two year Toxicology and Carcinogenesis Studies of Two Pentachlorophenol Technical-Grade Mixtures.

The two technical-grade mixtures of pentachlorophenol were taken from 1980's manufactured TGAI pentachlorophenol. The analysis reported was:

| $H_xCDD$ | Hexachlorobenzene (HCB) | TCDD |
|---|---|---|
| 10.1 ppm | 50 ppm | Not determined |

US EPA published a Notice in the Federal Register of Jan. 2, 1987 in which it was stated: " --- With the reduction of $H_xCDD$ levels and the specified limits of other contaminants---the Agency believes that the benefits of use of pentachlorophenol for wood preservation will exceed the risks of such use."

However, based on Agency concerns about the presence of other chlorinated dioxins [such as Polychlorinated dibenzo-p-dioxins (PCDDs) and Polychlorinated dibenzofuran (PCDBF)] in pentachlorophenol, an agreement was made between the wood preservative industry and the Agency in 1986 that:

1 Maximum $H_xCDD$ per batch released for shipment should not exceed 4 ppm;

2. Maximum average $H_xCDD$ of all batches sold during the month = 2 ppm;

3. Any detectable TCDD at a limit of detection should not be higher than one ppb and

4. There should be no increase in the Hexachlorobenzene (HCB) level in the TGAI production.

The following table provides a list of the microcontaminants in pentachlorophenol and Selective Physical Properties of the microcontaminants (pentachlorophenol included for comparison):

**TABLE IV-1: Pentachlorophenol microcontaminants and some of their chemical properties.**

| *General Chemical Formula* | Common Name | Vapor Pressure (Pa) | Water Solubility ($g/m^3$) | Henry's Law Constant($Pa-m^3/mol$) | *Log $K_{OW}$* |
|---|---|---|---|---|---|
| $C_6HCl_5O$ | Pentachloro phenol | 0.00415 | 14 | 0.079 | 5.05 |
| 2,3,4,7,8-PCDD | Pentachloro dibenzo-p-dioxin | - | - | - | - |
| 2,3,7,8-$H_x$CDD | Hexachloro dibenzo-p-dioxin | $5.1x10^{-9}$ | 0.00442 | 1.084 | 7.8 |
| 2,3,7,8-$H_p$CDD | Heptachloro dibenzo-p-dioxin | $7.5x10^{-10}$ | 0.0024 | 1.273 | 8.0 |
| 2,3,4,7,8-PCDF | Pentachloro dibenzo furan | | | | |
| 2,3,7,8-$H_x$CDF | Hexachloro dibenzo furan | $3.2x10^{-8}$ | 0.00825 | 1.454 | 7.0 |
| 2,3,7,8-$H_p$CDF | Heptachloro dibenzo furan | $4.7x10^{-9}$ | 0.00135 | 1.425 | 7.4 |
| OCDF | Octachloro dibenzo furan | $5.0x10^{-8}$ | 0.00116 | 0.191 | 8.0 |
| OCDD | Octachloro dibenzo-p-dioxin | $1.1x10^{-10}$ | 0.000074 | 0.684 | 8.2 |

Table IV-2 shows the concentrations of microcontaminants in more recent samples of pentachlorophenol.
The 1997/1998 production figures are per pentachlorophenol batch release for shipment. This information was provided by the registrants.

**Table IV-2[1]: Concentrations of microcontaminants in recent samples of pentachlorophenol.**

| Production Month | Max. $H_xCDD$ Measured | Max. Average $H_xCDD$[2] (weighted) | TCDD[3] | Hexachlorobenzene (HCB) |
|---|---|---|---|---|
| Nov. 1997 | 1.97 ppm | 1.29 ppm | N.D. | 52.4 ppm |
| Dec. 1997 | 1.48 ppm | 1.69 ppm | N.D. | 46.3 ppm |
| Jan. 1998 | 1.85 ppm | 1.36 ppm | N.D. | 51.5 ppm |
| Feb. 1998 | 2.06 ppm | 1.40 ppm | N.D. | 47.8 ppm |
| Mar. 1998 | 1.72 ppm | 1.36 ppm | N.D. | 67.4 ppm |
| Apr. 1998 | 1.72 ppm | 1.30 ppm | N.D. | 52.0 ppm |
| May. 1998 | 1.91 ppm | 1.41 ppm | N.D. | 58.0 ppm |
| Jun. 1998 | 1.79 ppm | 1.35 ppm | N.D. | 39.0 ppm |
| Jul. 1998 | 1.49 ppm | 1.29 ppm | N.D. | 54.6 ppm |
| Aug.1998 | 1.98 ppm | 1.23 ppm | N.D. | 57.1 ppm |

Notes:

1. Each batch is randomly selected near mid-month.
2. The weighing factor represents the weight of the batch released for shipment divided by the total weight of technical pentachlorophenol released for shipment.
3. N.D. stands for not detected at the detection limit of 1 ppb.

## B. Environmental Fate

### 1. Overview/summary of environmental fate of pentachlorophenol

Pentachlorophenol (PCP, Penta) has been used as a wood preservative since 1936.
There are approximately 60 million utility poles in service across the United States of which about 36 million are pentachlorophenol-treated (Malecki, 1992). It was estimated that 3% of utility poles are replaced each year with poles that were freshly treated with

pentachlorophenol. It was reported (Weinberg Group, 1998) that 10,000 metric tonnes of pentachlorophenol were used in 1990 and over 80% for wood preservation. This document is for the purpose of the reregistration of pentachlorophenol, and the reregistration of pentachlorophenol is for use primarily as a wood preservative for utility poles.

In order to comply with the reregistration requirements for pentachlorophenol, the Penta Task Force submitted the required studies. These studies were reviewed by the OPP and found acceptable for the active ingredient, pentachlorophenol. The environmental fate assessment for pentachlorophenol is based primarily on guideline studies that were submitted to the agency and satisfied requirements for registration for pentachlorophenol.

The reregistration issues for pentachlorophenol are unique with respect to most pesticides in having major issues/concerns with respect to the microcontaminants. Unfortunately, the guideline studies did not properly address the environmental fate of degradates/impurities of concern associated with pentachlorophenol such as the chlorinated dibenzodioxins and dibenzofurans. The registrant only submitted studies on pentachlorophenol, the registered pesticide, and did not submit guideline studies on the chlorinated dibenzodioxins stating that they were not "major" degradates. Therefore, the assessment of chlorinated dibenzodioxins will be based on information available from other sources including valid literature references.

After reviewing the available data from studies that meet guideline requirements, as well as data from the published literature it was concluded that there is sufficient data to assess the chemical stability and persistence of pentachlorophenol. We have sufficient hydrolysis, photodegradation on soil and in water and aerobic and anaerobic soil metabolism data to evaluate the rate of degradation of pentachlorophenol under environmental conditions.

From literature data one can conclude that the chlorinated dibenzodioxins are extremely persistent in soil with half lives of 15-100 years. On the other hand, pentachlorophenol is only moderately persistent with half lives in soil of a few months. It is also known that the chlorinated dibenzodioxins are hydrophobic with extremely low solubility in water, have a high adsorption to soils and high solubility in organic matter, and are extremely immobile in soils. On the other hand, pentachlorophenol is more soluble in water and more mobile in soils particularly with increased pH and the formation of the phenolate anion. The bioaccumulation of pentachlorophenol and chlorinated dibenzodioxins in fish is also well documented and the chlorinated dibenzodioxins have a very high bioaccumulation factor in fish with a very low depuration rate. Pentachlorophenol bioaccumulated moderately in fish (significantly lower than the chlorinated dioxins) and depuration was rapid reducing the bioaccumulation .

As was mentioned earlier, treated wood is a major use for pentachlorophenol but only one guideline leaching out of wood study is available. The study tested leaching out of treated wood immersed in water of pH 5, 7 and 9, seawater and fresh water and provides data for leaching in aquatic environment. A study on pentachlorophenol-treated wood provides data on leaching of pentachlorophenol out of treated wood under simulated annual rainfall. There

are studies covering soil analysis in the vicinity of utility poles but there is no data available on the levels of chlorinated dibenzodioxins leaching out of treated wood and in general it is difficult to have a quantitative assessment of the levels of pentachlorophenol and chlorinated dibenzodioxins that may leach and or volatilize out of treated utility poles placed in service under environmental conditions.

The Antimicrobials Division in the Office of Pesticide Programs should have quantitative data on the amounts of pentachlorophenol and chlorinated dibenzodioxins losses from treated wood as a result of leaching and volatilization. EPA should require studies on in service utility poles for which the treatment formulation and the method of application and the levels of pentachlorophenol and its microcontaminants when the poles are placed in service are clearly specified. Thereafter, the levels of pentachlorophenol and chlorinated dioxins remaining in the treated wood should be measured at specified time intervals and all volatiles and leachates should be monitored throughout the study periods. The pentachlorophenol-treated utility poles monitored should simulate various environmental conditions to which utility poles are generally exposed while in service. The environmental impact of pentachlorophenol and its contaminants assessment requires data on the levels of pentachlorophenol and chlorinated dioxins in utility poles placed in service at specified time intervals. Also, the level of losses as a result of volatilization and/or leaching should be measured as a function of time for each pole. Without such data, it is difficult to know how much of the pentachlorophenol and chlorinated dibenzodioxins leach and/or volatilize out of the utility poles placed in service and the potential contamination of soil, water and air.

The microcontaminants issue is important for the reregistration of pentachlorophenol and yet there is essentially little quantitative data on the level of release of CDD/CDF from utility poles while in service and the mode of release. The levels of CDD/CDF in utility poles presently placed in service are significantly lower than the levels decades ago. Major components are the Octachloro- , and the Heptachlorodibenzodioxins and the most toxic isomer the 2,3,7,8- tetrachlorodibenzodioxin is rarely detected in the technical Pentachlorophenol. The toxicity of the 2,3,7,8-is a standard for reporting levels of contaminants of other isomers and the levels of CDD are reported in TEQ (toxic equivalency).

Although no quantitative measurements are available, it is believed that most losses of CDD from in service utility poles result from volatility. The Weinberg Group reported for the Penta Task Force that most of the micro contaminants stay within the treated wood while in service. There are various reports on how much is being released from treated wood while in service. According to the Environment Canada (1998) Dioxins and Furans and Hexachlorobenzene Inventory, in service utility poles and account for a relatively small portion of CDD/CDF releases to air accounting for 2.4 g TEQ/year total. Poles treated before 1987 were held responsible for most of the release while only small amounts were released from poles that were treated after 1987 with purified pentachlorophenol. In another report (Eitzer and Hites, 1987) it was estimated a value of 44 g TEQ per year from volatilization of CDD/CDF. The Weinberg group estimated volatilization of 0.002 kg/year based on published wood data and

0.0005kg/year (0.5 grams/year). Since the Weinberg Group listed the EPA draft dioxin inventory provided emission estimates of 5 kg TEQ/year, they claim that pentachlorophenol contributions are less than 0.1%.

## 2. Guideline study description and summary

The following are summaries of guideline studies that satisfied the pentachlorophenol data requirements:

### a. Hydrolysis (161-1) 424811-01

Pentachlorophenol is stable in sterile water of pH 5, 7 and 9 and does not undergo any noticeable degradation at 25C for 30 days.

### b. Photodegradation in water (161-2) 428554-01

Pentachlorophenol photodegraded with half lives ranging from 13-20 minutes in sterile water at pH 5, 7 and 9 under simulated sunlight conditions (xenon lamp) and was stable in the dark under similar conditions. Dichloromaleic anhydride was the major degradate at all pHs reaching a maximum level of 64.5% of the applied at 80 minutes post treatment at pH 5, 100% after 110 minutes at pH 7 and 98.9% at 90 minutes at pH 9. Tetrachlororesorcinol, tetrachlorohydroquinone and tetrachlorocatechol were minor degradates.

### c. Photodegradation on soil (161-3) 419692-01

Pentachlorophenol degraded (photodegraded) slowly on soil with a half life of 37.5 days on a sandy loam soil under a simulated sunlight conditions (xenon lamp) and virtually no degradation was observed with the non exposed sample under similar conditions. After 30 days of irradiation 69.4% of the applied material was extractable from the soil and the extractable material was composed primarily of the parent pentachlorophenol. 19.1% was bound residues to soil and only 2% were volatile components. The registrant claimed in a March 6, 1995 addendum that all attempts to identify the photoproducts failed since multiple photoproducts were formed as a result of ring opening and dechlorinartion.

### d. Aerobic soil metabolism (162-1) 425943-02

Pentachlorophenol degraded with an observed half life of 7-14 days (63 days calculated first order half-life) in a sandy loam soil. The main degradates were multi isomers of tetrachlorophenol and trichlorophenols (dechlorination). Within a 12 month period, 25.7% of the applied material mineralized to form carbon dioxide and unextractable residues increased continuously to reach 64.0% of the applied.

e. **Anaerobic aquatic soil metabolism (162-3) 424368-01 and addendum of 8/16/94)**

Pentachlorophenol degraded under anaerobic aquatic conditions with a half life of 1-2 months at 25C. Pentachlorophenol degradation deviated clearly from a first order kinetics and underwent a slow degradation in the initial phase of the study with 83.5% remaining after 14 days and 72.2% at the one month interval. Then, degradation accelerated and only 5% of the pentachlorophenol remained at the 2 month interval. Various isomers of tetrachloro- and trichlorophenols were formed as degradates.

**f. Aerobic aquatic metabolism (162-4) 422886-01**

Pentachlorophenol degraded in a flooded sandy loam soil under aerobic aquatic conditions with a half life of 1-2 months at 25C. Mixtures of tetrachloro and trichlorophenol, as well as 3,4-dichlorophenol were major degradates. The 3,4-dichlorophenol reached a level of 36.6% of the applied at day 30 and bound residues reached a level of 40.9%.

**g. Mobility/Adsorption/Desorption (163-1) 426337-09 and 436270-00**

Pentachlorophenol showed Kd adsorption values of 5.72-10.8 in three sandy loam soils, and can be characterized as having low mobility in sandy loam soils. The following Kd values were obtained:

| | | | |
|---|---|---|---|
| Sandy loam | pH 5.9 | OM 0.4% | Kd=5.72 |
| Silt loam | pH 5.8 | high OM | Kd=8.3, |
| Sandy loam | pH 6.9 | OM 1.2% | Kd= 9.96, |
| | pH 7.5 | | Kd =2.0 |
| Sandy loam | pH 7.9 | OM 2.6% | Kd=10.8 |
| Silt loam | pH 4.2 | low OM | Kd=18 |
| | pH 6.6 | | Kd=1.3 |
| Clay loam | pH 5.3 | OM 3.7 | Kd= 74.6 |

The 1/n values were in the 0.81-0.88 range indicating good correlation to the Freundlich value.

The Kd values for pentachlorophenol indicate that it is immobile in clay soil having high adsorption coefficient. The adsorption of pentachlorophenol appeared to correlate well with the clay, the organic material content and the pH of the soils. Generally, pentachlorophenol has intermediate to low mobility in acidic and neutral soils. Adsorption to the soil increases with increased organic material content OM and with decreased pH. In alkaline (basic) soils the mobility of pentachlorophenol is increased due to the formation of phenolate anionic species and increased solubility in water. Therefore, in alkaline soils, particularly soils of low OM, downward mobility of pentachlorophenol is more likely to occur.

**h. Bioaccumulation in fish (165-4) 426337-10, 428554-02**

Pentachlorophenol residues at 2.5ug/L concentration bioaccumulated in bluegill sunfish with a bioaccumulation factor (BCF) of 190 for the edible tissue, 790 for the inedible tissue and 490 for the whole fish. Depuration was rapid with over 97% of the accumulated residues eliminated by day 14. At the 28 day of accumulation, 87-94% of the accumulated residues were identified as pentachlorophenol and 6-13% as glucoronide conjugates of pentachlorophenol.

**i. Aqueous availability from treated wood (166-1) 432050-01**

The following maximum and average leach rate in **mg** pentachlorophenol/kg leachate solution/ square inch wood surface/day were reported for southern pine wood of 11 inch length and one inch in diameter were treated with at 0.50 lbs/ cubic ft.

**TABLE IV-3: Leaching rates from pentachlorophenol treated wood at various pH levels**

| | Final concentration mg/l | Max Leaching rate x $10^{-2}$ mg/kg leachate/sq. inch/day | Average Leaching rate x $10^{-3}$ mg/kg leachate/sq. inch/day |
|---|---|---|---|
| pH 5 | 5.73 | 1.04 | 0.51 |
| pH 7 | 36.2 | 2.28 | 3.27 |
| pH 9 | 65.5 | 3.32 | 6.33 |
| 0.1N HCl | 1.73 | 0.6 | 0.17 |
| Seawater | 22.7 | 3.11 | 2.21 |
| purified water | 6.57 | 0.66 | 0.61 |

The data indicate that the average leach rate and the maximum leach rate increased with increased pH for pentachlorophenol

The environmental fate assessment for pentachlorophenol is based primarily on guideline studies that were submitted to the agency and satisfied requirements for registration for pentachlorophenol. All the required environmental fate data for pentachlorophenol was satisfied.

**3. Environmental Fate Assessment**

In general, the environmental fate and mobility of pentachlorophenol in soil and water will depend on the pH of the soil/water. The chemical behavior and the physical parameters pf pentachlorophenol will depend on whether it exists primarily as a phenol (more acidic

conditions or phenolàte anion (under basic conditions). Although pentachlorophenol is stable to hydrolytic degradation, it is not a persistent chemical being susceptible to several other modes of degradation. Pentachlorophenol is susceptible to microbial degradation under both aerobic and anaerobic conditions with half lives of less than two months. Photodegradation of pentachlorophenol is very rapid and eventually results in the destruction of the aromatic ring. Microbial degradation and exposure to sunlight results in dechlorination of the chlorinated ring and formation of the lower chlorinated phenols such as the tetra-, tri- and dichlorophenols. Pentachlorophenol mobility in soils depends heavily on the pH of the soils and the formation of the phenolate anion as the pH increases. The phenolate anion species is substantially more soluble in water, adsorbs less to the soil and is more likely to move into lower depths. Degradation of pentachlorophenol will reduce the likelihood of groundwater contamination and indications were that pentachlorophenol did not move significantly to lower depths in contaminated soils from utility poles. In addition, the amount leaching out of utility poles/square area/time is very small to pose risks to ground water. Pentachlorophenol is not likely to bioaccumulate significantly in fish and accumulated residues will depurate.

### a. Chemical and microbial degradation of Pentachlorophenol in water and soil.

Pentachlorophenol can be characterized as moderately persistent chemical. Pentachlorophenol is stable in water at pH 5-9 when it is not exposed to sunlight but will undergo rapid photodegradation in pH 5, 7 and 9 when exposed to natural sunlight at temperatures up to 36C with a half lives of 20 minutes. Several degradates were formed of which dichloromaleic anhydride is clearly the major degradate. Pentachlorophenol is also susceptible to microbial degradation in soil under aerobic and anaerobic conditions resulting in dechlorination and the formation of tetrachloro and trichlorophenol isomers. Degradation is soil under aerobic conditions was more rapid than under anaerobic conditions where half lives of 1-2 months were observed.

### b. Mobility of Pentachlorophenol in soil

In general, the mobility of pentachlorophenol with water as a carrier will depend on the pH of the soil/water and wood/water. Pentachlorophenol with a pKa of 4.74 is an acidic compound that will have its percentage of phenolate anion increase with increased pH. At pH 6 and above, most of the pentachlorophenol will exist in the phenolate ion. Since the phenolate anionic form has substantially higher solubility in water, the solubility of pentachlorophenol in water increases with increased pH. In addition, pentachlorophenol will be less soluble in organic material with increased pH. As a result of the increased solubility in water and the decreased adsorption to the soil, mobility of the phenolate anion in environmental pH above 6 can be substantial in the absence of degradation. However, microbial degradation under aerobic and anaerobic conditions is generally rapid enough to minimize contamination of ground water. Photodegradation cannot occur once the pentachlorophenol/phenolate anion

penetrates the soil and in not exposed to sunlight.

The adsorption/desorption coefficient which reflects the distribution of the chemical between water and soil shows lower adsorption coefficients (Kd values) in soils of higher pH and lower organic material content. Pentachlorophenol Kd values for a silt loam soil of low OM , pH 6.6, was 1.3 which was the lowest Kd values obtained for acidic soils. A sandy loam soil, pH 6.9, 1.2% OM and a sandy loam, pH 7.9, 2.6% OM provided Kd values of 10-11 for Pentachlorophenol. Since most agricultural soils are acidic, it is unlikely that pentachlorophenol with moderate to low mobility in acidic soils will move substantially to lower depths. Although movement in basic (alkaline) soils will be faster it is unlikely to reach very low depths and contaminate ground water. In addition to low movement, pentachlorophenol and its metabolites tetrachloro- and trichlorophenols will continue to degrade upon movement from aerobic soil to anaerobic soil depths with half lives of less than two months. The main defense against contamination of ground water will be microbial degradation under aerobic and anaerobic conditions.

Pentachlorophenol and its microcontaminants leaching out of utility poles will also depend on the method of application and the oil used as a solvent for the purpose of application. A major portion of the chemical loss occurs within the first year of application having the solvent as a carrier to the perimeter of the utility pole and downward. Contamination of soil found in the vicinity of utility poles at lower depths is also a result of downward movement of pentachlorophenol and its microcontaminants within the utility pole to the bottom of the pole with the carrier solvent as a result of gravity. This movement is also facilitated by the physical structure of the wood which is designed (by nature) to carry water and nutrients vertically. Therefore, contamination of soil in the vicinity of utility poles can also be as a result of downward movement within the pole. The lower part of the pole which becomes enriched by pentachlorophenol, microcontaminants and petroleum solvent can then contaminate both soil and water and the lower depths. Since movement can occur both through the soil and/or through the wood, it is unclear which mechanism is more responsible for the downward movement through the soil/water.

### c. Potential to contaminate ground and surface water

In considering the total amount of pentachlorophenol available for leaching from utility poles per area while in use, the relatively moderate mobility through the soil profile (with Kd values above 1) and the moderate degradation under aerobic and aerobic conditions (half lives of 1-2 months), contamination of water by pentachlorophenol and its metabolites should not be a concern. Pentachlorophenol adsorbed to soil may move in small quantities into surface water, it is not likely persist being exposed to sunlight and to aerobic microbial degradation. In summary, groundwater contamination should not occur from usage in utility poles. However, in places where a high soil concentration of pentachlorophenol may exist, such as in storage area for treated wood and when a large amount of treated wood is used, leaching into groundwater could be a concern particularly in soils of high pH (alkaline soils) since studies

that trace downward mobility under use conditions are not available.

Rain water collected in Burlington Ontario contained up to 10 ng/L of pentachlorophenol (CCREM, 1987). Surface water contained up to 5.69 ng/L pentachlorophenol and 1 ng/L tetrachlorophenols in the Bay of Quinte, which has a wood preservative site at the head of the bay. Concentrations decreased with increasing distance from the source (Fox and Joshi, 1984).

In 1976 it was noted by Arsenault that a typical effluent water from a Pentachlorophenol treatment facility contained 44 mg/L of pentachlorophenol and only 0.003 ng/L of OCDD which are due to the facts that OCDD is a micro contaminant and also that it has a substantially lower solubility in water than pentachlorophenol.

A study by the Electric Power Research Institute (EPRI) on 180 in use utility poles indicated movement of pentachlorophenol to lower depths and when samples were collected at 18, 30 and 48 inches pentachlorophenol was detected at levels above 100 ppb only in 5% of the cases. Over 85% of the samples showed less than 10 mg/kg levels. Generally, surface soil samples showed higher levels of Pentachlorophenol than subsurface soil samples and pentachlorophenol soil concentrations decreased exponentially with distance from the pole. It was not determined whether movement to lower depths is through the soil profile or through the wood to the bottom of the pole which then comes in contact with soil/water at lower depths.

### d. Discussion of special issues

#### i. Depletion of pentachlorophenol and its microcontaminants from treated utility poles

It is understood that pentachlorophenol and its microcontaminants are depleted from utility poles over time causing the poles to be more susceptible to decay. What is unclear is the level of loss of pentachlorophenol and its microcontaminants as a function of time. There are no quantitative data on the levels of pentachlorophenol and its microcontaminants when the utility poles are placed in service and at specified time intervals when in service. Generally, it was reported that losses from freshly treated poles are higher in the first year of service than in later years .

The mechanism of loss of preservatives and microcontaminants is unclear. It is unknown to what extent losses are due to volatilization, leaching out of the pole through the outside surface and/or the bottom of the pole or losses are due to degradation via microbes, sunlight, etc. It was proposed that depletion in the above ground portion of pentachlorophenol treated poles is volatilization of pentachlorophenol (Ruddick, 1991). Depletion of pentachlorophenol and its micro contaminants from utility poles by volatilization and leaching will be discussed below.

### a. Leaching Out of Treated Wood

The issue of leaching of pentachlorophenol and its microcontaminants out of treated wood is important in assessing potential exposure to this chemicals. According to a review by The Weinberg Group for the Penta Task Force, pentachlorophenol is applied to wood in a petroleum base carrier oil (P9 Oil/15% pentachlorophenol) by means of pressure or thermal treatment. The Weinberg Group in their report emphasize that possibly the main mechanism for leaching involves the downward migration of the oil along carrier along the longitudinal axis of the pole along with pentachlorophenol and its micro contaminants. This mechanism is called Gravitational Induced Downward Migration of Oil (GIDMO). Since the oil fraction dominates the pentachlorophenol fraction, the behavior of pentachlorophenol and its contaminants is influenced to a considerable extent by the behavior of oil.

Preservatives can leach from the pole above ground, migrate from the surface away and to deeper depths. Leaching into depth requires dissolution with water and can also occur from the pole butt in the soil or immersed in the water table in shallow water table.

According to the Weinberg Group Report for the Penta Task Force, aqueous leaching of pentachlorophenol from poles is only important for the first 10-12 days after treatment (Bellcore 1993) since the surface of the pole has to be replenished with pentachlorophenol from the inner layers of wood for leaching with water to occur. The report lists the following as factors affecting potential leaching of pentachlorophenol and microcontaminants from wood poles:

**TABLE IV-4: Factors affecting leaching of pentachlorophenol and microcontaminants from treated utility poles**

| Pole | Treatment | Environment |
|---|---|---|
| Wood type<br>lignin content<br>organic carbon content<br>age | Carrier oil used to apply pentachlorophenol, treatment method and contaminants concentrations | rainfall, pH of water, temperature, exposure to sunlight and soil characteristics such as OM |

The report goes on to say "Initially, we examined the literature to determine whether sufficient information on this factors is available to permit modeling of the leaching of micro contaminants from treated poles. Unfortunately, there are no published studies of the potential leaching of pentachlorophenol or its micro contaminants that address the above factors. Further, the published data that exist do not allow a consideration of which of these factors are important in influencing the rate of microcontaminants leaching from poles. The few sampling programs that have been reported typically did not collect key pole parameters (eg, organic carbon and oil levels in wood and soil) Moreover, the available data show no consistent relationship between the factors that could affect leaching and the presence of

pentachlorophenol or its micro contaminants in soil around poles. As a result, a model that could quantitatively predict the rate, or extent of leaching from pentachlorophenol-treated poles cannot, and has not been developed" (The Weinberg Group Inc, October 28, 1997)

The following are some of the studies that may provide some useful information on pentachlorophenol and its contaminants:

**Aqueous availability from treated wood (166-1) 432050-01**
(See table IV-3, above, for results)
The data indicate that the average leach rate and the maximum leach rate increased with increased pH for pentachlorophenol

One study (432050) was submitted to the agency and addressed only the leaching of pentachlorophenol from treated wood. As anticipated, leaching of pentachlorophenol from treated wood was dependent on the pH of the water solution and increased with increased pH. Average leaching of 0.51, 3.27 and 6.33 x $10^{3}$ mg/kg solution/sq. inch wood/day was obtained for pH 5,7 and 9 respectively.

Literature data (Cooper, 1991) indicate that pentachlorophenol in oil is rapidly depleted from the higher portion of the poles to below the ground area of the wood for the first few years. Laboratory field studies also show a relatively high rate of depletion from the wood surface and a leveling off with time. Rain water from pentachlorophenol in oil treated cedar maintained a relatively constant concentration of Pentachlorophenol at 0.3-0.7 $\mu$g/ml over a one year analysis period (Cserjesi, 1976). Test of depletion of pentachlorophenol from treated wood in France show 20-30% loses within the first year of service.

A study designed to measure leaching out of wood in storage areas measured leaching out of a bundle of 15 utility poles of Douglas fir of 16-27 inch diameters with natural rainfall in British Columbia (Whiticar, 1994). Pentachlorophenol release was relatively constant throughout the study period up to four months ranging from 1.57-2.85 mg/L of rainfall.

Buchanan, 1991 tested the leaching of utility poles similar to those used in service. Five class 5, Red Pine (Pinus Risonosa) were freshly treated with pentachlorophenol and there was no evidence of bleeding or surface deposits. Pressure treated poles generally receive 2.93 kg Pentachlorophenol/$m^{3}$ (CITW, 1989) . The size specification for class 5 utility poles is 48-52 cm circumference at the top, 86-97 cm bottom and a length of 12.2-13.7 meter. The leaching apparatus was a suspended 45 cm pole section of a mean volume of 0.0147 $m^{3}$ over a 15L bath. The base portion of each pole was capped with a 5cm stainless steel drip skirt and sealed with silicone to prevent leaching and wetting of the cut surface and to simulate in service. A small pump was used to recirculate the leaching solution at approximately 1.8 L/min through a perforated circular teflon tubing to allow a even distribution of the leachate and to simulate rainfall. The leaching solution simulated acid rain containing mineral salts, sulfiric and nitric acid of pH 4.2. The entire apparatus was covered with polyethylene plastic to maintain

constant humidity and minimize evaporation and was removed after each simulated rainfall event to allow the pole surface to dry. The study was conducted for 10 days at 20C. Leachate solutions were collected following the completion of each leaching event and 400 aliquots were extracted and analyzed GLC having a detection limit for pentachlorophenol at 0.1ug/L. The mean pentachlorophenol loss per leaching event was 23.3 mg/pole section with a total mean loss of 232 mg with a range of 159.7 to 330 and the maximum concentration of pentachlorophenol found in the study was 4.4 mg/L.

Based on a typical class 5 Red Pine pole average volume of $0.5m^3$ with retention levels of 2.93 kg Pentachlorophenol/$m^3$ for a total of 1.46kg/pole. If the mean Pentachlorophenol loss of 232.3 mg/pole section is multiplied by the total number of sections/pole assuming 10.7 meter poles above ground, then the total **annual loss/pole would be 6.6 gm pentachlorophenol/pole/year by leaching from the portion exposed above ground.**

A study on pentachlorophenol release onto soils adjacent to 31 utility poles in New York State describes the analysis of soil samples within proximity to the poles (EPRI, 1995). The study demonstrated that the concentration of pentachlorophenol decreased substantially with increasing distance from the utility poles. Pentachlorophenol concentration decreased by two orders of magnitude between 3 to 8 inch distance. Therefore most of the pentachlorophenol from the poles is within close proximity to the poles. At 30-40 inches away from the poles (one meter) levels of pentachlorophenol were below the detection limit. **Levels of pentachlorophenol in proximity to the poles were generally below 100mg/kg.**

A study by Gurprasad (1995), measuring contaminant soil concentrations in the vicinity of three utility poles in Edmonton, Canada, is limited by the small number of poles analyzed and by the lack of historical data on the age of the poles, the pentachlorophenol treatment method, the initial amount of pentachlorophenol/contaminants, etc.
Utility pole 1 was located in a light industrial area, appeared to have received butt-treatment-may have been encountering bleeding problem.
Utility pole 2 was located along a busy road in a medium industrial area and appeared to have been treated along its entire length
Utility pole 3 was in undeveloped area of Edmonton, appeared to receive butt treatment.

The data indicate that contamination of the soil are retained mainly within 20 cm from the pole and decrease substantially thereafter.

Vegetation in the vicinity of the poles did not accumulate dioxins to levels that can be detected.

**TABLE IV-5: Pentachlorophenol and microcontaminants contamination of soil at varying distances around 3 pentachlorophenol-treated utility poles**

| Chemical | pole 1 ug/g 2cm | pole 1 ug/g 20cm | pole 2 ug/g 2cm | pole2 ug/g 20cm | pole 3 ug/g 2cm | pole 3 ug/g 20cm |
|---|---|---|---|---|---|---|
| PCDD | nd | nd | nd | nd | nd | nd |
| HxCDD | 0.41 | 0.05 | 0.13 | nd | 0.07 | nd |
| HpCDD | 4.85 | 0.16 | 1.13 | O.1 | O.1 | 0.12 |
| OCDD | 28.3 | 0.78 | 4.62 | 0.16 | 0.99 | 0.42 |
| HCB | 0.14 | nd | 0.14 | nd | 0.16 | nd |
| Penta-chlorophenol | 6680 | 254 | 84 | 27 | 33 | nd |

nd for below 0.01 ug/g

One Canadian study (Wan, 1992) examined the release of Pentachlorophenol into streams in British Columbia from utility poles and railroad tiles which were generally treated in the 1980s and thereafter. The chlorophenols constituted 92% Pentachlorophenol, 7% tetrachlorophenol and 1% trichlorophenol and the tri- and tetrachlorophenols constituted a mixture of isomers. Sediment and water samples were collected from identical sites. Soil at the base of the utility poles registered a mean CP concentration of 2168 mg/kg (range 83-4600 mg/kg). Pentachlorophenol averaged 3792 ug/L (range 145-21,080) immediately adjacent to utility poles installed in , or located up to 1.0 meter away from a ditch. The study covered 14 utility ditches and 10 railroad ditches and sampling of the ditches were taken in March/April and November/December to coincide with periods of maximum seasonal runoff.

**TABLE IV-6: Pentachlorophenol levels in water samples from sites near utility pole rights-of-way**

| | CP, average ug/L | CP, range ug/L |
|---|---|---|
| at pole (within 0.1-1m) | 1408 | 11.8-3854 |
| 4 meter downstream | 13.6 | 1.0-34.8 |
| 3 meter upstream | nd | nd |

**TABLE IV-7: Pentachlorophenol concentration in water samples near railway rights-of-**

way

| | CP average ug/L | CP range ug/L |
|---|---|---|
| at pole (within 0.1-1m) | 225 | 1.0-750 |
| 4 meters downstream | 3.8 | 2.5-4.8 |
| 3 meters upstream | nd | nd |

**TABLE IV-8: Pentachlorophenol concentration in sediment samples near utility pole rights-of-way**

| | CP average mg/kg wet weight | CP range mg/kg wet weight |
|---|---|---|
| at pole (within 0.1-1m) | 139 | 0.4-828 |
| 4 meter downstream | 0.3 | 0.05-1.1 |
| 3 meter upstream | nd | nd |

**TABLE IV-9: Pentachlorophenol concentration in sediment samples near railway rights-of-way**

| | CP average mg/kg wet weight | CP range mg/kg wet weight |
|---|---|---|
| at pole (within 0.1-1m) | 49.7 | 1.1-72.8 |
| 4 meter downstream | 0.3 | 0.05-1.1 |
| 3 meter upstream | nd | nd |

A study conducted by the Electric Power Research Institute (EPRI, 1995) examined 168 in usage utility poles and soil samples were analyzed a distances from the poles. The analysis indicated that levels of Pentachlorophenol declined rapidly to non detect levels at distances of 30-40 inches from the poles. The detection limit for the determination of Pentachlorophenol in soil using a GC/ECD was 100 ug/kg (ppb).

The study also evaluated leaching of Pentachlorophenol into lower depths by the utility poles and found Pentachlorophenol residues were relatively constant from 18, 30 and 48 depths levels and averaged 20 mg/kg of soil near the utility pole. Maximum levels were above the 500 mg/kg soil level (500 ppb). Several pathways may exist for the movement of pentachlorophenol from the pole.

### b. Air Volatility

Pentachlorophenol has a relatively high vapor pressure of 1.1 x $10^{4}$ mm Hg at 25C. However, volatility from treated wood should be lower for the product to be effective for periods of 50 years and beyond. Photodegradation in air and dissipation may reduce exposure A photodegradation in air (43214601 was submitted to the agency but the study was of questionable quality. The chlorinated dibenzodioxins have substantially lower vapor pressure than pentachlorophenol and losses due to volatilization should therefore be even lower than those of pentachlorophenol.

Losses of pentachlorophenol and the chlorinated dibenzodioxins from treated wood were not measured quantitatively and it is not known what are the losses as a function of time. Some studies measured pentachlorophenol and CDD concentrations in air in proximity to utility poles treatment and storage facilities (Waitel, D.T.) but most studies cannot exclude potential air contamination from other sources including chlorinated dioxins reservoirs.

A report by the Weinberg group of May 28, 1998 which was prepared for the Penta Task Force claims that "the results of the modeling analysis showed volatilization is not a significant mode of release of either pentachlorophenol or microcontaminants from poles and that treated poles are negligible source of micro contaminants to the atmosphere. The total amount of PCDD/F toxic equivalents (TEQ) predicted to volatilize from in-service poles in the US was 0.002 kg/year based on published data and 0.0005 kg/year based on freshly treated pole data."

The Canadian study by Waitel was to confirm the presence of pentachlorophenol residues in air in central Canada, explore seasonal trends in atmospheric pentachlorophenol concentration and to identify possible sources of atmospheric pentachlorophenol. In the study, pentachlorophenol was detected at all five sampling sites with the highest concentrations in the vicinity of storage sites for freshly treated utility poles. The atmospheric concentration of pentachlorophenol were highest as the temperature increased during the months of July-August and conversely were lower during the winter months. The study does not provide any quantitative data on levels of Pentachlorophenol that volatilize from utility while in service and the levels of chlorinated dibenzodioxins that enter the atmosphere.

**c. Data for Aquatic Risk Assessment:**

Clearly, the available data is not suitable for a quantitative risk assessment having two major limitations:

a) Most of the available field studies do not provide quantitative data (material balance) on what was present in the pentachlorophenol treated wood when it was placed in service and what remained in the wood at specified time intervals and when the study was terminated.

b) Many of the utility poles used in studies were placed in service at least 10-20 years ago when different formulations and treatment methods were used and it is also likely that the % of

microcontamination in pentachlorophenol was higher. In general, information on the initial treatment levels was not available for most of the field studies. The EPRI study of in use utility poles claims that information on the treatment level was available for only 40% of the poles in the study.

We found out that contamination of soil in proximity of the utility poles occurred but we do not know how it got there, whether leached through the pole with the oil or leached with the water or possibly got there as a result of freshening, bleeding, etc. We know that contamination away from the pole is lessened substantially and that pentachlorophenol will not persist in the top soil exposed to photodegradation and aerobic soil metabolism. Yet, levels of pentachlorophenol were higher in surface soil samples than in samples taken from lower depths indicating very low movement.

Based on EPRI data technical grade pentachlorophenol consist of 85-90% pentachlorophenol, 4-8% tetrachlorophenol, 0.1% trichlorophenol and 2-6% other chlorophenols and micro contaminants of the chlorinated dibenzodioxins. Distillation of the technical grade pentachlorophenol results in 1-2% of pentachlorophenol, 5 % of the tetrachlorophenol and significant decrease in the chlorinated dibenzodioxins (mostly the octa and the hepta isomers) that have a higher boiling point (lower vapor pressure).

For a typical utility pole of 12 inch in diameter and 45 feet length and at a treatment rate of 0.6 lb/cubic feet, 40 pounds of pentachlorophenol are used

Clearly it is best to rely on recent data from poles that were treated by pentachlorophenol formulation and by method of applications that representative of present and future practices. Therefore, OPP may have to rely on the following three studies for aquatic risk assessment:

1) The Wan study titled "Utility and Railway Right of Way Contaminants in British Columbia : chlorophenols," is an aquatic field study that was conducted in 1990 on utility poles that were placed in service during 1980-1990. This published study covers a range of sites and 200 utility poles to provide useful information.

2) The Guideline 166-1 study provides valid information on the leaching of pentachlorophenol from treated wood immersed in solutions of pH 5, 7 and 9 at 25C. The study simulates leaching out of utility poles immersed in water and the data is reported in mg pentachlorophenol/kg leachate /sq inch of area utility pole/day. The study was conducted on a Southern Pine pole that was treated with pentachlorophenol at o.5 lbs/cubic ft of 11 inch length and 10 inch diameter. The poles were sealed at the top and placed in 75 cm x 32 cm x 47 cm tank of leaching solutions of pH 5, 7 , 9 , sea water and reagent grade water. The leaching solutions was circulated throughout the 30 day study period at 20C. Volatiles were trapped and all precautions were taken to prevent photodegradation.

The maximum leach rate occurred on day 1 for pH 5 and 7 and for fresh and sea water and on

day 3 for pH 9. The leaching rates were reported in mg pentachlorophenol/kg solution/sq inch/day and are reported in relationship to the area in cm square and the volume of the pole in cubic cm.
Values reported per square inch will be divided by 2.54 x 2.54=6.45

The total surface area of the treated wood is calculated as follows: 2 x 3.14x (5 x 2.54) x11x2.54=2228.38 $cm^2$ = 0.2228 $m^2$

the total volume of the solution is 75 x 32 x 47 =112800 $cm^3$ = 0.2811$m^3$

the total amount can be calculated by multiplying the volume of the solution by either the values in column 2 or 5.

**TABLE IV-10: Leaching rate of pentachlorophenol from treated utility poles in various solutions** .

| Leaching solution | Final concentration mg/l | maximum | average | Calculated leach amount in 30 days for the 11 inch pole in mg/kg soln | calculated total leach amount in 30 days for 11 inch pole in grams |
|---|---|---|---|---|---|
| pH 5 | 5.73 | $1.6 \times 10^{-3}$ | $0.80 \times 10^{-4}$ | 5.35 | 0.646 |
| pH 7 | 36.2 | $3.6 \times 10^{-3}$ | $5.1 \times 10^{-4}$ | 34.09 | 4.08 |
| pH 9 | 65.5 | $5.1 \times 10^{-3}$ | $9.8 \times 10^{-4}$ | 65.50 | 7.39 |
| 0.1N HCl | 1.73 | $0.92 \times 10^{-3}$ | $0.25 \times 10^{-4}$ | 1.67 | 0.195 |
| seawater | 22.7 | $4.8 \times 10^{-3}$ | $3.4 \times 10^{-4}$ | 22.72 | 2.56 |
| reagent water | 6.57 | $1.03 \times 10^{-3}$ | $0.95 \times 10^{-4}$ | 6.34 | 0.74 |

3) The Buchanan study in 1991 measured leaching and availability of pentachlorophenol from treated utility poles in Ontario, Canada. The value of annual loss of 6.6 grams pentachlorophenol /pole/year under yearly simulated rainfall appears to provide a realistic value for leaching from poles exposed above ground. The leaching value is substantially lower than the value calculated from the values in column 2 and 5 from the above table. Clearly, higher leaching values should be expected when poles are immersed in water solutions than when exposed to simulated rainfall.

Photodegradation and microbial degradation should play an important role in the degradation of any pentachlorophenol leaching out of utility poles. Exposure to sunlight on wood, on soil

and in water will reduce exposure to pentachlorophenol with time.

#### d. Formation of Chlorinated Dibenzodioxins (CDD) from Pentachlorophenol

**--THIS ISSUE WILL BE ADDRESSED IN A FORTHCOMING DOCUMENT.**

## C. Environmental Modeling/Drinking Water Assessment

### 1. Background

Pentachlorophenol is used as a wood preservative on utility poles. There are 36 million pentachlorophenol-treated utility poles in service across the United States. Because of the possible run off of pentachlorophenol from the utility poles, there are concerns about the environmental effects of pentachlorophenol on aquatics. The Risk Assessment and Science Support Branch (RASSB) has decided to calculate the Estimated Environmental Concentrations (EECs) using a combination of a model developed by Antimicrobials Division and the EPA's PRZM (Pesticide Root Zone Model) and EXAMS (Exposure Analysis Modeling System) models. The detailed descriptions of assumptions and inputs used in these models as well as the results are discussed in this report.

### 2. Modeling

#### a. Migration Flux of Pentachlorophenol from Pole Surfaces

It is assumed that the depletion of the pentachlorophenol in a utility pole follows first-order kinetics when the pole is in contact with rain. Thus,

$$dc/dt = -\mu c \qquad \text{(Eq. 1)}$$

Where:

$c$ = The pentachlorophenol concentration in wood (mg $cm^{-3}$)

$t$ = Raining time (day)

$\mu$ = Attenuation coefficient ($day^{-1}$)

By integration, equation 1 becomes:

$$c = c_o e^{-\mu t} \qquad \text{(Eq. 2)}$$

Where:

$c_o$ = the pentachlorophenol treatment retention or initial concentration in wood (mg $cm^{-3}$)

It is assumed that the depletion of pentachlorophenol only occurs at the surface of the pole at a depth of 1 cm. The migration flux of pentachlorophenol from wood surfaces is thus described by:

$$m = dc/dt = 1000\mu c_o e^{-\mu t} \quad \text{(Eq. 3)}$$

Where:

m = the migration flux or leaching rate of pentachlorophenol (ug $cm^{-2}$ $day^{-1}$)

To determine $\mu$ in Equation 3, one must conduct leaching experiments with utility poles. The standard Pentachlorophenol treatment retention for a utility pole is 0.45 pound per cubic feet or 7.215 mg $cm^{-3}$, as defined by the American Wood-Preserver's Association (Connor, 1994).

Taking the logarithm of both sides of Equation 2 yields Equation 4.

$$\ln (c/c_o) = -\mu t \quad \text{(Eq. 4)}$$

Based on Equation 4, a linear plot of $\ln (c/c_o)$ vs t would yield a slope which is equal to $\mu$. The linear regression analysis gives:

$$y = -.0028\ x - 0.03 \quad r^2 = 0.98 \quad \text{(Eq. 5)}$$

Substituting Equation 5 into Equation 3 gives the default equation for the Pentachlorophenol migration rate or flux from a utility pole:

$$\mathbf{m = 20.2\ e^{-0.0028t-0.03}}\ \text{ug cm}^{-2}\ \text{day}^{-1} \quad \text{(Eq. 6)}$$

The emission flux of wood preservative is a function of pH, salinity level, and treatment retention. It also depends on a number of other factors, such as the type of wood preservative, storage conditions, and the choice of wood types. Some of the factors simply can not be controlled by wood treaters. Therefore, it is suggested that whenever possible, the leaching experiment should be conducted for a batch of freshly treated utility pole and the corresponding migration rate equation should be obtained.

**b. Transport in Rain on the Surface of Utility Poles**

For this model, it is assumed that the rain falling on the top of a utility pole is evenly distributed onto the surfaces of a utility pole so that the velocity of rain which runs down a pole is determined by the intensity of rain. This assumption is reasonable within a certain range of rain intensity, considering hydrophilic and adsorptive properties of the surface of a wood pole. Based on this assumption, for an infinitesimal amount of rainfall, Equation 7 is used:

$$I\pi r^2 dt = (\pi^{®} + H)^2 - \pi r^2)\, dh \qquad \text{(Eq. 7)}$$

or $$I\pi r^2 dt = (2r + H)H\pi dh$$

Where:

I = Intensity of rain (m $day^{-1}$)

r = Radius of the utility pole (m)

t = Raining time (day)

H = The thickness of water film on the surface of utility poles (m)

h = Height of the utility pole (m)

dt = An incremental time (day)

dh = The incremental height of the utility pole (m)

Since $2r >> H$, Equation 7 can be simplified and converted to:

$$V = dh/dt = Ir/(2H) \qquad \text{(Eq. 8)}$$

Where:

V = The velocity at which the rain moves down a utility pole.

The time required for rain to reach the base of a utility pole (t') could be predicted by

$$t' = h/V = 2hH/(Ir) \qquad \text{(Eq. 9)}$$

The exposure time of rain water to the surface of the utility pole as it moves down the

pole is also represented by t'. The pentachlorophenol concentration at the base of a utility pole ($C_o$) can thus be predicted by:

$$C_o = (m\ t'\ 2\pi r\ dh)/(2\pi rH\ dh) \qquad \text{(Eq. 10)}$$

Where:

m = The migration rate of pentachlorophenol from a utility pole (ug $cm^2$ $day^{-1}$)

Substituting Equation 9 into Equation 10 yields:

$$C_o = 2mh/(Ir) \qquad \text{(Eq. 11)}$$

Using Equation 11, one can predict the concentration of pentachlorophenol in the rain water at the base of a utility pole as a function of time if the pentachlorophenol migration rate from pole surfaces and the rain intensity are known. In general, the height and the radius of a utility pole are fairly constant values. Equation 11 suggests that the concentration is proportional to the migration rate and the height of the utility pole, but inversely proportional to the intensity of rain and the radius of the pole.

The pentachlorophenol concentration at the base of the utility pole is closely related to rain intensity. The concentration can be as high as 300 ppm for low rain intensity (0-0.24 m $day^{-1}$) and as low as 13 ppm for high rain intensity (0.60 - 4.8 m $day^{1}$). Clearly, it is very important to define the averaged rain intensity in the proper manner.

When rain intensity-frequency data are available, the average rain intensity is defined as:

$$I_{av} = 0.12P_1 + 0.42P_2 + 2.7P_3 \qquad \text{(Eq. 12)}$$

Where:

$I_{av}$ = Averaged rain intensity (m $day^{-1}$)

$P_1$ = Fraction of rainfall in the range of low rain intensity (0 - 0.24 m $day^{1}$) (0.12 is midpoint of range)

$P_2$ = Fraction of rainfall in the range of medium rain intensity (0.24 - 0.6 m $day^{-1}$) (0.42 is midpoint of range)

$P_3$ = Fraction of rainfall in the range of high rain intensity (0.60 - 4.8 m/day) (2.7 is midpoint of range)

When rain intensity-frequency data are not available, a default value of 0.12 m $day^{-1}$ (the midpoint of the range for low rain intensity) is used for the averaged rain intensity. This conservative value was selected because it was assumed that the majority of rain falls at low rain intensity.

### c. Calculation of Pentachlorophenol concentrations at the base of the utility pole

Using the above discussion and equations, the calculated pentachlorophenol concentration at the base of each pole was 328 ppm.

### d. Assumptions and Calculations

The following assumptions were used to calculate concentration of Pentachlorophenol in soil:

1. The utility poles are placed in a 10 hectare (Ha) field planted with cotton in Mississippi delta.

2. There are two rows of utility poles in the field and the distance between the utility poles are 50 meters.

3. With the inter connections, it was assumed that a total of 20 utility poles will be placed in a 10 Ha field.

4. The area around the utility poles that pentachlorophenol spreads in the soil is a circle with a diameter of 1.0 meter. It was assumed that there was no downward movement of the pentachlorophenol into the soil.

5. The surface area around each pole was calculated to be 0.785 $m^2$ . The total area of the soil around all 20 poles in the fields contaminated with pentachlorophenol will be 15.70 $m^2$.

6. Using the ratio between the contaminated soil area (15.70 $m^2$ ) and the area of 10

Ha (100000 $m^2$), the applied concentration of pentachlorophenol was calculated to be 0.103 kg/Ha.

### e. PRZM3-EXAMS MODELS

A combination of PRZM3-EXAMS was used to calculate EECs for pentachlorophenol in the environment. As it was described in the above section, it was assumed that 100 percent of the chemical which leaches out of the pentachlorophenol-treated pole reach the soil. Furthermore it was assumed that the movement of the pentachlorophenol in the soil is lateral with no, or little, downward movement. It was also assumed that there was no volatilization of pentachlorophenol.

Tier II EEC uses a single high exposure site for each pesticide use of interest. The weather is simulated at the chosen site for 36 years so that the probability of an EEC occurring at those sites can be estimated. Only one application of pentachlorophenol was used in the scenario which is equal to the amount which leaches out of the utility pole after one time treatment with pentachlorophenol. The input data used to run PRZM3 is shown in Appendix . The field is located in Yazoo County, Mississippi. The soil series is Loring silt loam and is in Hydrologic group C, which has a high potential for runoff. The average yearly rainfall is 127.0 cm, average yearly runoff is 34.5 cm and average erosion rate is 215 Mg/Ha.

### f. Results

A summary of the results for the EECs simulated by PRZM3-EXAMS models are shown in Table IV-11. The results for instant, 96 hours, 21 day, 60 day, 90 day and yearly are reported in this table. The dissolved concentrations in water column and pore water as well as the concentrations in sediments and benthic organisms are also shown in this table. The PRZM3 model simulated the data for 36 years of the weather data. However, during the 36 years of simulation, pentachlorophenol was applied only once when the treated pole was installed into the ground. The application rate was equal to the maximum amount of the leaching rate from the treated pole as described in section III (A and B) of this report. This assumption is very conservative because all of the chemical will not leach out of the pole at once and leaching will be gradual through

the years. The data reported in Table IV-11 are not the average for the duration of the simulation run, but from the first year of the simulation which the model assumes the chemical was applied to the soil.

**Table IV-11. Estimated Environmental Concentrations (EECs) for Pentachlorophenol Leached out of Utility Poles.**

| Concentration | Instant | 96 Hours | 21 Day | 60 Day | 90 Day | Yearly |
|---|---|---|---|---|---|---|
| **Water Column Dissolved (ppb)** | 0.176 | 0.120 | 0.075 | 0.055 | 0.043 | 0.014 |
| **Pore Water Dissolved (ppb)** | 0.054 | 0.053 | 0.048 | 0.040 | 0.034 | 0.011 |
| **Benthic Sediment ($\mu$g/kg)** | 7.368 | 7.312 | 6.537 | 5.420 | 4.685 | 1.497 |
| **Benthic Organism ($\mu$g/g)** | 0.891 | 0.884 | 0.791 | 0.655 | 0.567 | 0.181 |

The EECs can be used to calculate the risk quotient using the following formula:

$$\text{Risk Quotient} = \frac{\text{EEC}}{\text{Toxicity Effect Level (e.g., } LC_{50})}$$

Where:

EEC = Estimated Environmental Concentrations

$LC_{50}$ = Lethal concentration for 50% of a test population.

**4. Ground and Drinking Water**

Pentachlorophenol is not mobile and has a low persistency in the environment. It dissipates through photo-degradation. After leaching out of the utility pole surface and reaching to the soil, pentachlorophenol is adsorbed to the soil particles. Pentachlorophenol has a very low solubility. Because of its affinity for soil particles, pentachlorophenol will not move downward into the ground water. Pentachlorophenol moves into surface waters adsorbed to the soil particles through runoff. Therefore, the effects of pentachlorophenol on ground and drinking water will be minimal.

## 5. Uncertainties and Limitations

- PRZM3-EXAMS models are developed for agricultural pesticides and use of these models to estimate the transport and fate of Pentachlorophenol involved several assumptions which may not be realistic when these models are used for their original purpose.

- The input scenario for the PRZM3 model is very conservative. The amount of the rainfall and runoff from the field in Mississippi Delta area is higher than average for other parts of the United States.

- The number of pentachlorophenol treated utility poles (20 poles)in a ten hectare field used in the scenario is high and very conservative.

- The pond used in the EXAMS model has no outlet. It is also assumed that all the runoff water from the field will end up in the pond. These assumptions makes this scenario very conservative.

- The concentration of pentachlorophenol leached out of the utility pole and entered into the soil is the maximum amount calculated as described in this report. These calculations assumes that most of the pentachlorophenol leaches out of the utility pole in a very short period of time after the pentachlorophenol treated pole is installed. However, in the real conditions in the field it takes several years for the pentachlorophenol to leach out of the surface of the wooden utility pole. Therefore, the results reported here are very conservative.

## V. SCIENCE ASSESSMENT - HUMANS

### A. Human Risk Assessment

#### 1. Hazard Assessment

The toxicological data base for Pentachlorophenol is **adequate** and will support reregistration eligibility. However, an acute inhalation toxicity study is required for the technical test material to assess acute inhalation hazards. A waiver was previously granted for a 90-day inhalation toxicity study, but the issue of acute inhalation toxicity still needs to be addressed.

#### a. Acute Toxicity

The following table summarizes the acute toxicity of pentachlorophenol. It is noted that the studies cited are older data, in which the test material may contain measurable concentrations of contaminants such as hexachlorodioxins.

| Guideline No. | Study Type | MRID # | Results | Toxicity Category |
|---|---|---|---|---|
| §81-1 (OPPTS 870.1100) | Acute Oral Toxicity | 00101715 | LD50 = 155 mg/kg (M); LD50 = 137 mg/kg (F) | II |
| §81-2 (OPPTS 870.1200 | Acute Dermal Toxicity | 00101715 | LD50 > 3980 mg/kg | IV |
| §81-3 | Acute Inhalation Toxicity | no acceptable study available | | |
| §81-4 (OPPTS 870.2400) | Primary Eye Irritation | 00101715 | Corneal involvement at day 7 post-instillation | II |
| §81-5 (OPPTS 870.2500) | Primary Dermal Irritation | 00101715 | Moderate irritation at 72 hours post-application | III |

| | | | | |
|---|---|---|---|---|
| §81-6 (OPPTS 870.2600 | Dermal Sensitization | 42594301 | no sensitization observed using Buehler method | N/A |

In addition to the above data submitted and reviewed by the Agency, numerous studies available in the scientific literature have also examined the acute toxicity of pentachlorophenol. For acute oral toxicity, the literature studies indicate an acute oral LD50 range of 100-400 mg/kg; acute dermal LD values in the range of 885 mg/kg and above, and acute inhalation LC50 values in the range of 0.21-2.2 mg/L. Literature data on eye irritation classify technical pentachlorophenol as a moderate to severe eye irritant with irreversible corneal opacity, while data on dermal irritation classify pentachlorophenol technical as a mild dermal irritant. These literature data are consistent with the above Agency reviewed data, with the exception of inhalation toxicity, for which no Agency data are available.

The Pentachlorophenol Task Force contends that waivers for both acute and subchronic inhalation testing were granted by EPA, and that an extensive effort was made to develop methods to conduct inhalation studies. This effort was without success, based on an inability to generate consistent chamber concentrations of pentachlorophenol. The Task Force has also cited label instructions (based on EPA Position Document 4 for Wood Preservatives) requiring the use of respirators and protective clothing for applicators entering pressure treatment cylinders and other related equipment contaminated with wood treatment formulation.

The Risk Assessment and Science Support Branch, Antimicrobials Division, has reviewed the documents in its possession regarding requests for waivers of inhalation toxicity data requirements, attempts at generating respirable atmospheres of pentachlorophenol, and conclusions reached in the Position Document 4 for Wood Preservatives. Several difficulties were apparently encountered in the attempt to generate respirable particles of pentachlorophenol. However, it is noted that in a letter from Argus Research Laboratories, a system was developed which "proved amenable to our generation of a concentrated atmosphere of respirable-sized particles of pentachlorophenol." It is not known with certainty that this test system was ever extensively tested; apparently, a preliminary study using concentrations of 0.15, 0.30, and 0.50 (units not specified), but concentrations of the test material apparently overlapped such that no dose-response relationships could be determined.

The concentrations used in the preliminary study were not apparently followed up to determine whether a dose-response could be obtained. These initial concentrations were somewhat closely spaced and may have led to the inability to distinguish a dose-response by the very doses selected for the preliminary study. The results of the preliminary study were apparently never fully reported to the Agency; mortality was reported, but to what extent this occurred is not known. Moreover, the registrant is in apparent possession of a system which can generate respirable particles of pentachlorophenol and did not specify the concentration at which

aggregation of particles occurred such that particles were no longer respirable. It is the position of the Antimicrobials Division that an acute inhalation toxicity study be conducted with pentachlorophenol based on the knowledge that a system exists to study inhalation toxicity.

### b. Subchronic Toxicity

**Available studies are adequate to satisfy subchronic testing requirements for this chemical.**

Subchronic Dermal Toxicity in Rats

In a 90-day dermal toxicity study (MRID # 43182301) pentachlorophenol (88.9% a.i.) was administered by dermal occlusion to groups of 10 male and female Sprague-Dawley rats for 6 hours/day, 5 days/week, at doses of 0, 100, 500, or 1000 mg/kg/day. No statistically significant differences in group mean body weight or weight gain were observed at any treatment level, nor were there any treatment related mortalities or clinical signs of toxicity. Dermal irritation consisting of erythema (grade 1-2) at 100 mg/kg/day and erythema (grade 1-4) at 500 and 1000 mg/kg/day was observed during weeks 2-5 of the study. The magnitude of the dermal irritation decreased after week 5. There was no dermal irritation observed in control rats. At the 500 and 1000 mg/kg/day dose levels, increased total white blood cell counts and absolute lymphocyte counts were observed in female rats. Decreased platelet count was observed at all dose levels in female rats. The hematological effects were ascribed to dermal irritation and not to systemic effects of the test chemical. Significant increases in alanine and aspartate aminotransferase activities were observed in male and female rats at the 500 and 1000 mg/kg/day dose levels, while increased cholesterol was observed at these dose levels in females only. Significant increases in absolute liver weight were observed at the 1000 mg/kg/day dose level for both males and females. The increase in liver weight was thought to be the result of enzyme induction. Kidney weights were increased at the 1000 mg/kg/day dose in males, and at the 500 and 1000 mg/kg/day dose levels in females. At necropsy, tan crusty areas, acanthosis of the epidermis, and chronic inflammation of the dermis was noted at the treatment site in all treatment groups of both sexes. An increased incidence of hepatocellular degeneration accompanied by chronic inflammation was observed at 500 and 1000 mg/kg/day in both males and females. Based on the results of this study, the Systemic LOEL = 500 mg/kg/day (enzyme induction and minimal to mild hepatocellular degeneration and chronic inflammation in males and females), and the Systemic NOAEL = 100 mg/kg/day.

This study is classified as **acceptable** and satisfies the guideline requirement (§82-3) for a subchronic dermal toxicity study in rats.

### c. Chronic Toxicity

**Available studies are adequate to satisfy chronic toxicity and carcinogenicity testing**

**requirements for this chemical.**

Chronic toxicity in dogs

In a chronic toxicity study, (MRID 43982701) pentachlorophenol (90.9% a.i.) was fed (gelatin capsules) to four beagle dogs/sex/dose at dose levels of 0, 1.5, 3.5, or 6.5 mg/kg/day for 52 weeks. At 6.5 mg/kg/day, one male and one female dog were sacrificed in extremis on days 247 and 305, respectively, due to significant clinical toxicity (significant weight loss, lethargy, marked dehydration, vomiting, icterus). Group mean body weight in surviving male dogs at the 6.5 mg/kg/day dose was decreased by 15% at week 13, and 21% at study termination. In females, a 19% decrease in group mean body weight was observed at week 13, and bodyweight remained significantly decreased until study termination. Decreased red cell count (16%), hemoglobin (9%), and hematocrit (8%), was observed in males at the 6.5 mg/kg/day dose at week 13. These decreases were also observed at week 26 and at necropsy. In females, significant decreases of 10-17% in these hematologic parameters were observed at 6.5 mg/kg/day from week 26 until study termination. Activities of alkaline phosphatase, alanine aminotransferase, and aspartate aminotransferase were significantly elevated for both sexes throughout the study at the 6.5 mg/kg/day dose. Gamma-glutamyltranspeptidase activity was increased in male dogs at week 13 by 45%. Absolute and relative liver weight in males and females was elevated by 32% and 49% over control at 6.5 mg/kg/day. Absolute and relative thyroid weight was also increased significantly in females at the 6.5 mg/kg/day dose. Gross stomach lesions consisting of multiple, raised mucosal foci were observed in increased incidence in all treated groups of male and female dogs with only one female control dog showing a similar lesion. Dark, discolored liver was also observed in increased incidence in male and female treated dogs, but a dose-response was observed only for males. Microscopically, increased incidence of lymphocytic mucosal inflammation was observed in the stomach of treated males and females. The lesion was present in all treated and control groups, but the severity of the lesion was increased, especially at the 3.5 and 6.5 mg/kg/day doses.

Although effects of toxicologic relevance were observed at the 1.5 mg/kg/day dose level (increased liver weight in female dogs, increased incidence of pigmentation of the liver, and increased incidence of lymphocytic mucosal inflammation of the stomach in both sexes), and therefore it can be concluded that a NOAEL was not established for this study, the 1993 EPA Rejection Rate Analysis - Toxicology document states that "in theory a NOAEL would not be necessary if: ...4) the RfD can be determined with the addition of an extra uncertainty factor from a LOEL (lowest-observed-effect-level)." From the effects observed at the 1.5 mg/kg/day dose level, it can be observed that the liver of dogs is also a target organ of pentachlorophenol induced toxicity, similar to that observed in other species. Thus, the endpoints in this present study can be used to support an RfD. Previously, the RfD was determined from a chronic toxicity study in rats in which pigmentation of the liver and kidneys was observed at a dose of 10 mg/kg/day. As similar effects were observed at a lower dose level in the present study, use of an extra uncertainty factor in determining the RfD would make the present study acceptable

for risk characterization purposes.

The present study is considered **acceptable** and satisfies the guideline requirement (§83-1; OPPTS 870.4100) for a chronic toxicity study in non-rodents.

**d. Carcinogenicity**

Carcinogenicity in Mice

In a study conducted by the National Toxicology Program, diets containing 100 or 200 ppm technical grade pentachlorophenol or 100, 200, or 600 ppm Dowicide EC-7 were administered to male and female B6C3F1 mice for 103 weeks. Average daily doses for mice receiving technical grade pentachlorophenol were 18 and 35 mg/kg/day for males, and 17 and 35 mg/kg/day for females. In the EC-7 dose groups, average daily doses were 18, 38, and 118 mg/kg/day for males, and 17, 34, and 114 mg/kg/day for females. Two groups of 35 male and 35 female mice served as controls. Both technical grade pentachlorophenol and Dowicide EC-7 contain 90% pentachlorophenol, but differ in the level of contaminants. These contaminants and their levels are summarized in the following table:

| Contaminant | PCP Formulation | |
|---|---|---|
| | EC-7 | Technical grade PCP |
| tetrachlorophenol | 9.4% | 3.8% |
| hexachlorobenzene | 65ppm | 50 ppm |
| TCDD | < 0.04ppm | non-detectable |
| HxCDD | 0.19ppm | 10.1ppm |
| HpCDD | 0.53ppm | 296ppm |
| OCDD | 0.69ppm | 1386ppm |
| PCDF | non-detectable | 1.4ppm |
| HxCDF | 0.13ppm | 9.9ppm |
| HpCDF | 0.15ppm | 88ppm |
| OCDF | non-detectable | 43ppm |

Reduced survival in male control mice in the technical grade pentachlorophenol dose groups was observed in this study, based on urinary tract infections resulting from injury sustained during fighting among gang-housed mice. Low-dose female mice in the EC-7 dose groups showed decreased survival vs controls. No other effects on survival were observed in this study. In all treated groups of both the EC-7 and technical grade dose groups, a variety of non-neoplastic liver pathology was observed. At the lowest dose, incidence of acute diffuse necrosis was 87% in technical grade treated males, 98% in EC-7 treated males, 90% in

technical grade treated females, and 42% in EC-7 treated females. The incidence of bile duct hyperplasia at the low dose of technical grade pentachlorophenol was 47% in males, but only 6.3% in males treated with EC-7 at the low dose. The incidence of clear cell foci was increased to 23% and 40% in technical grade and EC-7 treated males at the low dose, respectively. In females, clear cell foci were not observed at the low dose. The incidence of diffuse hematopoietic cell proliferation in males receiving technical grade pentachlorophenol was 17%, 65%, and 39% at the 0, 18, and 35 mg/kg/day dose levels, respectively, and was 6%, 31%, and 23% in the corresponding female dose groups, respectively. At the high dose of EC-7, male and female mice were observed with increased incidence (96%) of acute focal inflammation of the mucosal glands and focal metaplasia of the olfactory epithelium. In those mice receiving pure pentachlorophenol or EC-7, a metaplastic lesion of the nasal cavity consisting of an increase in the number of goblet cells and flattening of the epithelium was observed in increased incidence at the high dose.

Incidence of hepatocellular carcinoma was significantly increased (26%) in male mice at 35 mg/kg/day in the technical grade pentachlorophenol dose group (vs 7% in control), and was also significantly elevated in male mice at 38 and 118 mg/kg/day EC-7 (18 and 19% respectively vs 3% in control). Females were not observed with increased hepatocellular carcinoma in any dose group. The combined incidence of hepatocellular adenoma/carcinoma was significantly increased at all dose levels in male mice of both the technical grade and EC-7 pentachlorophenol dose groups (57% and 80% incidence in technical grade males at 18 and 35 mg/kg/day vs control; 42, 55, and 72% incidence in EC-7 males vs 18% in control, $p < 0.01$) with a significant dose-related trend. In females, the high dose EC-7 group was observed with increased incidence of combined hepatocellular adenoma/carcinoma combined (65% vs 3% in control, $p < 0.01$), also with a significant dose-related trend. Pheochromocytoma incidence was significantly increased in technical grade pentachlorophenol male mice at both the 18 and 35 mg/kg/day dose levels (24% and 52% respectively vs 0% in controls), as well as in the 38 and 118 mg/kg/day dose groups of males receiving EC-7 pentachlorophenol (54% and 96% respectively vs 3% in control. Female mice receiving technical grade pentachlorophenol showed increased incidence of pheochromocytoma at the high dose (78% vs 0% in control) with a significant dose-related trend. The combined incidence of hemangioma/hemangiosarcoma was significantly elevated in high dose female mice receiving either technical grade pentachlorophenol (12% vs 0% in control) or EC-7 pentachlorophenol (18% vs 0% in control) with a significant dose-related trend for both.

Under the conditions of this study, the NTP concluded that there was clear evidence of carcinogenicity for technical grade pentachlorophenol in male mice, based on increased incidence of adrenal medullary and hepatocellular neoplasms. There was some evidence of carcinogenic activity for female mice given technical grade pentachlorophenol, based on increased incidence of hemangiosarcomas and hepatocellular neoplasms. Clear evidence of carcinogenic activity was also concluded for pentachlorophenol EC-7, based on increased incidence of adrenal medullary and hepatocellular neoplasms in male mice, and increased incidence of adrenal medullary and hepatocellular neoplasms in female mice.

It is noted that the hemangiosarcomas, hepatocellular tumors, and adrenal tumors observed in this study formed the basis for the carcinogenic classification of pentachlorophenol by the Health Effects Division, Office of Pesticide Programs, as a B2 (probable) human carcinogen.

Chronic Toxicity / Carcinogenicity in Rats

In this study conducted by the National Toxicology Program, male and female Fischer 344 rats (50/sex/dose) were exposed to pentachlorophenol (approximately 99% pure, with no detectable levels of chlorinated dibenzodioxin, dibenzifuran, diphenyl ether, or hydroxydiphenylether) in feed for 105 weeks at dose levels of 200, 400, or 600 ppm (equivalent to 10, 20, and 30 mg/kg). Stop exposure groups of 60 male and 60 female rats received 1000 ppm pentachlorophenol (60 mg/kg) in feed for 52 weeks and then untreated diet until study termination at 2 years. At study termination, a significantly increased incidence of malignant mesothelioma originating from the tunica vaginalis was present in 1000 ppm males compared to controls, and the incidence exceeded the historical control range. Nasal squamous cell carcinomas were present in a single control male, three males at 200 pm, one male at 400 ppm, and 5 males at 1000 ppm. Historical control incidence was exceeded at 1000 ppm. At 7 months, an increased incidence of centrilobular hepatocyte hypertrophy and cytoplasmic hepatocyte vacuolization was observed in 1000 ppm males and females. At 2 years, 1000 ppm males were observed with increased incidence of chronic liver inflammation and basophilic focus, while male rats at all dose levels were observed with increased incidence of hepatodiaphragmatic nodule and cystic hepatocyte degeneration. Hepatodiaphragmatic nodules are developmental anomalies commonly observed in F344 rats, and increased incidences in this study were not exposure concentration related. Cystic degeneration was considered treatment related but is of uncertain biological relevance. Female rats at 2 years did not show dose-related increases in non-neoplastic changes of the liver. Under the conditions of this study, it was concluded that there was some evidence of carcinogenicity of pentachlorophenol in male Fischer 344 rats, based on increased incidences of mesothelioma and nasal squamous cell carcinoma. There was no evidence of carcinogenic activity of pentachlorophenol in female rats in this study. Statistical analysis of pentachlorophenol-induced tumors from the Science Analysis Branch, Health Effects Division (memorandum from Lori Brunsman, Statistician, to William Burnam, Chief, Science Analysis Branch, dated 1/20/99) showed a significant increasing trend as well as a statistical pair-wise significance ( $p < 0.01$) for mesotheliomas in multiple organs for males at the 1000 ppm dose level vs control. Male rats also had a significant trend and significant pair-wise difference ( $p < 0.05$) in incidence of nasal cavity squamous cell carcinomas vs control. There were no compound-related tumors in female rats.

**e. Developmental Toxicity**

**Available developmental toxicity studies are adequate to satisfy guideline requirements.**

In a developmental toxicity study, pregnant Crl:CD BR VAF/plus rats (25/dose) received oral administration of Pentachlorophenol (88.9%) at doses of 0, 10, 30 or 80 mg/kg/day during

gestation days 6 through 15. For maternal toxicity, the NOAEL was 30 mg/kg/day and the LOEL was 80 mg/kg/day based on reduced body weight gain during the dosing period (79% of control) and for the entire gestation period (88% of controls). Developmental toxicity observed at 80 mg/kg/day included: significant increase in the number of resorptions (mainly early) with a corroborative decrease in litter size; reduced mean fetal weights; significant increase in the number of fetuses with external, visceral and/or skeletal malformation/variations (hydrocephaly, diaphragmatic hernia, and dilation of the renal pelvis); and significant increase in the number of litters (22 of 23) that contained fetuses (6 of 22) with skeletal anomalies. The most common were vertebral structural variations and incomplete ossification. For developmental toxicity, the NOAEL was 30 mg/kg and the LOEL was 80 mg/kg/day based on increased resorptions, reduced fetal weight and skeletal malformations/variations.

In a study conducted by Schwetz et al. (Toxicol. Appl. Pharmacol 28: 151-161, 1974), doses of 0, 5, 15, 30, and 50 mg/kg/day commercial grade (88.4% a.i.) or purified (>98% a.i.) pentachlorophenol prepared in corn oil were administered to groups of pregnant Sprague-Dawley rats on gestation days 6-15 inclusive. For purified pentachlorophenol, the number of rats per group was as follows: control, 33 rats; 5 mg/kg, 15 rats; 15 mg/kg, 18 rats; 30 mg/kg/, 20 rats; 50 mg/kg, 19 rats. For the commercial grade of pentachlorophenol: 5 mg/kg, 18 rats; 15 mg/kg, 17 rats; 30 mg/kg, 19 rats; 50 mg/kg, 15 rats. Additional groups of rats were administered 0 or 30 mg/kg/day pentachlorophenol (type of a.i. not specified) on days 8-11 or 12-15 of gestation.

Maternal toxicity from purified pentachlorophenol was evidenced by decreased maternal weight gain at the 30 and 50 mg/kg dose groups for days 6-21 of gestation (74% decrease vs control). For the commercial grade, weight gain was decreased 43% at the 50 mg/kg dose, and by 22% at the 30 mg/kg dose. Weight gain appeared more significantly affected by purified pentachlorophenol. No other significant signs of maternal toxicity were observed. Fetal incidence of resorption was significantly increased at 30 and 50 mg/kg purified and commercial grade pentachlorophenol. The report stated that the fetal and litter incidence was also significantly increased at 15 mg/kg commercial grade pentachlorophenol, but the fetal incidence (7 and 8% at control and 15 mg/kg/day) as well as the litter incidence (55 and 64% at control and 15 mg/kg/day) did not appear biologically meaningful. Fetal body weight was reported significantly decreased for commercial grade pentachlorophenol at 30 and 50 mg/kg/day and at 30 mg/kg/day for purified pentachlorophenol, but actual values were not reported. Crown-rump length was significantly decreased at 30 mg/kg/day purified pentachlorophenol. The litter incidence of subcutaneous edema, lumber spurs, and supernumerary, lumber or fused ribs was significantly increased at 30 and 50 mg/kg commercial grade pentachlorophenol, but the data did not indicate a dose-response, i.e. the number of litters affected with subcutaneous edema, rib and vertebral abnormalities were greater at 30 mg/kg than at 50 mg/kg for the commercial grade of pentachlorophenol and for the purified pentachlorophenol. The number of litters at the high dose of purified pentachlorophenol was also severely limited (only 2 litters at this dose). Resorptions were

significantly increased when pentachlorophenol (both grades) was administered on gestation days 8-11, but not on gestation days 12-15.

Based on the results of this study, the Maternal NOAEL can be considered as 15 mg/kg/day, based on body weight effects, for both grades of pentachlorophenol. The Developmental NOAEL would appear to differ according to grade of pentachlorophenol used. Limited data for purified pentachlorophenol at the 50 mg/kg/dose hampers evaluation of a NOAEL and LOEL for this grade. For the commercial grade of pentachlorophenol, the NOAEL can be considered as 15 mg/kg/day, and the LOEL as 30 mg/kg/day, based on decreased fetal body weight and crown-rump length. The responses observed at the low dose of commercial grade pentachlorophenol for fetal anomalies (i.e. lumber spurs, anomalous ribs, subcutaneous edema) do not show a dose-response pattern at the higher doses of pentachlorophenol, and are also limited by reduced number of litters at the high dose.

In a study conducted by Welsh et al. (Fd. Chem. Toxic. 25(2): 163-172, 1987), groups of male and female Sprague-Dawley rats (20/group) were placed on diets containing purified pentachlorophenol at dose levels of 0, 60, 200, or 600 ppm (0, 4, 13, and 43 mg/kg/day reported for females; no actual intake data for males). These test diets were administered for 181 days. At the end of the subchronic phase, male and female rats were mated for teratological evaluation following subchronic exposure to pentachlorophenol. Body weight gain in maternal rats exposed to PCP was significantly decreased at the high dose (76% from control). Pregnancy rates were low in all dose groups (77.5, 55, 84.2, and 85 for the 0, 60, 200, and 600ppm dose groups, respectively) but, according to the report, there was no effect on fertility. Decreased number of viable fetuses (due to early death) was observed at the 600 ppm dose level of PCP, as well as an increase in total litter resorptions. Body weight of fetuses appeared decreased at the 200 ppm dose level; analysis at the 600 ppm dose level was not complete, due to an alteration in the sex ratio at this dose (100% male sex ratio at this dose as reported). There were no reported alterations in external or sternebral observations at any dose level tested in this study. Increased incidence of misshapen centra was reported at 200 ppm PCP, but the high dose could not be analyzed due to inadequate numbers of fetuses. The incidence of total skeletal variations was reported increased at the 200 ppm dose level for PCP. The results of this study demonstrate toxicity of PCP at 200 ppm (13 mg/kg/day) in the form of increased percentage of female rats with 2 or more resorptions. However, this study is hampered by a lack of fetal data at the high dose, and inconsistent and low percentages of pregnancy at each dose level of PCP tested. The data appear to point to a definitive toxic effect of PCP at the 43 mg/kg/day dose level on both maternal and fetal rats, with decreased weight gain in maternal rats, and decreased viable fetuses, increased early deaths , and increased resorptions at the high dose.

Developmental Toxicity in Rabbits

In a developmental toxicity study (MRID # 43091701), inseminated New Zealand White rabbits (20 rabbits/dose) were administered pentachlorophenol by gavage at doses of 0, 7.5,

15, and 30 mg/kg/day on gestation days 6-18 inclusive. Cesarean section examinations were performed on all surviving does on gestation day 29, followed by external, visceral, and skeletal examination of all fetuses. There were no maternal deaths or signs of maternal toxicity at any dose level. Significantly reduced weight gain was observed in the high dose group on gestation days 6-9 (loss of 40g vs gain of 20 g in controls), and in the mid and high dose groups for gestation days 9-12 (gain of 20 g at the mid and high dose vs gain of 50 g in controls). These changes were considered minimal as the differences in weight gain were equivalent to only 0.5-1% of mean body weight. A consistent reduction in mean food consumption (71-90% of control value on gestation days 9-12) was observed at the high dose. Weight gain was comparable for treated and control rabbits following the treatment interval. A slight but non-significant dose-related decrease in litter size was observed, corresponding to a decrease in implantations/litter for the treated does. There were no statistical differences in the number of treated litters vs control when the incidences for all observations of external, visceral, and skeletal effects were combined. Although the incidence of affected litters was not statistically significant, a significantly greater ($p < 0.01$) number of individual fetuses in the 15 mg/kg litters contained interfrontal ossification sites compared to controls. Based on the results of this study, the Maternal toxicity NOAEL = 15 mg/kg/day, and the Maternal toxicity LOEL = 30 mg/kg/day, based on minimally reduced body weight gain and consistent reductions in food consumption during treatment. The Developmental toxicity NOAEL = 30 mg/kg/day; a Developmental toxicity LOEL was not identified.

This study is classified as acceptable and satisfies the guideline requirement (83-3b) for a developmental toxicity study in non-rodents. It is noted that the guideline is minimally met, based on minimal maternal toxicity and no developmental toxicity. It is apparent that the rabbits could have tolerated higher doses of the test chemical. However, repeating the study would not necessarily add useful information

**f. Reproductive Toxicity**

In a 2-generation reproduction study (MRID 44464101), pentachlorophenol (88.9% a.i.; lot no. EL-604) in corn oil was administered to 30 male and female Sprague-Dawley rats/sex/dose by gavage at dose levels of 0, 10, 30, and 60 mg/kg/day. P1 male and female rats were given the test material once daily at least 70 days prior to cohabitation and continuing through the day before sacrifice. F1 generation rats may have been exposed *in utero* during gestation and via maternal milk during the postpartum period. The day after weaning, F1 generation rats were administered the test material by gavage at the same dose levels as P1 rats and were continued until one day prior to sacrifice. F2 pups were exposed possibly indirectly during maternal gestation or via maternal milk. In addition to standard parameters, estrous cycling and sperm morphology and function were measured. At 60 mg/kg/day pentachlorophenol, body weight and body weight gain of P1 and F1 parental rats was significantly decreased from control over the period of treatment pre-mating. Gestational and lactational body weights of P1 and F1 female rats were also significantly decreased. Fertility index and number of litters were decreased in both generations at 60 mg/kg/day pentachlorophenol. Days to vaginal patency

were significantly increased in P1 female pups, as was days to preputial separation in P1 male pups. Estrous cycling was not significantly affected in either P1 or F1 females, but in F1 males, the number of sperm observed with a broken flagellum was significantly increased at 60 mg/kg/day, and the average testicular spermatid count was significantly decreased at 60 mg/kg/day. Testis weight in F1 generation males was also decreased.

Decreased brain weight (4-8%) and increased liver weight (20-33%) were observed in both P1 and F1 male and female rats at 60 mg/kg/day. Macroscopic pathology (enlarged liver) and microscopic pathology (centrilobular hypertrophy, subacute inflammation, single cell necrosis, pigment deposition) were observed in increased incidence at 60 mg/kg/day. The single cell necrosis and pigment deposition were considered related to treatment. Mean litter size, number of live pups, and viability index were significantly reduced in the P1 and F1 pups. Decreased weight of the liver, brain, spleen, and thymus were observed in F2 pups at 60 mg/kg/day.

At 30 mg/kg/day, body weight and weight gain decreases of approximately 11% were observed in F1 female parental rats. Significant decreases in average testicular spermatid count and testis weight were observed in F1 male parental rats.

Based on the data in this study, the Systemic NOAEL = 10 mg/kg/day for male and female parental rats. The Systemic LOEL = 30 mg/kg/day for male and female rats, based on decreased body weight and weight gain in F1 generation parental rats, and adverse testicular effects in F1 male rats (decreased testis weight, decreased spermatid count).

The reproductive NOAEL = 10 mg/kg/day in this study. The reproductive LOEL = 30 mg/kg/day, based on decreased group mean litter weight.

This reproductive study in the rat is classified **unacceptable** and does not currently satisfy the guideline requirement for a 2-generation reproductive study (OPPTS 870.3800, formerly §83-4) in rats. The study may be upgraded upon receipt and review of the range-finding toxicity study, which contains data pertinent to evaluation of the homogeneity and stability of the dosing solutions used in the main study.

Agency's Position on Developmental Toxicity on Pentachlorophenol

In Position Document 4 (USEPA, 1984), the Agency required a teratogenicity / fetotoxicity warning on the labels for all uses of pentachlorophenol and salts of pentachlorophenol. This labeling was based on results obtained from literature studies using pentachlorophenol (Schwetz et al, 1974 [see above]; Larsen et al., Environmental Lett. 10(2): 121-128, 1975; Fahrig et al., In K.R. Rao (Ed.), Pentachlorophenol: Pentachlorophenol Chemistry, Pharmacology, and Environmental Toxicology, pp. 325-338, 1978) in which toxicity to the developing fetus was observed. These toxic responses (decreased body weight, fetal resorption increase, decreased crown-rump length) were observed in the presence of maternal toxicity, or were implied to be an indirect effect of maternal toxicity. The more recent data submitted and

reviewed by the Agency as summarized above supports this same conclusion. The Agency recognizes (Position Document 2/3, p. 261) that "pure pentachlorophenol is not a teratogen." However, it is recognized that the contaminants hexachlorodioxin and 2,3,7,8 tetrachlorodioxin are considered teratogenic chemicals. For this reason, and with the knowledge that hexachlorodioxin is a contaminant of pentachlorophenol, the warning labels on pentachlorophenol formulations are justified. This conclusion is supported by the October 29, 1998 meeting of the Health Effects Division Developmental and Reproductive Toxicity Science Advisory Review Committee, in which the issue of developmental toxicity of pentachlorophenol was presented as described above. There was consensus among the committee members that the developmental toxicity of pentachlorophenol is based upon the presence of the dioxin contaminants, and that pentachlorophenol itself is not teratogenic. It was also agreed by the committee that the teratogenicity warning should remain as part of the label for pentachlorophenol, based upon the presence of the dioxin contaminants.

**g. Mutagenicity**

The data used by the Agency in determining the mutagenicity of pentachlorophenol consists of studies performed and/or referenced by the National Toxicology Program as well as data submitted to the Agency and reviewed. According to the summary presented by the NTP (T.R. # 349), results of bacterial tests for induction of gene mutation or growth inhibition due to DNA damage are negative with the exception of one study using phenobarbital or 5,6-benzoflavone induced rat liver S9. A recent study from the literature (Gopalaswamy and Nair, Bull. Environ. Contam. Toxicol. 49: 300-305, 1992) reports positive mutagenicity of pentachlorophenol in the presence of S9 in an Ames assay.

Several studies have investigated structural chromosome aberrations produced by treatment with pentachlorophenol. In a Drosophila assay, no effect of pentachlorophenol was observed on chromosomal nondisjunction or chromosome loss. In an assay with Chinese hamster ovary cells, (NTP, T.R. 349, 1989, and Galloway et al., Environ. Molec. Mutagen. 10 [suppl. 10], 1987) pentachlorophenol was negative for structural chromosome aberrations in the absence of S9. In the presence of S9, chromosomal aberrations were distinctly increased at 100 $\mu$g/ml pentachlorophenol, the highest dose tested (33% of cells vs 3.0% in control), but a repeat experiment yielded only equivocal results (12% vs 3.0%). Sister chromatid exchange was considered weakly positive in the absence of S9 (at concentrations of 0, 1, 3, 10, and 30 $\mu$g/ml pentachlorophenol, SCE's/cell of 8.3, 8.2, 10.0, 9.0, and 9.4), and negative in the presence of S9. In a study by Ishidate (Data Book of Chromosomal Aberration Test In Vitro, Elsevier, 1988), pentachlorophenol at concentrations up to 60 $\mu$g/ml did not induce chromosomal aberrations in Chinese hamster lung fibroblasts. Changing to an intermittent treatment regimen and using concentrations of 240-300 $\mu$g/ml resulted in an increased fraction of cells (20-30%) with chromosomal aberrations in the absence and presence of exogenous metabolizing enzymes. Using human lymphocytes from healthy donors, Ziemson (Int. Arch. Occup. Environ. Health 59: 413-417) found no increase in sister chromatid exchange or chromosomal aberrations following

incubation with technical grade sodium pentachlorophenol up to 90$\mu$g/ml.

In a study submitted by the Pentachlorophenol Task Force and reviewed by the Agency (MRID # 43911301), CD-1 mice (5/sex/dose) were treated by gavage with pentachlorophenol (8.9% a.i.) at doses of 24, 60, or 120 mg/kg for male mice and 20, 50, or 100 mg/kg for female mice. Bone marrow cells were harvested at 24, 48, and 72 hours post-treatment. Triethylenemelamine was used as a positive control. Clinical signs of toxicity (prostration, ataxia, salivation, convulsions, and death) were observed in a preliminary study at doses of 500 mg/kg and above. In the main study, two males form the 24 hour time pint and 1 male from the 72 hour time point died approximately 8 hours after dosing. Examination of bone marrow cells showed no significant increase in the frequency of micronucleated polychromatic erythrocytes at any time point.

This study is classified as acceptable and satisfies the guideline requirement (84-2) for an in vivo cytogenetics assay.

Conclusions

The available evidence for gene mutations in bacterial systems suggests that pentachlorophenol is largely devoid of positive effects with the exception of one published report, in which a positive response was noted. Pentachlorophenol is also observed to be weakly clastogenic, with chromosomal aberrations observed using Chinese hamster ovary cells in the presence of rat liver S9. In contrast to the data for pentachlorophenol, data for the metabolite tetrahyhdroquinone (THQ) show positive effects on Chinese hamster V79 cells (increase in frequency of thioguanine-resistant mutants; Jansson and Jansson, Mutation Res. 260: 83-87), increase in micronuclei using V79 cells (Jansson and Jansson, Mutation Res. 279: 205-208), covalent binding to DNA (Witte et al., Mutation Res. 145: 71-75), and induction of DNA single-strand breaks (Witte et al., Mutation Res. 145: 71-75; Ehrlich, W.: Mutation Res. 244: 299-302). THQ has been thought to be involved in some or all of the mutagenic and carcinogenic effects of pentachlorophenol. Previous data suggesting that only rodent liver enzymes are capable of producing THQ are incorrect, as recent data have shown production of THQ both in vitro and in vivo.

**h. Metabolism**

The Agency has relied on data in the published scientific literature in support of the requirement for metabolism data on pentachlorophenol. The disposition and biotransformation of pentachlorophenol have been studies in a variety of species. Pentachlorophenol has been shown to be readily absorbed by oral, inhalation, and dermal routes of exposure (Reigner et al., Pharmaceut. Res. 9: 1053-1057, 1992; Yuan, JH et al., Xenobiotica 24(6): 553-560, 1994; Hoben, Bull. Environ.Contam. Toxicol 15: 466-474, 1976). Peak plasma levels have been shown to occur in the range of 1.3-6 hours in experimental animals in an oral dose range of 0.1-15 mg/kg, while in humans, maximum plasma concentration of pentachlorophenol occurred at 4 hours following a single 0.1 mg/kg oral dose. Absorption from dermal exposure has been shown

to range from 24% in rhesus monkeys (Wester et al., Fundam. Appl. Toxicol. 20: 68-71, 1993) to 40% in rats (EPA MRID # 00259257).

Biotransformation of pentachlorophenol has also been examined in several studies published in the scientific literature. Reigner et al. (Xenobiotica 21(12): 1547-1558, 1991) observed a 60% recovery in urine from intravenous or oral administration of 2.5 mg/kg pentachlorophenol, mainly as conjugated pentachlorophenol and conjugated tetrahydroquinone. In two earlier studies by Ahlborg (Arch. Toxicol 40: 45-53, 1978) and Braun (Toxicol. Appl. Pharmacol. 41: 395-406, 1977), glucuronides of pentachlorophenol were the major urinary metabolites observed after oral doses of 10 mg/kg and 100 mg/kg respectively. Pretreatment with phenobarbital in the study by Ahlborg et al. enhanced the formation of tetrahydroquinone. In other work by Rener and Hopfer (Renner, G. And Hopfer, C.: Xenobiotica 20: 573-582, 1990), 24 female Sprague-Dawley rats were treated with pentachlorophenol at 53 mg/kg/day for 28 days. The results of this study indicated that pentachlorophenol leads to tetrahydroquinone via 2,3,5,6-tetrachlorophenol as a main degradative pathway, and to trichlorohydroquinone via 2,3,4,6-tetrachlorophenol and 2,3,4,5-tetrachlorophenol via a minor pathway. Pentachlorophenol, tetrachlorophenol, and trichlorophenol can become conjugated with glucuronic acid or sulfate. While previous work in humans has shown the absence of formation of the tetrahydroquinone metabolite, newer published literature in dicates formation of this metabolite both in vitro (Mehmood et al., Chemosphere 33(4): 759-769, 1996) as well as in vivo.

The kinetics of elimination of pentachlorophenol have been published, with half-lives of elimination ranging from 5-6 hours in mice ( Reigner et al., Pharmaceut. Res. 9: 1053-1057, 1992) , 2-11 hours in rats (Reigner et al., Xenobiotica 21(12): 1547-1558, 1991; Braun et al., Toxicol. Appl. Pharmacol. 41: 395-406, 1977) and 72-84 hours in monkeys (Braun and Sauerhoff, Toxicol. Appl. Pharmacol. 38: 525-533, 1976). In humans, mean plasma half-life was calculated to be 30.2 ±4.0 hours following a 0.1 mg/kg oral dose to four male volunteers (Braun et al., in Toxicology and Occupational Medicine [W.B. Diechmann, ed.], pp. 289-296). Limited data on the volume of distribution of pentachlorophenol indicate that in contrast to rats, where the volume of distribution ranges from 116-268 ml/kg, humans show a higher volume of distribution (348 ml/kg). However, only one study was available with human data on volume of distribution.

### i. Neurotoxicity

The Pentachlorophenol Task Force has not committed to perform acute or subchronic neurotoxicity studies on pentachlorophenol. In discussions with the Pesticide Management Regulatory Agency, Health Effects Canada, it was determined that available published scientific literature would be reviewed in an attempt to characterize the neurotoxic potential of pentachlorophenol. The following is a summation of the literature data regarding neurotoxicity of pentachlorophenol.

**In vitro data**

Igisu et al. (1993) and Matsumura et al. (1997) have demonstrated, in vitro, that PCP (Sigma chemical; unknown purity but likely analytical grade) can inhibit human red cell acetylcholinesterase(AchE). Using isolated sciatic nerve-sartorius muscle preparations from toads, Montoya and Quevedo (Comp. Biochem. Physiol. 89C: 377-382) demonstrated a dose-dependent irreversible reduction of endplate potential at the neuromuscular junction using pentachlorophenol concentrations between 0.01-0.1mM. The origin and purity of the pentachlorophenol used in this study was not known.

Axonal conduction, using an *in vitro* preparation of toad de-sheathed sciatic nerve, was shown to be blocked (concentration-time dependent) irreversibly by pentachlorophenol (Sigma chemical; unknown purity but likely analytical grade) at concentrations ranging from 0.3 to 10 mM (Montoya et al., Comp. Biochem. Physiol. 89C: 377-382, 1988). In its ionized form, it appears that pentachlorophenol does not reach the site of action as effectively. Pentachlorophenol was more potent (approximately 2-fold) in causing axonal conduction block than procaine; The ED50 for pentachlorophenol was 1 mM. Pentachlorophenol was also able to cause a dose-time dependent ganglionic synaptic transmission block (also irreversible) at concentrations ranging from 0.003 to 0.03 mM. Pentachlorophenol is believed to have an effect during depolarization as it would interfere with $Ca^{++}$ influx (Montoya et Quevedo, Comp. Biochem. Physiol. 96C, 193-197, 1990).

**In vivo Data**

In a six month toxicity study using B6C3F1 mice (25 male mice and 10 female mice per dose group), diets containing 200, 600, or 1800 technical grade pentachlorophenol ; 200, 600, or 1200 ppm EC-7; 200, 600, or 1200 ppm DP-2; or 200, 500, or 1500 ppm pure pentachlorophenol were administered. The mice in this study were separated in to subgroups for determination of effects on behavior, histopathology, hematology, and clinical chemistry. Behavioral studies included 10 mice per sex per dose group. Examination for the presence of autonomic signs, pinnal, corneal, and righting reflexes; spontaneous motor activity; acoustical startle response; visual placement response; grip strength; and rotarod testing were performed. There were no treatment-related neurobehavioral effects observed at 5 weeks except for those mice receiving technical grade pentachlorophenol, in which a dose-dependent decrease in motor activity and rotarod performance was reported. After 26 weeks exposure, an increase in both motor activity and startle response was observed in female mice. No consistent effects were observed in any of the other behavioral parameters measured for any of the four grades of pentachlorophenol.

In a one-year toxicity study in dogs, (MRID # 43982701), pentachlorophenol (90.9% a.i.) was administered by gelatin capsule to groups of 4 beagle dogs/sex/dose at doses of 0, 1.5, 3.5, and 6.5 mg/kg/day for 52 weeks. There were no signs of a treatment-related effect on nervous system anatomy or function. In a 90-day dermal toxicity study in rats, groups of 10 Sprague-Dawley rats/sex/dose were administered 0, 100, 500, or 1000 mg/kg pentachlorophenol (88.9% a.i.) For 6 hours/day for 91 or 92 days. There were no clinical signs of neurotoxicity in this study, and no reported abnormalities of the brain, spinal cord, or pituitary.

Administration of technical grade pentachlorophenol in drinking water at a concentration of 20 mg/L for 14 weeks resulted in the presence of pentachlorophenol in the brain. Activity of acid protease was increased transiently during week 8 of exposure, superoxide dismutase increased transiently during week 14, and NADPH-diaphorase increased at 18 weeks. Glial cell superoxide dismutase was decreased relative to control at week 7 and 12.

Wistar rats were administered pentachlorophenol (unknown purity) in drinking water at approximately 0, 12, 40, and 118 mg/kg/day for 90-120 days. In rats given 40 mg/kg for 90 days and 118 mg/kg/ for 120 days, degenerative changes in approximately 10% of A and B nerve fibers of the sciatic nerve were observed, as was discontinuation of the myelin sheath by complete separation in several concentric rings, and variable loss of neurotubules, neurofilaments, and other axoplasmic components. It is noted that the doses at which these effects occurred (267 and 800 mg/L) are in excess of the solubility of pentachlorophenol (20 mg/L at 30 °C; 80 mg/L at 25 °C).

**Human Data**

In a study of workers exposed to pentachlorophenol in ambient air at concentrations ranging from 0.3-180 ng/m$^3$ at an average exposure duration of 16 years (range of 4-24 years), nerve conduction velocity of motor and sensory nerves was within normal range. Plasma and urinary levels of pentachlorophenol were stated as 38-1270 ng/L and 8-1224 ng/L, respectively. Clinical findings in chronically exposed individuals have included depression, chronic fatigue, dizziness, sleep disturbances, aggressive behavior, loss of appetite, headache, nausea, diarrhea, fear, thirst, and hyperthermia (Jorens et al., Neurotoxicology 12: 1-8, 1991). Cerbrospinal fluid in neurologic patients was observed to contain measurable levels of pentachlorophenol (0.75±0.49ppb).

In summary, the available data on pentachlorophenol indicate the potential for nervous system effects in vitro (decrease in neuromuscular and ganglionic conduction, inhibition of human red cell cholinesterase). In vivo animal data demonstrate some possible effects after subchronic administration. Clinical signs associated with pentachlorophenol exposure are suggestive of neurotoxicity, but not definitive. Pentachlorophenol is structurally related to known neurotoxicants such as hexachlorobenzene and hexachlorophene, which have ben demonstrated to cause swelling of the myelin sheath and/or convulsions after stimulation with auditory or physical stimuli.

**j. Immunotoxicity**

The immunotoxicity of pentachlorophenol has been investigated in several species, as reported in the scientific literature. Lactating cattle fed pentachlorophenol (0.2 mg/kg/day for 75-84 days followed by 2.0 mg/kg/day for 52-62 days) showed no statistically significant changes in T- and B-cell subpopulations, serum IgG, IgA, IgM, mitogen-induced lymphocyte blastogenesis, or antibody response to SRBC (Forsell et al., Tox. Environ. Health 50: 287-292, 1981). Kerkvliet et al. (Fundam. Appl. Toxicol. 2: 229-239, 1982) exposed B6 mice for 8 weeks to 86%

pentachlorophenol at doses of 0, 50, 250, and 500 ppm. All treated groups showed delayed peaks in splenic antibody production and serum antibody titers. The magnitude of the IgM and IgG response were decreased in a dose-dependent manner, with IgM being more sensitive. In mice exposed to 50 and 500 ppm pentachlorophenol (86% a.i.) For 10-12 weeks prior to tumor transplant, the incidence of progressive tumors increased from 35% in control to 67% and 82% at the 50 and 500 ppm dose levels, respectively. In contrast to the increased tumor susceptibility in pentachlorophenol treated mice, the incidence of viral-induced mortality was not altered by pentachlorophenol. Of interest is the observation that significant depression of T-lymphocyte cytolytic activity and enhancement of macrophage phagocytosis was seen in mice exposed to the 86% pure pentachlorophenol, but not in mice exposed to an analytical grade (99% purity) of pentachlorophenol.

White et al. (Agents and Actions 16: 387-392) exposed female mice to a technical grade of pentachlorophenol for 14 days at 10 mg/kg/day). Complement activity (classical C1 pathway and alternate pathway) was suppressed. Doses of 10 and 30 mg/kg/day did not produce any effect on complement activity. Complement activity at the 100 mg/kg/day dose was still reduced after a 30 day recovery period. Holsapple et al. (J. Toxicol. Environ. Health 20: 229-239) reported normal splenocyte antibody production after in vitro stimulation with antigens in mice treated with technical grade pentachlorophenol at 10, 30, or 100 mg/kg/day for 14 days or in mice treated with 100 mg/kg/day purified pentachlorophenol for 14 days. However, after in vivo immunization of mice treated with technical grade pentachlorophenol, a dose-related suppression of IgM antibody response was noted to sheep red blood cells. No such changes were reported in mice treated with the purified grade of pentachlorophenol.

The above results in mice are corroborated by results of a six-month study conducted in mice by the National Toxicology Program (1989). In that study, groups of 25 male B6C3F1 mice and groups of 10 female mice of the same strain received either technical grade pentachlorophenol (90.4% purity) at 200, 600, or 1800 ppm; Dowicide EC-7 (91% purity) at 200, 600, or 1200 ppm; pentachlorophenol DP-2 (91.6% purity) at 200, 600, or 1200 ppm; or pure pentachlorophenol (98.6% purity) at 200, 500, or 1500 ppm for 26-27 weeks. In addition to standard parameters, behavioral studies and body temperature measurements were performed on groups of 10 animals/sex at weeks 5 and 26. Behavioral measurements included examination for neurologic effects (righting reflex, spontaneous motor activity, acoustical startle response, visual placement response, grip strength, and rotarod testing). Mice designated for measurement of liver biochemistry were also examined for induction of liver AHH. It is important to note that in this study, the various dose groups were exposed to differing levels of chlorinated dibenzodioxins and chlorinated dibenzofurans., contaminants of pentachlorophenol. Among the effects of these agents are immunotoxic effects. The relevant exposures in this study are summarized in the following Table:

| MALES | Dose (ug/kg/day) from Technical Grade Pentachlorophenol | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1800 ppm |
| HxCDD | 0.3 | 0.8 | 2.6 |
| HpCDD | 8.4 | 25.3 | 75.9 |
| OcCDD | 40.7 | 119 | 356 |
| HxCDF | 0.3 | 0.8 | 2.5 |
| HpCDF | 2.4 | 7.5 | 22.7 |
| OcCDF | 1.2 | 3.7 | 11.0 |

| FEMALES | Dose (ug/kg/day) from Technical Grade Pentachlorophenol | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1800 ppm |
| HxCDD | 0.4 | 1.1 | 3.3 |
| HpCDD | 10.8 | 32.3 | 97.0 |
| OcCDD | 51.9 | 152 | 455 |
| HxCDF | 0.3 | 1.1 | 3.2 |
| HpCDF | 3.0 | 9.5 | 29.0 |
| OcCDF | 1.6 | 4.8 | 14.1 |

| MALES | Dose (ug/kg/day) from Pentachlorophenol DP-2 | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1200 ppm |
| HxCDD | 0.02 | 0.05 | 0.10 |
| HpCDD | 0.8 | 2.4 | 4.8 |
| OcCDD | 4.9 | 14.8 | 29.6 |
| HxCDF | 0.4 | 1.1 | 2.2 |
| HpCDF | 4.9 | 14.7 | 29.4 |
| OcCDF | 9.1 | 27.3 | 54.6 |

| FEMALES | Dose (ug/kg/day) from Pentachlorophenol DP-2 | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1800 ppm |
| HxCDD | 0.02 | 0.06 | 0.13 |
| HpCDD | 1.0 | 3.0 | 6.1 |
| OcCDD | 6.3 | 18.9 | 37.7 |
| HxCDF | 0.5 | 1.4 | 2.8 |
| HpCDF | 6.2 | 18.7 | 37.5 |
| OcCDF | 11.6 | 34.9 | 69.7 |

| MALES | Dose (ug/kg/day) from Dowicide EC-7 | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1200 ppm |
| HxCDD | 0.005 | 0.02 | 0.03 |
| HpCDD | 0.02 | 0.05 | 0.09 |
| OcCDD | 0.02 | 0.06 | 0.12 |
| HxCDF | 0.004 | 0.01 | 0.02 |
| HpCDF | 0.004 | 0.01 | 0.03 |
| OcCDF | -- | -- | -- |

| FEMALES | Dose (ug/kg/day) from Dowicide EC-7 | | |
|---|---|---|---|
| | 200 ppm | 600 ppm | 1200 ppm |
| HxCDD | 0.007 | 0.02 | 0.04 |
| HpCDD | 0.02 | 0.06 | 0.12 |
| OcCDD | 0.03 | 0.08 | 0.15 |
| HxCDF | 0.005 | 0.02 | 0.03 |
| HpCDF | -- | -- | -- |
| OcCDF | -- | -- | -- |

There were no detectable levels of hexa-, hepta, or octo-dibenzodioxins or dibenzofurans reported for pure pentachlorophenol.

From the above data, it is noted that the highest exposures to these contaminants occurred through technical grade pentachlorophenol and DP-2, particularly for the chlorinated dioxins. This correlates well with the observation that the most significant induction of AHH activity (units/mg protein) and cytochrome P-450 content (nmol/mg protein)occurred with these formulations of pentachlorophenol (page 252 of the study). This observation also correlated with the results of testing for plaque-forming cell response following immunization with sheep red blood cells and measuring hemagglutination titers. Technical grade pentachlorophenol exposure resulted in the most marked inhibition of this response (all doses tested), while the DP-2 formulation resulted in marked inhibition only at the highest dose. Dowicide EC-7 and pure pentachlorophenol did not affect this response. Thus, the degree of immunosuppression is consistent with the degree of exposure to dioxin and dibenzofuran contaminants in pentachlorophenol.

Human data on the effects of pentachlorophenol exposure on the immune system exist, but suffer form a lack of quality control. McConnachie et al. (Arch. Environ. Health 46: 249-253) examined lymphocyte phenotype frequencies, functional responses, serum imunoglobulin levels and antibodies in 10 families who were exposed to pentachlorophenol from manufacturer log-treated homes for periods ranging from 1-13 years. Individuals exposed to pentachlorophenol were observed with activated T-cells, autoimmunity, functional immunosuppression, B-cell dysregulation, and enhanced natural killer cell activity in females. No assessment was made for controlling medication, smoking habits, alcohol, or infection. Daniel et al (Arch. Environ. Health 50: 287-292) examined immune parameters in 188 patients who were exposed for more than 6 months to pentachlorophenol containing pesticides. Impaired in vitro stimulation of lymphocytes was observed in 65% of patients examined. Decreased CD4/CD8 T-lymphocyte ratio was observed in 11 patients who demonstrated abnormal lymphocyte stimulation to mitogens. However, factors which may have affected immune function in patients examined in this study were not controlled for. The effects of pentachlorophenol on the immune system in 32 workers exposed to pentachlorophenol was examined by Colosio (Arch. Environ. Health 48: 81-88, 1993). No significant effect on serum IgG, IgM, IgA, or complement factors C3 and C4 were observed, nor were any effects on mitogen stimulated blastogenesis of lymphocytes and quantification of lymphocyte subsets.

**Conclusions**

The immunotoxic effects of pentachlorophenol as studied by the NTP and others appear to indicate that the immunotoxic effects of pentachlorophenol are largely mediated by the chlorinated dioxin contaminants. Although data from human studies are limited, it is likely that the immune effects observed are also due in part to the contaminants present within pentachlorophenol. New data using the pure test chemical in *in vitro* human studies would add greatly to the understanding of the potential for immunotoxicity of pentachlorophenol in humans.

## 2. Dose Response Assessment

**a. Reference Dose (RfD) for Chronic Oral Exposure**

The RfD for pentachlorophenol was established at 0.0045 mg/kg/day, based on hepatotoxicity characterized as increases in liver weights and alkaline phosphatase activity, increased incidences of granular cytoplasmic pigment accumulation in the liver, and increased incidence of lymphocytic mucosal inflammation in the stomach. In determining the RfD, an uncertainty factor of 100 was applied to account for both interspecies extrapolation and intraspecies variability. In addition, an uncertainty factor of 3 under FIFRA was used for use of a LOAEL in determining the RfD. An additional UF of 10, which must be considered under the Food Quality Protection Act for protection of infants and children based on available developmental and reproductive toxicology data, was not employed for pentachlorophenol (see discussion below).

**b. Uncertainty Factor/FQPA Considerations**

The following evaluation of pentachlorophenol is provided to address FQPA considerations on the sensitivity of infants and children.

In a prenatal developmental toxicity study in Sprague-Dawley rats (at least 22/group; MRID # 43091702), pentachlorophenol (88.9% a.i.) Was administered by gavage in corn oil (5 ml/kg) at doses of 10, 30, or 80 mg/kg/day on gestation days 6-15. Cesarean sections were performed on gestation day 20. The maternal NOAEL was 30 mg/kg/day and the Maternal LOEL was 80 mg/kg/day, based on reduced body weight gains. The developmental NOAEL was also 30 mg/kg/day and the developmental LOAEL was 80 mg/kg/day, based on increased resorptions (mainly early) with corollary reductions in litter size, reduced fetal weight, and increased litter incidences of external, visceral, and/or skeletal malformations and variations. These findings included hydrocephaly, diaphragmatic hernia, dilatation of the renal pelvis, vertebral structural variations, and incomplete ossification of the sternebrae.

Older published literature data on developmental toxicity of pentachlorophenol in rats demonstrate NOAELs and LOAELs lower than that shown above. In a study by Schwetz et al. (Toxicol. Appl. Pharmacol 28: 151-161, 1974), doses of 0, 5, 15, 30, and 50 mg/kg/day commercial grade (88.4% a.i.) or purified (>98% a.i.) pentachlorophenol prepared in corn oil were administered to groups of pregnant Sprague-Dawley rats on gestation days 6-15 inclusive. For purified pentachlorophenol, the number of rats per group was as follows: control, 33 rats; 5 mg/kg, 15 rats; 15 mg/kg, 18 rats; 30 mg/kg/, 20 rats; 50 mg/kg, 19 rats. For the commercial grade of pentachlorophenol: 5 mg/kg, 18 rats; 15 mg/kg, 17 rats; 30 mg/kg, 19 rats; 50 mg/kg, 15 rats. Additional groups of rats were administered 0 or 30 mg/kg/day pentachlorophenol (type of a.i. not specified) on days 8-11 or 12-15 of gestation. Based on the results of this study, the Maternal NOAEL can be considered as 15 mg/kg/day, based on body weight effects, for both grades of pentachlorophenol. The Developmental NOAEL would appear to differ according to grade of pentachlorophenol used. Limited data for purified pentachlorophenol at the 50 mg/kg/dose hampers evaluation of a NOAEL and LOAEL for this grade. For the commercial

grade of pentachlorophenol, the NOAEL can be considered as 15 mg/kg/day, and the LOAEL as 30 mg/kg/day, based on decreased fetal body weight and crown-rump length. In a study by Welsh et al. (Fd. Chem. Toxic. 25(2): 163-172, 1987), male and female Sprague-Dawley rats were exposed to dietary pentachlorophenol at levels of 0, 4, 13, and 43 mg/kg/day for 181 days, through mating and pregnancy. Males were presumably sacrificed after mating, while females were sacrificed on day 20 of gestation. Body weight gain in high dose females was significantly decreased during gestation, but no effect was observed on pregnancy rate. Increased resorptions were also observed at the high dose. There were no specific external variations related to administration of pentachlorophenol at any dose level. Fetal data (excluding the high dose, due to small sample size). Crown-rump length and body weight were decreased at the 13 mg/kg/day dose level for female fetuses. This study concluded that pentachlorophenol demonstrated embryotoxicity, but not teratogenicity.

In a prenatal toxicity study in New Zealand White rabbits (20/group; MRID # 43091701), pentachlorophenol (88.9% a.i.) was administered in the diet at dose levels of 7.5, 15, or 30 mg/kg/day on gestation days 6-18. Cesarean section examinations were performed on all surviving does on gestation day 29, followed by external, visceral, and skeletal examination of all fetuses. There were no maternal deaths or signs of maternal toxicity at any dose level. Significantly reduced weight gain was observed in the high dose group on gestation days 6-9 (loss of 40g vs gain of 20 g in controls), and in the mid and high dose groups for gestation days 9-12 (gain of 20 g at the mid and high dose vs gain of 50 g in controls). These changes were considered minimal as the differences in weight gain were equivalent to only 0.5-1% of mean body weight. A consistent reduction in mean food consumption (71-90% of control value on gestation days 9-12) was observed at the high dose. Weight gain was comparable for treated and control rabbits following the treatment interval. A slight but non-significant dose-related decrease in litter size was observed, corresponding to a decrease in implantations/litter for the treated does. There were no statistical differences in the number of treated litters vs control when the incidences for all observations of external, visceral, and skeletal effects were combined. Although the incidence of affected litters was not statistically significant, a significantly greater ($p < 0.01$) number of individual fetuses in the 15 mg/kg litters contained interfrontal ossification sites compared to controls. Based on the results of this study, the Maternal toxicity NOAEL = 15 mg/kg/day, and the Maternal toxicity LOAEL = 30 mg/kg/day, based on minimally reduced body weight gain and consistent reductions in food consumption during treatment. The Developmental toxicity NOAEL = 30 mg/kg/day; a Developmental toxicity LOAEL was not identified.

**Summary of reproductive and developmental toxicity:**

The developmental and reproductive toxicity of pentachlorophenol has been summarized above, based on available submitted studies reviewed by the Agency, as well as scientific data from the open literature.

**Recommendation for a developmental neurotoxicity study:**

The Health Effects Division's Hazard Identification Science Advisory Committee determined that, based on a weight-of-the-evidence review of available data, that a developmental neurotoxicity study is not recommended for pentachlorophenol. The following information was considered in reaching this determination:

1) Evidence in support of requiring a developmental neurotoxicity study:

- Minimal clinical evidence of non-specific neurobehavioral effects such as salivation, ataxia, or convulsions.

2) Evidence against the requirement for a developmental neurotoxicity study:

- Based on the available data, there is no evidence of frank, unequivocal neurotoxicity in the database, including changes in brain weight or incidence of neuropathology in the central nervous system tissues (nonperfused). The nervous system has not been generally considered as a target tissue.

3) No evidence of abnormalities in the development of the fetal nervous system were observed in the prenatal developmental toxicity studies in either rats or rabbits at maternally toxic oral doses up to 30 mg/kg/day. Cited incidences of hydrocephaly in the prenatal study in rats were included in only 2 fetuses of 2 litters and were within published historical control ranges.

**c. Special Sensitivity to Infants and Children**

Under the Food Quality Protection Act (FQPA), P.L. 104-170, which was promulgated in 1996 as an amendment to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and the Federal Food, Drug and Cosmetic Act (FFDCA), the Agency was directed to "ensure that there is a reasonable certainty that no harm will result to infants and children" from aggregate exposure to a pesticide chemical residue. The law further states that in the case of threshold effects, for purposes of providing this reasonable certainty of no harm, "an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children. Notwithstanding such requirement for an additional margin of safety, the Administrator may use a different margin of safety for the pesticide residue only if, on the basis of reliable data, such margin will be safe for infants and children."

Although no food uses have been identified for pentachlorophenol, dietary monitoring data assembled by the Food and Drug Administration indicate the presence of pentachlorophenol in certain food items (i.e. milk, pears, pork). In addition, indirect food additive uses under regulation by FDA have also been identified for pentachlorophenol. Therefore, pursuant to the language and intent of the FQPA directive regarding infants and children, the applicable toxicity

database for pentachlorophenol was evaluated by the Hazard Identification Science Advisory Committee.

**Adequacy of data:** The data included developmental toxicity studies in the rat and rabbit, as well as a two-generation reproduction toxicity study in rats. All of these studies have been reviewed and judged to be acceptable for regulatory purposes. Based on a weight of the evidence determination, the Committee did not recommend a developmental neurotoxicity study in rats.

**Susceptibility issues:** In the developmental toxicity studies in rats and rabbits as well as in the two-generation reproductive toxicity study in rats, there was no indication of increased sensitivity of young animals to pre- and/or post-natal exposure to pentachlorophenol. However, as noted above, contaminants of pentachlorophenol formulations have been demonstrated to be teratogenic agents. Based on this information, labeling was agreed to in Position Document 4, issued July 1984, that all registrants of products containing pentachlorophenol or its salts would incorporate language stating that the U.S. EPA has determined that pentachlorophenol can produce defects in the offspring of laboratory animals, and that exposure to pentachlorophenol during pregnancy should be avoided.

It is reasonable to continue to employ this labeling language, even in light of the more recent developmental and reproductive toxicity data for pentachlorophenol, based on the knowledge that pentachlorophenol will never be completely free from dioxin and furan contaminants.

### d. Carcinogenicity Classification

Since the time of the last Peer Review for pentachlorophenol, a new study by the National Toxicology Program has been conducted and reviewed by the Agency. As summarized above, there was evidence of carcinogenicity in male rats at 1000 ppm, as shown by an increased incidence of malignant mesothelioma of the tunica vaginalis, and an increased incidence of nasal squamous cell carcinomas at the 1000 ppm dose. The presence of tumors in the stop-dose group but not any of the longer term dose groups supports the hypothesis originally put forth in the NTP report that shorter term high dose exposure results in a more potent tumorigenic response. This observation is relevant to the proposed mechanism for production of tumors by pentachlorophenol, in which it is proposed that the tumorigenic response may be based upon a non-linear mechanism, as summarized below.

The occurrence of mesotheliomas as well as squamous cell carcinomas could be related to the capability of pentachlorophenol to cause oxidative damage to cells. In a study by Umemura et al. (Fundam. Appl. Toxicol., 1996), cell proliferation in hepatocytes of mice treated with doses of 41, 86, and 200 mg/kg/day for either 2 or 4 weeks was examined. Levels of 8-hydroxy-deoxyguanosine in hepatic nuclear DNA (an indicator of oxidative DNA damage) were significantly increased vs control at both 2 and 4 weeks of treatment with pentachlorophenol. Absolute and relative liver weights were also significantly increased in a dose-related fashion

after 2 and 4 weeks of treatment, concomitant with increases in hepatic DNA content. Labeling indices using bromodeoxyuridine as a marker for cell proliferation showed increased labeling indices at 2 and 4 weeks at all dose levels vs control. The results of this study suggest that oxidative stress brought on by pentachlorophenol administration (as shown by the increase in 8-hydroxy-deoxyguanosine levels) may lead in turn to cellular proliferation, which, if sustained (as shown by the apparent increase at both 2 and 4 weeks of treatment), may lead to tumorigenesis in the liver of mice.

These results are corroborated by the observations of Swenberg, who examined the DNA in tissue samples from rats in the stop-exposure group. A two-fold increase in 8-hydroxy-deoxyguanosine levels were found in the stop-exposure group of rat livers as compared to control. The pentachlorophenol metabolites tetrachloro-1,4-benzoquinone and tetrachloro-1,2-benzosemiquinone were observed to be bound to the liver proteins in male rats in the stop-exposure dose group.

Other investigators have also reported oxidative damage from administration of pentachlorophenol. In a study by Suzuki et al. (Biol. Pharm. Bull. 20: 271-274, 1997) the ability of several different pesticides to induce lipid peroxidation was examined using hepatocytes isolated from male Wistar rats. Cytotoxicity, as evaluated by release of LDH into the cell medium, was increased by 20-35% following incubation with pentachlorophenol (1mM). Cellular phospholipid hydroperoxide levels were increased 5 fold by pentachlorophenol, while cellular glutathione was almost virtually eliminated by pentachlorophenol treatment. The results of this study suggest that peroxidative damage to cellular membrane phospholipids may underlie the cytotoxicity induced by pentachlorophenol.

The apparent difference in tumorigenic response in mice vs rats could be explained by observations regarding production of a unique metabolite in mice as well as the nuclear DNA binding ability of reactive pentachlorophenol metabolites in mice vs. rats. In a study by Lin et al. ( Toxicol. and Appl. Pharmacol 145: 399-408, 1997) experiments were conducted in B6C3F1 mice to determine whether the types and quantities of adducts differed between rats and mice. Kinetic constants from in vitro studies were also used to estimate tissue doses of pentachlorophenol-derived quinones in the livers of both rats and mice. Subcellular doses to liver cytosol and acid-soluble nuclear proteins were differentiated. Both rats and mice were given a single oral dose of 20mg/kg pentachlorophenol, and sacrificed at 0.5, 1, (mice only), 2, 4, 8, 24, 48, 168, and 336 hours after dosing.

Estimation of second-order rate constants for tetrachloro-1,2-benzoquinone and tetrachloro-1,4-benzoquionone and the corresponding quinone adducts showed differences in second-order reaction rate constants, the rates being statistically significant between rats and mice for virtually all reaction rates. Study of the time course of adduct formation showed that quinone adducts reached maximum values earlier in mice (0.5-4 hrs. than in rats (8-24 hours), indicating faster biotransformation of pentachlorophenol in mice vs. rats. While rat liver cytosol showed higher amounts of binding of total tetrachloro-1,4-benzoquinone based quinones, the estimated total

dose of quinones to mouse liver nuclei was about 4-fold greater than that to rat liver nuclei. In addition, the unique presence of tetrachloro-1,2-benzoquinone in mouse liver proteins but not rat liver proteins, coupled with the tumorigenic response in mice vs rats, suggests a role for this quinone metabolite in pentachlorophenol mouse liver tumorigenesis.

Thus, there is a body of experimental evidence pointing to a mechanism for pentachlorophenol-induced tumorigenesis involving generation of quinone and semiquinone metabolites, oxidative damage to cell membrane components as well as cell DNA, and sustained cell proliferation. These responses to pentachlorophenol are expected to occur only at higher exposures to the chemical as the mechanism is not one of direct DNA toxicity but one involving cellular defense mechanisms against oxidative stress and/or cell proliferation.

The Health Effects Division's Mechanism of Toxicity Science Advisory Review Committee met on October 20th, 1998 to discuss carcinogenicity and mechanistic data for the wood preservative chemical pentachlorophenol. The data consisted of summaries of rat and mouse carcinogenicity studies conducted by the National Toxicology Program, as well as published scientific studies on the potential mode of action of tumor induction by pentachlorophenol.

Previously, the Health Effects Division's Carcinogenicity Peer Review Committee classified pentachlorophenol as a B2 carcinogen, based upon combined incidence of hemangiosarcomas, liver adenomas/carcinomas, and adrenal pheochromocytomas observed in male and female mice from the NTP study. Since then, information on the carcinogenicity of pentachlorophenol in rats (NTP Technical Report No. 483) as well as information on the mechanism whereby pentachlorophenol may induce hepatic tumors has become available. In the rat study, significantly increased incidence of malignant mesothelioma of the tunica vaginalis was observed at 60 mg/kg/day as well as an increased incidence of nasal squamous cell carcinoma at 60 mg/kg/day. It is noted that the 60 mg/kg/day dose group received pentachlorophenol in the diet only for 52 weeks, followed by a 52 week control diet intake period. Tumors were not observed in increased incidence at lower doses (10, 20, and 30 mg/kg/day), in contrast to the mouse. The results of the rat study supported the hypothesis put forth by the NTP that shorter term, higher dose exposure to pentachlorophenol would result in a more potent tumorigenic response compared to lower dose, longer term exposure.

Scientific literature has become available which has reported on a possible mode of action for pentachlorophenol induced liver tumors, as well as the apparent sensitivity of mice vs rats. Studies conducted by both Umemura et al. (Fundam. Appl. Toxicol., 1996) as well as Swenberg (NTP Technical Report 483) showed increased levels of 8-hydroxy-deoxyguanosine in hepatic nuclear DNA from mice treated with 41, 86, or 200 mg/kg/day pentachlorophenol for 2 or 4 weeks as well as in rat liver DNA from those rats in the 60 mg/kg/day dose group. Elevation of 8-hydroxy-deoxyguanosine is an indicator of oxidative DNA damage.

Suzuki et al. (Biol. Pharm. Bull. 20: 271-274, 1997) reported increased LDH leakage from hepatocytes isolated from male Wistar rats and exposed to 1 mm pentachlorophenol, 5-fold

elevation of cellular phospholipid hydroperoxide, and virtual elimination of cellular glutathione following pentachlorophenol incubations.

Lin et al. (Toxicol. Appl. Pharmacol. 145: 399-408, 1997) reported that the estimated dose of quinone metabolites to mouse hepatic nuclei was approximately 4-fold greater than that observed in rat liver nuclei, and that time to peak quinone adduct formation was shorter for mice vs rats. The unique metabolite tetrachloro-1,2-benzoquinone was also observed in mouse liver proteins but not rat liver proteins. These data support the observed greater sensitivity of mice to tumor induction by pentachlorophenol based on greater accumulation of quinone metabolites in liver nuclei, faster biotransformation of pentachlorophenol to toxic quinone metabolites, and the presence of a unique quinone metabolite in mouse liver.

The Mechanism of Toxicity Committee considered all of the above data in the October 20th meeting and reached the following conclusions:

1) The data presented in support of a mode of action for induction of liver tumors by pentachlorophenol were considered to be suggestive but not conclusive.

2) Pentachlorophenol is largely devoid of positive mutagenic effects with the exception of one published report, in which a positive response was noted. Pentachlorophenol is also observed to be weakly clastogenic, with chromosomal aberrations observed using Chinese hamster ovary cells in the presence of rat liver S9. In contrast to the data for pentachlorophenol, data for the metabolite tetrahyhdroquinone (THQ) show positive effects on Chinese hamster V79 cells (increase in frequency of thioguanine-resistant mutants, increase in micronuclei using V79 cells, covalent binding to DNA , and induction of DNA single-strand breaks.

3) Data were lacking to explain the potential mode(s) of action for the pentachlorophenol induced pheochromocytomas and hemangiosarcomas.

4) The Committee agreed that the available data are insufficient to warrant reconsideration of the carcinogenicity classification for pentachlorophenol at this time. Thus, **the classification for pentachlorophenol will remain as Group B2.**

**e. Dermal absorption**

A value of 40% dermal absorption for pentachlorophenol will be used, based on a submitted study from the scientific literature accepted by the Agency.

**f. Other Toxicological Endpoints**

**Toxicity End-Point Selection**

**i. Acute Dietary (one day)**

For acute dietary risk assessment, the developmental toxicity study in rabbits was selected (MRID # 43091702), using a NOAEL of 30 mg/kg/day, based on increased resorptions, reduced fetal weight, and skeletal malformations / variations observed at 80 mg/kg/day (LOAEL). The skeletal malformations / variations are presumed to occur after a single exposure (dose) and are thus considered to be appropriate for this risk assessment. This risk assessment is required for pentachlorophenol.

The Health Effects Division Hazard Identification Science Advisory Committee determined that the 10x factor to account for enhanced sensitivity of infants and children (as required by FQPA) should be removed. For acute dietary risk assessment, a Margin of Exposure (MOE) of 100 is required for protection of the general U.S. population including infants and children from exposure.

### ii. Chronic Dietary

For chronic dietary risk, a chronic toxicity study in dogs (MRID # 43982701) was selected, with a LOAEL of 1.5 mg/kg/day, based on hepatotoxicity characterized as increases in liver weights and alkaline phosphatase activity, increased incidences of granular cytoplasmic pigment accumulation in the liver, as well as increased incidence of lymphocytic mucosal inflammation in the stomach.

For chronic risk assessment, an uncertainty factor of 300 was applied to derive the Reference Dose. The uncertainty factor of 300 included a 10x factor for interspecies variation, a 10x factor for intraspecies variation, and 3x for use of a LOAEL (under FIFRA).

### iii. Occupational Exposure

1. Dermal Absorption

For pentachlorophenol, a dermal absorption factor of 40% is used for correction of oral to dermal dosing. This value is based on a dermal absorption study (MRID # 00259257) in which radiolabelled pentachlorophenol (5% solution in P-9 oil) was applied to the skin of young adult Sprague-Dawley rats for 0.25, 1, 4, 8, and 24 hours. At 8 hours post-application, the calculated absorption was 40%. At 24 hours post-application, the calculated absorption was 60%.

2. Short Term Dermal (1-7 days)

For short term occupational exposure, the developmental toxicity study in rats (MRID # 43091702) was selected, using the developmental NOAEL of 30 mg/kg/day, based on increased resorptions, reduced fetal weight and skeletal malformations / variations at 80 mg/kg/day. Since an oral NOAEL was selected, a dermal absorption rate of 40% should be used for correcting the oral dose to a dermal dose for risk assessments. This risk assessment is required for

pentachlorophenol.

Although a 90-day dermal toxicity study in rats (MRID # 43182301) was available, it was not considered appropriate for this risk assessment because of the concern for developmental effects that were seen in rats which were not observed in the 90-day dermal toxicity study.

3. Intermediate Term Dermal (7 days to several months)

For intermediate term occupational exposure, the developmental toxicity study in rats (MRID # 43091702) was selected, using the developmental NOAEL of 30 mg/kg/day, based on increased resorptions, reduced fetal weight and skeletal malformations / variations at 80 mg/kg/day. Since an oral NOAEL was selected, a dermal absorption rate of 40% should be used for correcting the oral dose to a dermal dose for risk assessments. This risk assessment is required for pentachlorophenol.

4. Long Term Dermal (Several Months to Life Time)

For long term occupational exposure, the chronic toxicity study in dogs (MRID # 43982701) was selected, as described above under chronic dietary exposure. It is noted that the chronic dog study was used for establishing the RfD for pentachlorophenol.

5. Inhalation Exposure (any time period)

One inhalation toxicity study was available for the technical material in the scientific literature which was considered unacceptable for regulatory purposes, while studies in the toxicology one-liner database with pentachlorophenol formulations were all considered unacceptable by the Agency. Due to the lack of acceptable data, it was determined that pentachlorophenol be placed in Toxicity Category I for inhalation toxicity. Thus, a respirator must be worn during use of pentachlorophenol. Also, a data call in for acute and subchronic inhalation toxicity studies were recommended for the technical test material.

**iv. Margin of Exposure for Occupational/Residential Exposures**

For short and intermediate term dermal risk assessment, a Margin of Exposure (MOE) of 100 is adequate because a NOAEL was used for these risk assessments. Fore long-term dermal risk, a MOE of 300 is required due to use of a LOAEL for this risk assessment. The additional uncertainty factor is applied under FIFRA for lack of a NOAEL, as risk assessments under FQPA are not applied to occupational exposure.

## v. Summary of Toxicological Endpoints for Risk Assessment

| Summary of Toxicological Endpoints to be used for Risk Assessment of Pentachlorophenol | | | |
|---|---|---|---|
| Exposure Scenario | Dose (mg/kg/day) | Endpoint | Study |
| Acute Dietary | Developmental NOAEL = 30 | increased resorptions, reduced fetal weight, and skeletal malformations | Developmental - rat |
| | UF = 100 | **Acute RfD = 0.3 mg/kg/day** | |
| Chronic Dietary | LOAEL = 1.5 | Incr. liver weight and alk. phos. activity; increased incidence of granular cytoplasmic pigment accumulation in the liver | Chronic Toxicity - Dog |
| | UF = 100, plus 3x under FIFRA | **Chronic RfD = 0.0045 mg/kg/day** | |
| Carcinogenicity | q1*= 1.2 X $10^{-1}$ | Pentachlorophenol is classified as a B2 (probable human carcinogen) using a linear low-dose extrapolation model . | |
| Short-term (Dermal) | NOAEL = 30 | increased resorptions, reduced fetal weight, skeletal malformations | Developmental - Rat |
| Intermediate-term (Dermal) | NOAEL = 30 | increased resorptions, reduced fetal weight, and skeletal malformations | Developmental - Rat |
| Long-term (Dermal) | LOAEL = 1.5 | Incr. liver weight and alk. phos. activity; increased incidence of granular cytoplasmic pigment accumulation in the liver | Chronic Toxicity - Dog |
| Inhalation (any time period) | NO ADEQUATE STUDY AVAILABLE | | |

## 3. Dietary Exposure and Risk Assessment/Characterization

### a. Dietary Exposure (Food Source)

#### i. Directions for Use

AD evaluated the registered use patterns for pentachlorophenol as it relates to the residue chemistry data requirements to support the wood preservative use. The use pattern of Pentachlorophenol (PCP) has seen major changes in approximately the last twenty years from being a multipurpose pesticide applied as a herbicide, bactericide, algicide, fungicide, mollucsides and insecticide to mostly as a wood preserving pesticide.

PCP is a polar molecule and from an environmental point of view moderately water soluble ( 42 ppm.) and it has a tendency to be released to all environmental compartments ( see product chemistry and environmental fate chapters). It was, therefore, present in all the environmental compartments. Consequently, it is important to determine the route PCP takes for human exposure. Molly-A. Hattemer-Frey and Curtis Travis ( 1989) used models to determine the environmental partitioning of PCP in the six environmental compartments ( air, soil, water, sediment, suspended sediment in water and biota in water. The conclusions drawn from these studies were: PCP partitions mainly into soil ( 96.5%) and food chain ( fruits, vegetables, grains) and 99.9% of human exposure is through food chain. The toxicity and oncogenicity of PCP and some of the micro contaminants ( dioxins) have long been established. It was the presence of PCP and its micro contaminants on food related items that started the process of restricting the use of PCP. The Food and Drug Administration in its Memoranda of 1975, on Total Diet Samples ( market basket survey) reported the presence of PCP up to 0.49 ppm. concentration ( four times as high as previously reported) in candy bars which was the result of using the specific wrappers; the presence of PCP was detected in gelatin, the suspected source of which was PCP-treated hides used as raw materials for gelatin production. Multipurpose uses of PCP were slowly phased out particularly as a food additive or on food related items by US FDA.

In Feb.1981, the US EPA issued a Position Document 2/3 which proposed "canceling or denying registration of spray formulations of PCP containing < 5%, prohibiting indoor applications of the wood preservatives, most indoor use of PCP treated wood and uses of wood preservative that are likely to contaminate food, feed or drinking water."

The US EPA in its Position Document 4 ( February 1984) imposed further restrictions in the usage of PCP so that it would be used by Certified Applicators only. The Position Document 4 ( PDP 4) also mandated that a Chemical Information Sheet ( CIS ) must be distributed by the manufacturer of PCP to the buyer/applicator, and the CIS should include the statement that PCP treated wood would not be allowed to come in contact with food/feed/public drinking water and with drinking water for animals. In addition, the purchaser will not use the PCP treated wood for interior purposes or in barns where domestic animals might lick the wood or

rub themselves on the treated wood.

### ii. Meat, Milk, Poultry, Eggs

Progressively mandatory restrictive use of PCP imposed by the Agency by 1988 has resulted in a dramatic decline of PCP contamination in dairy and non-dairy foods as borne out by the following numbers:

1. According to the US FDA 's Total Diet Study ( TDS ) for non-milk foods, the US daily intake of PCP was: 0.70 $\mu$g in 1972-73, 0.80 $\mu$g in 1974 while for 1981/82 it was 0.023$\mu$g and 0.079 $\mu$g ( Philip Howard, 1991).

2. In 1988, the TDS for PCP intake was ( in $\mu$g/kg) 0.0004, 0.0002 and 0.0003 for 6-11 month old infant, 14-16 year old male and 60-65 year old male respectively. ( Philip Howard, 1991).

3. For milk products , in 1987-88, the highest levels of PCP reported were 0.009 and 0.013 ppm. and in 1988/89, there were no PCP levels reported in any milk or milk fortified products.

4. Hatter-Frey/ Travis models yielded a background level of PCP to be 16.6 $\mu$g/day, based on which the daily dose per unit body weight for young children, adult females and adult males of 20, 60 and 70 kg would be 0.83, 0.28 and 0.24 $\mu$g/kg-day respectively. Cal EPA using model TAS obtains similar numbers, based on milk consumption, for the same three demographic groups.

5. FDA does not conduct any TDS on the non-milk foods like cereal, alfalfa or grass, or grain forages etc. after PCP was banned for agricultural use and hence, no residue numbers are available for such items.

## b. Dietary Exposure (Drinking Water Source)

### i. Surface Water Estimates

Surface water runoffs from the PCP treated utility poles may be a possible source for PCP or its transformation products in drinking water or in foods. The Agency has used the PRZM3-EXAMS model to estimate the environmental concentrations (EC's) for PCP The basis of the model and calculations is the scenario: Poles treated with PCP are located in a field in Mississippi Delta . This field is planted with cotton. The runoff from this goes to a standard pond.

Estimated environmental concentrations in various environmental compartments are as follows:

| Environmental Compartment | Yearly estimate |
|---|---|
| Water Column Dissolved Conc.. (ppb) | 0.014 |
| Pore Water Dissolved Conc.. ( ppb) | 0.011 |
| Benthic Sediment Conc.. ( mg/kg) | 0.0015 |
| Benthic Organism Conc.. ( $\mu$g/g) | 0.181 |

Human exposure to PCP through drinking water is much lower than the background PCP concentrations.

Using the PRZM3-EXAMS model and the available environmental fate data for pentachlorophenol, RASSB calculated the following Estimated Environmental Concentrations (EECs) for residues of pentachlorophenol in surface water as follows:

Acute or instant EECs: 0.176 ppb
Chronic (yearly) EECs: 0.014 ppb

**ii. Ground Water Estimates**

EEC's for groundwater were not calculated. Based upon the physical / chemical characteristics of pentachlorophenol and available, but limited, monitoring data, groundwater EEC values are not expected to add significantly to the risk assessment for pentachlorophenol.

**iii. Environmental Fate Assessment**

In the case of pentachlorophenol, acute exposure is expected to occur from both food and water sources. However, based on the results of PRZM3-EXAMS modeling using very conservative assumptions, exposure to pentachlorophenol in drinking water is expected to be negiligible. This is based on the observation that pentachlorophenol demonstrates high affinity for soil particles and is not mobile once introduced into the soil. In cases where pentachlorophenol gets in to surface water, at least 80 percent is found to sequester into sediment. Once attached to soil or in sediment, pentachlorophenol is subject to photodegradation, thereby further reducing exposure.

**c. Dietary Risk Assessment and Characterization (Food Sources)**

**i. Acute Dietary Risk**

Based on U.S. Food and Drug Administration regulatory monitoring data for fiscal years 1985-1991, detectable residues of pentachlorophenol were observed mainly in milk, with detection also observed in grape jelly,raw pears, and pork. These data were reported in the California Environmental Protection Agency's Risk Characterization Document for Pentachlorophenol, and are used in the dietary risk assessment for pentachlorophenol in the present document. An acute dietary exposure analysis wasconducted for pentachlorophenol using the data as presented in CAl EPA's risk characterization document. Results for exposures at the 95th percentile are summarized in the following table:

| Acute Dietary Risk Analysis for Pentachlorophenol | | | |
|---|---|---|---|
| Population Subgroup | 95th Percentile Daily Exposure (ug/kg/day) [a] | Percent Acute RfD | MOE [b] |
| non-nursing infants | 1.19 | 0.39% | 25,210 |
| children ages 1-6 | 0.88 | 0.29% | 34,090 |
| children 7-12 | 0.48 | 0.16% | 62,500 |
| males, 20+ | 0.15 | 0.05% | 200,000 |
| females, 20+ | 0.14 | 0.05% | 214, 285 |

a- not pregnant, not nursing

b- acute exposure analysis based on the 95th percentile daily consumption profiles for whole milk consumed in any form. Milk was asumed to contain pentachlorophenol at 0.013 ppm, the maximum level detected by USFDA in 1987-88.

### ii. Chronic Dietary Risk

A chronic dietary exposure analysis was conducted for pentachlorophenol using the data as presented in CAl EPA's risk characterization document. Results for exposures using the calculated Annual Average Daily Dose are summarized in the following table:

| Chronic Dietary Risk Analysis for Pentachlorophenol | | | |
|---|---|---|---|
| Population Subgroup | Annual Average Daily Dose ($\mu$g/kg/day) [a] | Percent Chronic RfD | MOE [b] |
| non-nursing infants | 0.11 | 2.2% | 13, 636 |
| children ages 1-6 | 0.12 | 2.4% | 12,500 |
| children 7-12 | 0.070 | 1.4% | 21,428 |
| males, 20+ | 0.018 | 0.36% | 83,333 |
| females, 20+ | 0.017 | 0.34% | 88,235 |

a- not pregnant, not nursing

b-chronic exposure analysis based on the annual average daily consumption profiles for whole milk consumed in

any form. Milk was assumed to contain pentachlorophenol at 0.005 ppm, half of the FDA reporting cutoff since 1989.

Chronic aggregate risk for pentachlorophenol will include risk estimates associated with exposure through food and registered residential uses. As shown below, exposure to pentachlorophenol in food results in an exposure which represents up to 2.4% of the chronic RfD for children aged 1-6, the most exposed subpopulation according to this analysis. As stated previously, drinking water exposure is considered negligible for pentachlorophenol.

### iii. Carcinogenic Risk

A lifetime exposure analysis was conducted for pentachlorophenol, using published data on indoor exposure to pentachlorophenol from treated lumber in log homes as well as an older study published which examined soil concentrations of pentachlorophenol around treated poles. It is noted that both studies are older, and that the use of PCP for treatment of logs used in home construction is no longer allowed. Therefore, the data are limited with respect to the amount of data available and indoor exposure to PCP is likely to be significantly lower currently than in the past, based on restrictions applied to use of PCP after 1984.

### iv. Drinking Water Risk

Based on the acute and chronic dietary exposure estimates presented in the tables above ("Acute Dietary Risk Analysis for Pentachlorophenol" and "Chronic Dietary Risk Analysis for Pentachlorophenol"), drinking water levels of comparison (DWLOC) were calculated using the formula below. A human health DWLOC is the concentration of a pesticide in drinking water which would result in unacceptable aggregate risk, after having already factored in all food exposures and other non-occupational exposures for which OPP has reliable data.

$$DWLOC_{acute} = \frac{[\text{acute water exposure (mg/kg/day)} \times \text{(body weight)}]}{[\text{consumption (L)} \times 10^{-3}\ \text{mg}/\mu\text{g}]}$$

where acute water exposure (mg/kg/day) = acute RfD - acute food exposure (mg/kg/day)

$$DWLOC_{chronic} = \frac{[\text{chronic water exposure (mg/kg/day)} \times \text{(body weight)}]}{[\text{consumption (L)} \times 10^{-3}\ \text{mg}/\mu\text{g}]}$$

where acute water exposure (mg/kg/day) = RfD - chronic food exposure (mg/kg/day)

The Agency's default body weights and consumption values used to calculate DWLOCs are as follows: 70 kg/2L (adult male); 60 kg/2L (adult female); 10 kg/1L (child). It is noted that a dietary exposure figure for the U.S. population was not available, so separate DWLOCs were calculated for the subpopulations as outlined in the dietary exposure and risk section.

**Acute DWLOC**

For adult males and females, the acute DWLOC was calculated as 10,465 ppb, while for children ages 1-6, the acute DWLOC was calculated as 2990 ppb.

**Chronic DWLOC**

For adult males, the chronic DWLOC was calculated as 174 ppb, and for adult females, 149 ppb. For infants and children ages 1-6, the chronic DWLOC was calculated as 49 ppb.

## 4. OCCUPATIONAL AND RESIDENTIAL EXPOSURE AND RISK ASSESSMENT/CHARACTERIZATION

### a. Occupational and Residential Exposures

#### i. Handler Exposures

EPA has determined that there are potential exposures to mixers, loaders, applicators, and other handlers during typical use-patterns associated with pentachlorophenol and from use in commercial, industrial, and residential settings. As a restricted use chemical, pentachlorophenol can only be applied by a certified applicator, primarily in commercial and industrial settings. However, certified applicators conduct groundline remedial treatment applications to utility poles in residential settings. The following types of handler exposures have been identified:

<u>Primary Occupational Handlers- Mixer/Loaders</u>

(1a) mixing/loading crystalline technical grade product to make ready-to-use product;
(1b) mixing/loading crystalline technical grade product to make concentrated product;
(2a) mixing/loading liquid formulation at joinery mills; and
(2b) mixing/loading liquid formulation at pressure treatment plants;

<u>Secondary Occupational Handlers - Applicators</u>

(3a) applying liquid formulation at joinery mills - by dipping;
(3b) applying liquid formulation at joinery mills - using an airless sprayer;
(3c) applying liquid formulation at joinery mills - using a low pressure handwand;
(3d) applying liquid formulation at joinery mills - by brushing;
(4) applying liquid formulation at pressure treatment plants - helpers/switchmen; and
(5) applying grease formulation for groundline remediation of utility poles- by brushing.

Table 1 provides a description of exposure scenarios for primary and secondary occupational handlers.

**Table 1. Exposure Scenarios for Primary and Secondary Occupational Handlers**

| Exposure Scenario | | Scenario Description |
|---|---|---|
| | ***Primary Handlers*** | |
| (1a) | Mixing/loading crystalline technical grade product to make ready-to-use product | Scenario pertains to a formulating facility or wood pressure treating facility (e.g., manufacturing telephone poles). Crystalline penta block is loaded and mixed with solvent at the correct use dilution to make a liquid ready-to-use product. The mixing usually occurs in a closed system. Potential exposure to workers results from open loading of the crystalline block and handling of ready-to-use product after mixing with a solvent .No PHED data are available for crystalline block. Granular data from PHED are used as surrogates. |
| (1b) | Mixing/loading crystalline technical grade product to make a concentrated product | Scenario also pertains to a formulating facility or wood pressure treatment facility. Crystalline penta block is loaded and mixed with solvent to make a concentrated product used as a formulation intermediate. The mixing usually occurs in a closed system. Potential exposure to workers results from open loading of crystalline block and handling of concentrated liquid product after mixing with a solvent. No PHED data are available for a crystalline block. Granular data from PHED are used as surrogates. |
| (2a) | Mixing/loading liquid formulation at joinery mills | Scenario pertains to a joinery mill in which milled wood (millwork) (e.g., doors, windows, and architectural moldings) is treated. Mechanical pumps are used to pump ready-to-use (prepared from concentrate) preservative into the vat. Potential exposure to workers occurs while mixing/loading liquids. PHED data for liquids are used. |
| (2b) | Mixing/loading liquid formulation at pressure treatment plants | Scenario pertains to a wood pressure treatment plant. Liquid ready-to-use PCP is prepared from concentrate and loaded into the retort using a mechanical pump. Potential exposure occurs while pumping liquid into the retort. PHED data for liquids are used. |
| | ***Secondary Handlers/Applicators*** | |
| (3a) | Applying liquid formulation at joinery mills - dipping | Scenario occurs at a joinery mill. Ready-to-use liquid is applied to millwork through processes such as bundle dipping, in-line dipping, flood coating, and vacuum treatment in which a bundle of millwork pieces are mechanically dipped into a vat of PCP wood preservative. Potential exposure to workers was estimated using exposure concentrations identified in the available literature. |
| (3b) | Applying liquid formulation at joinery mills - airless spraying | Although spraying of wood preservative may be a diminished use pattern at joinery mills, the LUIS[4] profile and several registered PCP labels (EPA Reg. 7234-60, 7234-61) still list spraying of seasoned and unseasoned wood as a registered PCP use. Potential exposure to workers was estimated using PHED data for an airless sprayer as surrogate data. |
| (3c) | Applying liquid formulation at joinery mills - low pressure handwand | Low pressure handwand application on millwork was estimated using PHED data. This scenario is considered a diminished use pattern (see above). |
| (3d) | Applying liquid formulation at joinery mills - brushing | Brushing millwork with PCP wood preservative was estimated using PHED data. Brushing millwork may be a diminished use pattern, but it is still registered according to the LUIS[4] profile and the PCP labels (EPA Reg. 7234-60 and 7234-61). |

| Exposure Scenario | | Scenario Description |
|---|---|---|
| (4) | Applying liquid formulation at pressure treatment plants - helpers/switchmen | Scenario includes exposure to workers opening retorts at a pressure treatment facility to load untreated wood into the retort vessel at the beginning of a treatment cycle, and to unload treated wood from the retort at the end of the treatment cycle. Only inhalation data are available from the available literature. |
| (5) | Applying grease formulation for groundline remediation of utility poles - brushing | Scenario pertains to brushing grease formulation onto utility poles or other wood products for groundline remedial treatment. PHED data were used because of the lack of any chemical-specific data. |

## Handler Data and Assumptions

In the course of development of this Reregistration Eligibility Decision Document (RED), both chemical-specific handler data identified from pertinent literature sources, as well as data from the Pesticide Handlers Exposure Database (PHED) were used in conjunction with labeling and estimates from industry representatives of amounts handled to predict exposures.

### a. Chemical-Specific Handler Exposure Data

#### *Dermal Exposure Study*

One dermal study was used to provide chemical-specific handler exposure data in support of the reregistration of pentachlorophenol (Fenske et al., 1987).[5] Fenske et al. (1987) assessed dermal 2,3,4,6-tetrachlorophenol (TCP) and pentachlorophenol (PCP) exposure to gloved joinery mill workers. Fenske et al. (1987) conducted a quantitative assessment of dermal exposure among nine timber mill workers. A fluorescent tracer was included in a sapstain control application of an aqueous tetrachlorophenol (TCP) formulation in a planing mill. The wood passing through the mill was treated with Parmatox 100®, a formulation consisting of 20 percent 2,3,4,6-tetrachlorophenol (TCP), 3 percent pentachlorophenol (PCP), and less than 0.4 percent other chlorophenol isomers.[5]

The study examined exposures during wood treatment for sapstain control at a planer mill in the central lumber-producing region of the Olympic Peninsula of Washington State. Workers were exposed to the TCP formulation after the lumber had been treated.

Specific work related activities included grading, pulling, and stacking freshly treated lumber on an assembly line process whereby, lumber moves on a chain-driven system. The wood had already been treated in a closed spray system typical of mills in the area. The job required continual hand contact in which a 30-foot long 4-inch x 4-inch board is pulled from the assembly line and stacked. In addition, the job included handling wood on the assembly line for occasional trimming (e.g., grading).[5] Workers wore polyvinyl chloride (PVC) gloves during their work day, as well as vinyl aprons. Underneath the gloves, workers wore thick cotton glove monitors. Workers were asked to wear these gloves up to 45 minutes and then the cotton gloves would be replaced. Following the work day, workers' exposure was examined using video imaging analysis to quantitate dermal fluorescence. The mean dermal exposure of hands and forearms to TCP using the fluorescent techniques was 178 $\mu$g/hr $\pm$ 42.2. The author of this study recommended that the data in the study be considered as a preliminary step towards an accurate assessment of dermal exposure to chlorophenols in timber mills. The author (Fenske et al., 1987) also noted that the sample size was small, the study was limited to one mill, and the fluorescent technique had only been recently developed at the time of the study.[5]

In addition to the data gaps mentioned by the author, several other data gaps may limit the usefulness of the data for registration of pentachlorophenol. The data gaps according to Series 875 Guidelines include:

- A typical end-use product was not represented. The end-use product examined was a mixture of pentachlorophenol (3 %) and TCP. Typical registered pentachlorophenol products contain 5% pentachlorophenol. Based on pentachlorophenol product labeling ready-to-use (RTU) solutions of 10 % ai are also currently registered.

- The study was done in 1987. Typical worker-related activities in joinery mills may be different in 1998. Industry information indicates that pentachlorophenol use in joinery mills for sapstain control is a diminished use.[6]

- Proper quality assurance/quality control (QA/QC) information is lacking (lack of fortifications, high deviations in reported study, insufficient field recovery information, lack of field blanks and control populations, lack of storage stability data, and lack of proper laboratory recovery information).

- An insufficient number of replicates and data from only one lumber mill test site are available.

The Fenske et al. (1987) study was used in California's PCP Risk Characterization Document by Brodberg and Thonginthusak (1995) to address potential human health risks from pentachlorophenol exposure in occupational scenarios involving pressure treatment of lumber and logs.[7] The Fenske et al. (1987) data, along with some of the assumptions used in the Brodberg and Thonginthusak (1995) report, were used in this assessment to develop hourly dermal exposures for the following scenario: (3a) applying liquid formulation at joinery mills by dipping. Since no PHED data were appropriate for this exposure scenario, the data in this study were the only information available. According to Brodberg and Thonginthusak (1995), the Fenske et al. (1987) dermal exposure data were based on TCP gloved hand and forearm data in which pentachlorophenol exposure was observed as one-tenth the TCP exposure; the formulation used in the study was 3% pentachlorophenol and the solution was diluted 20 fold.[5,7] Therefore, the hourly dermal exposure to pentachlorophenol for this scenario was adjusted as follows.

$$\textit{Hourly Dermal Exposure}\left(\frac{mg\ ai}{hour}\right) = \textit{PCP Exposure}\left(\frac{\mu g\ ai}{hour}\right) \times CF\ (mg/\mu g) \times DF \times FC$$

Pentachlorophenol Exposure = 10% x 178 $\mu$g/hr TCP
Conversion Factor (CF) = 1 mg per 1000 $\mu$g
Dilution Factor (DF) = 20 fold
Formulation Correction (FC) = ratio (5/3) developed to estimate dermal exposures of a 5% ai RTU from a 3% ai concentration and ratio (10/3) to estimate dermal exposures of a 10% ai RTU formulation in Fenske et al. (1987)[5]

The hourly dermal exposure value was used in conjunction with the exposure duration and body weight to calculate the dermal dose.

***Inhalation Exposure Study***

Two inhalation studies were used to provide chemical-specific handler exposure data in support of reregistration. The specific scenarios that use air concentration data from these studies include: (3a) applying liquid formulation at joinery mills by dipping, and (4) applying liquid formulation at pressure treatment plants - helpers/switchmen. No PHED data were appropriate for either of these scenarios.

Air concentration data to support scenario (3a) comes from the study entitled "Chlorophenol exposure in sawmills" (Kauppinen and Lindroos, 1985).[8] The study measured air concentrations of trichlorophenols, tetrachlorophenols, and pentachlorophenol when dipping wood in vats near work sites at joinery mills. The mean air concentration of 55 $\mu g/m^3$ for trough dipping of wood was used to estimate exposures to joinery mill workers during dipping operations. An exposure period of 8 hours (entire work day) was assumed. The mean air concentration of 55 $\mu g/m^3$ was lower than mean air concentration for indoor vat-dipping (64 $\mu g/m^3$), but higher than outdoor vat-dipping (24 $\mu g/m^3$), and was selected as a reasonable exposure point concentration for this scenario.[8]

The study performed by Kauppinen and Lindroos (1985) was conducted in 10 Finnish sawmills, where a chlorophenol salt formulation was used for the blue stain control of sawed wood. The formulation used for the study contained 10 to 20 % 2,4,6-trichlorophenol (TCP) and about 5 % pentachlorophenol (PCP). The workers were exposed to the TCP/PCP solution after dipping of the boards was conducted. Workers that pulled wood after the dipping process was conducted, were examined in this study.[8] Air sampling was conducted measuring chlorophenols in the air using an impinger containing 25 mL toluene as the absorbing liquid. The pumps used were either electric-circuit driven or battery operated (MSA Monitaire Sampler), with a sampling rate of 1 L/min. After air sampling was conducted, samples were analyzed with a gas chromatograph with an electron capture detector.[8] As in the case with the other studies used to support Pentachlorophenol registration, the usefulness of the data is limited by uncertainties associated with the validity of the data, as well as its conformance to Series 875 standards.[9] The data gaps are listed below.

- Study was conducted in Finland, and does not represent U.S. sawmills.
- Recovery data was not reported.
- Limits of Quantification (LOQs) were not available.
- Sample storage, and laboratory QA/QC were not discussed.
- High variability in data.

The air concentration data used for scenario (4) applying liquid formulation at pressure treatment plants - helpers/switchmen, is the maximum measured air concentration (197.2 $\mu g/m^3$) of pentachlorophenol for a switchman at a wood pressure treatment facility as cited in National Institute for Occupational Safety and Health (NIOSH) (1983) and Appleton (1983).[10,11] The maximum air concentration was used as a conservative estimate of the exposure point concentration for individuals who would be exposed to pentachlorophenol during activities such as opening and closing retort doors, or coming in close proximity to the retort during periods when the doors are open. An exposure duration of 1 hour/day was assumed. This is based on the assumption that retort doors are opened twice a day for 30 minutes at a time.[6] The data from the NIOSH (1983) report were from government commissioned studies during the period of 1975 through 1980 for the purpose of estimating exposure to pentachlorophenol during wood preservative uses. Based on the Agency review, the studies were found to be of unacceptable quality for the purposes of registration.[11] However, due to the lack of acceptable air monitoring data for pentachlorophenol-related uses, these studies were used to provide an estimate of exposure. The NIOSH (1983) study measured air concentrations in the breathing zones of workers conducting various tasks involving pentachlorophenol at pressure treatment plants. The two types of workers measured were a pressure treatment operator who was exposed to concentrations of 10.0 to 22.8 $\mu g/m^3$ during his different tasks and a switchman who was exposed to concentrations of 16.7 to 197.2 $\mu g/m^3$. Note that the results used for this assessment came from a study conducted at several different wood preservative sites and a detailed description of these sites was not provided. Missing details include the actual pentachlorophenol labeled product used with the corresponding percent ai, details of the sampling and analytical methods, and quality control procedures. Because the percent ai was not reported, calculation of typical and maximum exposures (i.e., based on either a 5 or 10 % PCP solution) could not be calculated in this assessment. It should be noted that Appleton's 1983 review of this study indicated that the scientific validity of the results is

highly questionable. "Quality control procedures appear to be non-existent in this study. Agreement between sampling results obtained from the NIOSH and DOW methods was not good and the results were highly variable. Reagent and method blanks should have been prepared and breakthrough volumes determined. There were no field and laboratory spikes to check the recovery of the methods, nor were detection or quantitation limits given for the methods." Appleton (1983) recommended that "based on the lack of scientific validity, this study is not acceptable for use in the registration of PCP." However, based on the lack of other acceptable data, these data were used as a preliminary range-finder for the exposure/risk assessment with the understanding that the results are highly uncertain.[10,11]

**b. PHED Data**

The *Pesticide Handlers Exposure Database (PHED) Version 1.1* was used to estimate exposures for all other handler scenarios.[12] PHED was designed by a task force consisting of representatives from the U.S. EPA, Health Canada, the California Department of Pesticide Regulation, and member companies of the American Crop Protection Association. PHED is a generic database containing measured exposure data for workers involved in the handling or application of pesticides in the field (i.e., currently contains data for over 2,000 monitored exposure events). The basic assumption underlying the system is that exposure to pesticide handlers can be calculated using the monitored data because exposure is primarily a function of the physical parameters of handling and application process (e.g., packaging type, application method, and clothing scenario). PHED also contains algorithms that allow the user to complete surrogate, task-based exposure assessments beginning with one of the four main data files contained in the system (i.e., mixer/loader, applicator, flagger, and mixer/loader/applicator).[12]

Users can select data from each major PHED file and construct exposure scenarios that are representative of the use of the chemical. However, to add consistency to the risk assessment process, the EPA, in conjunction with the PHED Task Force, has evaluated all data within the system and developed surrogate exposure tables that contain a series of standard unit exposure values for various exposure scenarios. These standard unit exposure values are based on the "best fit" values calculated by PHED. PHED calculates "best fit" exposure values by assessing the distributions of exposures for each body part included in

data sets selected for the assessment (e.g., chest or forearm) and then calculating a composite exposure value representing the entire body. PHED categorizes distributions as normal, lognormal, or in any "other" category. Generally, most data contained in PHED are lognormally distributed or fall into the PHED "other" distribution category. If the distribution is lognormal, the geometric mean for the distribution is used in the "best fit" exposure value. If the data are an "other" distribution, the median value of the data set is used in the calculation of the "best fit" exposure value. As a result, the surrogate unit exposure values that serve as the basis for this assessment generally range from the geometric mean to the median of the selected data set. PHED unit exposure data used in this assessment represent the estimated level of exposure expected per unit amount of pesticide handled and are reported in units of mg exposure/lbs ai handled.[12]

**c. Estimated Amount Handled**

Table 2 provides the assumptions used to estimate the amount of Pentachlorophenol handled per day. The sources for these assumptions are presented in the bulleted items below. The estimated amounts handled per day were used in conjunction with PHED data or data from chemical-specific handler studies to yield exposure dose estimates for handlers in various industrial settings.

The following assumptions were used in order to complete the daily amount handled for this assessment:

- Members of the Penta Task Force estimated that the concentration of the typical ready-to-use (RTU) liquid concentrate used in the utility pole wood pressure treatment industry is 5 % active ingredient (ai). This was verified on the following labels: EPA Reg. Nos. 1022-15, 1022-16, and 1022-356). The amount of liquid concentrate RTU made from crystalline block pentachlorophenol is estimated at 7,000 gallons per day based on a personal communication with industry representatives.[6] The density of the liquid is estimated to be 7 lb/gal and was verified as a mid-range density value on the following labels: EPA Reg. Nos. 1022-15, 1022-16, and 1022-356.[6]

- Members of the Penta Task Force estimated that the concentration of the typical pentachlorophenol liquid concentrate used in the wood pressure treatment industry is 40 % ai. This was verified on the following labels, EPA

Reg. Nos. 1022-15 and 1022-120; the amount of technical liquid concentrate made from crystalline block pentachlorophenol is estimated at 15,000 gallons per day. The density is estimated to be 7 lb/gal, and was verified as a mid-range density value on the following labels (EPA Reg. Nos. 1022-15, 1022-16, and 1022-356).[6]

- From Agency review of current pentachlorophenol product labeling, there is a 48 % ai soluble concentrate registered and a ready-to-use product with 10 % ai. These values were used to estimate the maximum amounts handled.[13]

- In some studies the percent ai used in the wood treatment process was 5 % ai. In order to obtain the maximum labeled rate, the study results had to be corrected to account for a 10 % ai solution.[13]

- EPA estimated that the typical amounts of liquid formulation handled at joinery mills is 1,000 gallons and that the typical amounts for use or application on utility poles via pressure treatment retorts is 5,000 gallons.[6]

- Typical Agency upper bound estimates of the amount of liquid used per day in an airless sprayer and low pressure handwand is 50 gallons.

- Typical Agency upper bound estimates of the amount of liquid used per day for painting with a paint brush is 5 gallons.

- Members of the Penta Task Force estimated that a typical crew (three people) can brush or swab 20 utility poles per day (assuming that is the only task for the day) for groundline treatment of utility poles.[6]

**Table 2. Exposure Estimates/Assumptions of Pentachlorophenol for Daily Amount Handled**

| Exposure Scenario | Pounds ai used |
|---|---|
| | ***Primary Handlers - Mixer/Loaders*** |
| (1a) Mixing/Loading Crystalline Technical Grade Product to Make Ready to Use Product (5% ai - typical; 10% - maximum) | Assumes 7,000 gallons of a 5% (typical) or 10% (maximum) ready-to-use PCP product prepared per day from a technical grade PCP using a 7 lb/gal density. Calculated as follows: 7,000 gallons x 0.05 (typical) or 0.10 (maximum) x 7 lb/gal, the total active ingredient is 2,450 lb ai handled/day (typical) or 4,900 lb ai handled/day (maximum). |
| (1b) Mixing/Loading Crystalline Technical Grade Product to Make Concentrated Product (40% ai - typical; 48% - maximum) | Assumes 15,000 gallons of a 40% (typical) or 48% (maximum) concentrated PCP product prepared per day from a technical grade PCP using a 7 lb/gal density. Calculated as follows: 15,000 gallons x 0.4 (typical) or 0.48 (maximum) x 7 lb/gal, the total active ingredient is 42,000 lb ai handled/day (typical) or 50,400 lb ai handled/day (maximum). |
| (2a) Mixing/Loading Liquid Formulation at Joinery Mills (40% diluted to 5% typical; 48% diluted to 10% maximum) | Assumes 1,000 gallons of a 5% (typical) or 10% (maximum) ready-to-use PCP product prepared from a 40% (typical) or 48% (maximum) concentrated PCP product using a 7 lb/gal density. Calculated as follows: 1,000 gallons x 0.05/0.4 (typical) or 0.1/0.48 (maximum) x 7 lb/gal, the total active ingredient is 875 lb ai handled/day (typical) or 1,460 lb ai handled/day (maximum). |
| (2b) Mixing/Loading Liquid Formulation at Pressure Treatment Plants (40% diluted to 5% typical; 48% diluted to 10% maximum) | Assumes 5,000 gallons of a 5% (typical) or 10% (maximum) ready-to-use PCP product prepared from a 40% (typical) or 48% (maximum) concentrated PCP product using a 7 lb/gal density. Calculated as follows: 5,000 gallons x 0.05/0.4 (typical) or 0.1/0.48 (maximum) x 7 lb/gal), the total active ingredient is 4,375 lb ai handled/day (typical) or 7,290 lb ai handled/day (maximum). |
| | ***Secondary Handlers - Applicator*** |
| (3a) Applying Liquid Formulation at Joinery Mills - Dipping | Not needed in assessment; chemical-specific data used. |
| (3b) Applying Liquid Formulation at Joinery Mills - Airless Spraying | Assumes 50 gallons of spray used per day and a maximum usage of 3.6 lb ai per 10 gallons. The total active ingredient is 18 lb ai handled/day. |
| (3c) Applying Liquid Formulation at Joinery Mills - Low Pressure Handwand | Assumes 50 gallons of spray used per day and a maximum usage of 3.6 lb ai per 10 gallons. The total active ingredient is 18 lb ai handled/day. |
| (3d) Applying Liquid Formulation at Joinery Mills - Brushing | Assumes 5 gallons of liquid wood preservative per day and a maximum usage of 3.6 lb ai per 10 gallons. The total active ingredient is 1.8 lb ai handled/day. |
| (4) Applying Liquid Formulation at Pressure Treatment Plants - Helpers/Switchmen | The agency has no data at this time. Dermal exposure not assessed. Not needed in inhalation assessment; chemical-specific data used. |

| Exposure Scenario | Pounds ai used |
|---|---|
| (5) Applying Grease Formulation for Groundline Remediation of Utility Poles - Brushing | Assumes 20 poles treated per day and a maximum usage of 3.8 lb ai per pole. The total active ingredient is 76 lb ai handled/day. |

### ii. Post-application Exposures

The Agency is concerned about potential post-application exposures to pentachlorophenol. According to information obtained from the Agency for Toxic Substances and Disease Registry (ATSDR), soil half-lives are usually on the order of 2-4 weeks. Photolysis in water under laboratory ultraviolet (UV)-light irradiation indicates an estimated half-life of about 100 hours at pH 3.3 and 3.5 hours at pH 7.3. Atmospheric pentachlorophenol is probably photolyzed in the absence of water, although mechanisms for this reaction are not well known. Since the pentachlorophenol is not rapidly degraded, and exhibits moderate toxicity, potential post-application scenarios may be of concern.[15]

The Agency has determined that there are potential exposure concerns relating to post-application exposure to individuals following pentachlorophenol applications in commercial, industrial, and residential settings. The potential individual post-application exposures are outlined below:

***Occupational Postapplication Exposure***

(1) pressure treatment facility yardman;
(2) pressure treatment facility retort maintenance;
(3) pressure treatment facility QA/QC inspector;
(4) other activities adjacent to a pressure treatment plant;
(5) pressure treatment facility storage yard worker/distributer; and,
(6) pole installers.

***Residential/Homeowner Post-application Exposure***

(1) homeowner incidental ingestion and dermal contact with soil contaminated with pentachlorophenol (e.g., soil contaminated by PCP treated utility poles) (child);

(2) outdoor homeowner dermal contact with industry pressure treated wood products (e.g., utility poles, posts, decks, shingles, fencing, lumber, piers, etc.) (adult);

(3) outdoor homeowner incidental hand-to-mouth and dermal contact with industry pressure treated wood products (e.g., utility poles, posts, decks, shingles, fencing, lumber, piers, etc.) (child).

## b. Occupational and Residential Risk Assessment/Characterization

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

### i. Summary of Toxicity Concerns Relating to Occupational and Residential Exposures

### a. Acute Toxicology

The toxicological data base for Pentachlorophenol [2,3,4,5,6- pentachlorophenol] is adequate and will support re-registration eligibility. Toxicity categories for Pentachlorophenol are shown in Table 3. In the case of Pentachlorophenol there were no acceptable guideline inhalation toxicity studies with which to determine toxicity by the inhalation exposure route. In the absence of inhalation data, OPP/HED's Hazard Identification Assessment Review Committee determined that Pentachlorophenol should be placed in Category I for inhalation toxicity. Pentachlorophenol is not classified as a dermal sensitizer. Table 3 summarizes these toxicity findings.[1,2]

**Table 3. Acute Toxicity Categories for Pentachlorophenol**

| Study | Results | Toxicity Category |
|---|---|---|
| Acute Oral Toxicity | $LD_{50}$ = 155 mg/kg (male)<br>$LD_{50}$= 137 mg/kg (female) | II |
| Acute Dermal Toxicity | $LD_{50}$ > 3,980 mg/kg | IV |
| Acute Inhalation Toxicity | No guideline study available to determine a $LD_{50}$. | I |
| Primary Eye Irritation | Corneal involvement at day 7 post-instillation. | II |

| Study | Results | Toxicity Category |
|---|---|---|
| Primary Dermal Irritation | Moderate irritation at 72 hrs. post-application. | III |
| Dermal Sensitization | No dermal sensitization. | NA |

## Selection of Toxicological Endpoints

The pentachlorophenol Hazard Identification Assessment Review Committee report (dated December 8, 1997) indicates that there are toxicological endpoints of concern for pentachlorophenol. Table 4 summarizes these endpoints. A more detailed discussion of the toxicological endpoints is provided in the Hazard Assessment section of this RED.

**Table 4. Toxicological Endpoints for Assessing Occupational and Residential Exposures/Risks**

| Study | Endpoint | Recommended MOE |
|---|---|---|
| Acute Dietary Exposure | NOAEL = 30 mg/kg/day | 100 |
| Chronic Dietary Exposure - Reference Dose (RfD) | LOAEL = 1.5mg/kg/day | 300 |
| Short-term Dermal Exposure (1-7 days) | NOAEL = 30 mg/kg/day | 100 |
| Intermediate-term Dermal Exposure | NOAEL = 30 mg/kg/day | 100 |
| Long-term Dermal Exposure | LOAEL = 1.5mg/kg/day | 300 |
| Short- and Intermediate-term Inhalation Exposure | No endpoint selected. Agency waived acute & 90-day inhalation toxicity studies. | Not selected |
| Oral Cancer Slope Factor[3] | 0.12 (mg/kg/day)[1] | NA |

NA- Not Applicable.

### ii. Handler Risk Assessment and Characterization

#### a. Handler Exposure and Non-Cancer Risk Calculations

Handler exposure assessments are completed by EPA using a baseline exposure scenario and, if required, increasing levels of risk mitigation [personal protective equipment (PPE) and engineering controls] to achieve an appropriate margin of exposure (MOE) or cancer risk. The baseline scenario generally represents a handler wearing long pants, a long-sleeved shirt, no respirator, and no chemical-resistant gloves (there are exceptions pertaining to the use of gloves and these are noted). PPE scenarios generally represent handlers wearing double layer clothing, gloves, and a respirator. Engineering controls generally represent the use of closed systems for mixing/loading the Pentachlorophenol. Table 5 presents the exposure/risk calculations for each exposure scenario.

**Table 5. Handler Short-term, Intermediate-term, and Chronic (Long-term) Dermal Doses/Risks, and Inhalation Doses for Pentachlorophenol (PCP)**

| Exposure Scenario[a] | Daily Amount Handled (lbs ai/day)[b] | Dermal | | | | | Inhalation | | |
|---|---|---|---|---|---|---|---|---|---|
| | | PHED Dermal Unit Exposure (mg/lb ai)[c] | Short- and Intermediate-term Dermal Dose (mg/kg/day)[d] | Chronic Dermal Dose (mg/kg/day)[e] | Short- and Intermediate-term Dermal MOE[f] | Chronic Dermal MOE[g] | PHED Inhalation Unit Exposure (ug/lb ai)[c] | Monitored Air Concentration (ug/m$^3$)[h] | Inhalation Dose (mg/kg/day)[i] |
| ***Primary Handlers - Mixer/Loaders*** | | | | | | | | | |
| (1a) M/L Crystalline Technical Grade Product to Make Ready-to-Use Product (5% ai - typical; 10% - maximum) | 2,450 (typical) 4,900 (max) | 0.00017 Closed Mixing | 0.0028 (typical) 0.0056 (max) | 0.0024 (typical) 0.0048 (max) | 11,000 (typical) 5,400 (max) | Not Applicable; Exposure <180 days/year | 0.034 | -- | 0.0012 (typ.) 0.0024 (max.) |
| (1b) M/L Crystalline Technical Grade Product to Make Concentrated Product (40% ai - typical; 48% - maximum) | 42,000 (typical) 50,400 (max) | 0.00017 Closed Mixing | 0.048 (typical) 0.057 (max) | 0.041 (typical) 0.049 (max) | 630 (typical) 530 (max) | Not Applicable; Exposure <180 days/year | 0.034 | -- | 0.020 (typ.) 0.024 (max.) |
| (2a) M/L Liquid Formulation at Joinery Mills (40% diluted to 5% typical; 48% diluted to 10% maximum) | 875 (typical) 1,460 (max) | 0.0086 Closed Mixing | 0.050 (typical) 0.084 (max) | 0.043 (typical) 0.072 (max) | 600 (typical) 360 (max) | 35 (typical) 21 (max) | 0.083 | -- | 0.0010 (typ.) 0.0017 (max.) |
| (2b) M/L Liquid Formulation at Pressure Treatment Plants (40% diluted to 5% typical; 48% diluted to 10% maximum) | 4,375 (typical) 7,290 (max) | 0.0086 Closed Mixing | 0.25 (typical) 0.42 (max) | 0.22 (typical) 0.35 (max) | 120 (typical) 72 (max) | Not Applicable; Exposure <180 days/year | 0.083 | -- | 0.0052 (typ.) 0.0086 (max.) |
| ***Secondary Handlers - Applicators*** | | | | | | | | | |
| (3a) Applying Liquid Formulation at Joinery Mills - Dipping | -- | No PHED Data Available; 0.593 mg/hr * 8 hrs/day (typ.) and 1.19 mg/hr (max.) * 8 hrs/day | 0.032 (typ.) 0.063 (max.) | 0.027 (typ.) 0.054 (max.) | 950 (typ.) 473 (max.) | 55 (typ.) 28 (max.) | -- | 55 Exposure Time = 8 hr/day[k] | 0.0079 (typ.) 0.016 (max.) |
| (3b) Applying Liquid Formulation at Joinery Mills - Airless Spraying | 3.6 lb ai/10 gal * 50 gal/day | 38 (Baseline) | 4.6 | 3.9 | 6.6 | <1 | 830 (Baseline) | -- | 0.21 |
| | | 14 (PPE) | 1.7 | 1.4 | 18 | 1.0 | 83 (PPE) | | 0.021 |
| (3c) Applying Liquid Formulation at Joinery Mills - Low Pressure Handwand | 3.6 lb ai/10 gal * 50 gal/day | 100 (Baseline) | 12 | 10 | 2.5 | <1 | 30 (Baseline) | -- | 0.0077 |
| | | 0.37 (PPE) | 0.044 | 0.038 | 680 | 39 | 3.0 (PPE) | | 0.00077 |
| (3d) Applying Liquid Formulation at Joinery Mills - Brushing | 3.6 lb ai/10 gal * 5 gal/day | 180 (Baseline) | 2.2 | 1.9 | 14 | <1 | 280 (Baseline) | -- | 0.0072 |
| | | 22 (PPE) | 0.26 | 0.23 | 110 | 6.6 | 28 (PPE) | | 0.00072 |

| Exposure Scenario[a] | Daily Amount Handled (lbs ai/day)[b] | Dermal | | | | | Inhalation | | |
|---|---|---|---|---|---|---|---|---|---|
| | | PHED Dermal Unit Exposure (mg/lb ai)[c] | Short- and Intermediate-term Dermal Dose (mg/kg/day)[d] | Chronic Dermal Dose (mg/kg/day)[d] | Short- and Intermediate-term Dermal MOE[f] | Chronic Dermal MOE[g] | PHED Inhalation Unit Exposure (ug/lb ai)[c] | Monitored Air Concentration (ug/m$^3$)[h] | Inhalation Dose (mg/kg/day)[i] |
| (4) Applying Liquid Formulation at Pressure Treatment Plants - Helpers/Switchmen | No Data | No Data | -- | -- | -- | -- | -- | 197 Exposure Time = 1 hr/day[j] | 0.0035 |
| (5) Applying Grease Formulation for Groundline Remediation of Utility Poles - Brushing | 3.8 lb ai/pole * 20 poles treated/day | 180 (Baseline) | 91 | 78 | <1 | <1 | 280 (Baseline) | -- | 0.30 |
| | | 22 (PPE) | 11 | 9.6 | 2.7 | <1 | 28 (PPE) | | 0.030 |

[a] Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.

[b] Primary handler - mixer/loader amounts handled/day based on personal communications with industry representatives, as described in Table 2.
Secondary handler - applicator amounts handled/day based on the amount of ai applied per gallon or per pole, as indicated on the PCP labels and/or LUIS (10/20/97) report. Number of gallons used per day based on EPA standard assumptions for the development of REDs, and the number of poles treated per day based on personal communications with industry contacts, as discussed in the text.

[c] PHED Version 1.1 used; for mixer/loaders, data from the mixer/loader file used; for applicators, PHED data from mixer/loader/applicator file used. Scenario descriptors and data confidence are as follows:

Scenarios (1a) and (1b): data for granular used; single layer clothing, no gloves, closed mixing, no respirator; hands = 10 replicates, all grades, low confidence; dermal = 33-78 replicates, ABC grades, low confidence; inhalation = AB grades, 58 replicates, high confidence; baseline data times 0.02 to account for 98% protection from closed mixing.

Scenarios (2a) and (2b): liquid formulation, mechanical transfer; single layer clothing, gloves, closed mixing, no respirator; hands = 31 replicates, AB grades, high confidence; dermal = 16-22 replicates, AB grades, high confidence; inhalation = 27 replicates, AB grades, high confidence.

Scenario (3a): No PHED data available.

Scenario (3b): baseline - single layer clothing, no gloves, open mixing, no respirator; hands = 15 replicates, B grade, high confidence; dermal = 15 replicates, B grade, high confidence; inhalation = C grade, 15 replicates, medium confidence; dermal PPE - double layer clothing, gloves; hand data = same data as baseline with a 90% protection factor to account for gloves; dermal = same data as baseline with a 50% protection factor to account for double layer of clothing; inhalation PPE assumes respirator provides 90% protection.

Scenario (3c): baseline - single layer clothing, no gloves, open mixing, no respirator; hands = 70 replicates, all grades, low confidence; dermal = 9-80 replicates, ABC grades, low confidence; inhalation = ABC grades, 80 replicates, medium confidence; dermal PPE - double layer clothing, gloves; hands = 10 replicates, ABC grade, low confidence; dermal = same data as baseline with a 50% protection factor to account for double layer of clothing; inhalation PPE assumes respirator provides 90% protection.

Scenario (3d): baseline - single layer clothing, no gloves, open mixing, no respirator; hands = 15 replicates, AB grades, low confidence; dermal = 14-15 replicates, C grade, low confidence; inhalation = C grade, 15 replicates, medium confidence; dermal PPE - double layer clothing, gloves; hand data = same data as baseline with a 90% protection factor to account for gloves; dermal = same data as baseline with a 50% protection factor to account for double layer of clothing; inhalation PPE assumes respirator provides 90% protection.

Scenario (4): No PHED data available.

Scenario (5): baseline - single layer clothing, no gloves, open mixing, no respirator; hands = 15 replicates, AB grades, low confidence; dermal = 14-15 replicates, C grade, low confidence; inhalation = C grade, 15 replicates, medium confidence; dermal PPE - double layer clothing, gloves; hand data = same data as baseline with a 90% protection factor to account for gloves; dermal = same data as baseline with a 50% protection factor to account for double layer of clothing; inhalation PPE assumes respirator provides 90% protection.

[d] Short- and Intermediate-term Dermal Dose = [PHED Unit Exposure (mg/lb ai) * Amount of ai handled per day (lbs ai/day) * Absorption Factor (0.40)] / Body Weight (60 kg) or [Hourly Dermal Exposure (mg/hr) * Exposure Time (hr/day) * Absorption Factor (0.40)]/Body Weight (60 kg).

Chronic Dermal Dose = [PHED Unit Exposure (mg/lb ai) * Amount of ai handled per day (lbs ai/day) * Absorption Factor (0.40)] / Body Weight (70 kg) or [Hourly Dermal Exposure (mg/hr) * Exposure Time (hr/day) * Absorption Factor (0.40)]/Body Weight (70 kg).
Short- and Intermediate-term Dermal MOE = Short- and Intermediate-term NOAEL (30 mg/kg/day) / Short- and Intermediate-term Dermal Dose (mg/kg/day).
Chronic Dermal MOE = Chronic NOAEL (1.5 mg/kg/day) / Chronic Dermal Dose (mg/kg/day).
Air Concentration data based on monitoring studies found in the literature, as follows:

Scenario (3a): Kauppinnen and Lindroos (1985).

Scenario (4): NIOSH (1983).

Inhalation dose (all time periods)= [PHED Unit Exposure (ug/lb ai) * Conversion Factor (0.001 mg/ug) * Amount of ai handled per day (lbs ai/day) / Body Weight (70 kg); or [Air Concentration ($ug/m^3$) * Conversion Factor (0.001 mg/ug) * Inhalation Rate (1.25 $m^3$/hr) * Exposure Duration (hr/day) * FC (unitless; to convert air concentrations from use of 5% PCP solutions to concentrations from use of 10% PCP) / Body Weight (70 kg)].
Hourly dermal exposure based on TCP gloved hand and forearm data from Fenske et al. (1987) in which PCP exposure was observed as one-tenth the TCP exposure; the formulation used in the study was 3% PCP (instead of 5% or 10%) and the solution was diluted 20 fold (Brodberg and Thongsinthusak, 1995). Thus, 178 ug/hr was adjusted as follows: 178 ug/hr TCP exposure * 0.1 = 17.8 ug/hr PCP exposure; 17.8 * 20 * 5/3 = 593 ug/hr or 0.593 mg/hr for typical uses and 17.8 * 20 * 10/3 = 1,187 $\mu$g/hr or 1.19 mg/hr for maximum uses.
Based on the assumption that dipping and related activities could be carried out over an entire workday. Typical concentration based on 5% PCP, as used in study. Maximum exposure based on adjustment to 10% PCP.
Based on the assumption that exposure to PCP in pressure treatment cylinders occurs only when the doors are opened, and that opening occurs once or twice per day for about 30 minutes. These assumptions are based on information provided by industry contacts, as described in the text. The percent of ai in solution not reported in study. Thus, it is not known whether this is a typical or maximum value.

***Inhalation Dose***

The daily inhalation dose was calculated using a 60 kg body weight for both short-term and intermediate-term exposure and 70 kg body weight for chronic exposures.[1] For scenarios for which no chemical-specific information was available, PHED data were used as surrogates to assess doses. Daily inhalation dose was calculated as follows:

$$\text{Daily Inh. Dose}\left(\frac{\text{mg ai}}{\text{kg/day}}\right) = \text{PHED Unit Exp.}\left(\frac{\mu\text{g}}{\text{lb ai}}\right) \times \text{CF } (\text{mg}/\mu\text{g}) \times \text{amt. handled}\left(\frac{\text{lbs ai}}{\text{day}}\right) \times \left(\frac{1}{\text{Body Weight (kg)}}\right)$$

PHED Unit Exp ($\mu$g/lb) = Values obtained from PHED Version 1.1;
CF (0.001 mg/$\mu$g) = conversion factor ;
Amount handled (lb ai/day) = values from Table 2;
Body Weight (kg) = 60 kg for short- and intermediate-term and 70 kg for chronic.

Chemical-specific data were available for scenarios (3a) and (4). For these scenarios, specific inhalation studies which measured air concentrations in joinery mills (Kauppinnen and Lindroos,1985) and pressure treating facilities (NIOSH, 1983) were used to derive an inhalation dose. The inhalation dose from an air concentration was calculated as follows:

$$\text{Daily Inh. Dose}\left(\frac{\text{mg ai}}{\text{kg/day}}\right) = \text{Air Concentration}\left(\frac{\mu\text{g}}{\text{m}^3}\right) \times \text{CF } (\text{mg}/\mu\text{g}) \times \text{IR}\left(\frac{\text{m}^3}{\text{hr}}\right) \times \text{ED}\left(\frac{\text{hr}}{\text{day}}\right) \times \left(\frac{1}{\text{Body Weight (kg)}}\right) \times \text{FC (unitless)}$$

Air Concentration = Values obtained from studies (Kauppinnen and Lindross, 1985; and NIOSH, 1983); [8,10]
CF (0.001 mg/$\mu$g) = conversion factor;
ED (hr/day) =8 hours for joinery mills and 1 hour for helper/switchmen in pressure treatment plants (Personal Communication with members of the Penta Task Force, 1998)[6] ;
Body Weight (kg) =60 kg for short- and intermediate-term and 70 kg for chronic;
FC = ratio (10/5) developed to estimate inhalation exposures of a 10% ai ready-to-use (RTU) solution from a 5% ai concentration. No FC was needed to estimate typical concentrations using

a 5% ai RTU. Note that this ratio was used for scenario (3a) in which a known 5% pentachlorophenol solution was used. However, for scenario (4), the % pentachlorophenol was not reported. Thus, the air concentration from this study was used directly. It is not known whether this represents a typical or maximum value.

***Dermal Dose***

The daily dermal dose was calculated using a 60 kg body weight for both short-term and intermediate-term exposure and 70 kg body weight for chronic exposures. For scenarios for which no chemical-specific information was available, PHED data were used as surrogates to assess doses. Daily dermal dose was calculated as follows:

$$\text{Daily Dermal Dose}\left(\frac{\text{mg ai}}{\text{kg/day}}\right) = \text{PHED Unit Exp.}\left(\frac{\text{mg}}{\text{lb ai}}\right) \times \text{amt. handled}\left(\frac{\text{lbs ai}}{\text{day}}\right) \times \left(\frac{1}{\text{Body Weight (kg)}}\right) \times \text{ABS (\%)}$$

PHED Unit Exp (mg/lb ai ) = values obtained from (PHED, 1998)[12] ;
CF (mg/$\mu$g) = conversion factor ;
Amount handled (lb ai/day) = values from Table 2 ;
Body Weight (kg) = 60 kg for short- and intermediate-term and 70 kg for chronic[1] ;
ABS = 40% absorption[1] .

Chemical-specific data were available for scenario (3a). The dermal dose was calculated as follows:

$$\text{Daily Dermal Dose}\left(\frac{\text{mg ai}}{\text{kg/Day}}\right) = \text{Hourly Dermal Exposure}\left(\frac{\text{mg ai}}{\text{hr}}\right) \times \text{ET (hrs)} \times \left(\frac{1}{\text{Body Weight (kg)}}\right) \times \text{ABS (\%)}$$

Hourly Dermal Exposure = Calculations outlined in section 4.b.2.a;
Exposure Time = 8 hours;
Body Weight (kg) = 60 kg for short- and intermediate-term and 70 kg for chronic[1];
ABS = 40% absorption[1].

The calculations of the daily dermal dose of pentachlorophenol received by handlers were used to calculate the short-term, intermediate-term, and chronic dermal MOEs. The daily dermal MOE was calculated using a NOAEL of 30 mg/kg/day for short- and intermediate-term exposure, and a NOAEL of 1.5 mg/kg/day for chronic exposures.[1] The following formula describes the calculation of a dermal MOE:

$$Dermal\ MOE = \left( \frac{NOAEL\ (mg/kg/day)}{Dermal\ Dose\ (mg/kg/day)} \right)$$

The acceptable MOE target for total short- and intermediate-term exposure for dermal was 100 and the acceptable MOE for chronic exposure was 300.[1]

**b. Handler Exposure and Cancer Risk Calculations**

The lifetime average daily dose was calculated by adding the chronic dermal and inhalation doses and accounting for exposure frequency, exposure duration, and lifetime. Exposure duration was assumed to be 40 years and is the standard value used by OPP to represent a working lifetime. This is assumed to be a conservative value. Lifetime is assumed to be 75 years. This is the recommended value for the U.S. population, as cited in EPA's Exposure Factors Handbook (U.S. EPA, 1997).[14] Exposure frequency is scenario-specific. Table 6 details the exposure frequency values and handler cancer risk estimates. Exposure frequencies were based on Agency estimates and information provided by industry contacts.[6] For example, Scenario (1a) M/L Technical Grade Product to Make Ready-to-Use Product, is assumed to occur once per month; Scenario (1b) M/L Technical Grade Product to Make Concentrated Product is assumed to occur 10 times per month or twice per week. This is based on information from industry contacts that indicated that the formulation of concentrated product occurs much more frequently than ready-to-use product.[6] Scenario (2b) M/L Liquid Formulation at Pressure Treatment Plants assumes that retorts are filled once per week, and that residual pentachlorophenol in the retort is reused for subsequent retort loads. All other handler scenarios assume an exposure frequency of 250 days per year (i.e., 5 days per week, 50 days per year). This is a standard Agency assumption for days worked per year. The following formula describes the calculation of the lifetime average daily dose:

$$\text{Lifetime Average Daily Dose}\left(\frac{\text{mg ai}}{\text{kg/day}}\right) = \left(\frac{[\text{Chronic Dermal Dose (mg/kg/day)} + \text{Inhalation Dose (mg/kg/day)}] \times \text{Exposure Frequency (days/yr)} \times \text{Exposure Duration (yrs)}}{365\ \text{days/yr} \times \text{Lifetime (yrs)}}\right)$$

It should be noted that, based on EPA standard practice, cancer risks were assessed using only the typical assumptions for the amount of ai handled per day.

Risk was calculated by multiplying the lifetime average daily dose times the cancer slope factor (e.g., 0.12 mg/kg/day)$^{-1}$ using the following formula:

$$\text{Risk} = \text{LADD}\left(\frac{\text{mg ai}}{\text{kg/day}}\right) \times \text{Cancer Slope Factor}\left(\frac{1}{(\text{mg/kg/day})}\right)$$

**Table 6. Handler Cancer Risks for Pentachlorophenol (PCP)**

| Exposure Scenarios[a] | Chronic Dermal Dose (mg/kg/day)[b] | Inhalation Dose (mg/kg/day)[b] | Exposure Frequency (days/year)[c] | Exposure Duration (years)[d] | Lifetime (years)[d] | LADD (mg/kg/day)[e] | Cancer Risk[f] |
|---|---|---|---|---|---|---|---|
| ***Primary Handlers - Mixer/Loaders*** | | | | | | | |
| (1a) Mixing/Loading Crystalline Technical Grade Product to Make Ready-to-Use Product (5% ai) | 0.0024 | 0.0012 | 12 | 40 | 75 | 0.000063 | $7.6 \times 10^{-6}$ |
| (1b) Mixing/Loading Crystalline Technical Grade Product to Make Concentrated Product (40% ai) | 0.041 | 0.020 | 120 | 40 | 75 | 0.011 | $1.3 \times 10^{-3}$ |
| (2a) Mixing/Loading Liquid Formulation at Joinery Mills (40% diluted to 5%) | 0.043 | 0.0010 | 250 | 40 | 75 | 0.016 | $1.9 \times 10^{-3}$ |
| (2b) Mixing/Loading Liquid Formulation at Pressure Treatment Plants (40% diluted to 5%) | 0.22 | 0.0052 | 50 | 40 | 75 | 0.016 | $2.0 \times 10^{-3}$ |
| ***Secondary Handlers - Applicators*** | | | | | | | |
| (3a) Applying Liquid Formulation at Joinery Mills - Dipping | 0.027 | 0.0079 | 250 | 40 | 75 | 0.013 | $1.5 \times 10^{-3}$ |
| (3b) Applying Liquid Formulation at Joinery Mills - Airless Spraying | 3.9 | 0.21 | 250 | 40 | 75 | 1.5 | $1.8 \times 10^{-1}$ |
| | 1.4 | 0.021 | | | | 0.52 | $6.2 \times 10^{-2}$ |
| (3c) Applying Liquid Formulation at Joinery Mills - Low Pressure Handwand | 10 | 0.0077 | 250 | 40 | 75 | 3.6 | $4.4 \times 10^{-1}$ |
| | 0.038 | 0.00077 | | | | 0.014 | $1.7 \times 10^{-3}$ |
| (3d) Applying Liquid Formulation at Joinery Mills - Brushing | 1.9 | 0.0072 | 250 | 40 | 75 | 0.70 | $8.4 \times 10^{-2}$ |
| | 0.23 | 0.00072 | | | | 0.084 | $1.0 \times 10^{-2}$ |
| (4) Applying Liquid Formulation at Pressure Treatment Plants - Helpers/Switchmen | -- | 0.0035 | 250 | 40 | 75 | 0.0013 | $1.5 \times 10^{-4}$ (inhal. only) |
| (5) Applying Grease Formulation for Groundline Remediation of Utility Poles - Brushing | 78 | 0.30 | 250 | 40 | 75 | 29 | $3.4 \times 10^{0}$ |
| | 9.6 | 0.030 | | | | 3.5 | $4.2 \times 10^{-1}$ |

[a] Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.
[b] Taken from Table 5.

[c] Based on personal communications with industry contacts and assumptions regarding work frequency (i.e., 250 days is equivalent to full time work; 5 days per week, 50 weeks per year).
[d] Based on data from EPA's Exposure Factors Handbook; U.S. EPA, 1997.
[e] Lifetime Average Daily Dose (LADD) = [(Chronic Dermal Dose (mg/kg/day) + Inhalation Dose (mg/kg/day) * Exposure Frequency (days/yr) * Exposure Duration (yrs)] / [365 days/yr * Lifetime (yrs)].
[f] Risk = LADD (mg/kg/day) * Cancer Slope Factor (0.12 mg/kg/day$^{-1}$; IRIS; U.S. EPA, 1998).

## c. Handler Non-Cancer Risks from Dermal Exposures to Pentachlorophenol

Acute, sub-chronic, and chronic toxicity endpoints related to dermal exposures to pentachlorophenol have been identified. A MOE of greater than 100 for pentachlorophenol is considered to indicate no risk concern for short-term and intermediate-term exposures, and a MOE of greater than 300 for pentachlorophenol is considered to indicate no risk concern for chronic exposures. The results presented in Table 5 are summarized in the following bulleted categories.

***Handler Scenarios with Non-Cancer Risk Concerns (Short-term and Intermediate-term Risk)***

The calculations of short-term and intermediate-term risks indicate that dermal MOEs are more than 100 at **baseline** for the following scenarios:

- (3a) Applying liquid formulation at joinery mills - dipping.

The calculations of short-term and intermediate-term risks indicate that dermal MOEs are more than 100 **with additional PPE** for the following scenarios:

- (3c) Applying liquid formulation at joinery mills - low pressure handwand.
- (3d) Applying liquid formulation at joinery mills - brushing.

The calculations of short-term and intermediate risks indicate that dermal MOEs are more than 100 **with additional Engineering Controls** for the following scenarios (note that production of pentachlorophenol uses only engineering controls):

- (1a) Mixing/loading crystalline technical grade product to make ready-to-use product (5% ai) (closed mixing).
- (1b) Mixing/loading crystalline technical grade product to make concentrated product (40% ai) (closed mixing).

- (2a) Mixing/loading liquid formulation at joinery mills (closed mixing).

- (2b) Mixing/loading formulation at pressure treatment plants (closed mixing using typical industry estimates).

The calculations of short-term and intermediate-term risk indicate that the dermal MOEs are less than 100 despite the maximum mitigation measures for the remainder of the scenarios.

- (2b) Mixing/loading formulation at pressure treatment plants (closed mixing using maximum estimates).

- (3b) Applying liquid formulation at joinery mills - airless spraying.

- (5) Applying grease formulation for groundline remediation of utility poles - brushing.

***Data Gaps***

Data gaps exist for the following scenario:

- (4) Applying liquid formulation at pressure treatment plants - helpers/switchmen.

***Handler Scenarios with Non-Cancer Risk Concerns (Chronic Risk)***

The calculations of chronic-term risks indicate that dermal MOEs are less than 300 at **baseline, with additional PPE and Engineering Controls** for all of the applicable scenarios assessed. The calculations of chronic-term risk indicate that the MOEs did not equal or exceed the 300 threshold despite the maximum mitigation measures for these scenarios.

The following scenarios were not assessed because the exposure was less than 180 days/year:

- (1a) Mixing/loading crystalline technical grade product to make ready-to-use product (5% ai):

- (1b) Mixing/loading crystalline technical grade product to make ready-to-use product (40% ai).

- (2b) Mixing/loading liquid formulation at pressure treatment plants.

***Data Quality and Confidence in Assessment***

Several issues must be considered when interpreting the occupational exposure risk assessment. These include:

- Several handler assessments were completed using "low quality" PHED data due to the lack of a more acceptable data set.
- Several generic protection factors were used to calculate handler exposures. These protection factors have not been completely evaluated and accepted by the Antimicrobials Division.
- Factors used to calculate daily exposures to handlers (e.g., amount handled) were based on personal communications with industry representatives.

**d. Handler Non-Cancer Risks from Inhalation Exposures to Pentachlorophenol**

No toxicity endpoints have been identified for inhalation exposure.

**e. Handler Cancer Risks from Dermal and Inhalation Exposures to Pentachlorophenol**

***Handler Scenarios with Cancer Risk Concerns***

Carcinogenic endpoints related to dermal and inhalation exposures to pentachlorophenol have been identified. A risk at the $10^{-6}$ to $10^{-5}$ range indicates only a limited risk concern. The results in Table 6 are summarized in the following bulleted categories.

The following scenario indicates a cancer risk concern **at baseline** and **with PPE** that is below $10^{-5}$:

- (1a) Mixing/loading crystalline technical grade product to make ready-to-use product (5% ai).

The remainder of the scenarios are greater than $10^{-5}$ even with maximum mitigation measures.

Data gaps exist for the following scenario:

- (4) Applying liquid formulation at pressure treatment plants - helpers/switchmen.

### iii. Post-application Risk Assessment and Characterization

**Occupational Post-application Data and Assumptions, Exposures and Risks**

In the course of development of this RED, chemical-specific handler data identified from pertinent literature sources were used in conjunction with both industry and Agency estimates of exposure parameters to predict exposures. Tables 7 and 8 include the exposure/risk calculations for non-cancer and cancer risks for each exposure scenario.

### a. Chemical-Specific Post-application Exposure Data

***Dermal Exposure Studies***

Two dermal studies were used to provide chemical-specific post-application dermal exposure data in support of the re-registration of pentachlorophenol. The first study (Fenske et al., 1987)[5] was used in *California's PCP Risk Characterization Document* (Estimation of exposure of persons in California to pesticide products containing pentachlorophenol by Brodberg and Thonginthusak, 1995) to address potential human health risks from pentachlorophenol exposure in occupational scenarios involving pressure treatment of lumber and logs.[7] The Fenske et al. (1987) data, along with some of the assumptions used in the Brodberg and Thonginthusak report, were used in this document to develop hourly dermal exposures for the following post-application scenario:[5,7] (1) pressure treatment facility yardman. Calculations for the hourly dermal exposures are identical to those in the handler section of this

document. The exposure duration for a pressure treatment facility yardman is assumed to be 2 hours/day. This represents one-fourth of a workday and assumes that a worker conducts other tasks for the remainder of the day.

The second chemical-specific dermal post-application study was entitled "Occupational Exposure of Electrical Utility Linemen to Pentachlorophenol."[16] The purpose of the study was to characterize chronic or long-term exposure of pentachlorophenol to lineman by examining pentachlorophenol levels on gloves. Occupational exposure data and other factors such as age and work experience were considered. The study used both biological monitoring measurements of pentachlorophenol (e.g., using urine samples) and measurement of pentachlorophenol residues found on the gloves of utility linemen. The activities of the electrical utility linemen include frequent climbing of new or in-service pentachlorophenol-treated poles, which require significant skin contact to pentachlorophenol containing oils that run down the surface of the utility pole.[16] No mention of the exact concentration (% ai) of pentachlorophenol in the pressure-treated utility poles was mentioned in the study; since the percent ai was not reported, typical and maximum exposures could not be calculated. Utility poles are typically pressure-treated with 3.8 lb pentachlorophenol/pole.[17]

The study examined 23 utility linemen workers and used 5 administrative workers as controls. The study measured workers' exposure for over 150 days. Since the urinary elimination half-life of pentachlorophenol was observed to be 15 days, the sampling frequency was set at once every four weeks. Approximately 10 of the workers exchanged gloves after each four week monitoring period. The other utility workers could exchange gloves based on their own judgement.[16] For the purposes of this assessment, it was assumed that the concentrations on the gloves represented a single exposure period. This is a conservative assumption and may overestimate exposure. However, because it was not known whether glove residues from multiple exposure events had actually accumulated on the gloves or whether residues had dissipated from previous exposure events, the concentration at the time of sampling was used as a conservative estimate of the exposure period being evaluated here.

Horsehide gloves were used for line work. The level of pentachlorophenol on gloves was determined by using the following method. Three areas of 0.64 $cm^2$ were stamped out from

the thumb, index finger and palm area. The stamped glove material was placed in a vial in contact with a solution containing toluene and an internal standard and agitated in an ultrasonic bath for 10 minutes. The extracting solution was further diluted and analyzed by using gas chromatography.[16]

The overall geometric mean concentration of pentachlorophenol residues reported during periodic measurement of workers' gloves was 29.6 $\pm$ 1.74 and the maximum geometric mean reported was 41.5 $\mu g/cm^2$. There was an increase in exposure during the warmer summer days and exposure to pentachlorophenol tended to be greater with less qualified workers.[16]

The most conservative value reported (e.g., maximum geometric mean of reported glove residue concentrations) was used in the exposure calculations. The calculation of the dermal dose used in Table 7 is presented below. Calculation of dermal MOEs and cancer risks are analogous to the calculations identified in the handler sections (Section 4.b.3.a and 4.b.3.b, respectively).

$$Daily\ Dermal\ Dose\left(\frac{mg\ ai}{kg/Day}\right) = PCP\ Residue\left(\frac{mg\ ai}{cm^2}\right) x\ SA\ (cm^2)\ x\left(\frac{1}{Body\ Weight\ (kg)}\right) x\ ABS\ (\%)$$

PCP Residue ($mg/cm^2$) = value reported in study;[16]
SA ($cm^2$) = 612 $cm^2$ [residues are transferred to the palms of the hands and forearms with gloves and long-sleeves providing 90% protection to the hands and 50% protection to the arms (420 $cm^2$ (palms) x 0.1 + 1,140 $cm^2$ (forearms) x 0.5) based on information cited in U.S. EPA 1997 [12]];
Body Weight (kg) = 60 kg for short- and intermediate-term and 70 kg for chronic;[1]
ABS= 40% absorption.[1]

Short- and intermediate-term dermal and chronic dermal MOEs were calculated, as described earlier for handlers, and are presented in Table 7.

***Inhalation Exposure Studies***

The Penta Task Force submitted five workplace exposure studies in support of registration.[10] These were all government commissioned studies during the period of 1975 through 1980 for the purpose of estimating exposure to pentachlorophenol during wood preservative uses.[10] Based on the Agency review, the studies were found to be of unacceptable quality for the purposes of

registration.[11] However, due to the lack of acceptable air monitoring data for pentachlorophenol-related occupational uses, these studies were used to provide an estimate of exposure.

One of the studies entitled "Industrial Hygiene Surveys of Occupational Exposure to Wood Preservative Chemicals" reviewed by Appleton in 1983 was used to evaluate inhalation exposures for the following scenarios [10,11]:

- (1) Pressure Treatment Facility Yardman;
- (3) Pressure Treatment Pole Inspector;
- (4) Other Activities Adjacent to a Pressure Treatment Plant;
- (5) Pressure Treatment Facility Storage Yard Worker/Distributer.

The study was conducted to assess worker exposures to wood preservative chemicals at eleven wood pressure treatment plants and two manufacturing plants. Airborne exposure levels and methods of exposure control were evaluated using NIOSH and alternate sampling and analytical procedures. Personal breathing zone concentrations for yardmen at a typical pressure treatment plant ranged from 5.1 to 14.5 $\mu g/m^3$. Air concentrations adjacent to the plant were at a level of 1.2 $\mu g/m^3$, and were 7.0 $\mu g/m^3$ in a pole storage area 50-60 feet downwind. At a non-pressure treatment plant, a pole inspector's air exposure concentration was 170.1 $\mu g/m^3$. The maximum air concentration for each of these worker populations was used in the exposure calculations. Because no information was available on the percent of pentachlorophenol in the formulation used, it was not possible to characterize these data as typical or maximum.[10,11]

Appleton's (1983) review of this study indicated that the scientific validity of the results is highly questionable. "Quality control procedures appear to be non-existent in this study. Agreement between sampling results obtained from the NIOSH and DOW methods was not good, and the results obtained were highly variable. Reagent and method blanks should have been prepared and breakthrough volumes determined. There were no field and laboratory spikes to check the recovery of the methods, nor were detection or quantitation limits given for the methods."[11] Appleton (1983) recommended that "Based on the lack of scientific validity, this study is not acceptable for use in the registration of PCP."[11] However, based on the lack of other acceptable data, these data were used as a preliminary range-finder for the exposure/risk assessment with the understanding that the results are highly uncertain.

The second study used to estimate exposure was Brodberg and Thonsinthusak (1995).[7] Brodberg and Thongsinthusak (1995) used the data above in conjunction with another data source to estimate the mean air exposure concentration for a retort maintenance worker in the pressure treating industry.[7] This mean value was used in estimating exposure for Scenario (2) Pressure treatment facility retort maintenance worker.

Inhalation dose was calculated from an air concentration as follows:

$$\text{Daily Inh. Dose}\left(\frac{mg\ ai}{kg/day}\right) = \text{Air Concentration}\left(\frac{ug}{m^3}\right) \times CF\ (mg/ug) \times IR\left(\frac{m^3}{hr}\right) \times ED\left(\frac{hr}{day}\right) \times \left(\frac{1}{\text{Body Weight (kg)}}\right)$$

Air Concentration = Values obtained from studies; [7,10,11]
CF (0.001 mg/μg) = conversion factor;
IR ($m^3$/hr) = inhalation rate; 1.25 $m^3$/hr;[14]
ED (hr/day) =2 hours for pressure treatment facility yardmen; 1 hour for pressure treatment facility retort maintenance and pressure treatment facility pole inspector; 3 hours for activities adjacent to a pressure treatment plant; and 8 hours for pressure treatment facility storage yard worker/distributor; [6]
Body Weight (kg) = 60 kg for short- and intermediate-term and 70 kg for chronic. [1]

Note that no toxicity values were available to predict inhalation MOEs. Postapplication inhalation exposures are presented in Table 7.

**b. Modeling Approaches**

Because no dermal data were identified in the available literature for a pressure treatment facility QA/QC inspector, the DERMAL model was used to provide an estimate of dermal exposure. The DERMAL model is frequently used by the U.S. EPA's Office of Pollution Prevention and Toxics (OPPT) for estimates of exposure to chemical substances during use of consumer products.[18,19] The model calculates dermal exposure using the film thickness approach. This method assumes that contact with a formulated product results in the presence of a film of material on the surface of the skin. Using the weight fraction of the ai in the product, and

assumptions regarding the film thickness of pentachlorophenol, the surface area of the skin exposed and the density of pentachlorophenol, dermal exposures were calculated. For the purposes of this assessment, the film thickness of pentachlorophenol on the skin was assumed to be the same as that used for motor oil in the dermal model. This is believed to be a reasonable assumption because pentachlorophenol is an oil-based product. The density for motor oil was also used. This value is consistent with the density provided on pentachlorophenol labels (i.e., 7 lbs/gal). The palms of the hands were assumed to be exposed. The DERMAL model exposure calculation is identified below:

$$Dermal\ Dose\left(\frac{mg\ ai}{kg/day}\right) = SA/BW\left(\frac{cm^2}{kg}\right) x\ FT(cm)\ x\ D\left(\frac{g}{cm^3}\right) x\ WF(unitless)\ x\ ABS(\%)\ *\ CF\ mg/g$$

SA/BW ($cm^2/kg$) = 7.8 $cm^2/kg$ skin surface area per body weight;[19]
FT (cm) = 1.588 E-02 cm film thickness of PCP on skin; [19]
D ($g/cm^3$) = 0.84 $g/cm^3$ density;
WF (unitless) = 0.05 and 0.1 weight fraction of ai in PCP product;
ABS (%) = 40% absorption factor;[1]
CF = conversion factor (1,000 mg/g).

**Table 7. Post-application Short-term, Intermediate-term, and Chronic (Long-term) Dermal Doses/Risks, and Inhalation Doses for Pentachlorophenol (PCP)**

| Exposure Scenario[a] | Dermal | | | | | Inhalation | |
|---|---|---|---|---|---|---|---|
| | Dermal Exposure Data | Short- and Intermediate-term Dermal Dose (mg/kg/day) | Chronic Dermal Dose (mg/kg/day) | Short- and Intermediate-term Dermal MOE[b] | Chronic Dermal MOE[c] | Monitored Air Concentration ($ug/m^3$)[d] | Inhalation Dose (mg/kg/day)[e] |
| (1) Pressure Treatment Facility Yardman (i.e., unloading trams of treated wood) | 0.593 mg/hr (typ.) * 2 hrs/day[f]<br>1.19 mg/hr (max.) * 2 hrs/day | 0.0079 (typ.)[g]<br>0.016 (max.) | 0.0068 (typ.)[h]<br>0.014 (max.) | 3,800 (typ.)<br>1,900 (max.) | 220 (typ.)<br>110 (max.) | 14.5<br>Exposure Time = 2 hrs/day | 0.00052 |
| (2) Pressure Treatment Facility Retort Maintenance | No Dermal Data Available | -- | -- | -- | -- | 23.7<br>Exposure Time = 1 hr/day | 0.00042 |
| (3) Pressure Treatment Facility QA/QC Inspector (i.e., QA/QC) | [i] | 0.21 (typ.)[i]<br>0.42 (max.) | 0.21 (typ.)[i]<br>0.42 (max.) | 140 (typ.)<br>70 (max.) | 7.2 (typ.)<br>3.6 (max.) | 170<br>Exposure Time = 1 hr/day | 0.0030 |
| (4) Other Activities Adjacent to a Pressure Treatment Plant (i.e., non-wood handling employees) | No Dermal Data Available | -- | -- | -- | -- | 1.2<br>Exposure Time = 3 hrs/day | 0.000064 |
| (5) Pressure Treatment Facility Storage Yard Worker/Distributor | No Dermal Data Available | -- | -- | -- | -- | 7.0<br>Exposure Time = 8 hrs/day | 0.0010 |
| (6) Pole Installers | 25.3 mg/day[j] | 0.17[j] | 0.15[j] | 180 | 10 | No Inhalation Data Available | — |

[a] Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.

[b] Short- and Intermediate-term Dermal MOE = Short- and Intermediate-term NOAEL (30 mg/kg/day) / Short- and Intermediate-term Dermal Dose (mg/kg/day).

[c] Chronic Dermal MOE = Chronic NOAEL (1.5 mg/kg/day) / Chronic Dermal Dose (mg/kg/day).

[d] Air Concentration data based on monitoring studies found in the literature, as follows:
Scenario (1), (3), (4), and (5): NIOSH (1981).
Scenario (2): Brodberg and Thongsinthusak (1995).

[e] Inhalation dose = [Air Concentration ($ug/m^3$) * Conversion Factor (0.001 mg/ug) * Inhalation Rate (1.25 $m^3$/hr) * Exposure Duration (hr/day) * 1.0 (100% Absorption)] / Body Weight (70 kg)].

[f] Hourly dermal exposure based on TCP gloved hand and forearm data from Fenske (1987) in which PCP exposure was observed as one-tenth the TCP exposure; the formulation used in the study was 3% PCP (instead of 5% or 10%) and the solution was diluted 20 fold (Brodberg and Thongsinthusak, 1995). Thus, 178 ug/hr was adjusted as follows: 178 ug/hr TCP exposure * 0.1 = 17.8 ug/hr PCP exposure; 17.8 * 20 * 5/3 = 593 ug/hr or 0.593 mg/hr for typical uses and 17.8 * 20 * 10/3 = 1,187 $\mu$g/hr or 1.19 mg/hr for maximum uses.

[g] Short and Intermediate Dermal Dose=(0.593 mg/hr*2 hr/day * Abs (40%))/60 kg for typical uses and (1.19 mg/hr * 2 hr/day * Abs (40%))/60 kg.

[h] Chronic Dermal Dose= (0.593 mg/hr * 2 hr/day * Abs (40%))/70 kg for typical uses and (1.19 mg/hr * 2 hr/day * Abs (40%))/70 kg.

[i] OPPT's film thickness approach used to estimate dermal doses: Dermal Dose = 0.78 $cm^2$/kg skin surface exposed * 1.588E-2 cm film thickness of PCP on skin * 0.84 $g/cm^3$ (density of PCP formulation) * 0.05 or 0.1 (weight fraction of ai in PCP product) * 0.40 (absorption fraction) * 1,000 mg/g.

[j] The PCP residue concentration (e.g., maximum geometric mean) of 41.5 $\mu g/cm^2$ PCP was transferred onto the gloves of a representative sample of utility pole linemen (Thind et al., 1991). It is assumed the PCP residues in linemen would be transferred to the palms of the hands and forearms with gloves and long-sleeves providing 90% protection to the hands and 50% protection to the arms (420 $cm^2$ * 0.1 (palms) +1,140 $cm^2$ * 0.5 (forearms) = 612 $cm^2$ surface area exposed). The amount of PCP available per day would equate to 25.3 mg/day (41.5 $\mu g/cm^2$ * 0.001 mg/$\mu$g * 612 $cm^2$/day). Dermal dose= [25.3 mg/day x 40% absorption]/[Body weight (60 kg for short- and intermediate-term, or 70 kg for chronic]. Note that a ratio of (10/5) is used to correct for a 10% ai RTU solution.

**Table 8. Post-application Cancer Risks for Pentachlorophenol (PCP)**

| Exposure Scenario[a] | Chronic Dermal Dose (mg/kg/day)[b] | Inhalation Dose (mg/kg/day)[b] | Exposure Frequency (days/year)[c] | Exposure Duration (years)[d] | Lifetime (years)[d] | LADD (mg/kg/day)[e] | Cancer Risk[f] |
|---|---|---|---|---|---|---|---|
| (1) Pressure Treatment Facility Yardman (i.e., unloading trams of treated wood) | 0.0068 | 0.00052 | 250 | 40 | 75 | 0.0026 | $3.2 \times 10^{-4}$ |
| (2) Pressure Treatment Facility Retort Maintenance | -- | 0.00042 | 50 | 40 | 75 | 0.000031 | $3.7 \times 10^{-6}$ (inhal. only) |
| (3) Pressure Treatment Facility Pole Inspector (i.e., QA/QC) | 0.21 | 0.0030 | 250 | 40 | 75 | 0.078 | $9.3 \times 10^{-3}$ |
| (4) Other Activities Adjacent to a Pressure Treatment Plant (i.e., non-wood handling employees) | -- | 0.000064 | 250 | 40 | 75 | 0.000023 | $2.8 \times 10^{-6}$ (inhal. only) |
| (5) Pressure Treatment Facility Storage Yard Worker/Distributor | -- | 0.0010 | 250 | 40 | 75 | 0.00037 | $4.4 \times 10^{-5}$ (inhal. only) |
| (6) Pole Installer | 0.15 | -- | 250 | 40 | 75 | 0.055 | $6.6 \times 10^{-3}$ (dermal only) |

[a] Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.
[b] Taken from Table 7.
[c] Based on personal communications with industry contacts and assumptions regarding work frequency (i.e., 250 days is equivalent to full time work; 5 days per week, 50 weeks per year).
[d] Based on data from EPA's Exposure Factors Handbook; U.S. EPA, 1997.
[e] Lifetime Average Daily Dose (LADD) = [(Chronic Dermal Dose (mg/kg/day)] + Inhalation Dose (mg/kg/day)} * Exposure Frequency (days/yr) * Exposure Duration (yrs)] / [365 days/yr * Lifetime (yrs)].
[f] Risk = LADD (mg/kg/day) * Cancer Slope Factor (0.12 $mg/kg/day^{-1}$; IRIS; U.S. EPA, 1998).

### c. Post-application Cancer Risk Calculations

Post-application cancer risks were calculated in the same manner as for handlers. The exposure durations and lifetime values used were the same as for handlers. Exposure frequency was assumed to be 250 days/year (i.e., standard annual working frequency) for all scenarios except (2) Pressure Treatment Retort Maintenance. Retort maintenance was assumed to occur once per week (i.e., 50 times/year). Estimated cancer risks from dermal and inhalation post-application exposures are presented in Table 8.

## Occupational Post-application Risk Assessment and Characterization

### a. Post-application Non-Cancer Risks from Dermal Exposure to Pentachlorophenol

Acute/subchronic and chronic toxicity endpoints related to dermal exposures to pentachlorophenol have been identified. A dermal MOE grater than 100 for pentachlorophenol is considered to indicate no risk concern for short-term and intermediate-term exposures, and a dermal MOE greater than 300 for pentachlorophenol is considered to indicate no risk concern for chronic exposures. The results of Table 7 are summarized in the following bulleted categories.

***Occupational Post-application Scenarios with Non-Cancer Risk Concerns (Short- and Intermediate-Term Risk)***

The calculations of short-term and intermediate-term risks indicate that a dermal MOEs are more than 100 at **baseline** for the following scenarios:

- (1) Pressure Treatment Facility Yardman;
- (3) Pressure Treatment Facility QA/QC Inspector (typical);
- (6) Pole Installers.

The calculations of short-term and intermediate-term risks indicate that dermal MOEs are less than 100 at **baseline** for the following scenarios:

- (3) Pressure Treatment Facility QA/QC Inspector (maximum)

Data gaps exist for the following scenarios:

- (2) Pressure Treatment Facility Retort Maintenance;
- (4) Other Activities Adjacent to a Pressure Treatment Plant;
- (5) Pressure Treatment Facility Storage Yard Worker/Distributor.

***Occupational Post-application Scenarios with Non-Cancer Risk Concerns (Chronic Risk)***

The calculations of chronic risks indicate that dermal MOEs are less than 300 at baseline for the following scenarios:

- (1) Pressure Treatment Facility Yardman ;
- (3) Pressure Treatment Facility QA/QC Inspector;
- (6) Pole Installers.

Data gaps exist for the following scenarios:

- (2) Pressure Treatment Facility Retort Maintenance;
- (4) Other Activities Adjacent to a Pressure Treatment Plant;
- (5) Pressure Treatment Facility Storage Yard Worker/Distributor.

**b. Post-application Non-Cancer Risks from Inhalation Exposures to Pentachlorophenol**

No toxicity endpoints have been identified for inhalation exposure.

**c. Post-application Cancer Risks from Dermal and Inhalation Exposures to Pentachlorophenol**

***Occupational Post-application Scenarios with Cancer Risk Concerns***

Carcinogenic endpoints related to dermal and inhalation exposures to pentachlorophenol have been identified. A risk at the E-6 to E-5 range indicates a limited risk concern. The results of Table 8 are identified in the following bulleted categories.

The following scenarios indicate a cancer risk concern below $10^{-5}$:

- (2) Pressure Treatment Facility QA/QC Inspector (inhalation only);
- (4) Activities Adjacent to a Pressure Treatment Plant (inhalation only).

The remainder of the scenarios are greater than the $10^{-6}$ to $10^{-5}$ range.

**Residential Post-application Exposure and Risk Characterization**

The Agency is concerned that there are potential exposure concerns relating to post-application exposure to individuals following pentachlorophenol applications in residential settings. The potential residential post-application exposure pathways are outlined below:

(1) homeowner incidental ingestion and dermal contact with soil contaminated with Pentachlorophenol (e.g., soil contaminated by PCP-treated utility poles) (child);

(2) outdoor homeowner dermal contact with industry pressure-treated wood products (e.g., utility poles, posts, decks, shingles, fencing, lumber, piers, etc.) (adult);

(3) outdoor homeowner hand-to-mouth and dermal contact with industry pressure-treated wood products (e.g., utility poles, posts, decks, shingles, fencing, lumber, piers, etc.) (child).

**a. Residential Post-application Data and Assumptions, and Exposure and Risk Calculations**

No chemical-specific post-application studies conforming to OPPTS Test Guidelines Series 875 guidelines were available; however, two studies (see subsection 8a) were found from scientific literature sources and were used to provide estimates of exposure for the following exposure pathways: (1) homeowner contact with soil contaminated with pentachlorophenol (child); (2) outdoor homeowner contact with industry pressure treated wood products (adult); and (3) outdoor homeowner contact with industry pressure treated wood products (child).

Tables 9 and 10 include the exposure/risk calculations for non-cancer and cancer risks for each scenario. The calculations are described in the exposure estimates subsection.

**Table 9. Residential Post-application Short-term, Intermediate-term, and Chronic (Long-term) Dermal Doses/Risks, and Ingestion Doses for Pentachlorophenol (PCP)**

| Exposure Scenario[a] | Dermal | | | | | Ingestion | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Exposure Data[b] | Short- and Intermediate-term Dermal Dose[c] (mg/kg/day) | Chronic Dermal Dose[d] (mg/kg/day) | Short- and Intermediate-term Dermal MOE[e] | Chronic Dermal MOE[f] | Short- and Intermediate-term Oral Dose[c] (mg/kg/day) | Chronic Dose[d] (mg/kg/day) | Short- and Intermediate-term Oral MOE[e] | Chronic Oral MOE[f] |
| (1) Contact With Soil Contaminated with PCP From a Utility Pole (Child) | CS= 3.4 mg/kg (min) and 654 mg/kg (max) | 2.17E-04 (min)<br>4.18E-02 (max) | 2.17E-04 (min)<br>4.18E-02 (max) | 1.38E+05 (min)<br>7.18E+02 (max) | 6.90E+03 (min)<br>3.59E+01 (max) | 2.27E-05 (min)<br>4.36E-03 (max) | 2.27E-05 (min)<br>4.36E-03 (max) | 1.32E+06 (min)<br>6.88E+03 (max) | 6.62E+04 (min)<br>3.44E+02 (max) |
| (2) Outdoor Residential Contact With Industry Pressure-Treated Wood Products (e.g.,utility poles, fencing, porches, shingles, steps, and decks) (Adults) | Cwipe= 8.96E-04 ug/cm2 (min) 1.87E-03 ug/cm2 (max) | 6.27E-07 (min)<br>1.31E-06 (max) | 5.38E-07 (min)<br>1.12E-06 (max) | 4.78E+07 (min)<br>2.29E+07 (max) | 2.79E+06 (min)<br>1.34E+06 (max) | NA | NA | NA | NA |
| (3) Outdoor Residential Contact With Industry Pressure-Treated Wood Products (e.g.,utility poles, fencing, porches, shingles, steps, and decks) (Child) | Cwipe= 8.96E-04 ug/cm2 (min) 1.87E-03 ug/cm2 (max) | 9.87E-06 (min)<br>2.06E-05 (max) | 9.87E-06 (min)<br>2.06E-05 (max) | 3.04E+06 (min)<br>1.46E+06 (max) | 1.52E+05 (min)<br>7.28E+04 (max) | 1.30E-04 (min)<br>2.72E-04 (max) | 1.30E-04 (min)<br>2.72E-04 (max) | 2.30E+05 (min)<br>1.10E+05 (max) | 1.15E+04 (min)<br>5.51E+03 (max) |

NA- Not applicable. Scenario not of concern.

a Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.
b Refer to Section 8a for data used to provide exposure estimates.
c Refer to Section 8b for exposure estimates - non cancer calculations for scenario-specific calculations
d Short- and intermediate-term and chronic dose are identical with respect for children; however, for adults, the body weights are adjusted based on toxicity (see Section 8b - Dermal Exposure to PCP Treated Wood Products).
e Short- and intermediate-term (dermal/oral) MOE - short- and intermediate-term NOAEL (30 mg/kg/day) / short- and intermediate-term (dermal/oral) dose (mg/kg/day).
f Chronic (dermal/oral) MOE = chronic (dermal/oral) NOAEL (1.5 mg/kg/day) / chronic (dermal/oral) dose (mg/kg/day).

Refer to Section (8) (b) for formulas used to calculate exposure doses and MOEs.

**Table 10. Residential Post-application Cancer Risks for Pentachlorophenol (PCP)**

| Exposure Scenarios[a] | Chronic Dermal Dose (mg/kg/day)[b] | Chronic Oral Dose[b] (mg/kg/day) | Exposure Frequency[c] (days/year) | Exposure Duration[d] (years) | Lifetime[d] (years) | LADD[e] (mg/kg/day) | Cancer Risk[f] |
|---|---|---|---|---|---|---|---|
| (1) Contact With Soil Contaminated with PCP (Child) | 2.17E-04 (min)<br>4.18E-02 (max) | 2.27E-05 (min)<br>4.36E-03(max) | 365 | 3 | 75 | 9.6E-06 (min)<br>1.9E-03 (max) | 1.2E-06 (min)<br>2.2E-04 (max) |
| (2) Outdoor Residential Contact With Industry Pressure-Treated Wood Products (e.g., utility poles, fencing, porches, shingles, steps, and decks) (Adults) | 5.38E-07 (min)<br>1.12E-06 (max) | NA | 365 | 30 | 75 | 2.2E-07 (min)<br>4.5E-07 (max) | 2.6E-08 (min)<br>5.4E-08 (max) |
| (3) Outdoor Residential Contact With Industry Pressure-Treated Wood Products (e.g.,utility poles, fencing, porches, shingles, steps, and decks) (Child) | 9.87E-06 (min)<br>2.06E-05 (max) | 1.30E-04 (min)<br>2.72E-04 (max) | 365 | 3 | 75 | 5.6E-06 (min)<br>1.2E-05 (max) | 6.7E-07 (min)<br>6.4E-06 (max) |

NA- Not applicable. Scenario not of concern.

a Exposure scenarios based on EPA/OPP/AD's Use Profile for PCP.
b Taken from table 9.
c Based on data from EPA's Risk Assessment Guidance for Superfund (U.S. EPA, 1989).
d Based on data from EPA's Exposure Factors Handbook (U.S. EPA, 1997).
e Lifetime average daily dose (LADD) = [chronic dermal dose (mg/kg/day) + inhalation dose (mg/kg/day)] * exposure frequency (days/yr) * exposure duration (yrs) / [365 days/yr * lifetime (yrs)].
f Risk = LADD (mg/kg/day) * cancer slope factor $(0.12\ mg/kg/day)^{-1}$. [Note: Cancer slope factor from IRIS, 1998; U.S. EPA, 1998.]

Refer to Section (8) (c) for formulas used to calculate LADD and cancer risk.

## b. Chemical-Specific Post-application Exposure Data

*Summary of Existing Data Found in the Available Literature*

Most of the studies found in the available literature that address residential exposure pertained to indoor exposure from pentachlorophenol-treated lumber in log homes. The use of pentachlorophenol-treated lumber in the construction of log homes is no longer an allowed use.[17] This use pattern is currently banned as a result of the 1984/1986 "Rebuttable Presumption Against Registration" (RPAR) proceedings on wood preservatives.[17] However, a brief description of these studies is provided in the following paragraphs as a qualitative indicator of exposure that might occur from indoor homeowner contact with pentachlorophenol-treated products. Data from some of these studies were also used in quantitative estimates of exposure, as appropriate. It should be noted that two coats of sealant must be applied to interior structures of pentachlorophenol-treated wood used for indoor structures. Thus, the exposure values reported in studies where no sealant was used would be expected to be higher than when this protective measure is used. In addition, the amount of pentachlorophenol-treated wood in a log home is likely to be substantially greater than in a house built with pentachlorophenol-treated wood beams that have been properly sealed with two coats. Other studies dealt with pentachlorophenol in outdoor soil or household dust. These studies were not submitted by the registrant, but were found in the scientific literature. The following two paragraphs provide previous data for log homes.

Based on an exposure/risk assessment for pentachlorophenol conducted by California's Department of Pesticide Regulations, "Exposure in pre-existing log homes constructed of pressure-treated logs is estimated from the mid-point of the range for these homes (0.5 - 10 $\mu g/m^3$). For rest activity levels (respiration of 0.72 $m^3$/hr for a 75.9 kg male), this would result in a 15 hour exposure and a dosage of 0.71 $\mu$g/kg/day."[7]

ATSDR (1994) reported the following: "Pentachlorophenol was detected at a geometric mean concentration of 0.080 ng/L (7.3 ppb) in 62 of 63 air samples taken in 21 log homes treated with the compound. The homes, all located in Kentucky, were categorized into six treatment types: (1) "never treated"; (2) external treatment; (3) manufacturer treated; (4) treated and sealed; (5) treated, sealed, and neutralized; (6) treated and neutralized. Concentrations in 'never treated' homes, which were lower than those in treated homes, were believed to be the result of application of pentachlorophenol to logs during storage to prevent fungal growth. Treated logs were found to be the source of pentachlorophenol in indoor air; air concentrations

were highly correlated with pentachlorophenol concentrations in wood cores (geometric mean 15,900 ng/g wood) and log surface wipes (geometric means 89.6 and 187 ng/100 $cm^2$). Concentrations of pentachlorophenol in older structures built with pressure-treated wood brushed with pentachlorophenol were reported to range from 0.5 to 10 $\mu g/m^3$. Use of sealers decreased this concentration by 85 percent. Indoor air concentrations for interiors of structures built with industrially dipped non-pressure-treated wood were reported to contain levels of pentachlorophenol that ranged from 34 to 104 $\mu g/m^3$. Logs used for home construction are no longer treated with pentachlorophenol."[15]

Note that the above study mentioned that log surface wipe samples were also collected and analyzed. Because log homes used pentachlorophenol-treated logs, it was assumed that the log surface wipes could be used as an indicator of residues left on other pentachlorophenol- treated wood products (e.g., fencing, decking, porches, shingles, and decks).

Homeowners could also come into contact with pentachlorophenol in soil near pressure-treated lumber or utility poles (i.e., Scenario 1). One study in the existing literature estimated soil concentrations associated with this use. Pentachlorophenol concentrations of 3.4 to 654 ppm were found within 12 inches of treated poles.[15,20] These data were collected in 1976. Therefore, it was uncertain whether the data were representative of current conditions. However, recent data from a 1995 study confirms these initial estimates and actually reports some results that are higher. [21] Thus, the earlier source, coming from a peer reviewed document, was used as an estimate of soil concentrations for Scenario 1.

Other studies provided estimates of household surface dust concentration, but were not suitable to provide estimates of human exposure from specific pentachlorophenol uses. As reported by Coad and Newhook (1992), "Pentachlorophenol measurements in household surface dust have been reported in dwellings with no known indoor pentachlorophenol wood preservative applications or treatments. The median concentration in 41 "control" homes in Germany was 0.008 $\mu g$ PCP/g dust."[22] Another study reported "a mean of 4.8 ppm PCP in the household dust of four Seattle residences; the decks outside of two of these homes were treated with PCP preservatives 3 and 5 years previously." A third study reported that "PCP was recently detected in the household dust of all of nine North Carolina homes surveyed, with an average of 0.83 $\mu g/g$ dust."[22] "The grand mean of 0.58 $\mu g$ PCP/g dust, incorporating all reported PCP concentrations in American and European household dust, was chosen to represent the levels of PCP dust contamination likely to be found in the average Canadian home."[22] These data represent indoor PCP concentrations from a variety of sources and cannot be linked to specific pentachlorophenol

use scenarios. Thus, they cannot be used to estimate residential exposures and risks from labeled uses being considered for reregistration. Additional data are needed to adequately evaluate residential exposure and risk from pentachlorophenol use.

Only one source of residential outdoor inhalation exposure data was identified in the available literature. This is assumed to be a negligible exposure route. "Inhalation of estimated ambient levels of pentachlorophenol in the atmosphere has an associated exposure level of 6 $\mu$g/day for the general population. Subpopulations in the vicinity of pentachlorophenol sources and workers, may be exposed to significantly higher levels."[15] The short- and intermediate-term dose is calculated to be 1 x $10^{-4}$ mg/kg/day, which is about 5-times lower than the dose reported for a pressure treatment facility yardman who unloads trams of wood (5.2 x $10^{-4}$ mg/kg/day), and similar to the nonwood handling employee exposure (6.4 x $10^{-5}$ mg/kg/day). This study could not be used as an estimate of outdoor residential exposure because it was not specific to any particular use scenario. Instead, it provided data on ambient levels of pentachlorophenol in the atmosphere to which the general population may be exposed.

***Data Used to Provide Exposure Estimates***

In addition to the studies described above, the following studies were reviewed for information pertaining to the post-application residential scenarios being evaluated in this assessment. Data from these studies were used to provide exposure concentrations used in Tables 9 and 10 for Scenarios 1-3:

Arsenault (1976) described a study in which pentachlorophenol was measured in the soil. The concentrations reported in this study were used as a soil concentration estimate for scenario (1). In the Arsenault (1976) study, "soil samples were collected from the base of 30 pentachlorophenol-treated utility poles. Approximately three samples of soil were taken from around the base of the pole at distances of 1 inch, 12 inches, and 60 inches from the pole. Four samples were composited and analyzed as a single sample. Wood samples were also taken from the pole to serve as a comparison. The soil samples were air dried. Aliquots were extracted by a hexane-acetic acid procedure in a Soxhelt extractor for four hours. The solvent was further extracted with a 1 percent caustic solution to remove impurities. The acidified water-pentachlorophenol solution was then further extracted with fresh hexane solution. The hexane was evaporated to dryness using nitrogen gas and the sample analyzed by GC."[20]

The results of the analysis showed that the pentachlorophenol concentration within 1 inch of the pole averaged 658 ppm (one value was as high as 9,500 ppm). At a distance of 12 inches from the poles, the soil had an average concentration of only 3.4 ppm with the maximum being 40 ppm.

In another related study, EPRI (1995) collected samples from 31 pole sites in the state of New York. "These included a range of soil types, wood types, pole sizes, and in-service ages. Approximately 40 to 50 soil samples were collected from each wood pole site in this study and were examined for pentachlorophenol. Methods of analysis followed EPA and ASTM standards. Soil samples were collected from four distances away from the edge of a pole: approximately 3, 8, 18, and 30 inches. Wood samples were taken at various soil depths."[21]

The results of this study indicate that the maximum soil pentachlorophenol concentrations at most pole sites were less than 100 ppm; however, a maximum concentration of at least 100 ppm was observed at nine (29 percent) of the poles sites. Note that the highest pentachlorophenol concentration was 1,900 ppm. The maximum pentachlorophenol concentration of soil at each pole site (if all soil samples were averaged) within 3 inches was 510 ppm and the maximum pentachlorophenol concentration of soil at each pole site (if all soil samples were averaged) within 8 inches was 72 ppm.[21]

Note that these EPRI (1995) results are similar to the results of the Arsenault (1976) study. The data from Arsenault (1976) was used in the exposure risk calculations in this document because of the larger range and the larger geographic scope of the study. Note that some QA/QC methods (percent recovery, storage of samples, storage stability of samples, and field and method blanks) were not reported.

EPA (1986) described a study in which 12 to 21 surface wipe samples were collected from inside 21 homes in Kentucky. Two separate sets of wipe samples were collected and analyzed. One set of wipe samples was collected adjacent to a wood core sample and the second set of wipe samples was collected from likely exposure surfaces inside the log homes. The collection of a wipe sample was as follows: a Whatman Smear Tab was saturated with methanol and wiped over a 100 $cm^2$ area. After the wipe, the wipe sample was collected in a 1 oz. bottle with a screw top. The samples were placed in a cooler with dry ice and were air freighted to a laboratory. A field blank sample was analyzed from every third house. The field blanks were spiked to obtain a range of 700 to 900 ug/sample.[24]

The wipe samples were extracted using hexane and were analyzed using a packed column GC with an electron capture device. Geometric means of 89.6 for the exposure samples (min) and 187 ng/100 $cm^2$ for the adjacent samples (max) were reported. The QA/QC for this study appeared to be acceptable except for the lack of percent recovery information. The log surface wipe samples were used as an estimate of exposure for other wood products (decks, fencing, etc.) For the purposes of this report, however, the major limitations of using the results of this study are as follows:

- Since pentachlorophenol use in log homes has been banned, it is unknown whether the amount of pentachlorophenol applied to the logs is the same as other wood products.

- The wipe sampling was conducted inside. It is unknown what the results of wipe sampling conducted on the outside of the building would have been.

**c. Exposure Estimates - Non-Cancer Calculations**

The following section identifies the assumptions, exposure calculations and risk calculations used to evaluate the non-cancer risks of residential exposure of pentachlorophenol. The overall results are displayed in Table 9. In each scenario, specific calculations of exposure dose (e.g., average daily dose) are explained as well as the general calculation for MOEs. Note that the chronic doses for adults are calculated differently from the short- and intermediate-term doses because of different toxicity endpoints (e.g., short- and intermediate toxicity endpoints specific to females) which results in a change of body weight selected for the calculation as 70 kg not 60 kg. The short- and intermediate-term and chronic doses for children are identical.

***Homeowner Contact With Soil Contaminated With Pentachlorophenol Around a Utility Pole***

The calculations of ingestion and dermal contact with soil contaminated with pentachlorophenol (child) around a utility pole are described in the equations below. Note that no specific inhalation studies for soil around utility poles could be identified. Contribution to the overall risk to children through inhalation of soil particles would be expected to be negligible. In addition, the adult exposure to soil contaminated with PCP was not assessed because the risk concerns for children are expected to be higher than for adults because children ingest more soil, and have lower body weights. The ingestion and inhalation pathways for an adult exposed to PCP in soil would be negligible, and dermal exposure would be confined to the hands and feet.

Children could potentially play in the soil and place dirt on their hands, arms, feet, and legs. Exposure was estimated using PCP concentrations in the soil, as reported by Arsenault (1976).[15,20]

***Ingestion of Soils***

The average daily dose (ADD) for ingestion was estimated using the following general equation:

$$ADD = \frac{CS \times IR \times CF \times FI}{BW}$$

where:

| | |
|---|---|
| ADD= | average daily dose (mg/kg-day); |
| CS= | concentration of PCP in soil (mg/kg); |
| IR= | ingestion rate (mg/day); |
| CF= | conversion factor (kg/mg); |
| FI= | fraction ingestion (%); and |
| BW= | body weight (kg). |

The minimum and maximum concentrations of PCP in soil (CS) are 3.4 and 654 ppm (mg/kg), respectively.[15] The ingestion rate (IR) of 100 mg/day is the mean rate for children.[14] A conversion factor (CF) of $10^{-6}$ was applied to account for converting kg to mg. Children (3-years old) weigh 15 kg.[14] Children at this age are assumed to be the highest exposed individuals because of a very high ingestion rate of soil compared to body weight. The fraction of ingestion (FI) was assumed to be 100 %. Note that the FI is a conservative estimate because the child is assumed to ingest dirt at the PCP concentration from around a utility pole. This may not be entirely realistic, and would represent a worst case condition.

***Dermal Contact of Soils***

The average daily dose (ADD) for dermal contact with soils around a utility pole was calculated as follows:

$$ADD = \frac{CS \times CF \times SA \times AF \times ABS}{BW}$$

where:

ADD= average daily dose (mg/kg-day);
CS= concentration of PCP in soil (mg/kg);
CF= conversion factor (kg/mg);
SA= surface area ($cm^2$/day);
AF= adherence factor ($mg/cm^2$);
ABS= dermal absorption (%); and,
BW= body weight (kg).

The minimum and maximum concentrations of PCP in soil (CS) would be 3.4 to 654 ppm (e.g., mg/kg).[15, 20] A conversion factor (CF) of 1E-06 was applied to account for converting kg to mg. The surface area (SA) was assumed to be 1,652.5 $cm^2$.[14] It was assumed that children playing near a utility pole could possibly be exposed on the legs, hands, and arms while crawling, which would be 25% of the total skin surface area for children (90th percentile for children 3 yrs old).[14] The adherence factor (AF) of 1.45 $mg/cm^2$ is a commonly used AF for commercial potting soil.[23] The dermal absorption (ABS) of 40 % is provided by EPA.[1] The mean body weight (BW) of children age 3 is 15 kg.[14]

***Outdoor Residential Contact With Industry Pressure-Treated Wood Products***

Limited dermal residue exposure data exists for utility poles, fencing, porches, shingles, steps, and decks. Homeowners may come in contact with these surface in their daily activities. Because of the lack of appropriate data, log surface wipes (geometric means 89.6 and 187 ng/100 $cm^2$) from an EPA study examining pentachlorophenol in log homes was used as "surrogate" exposure data to estimate residential exposure to utility poles, fencing, porches, shingles, steps, and decks.[15, 24]

The dermal pathway was the only pathway calculated for adult exposure to industrially treated wood products (e.g., scenario 2). It would not be expected that adults would ingest residues from treated wood (e.g., hand-to-mouth transfer). Worst case risks for oral ingestion can be examined by using a childhood exposure scenario (e.g., Scenario 3). Child exposure to treated wood products would be expected to be the most conservative estimate of the overall risk.

***Ingestion of Pentachlorophenol from Pressure-Treated Wood Products Due To Hand-To-Mouth Transfer***

The average daily dose (ADD) for ingestion of pentachlorophenol residues due to hand-to-mouth transfer from treated wood was calculated as follows:

$$ADD = \frac{Cwipe \times SA \times FQ \times ET}{BW}$$

where:

ADD= average daily dose (mg/kg-day);
$C_{wipe}$= concentration of log surface wipe samples (mg/cm$^2$);
SA= surface area of hands (cm$^2$/event);
FQ= frequency (events/hr);
ET= exposure time (hr/day); and,
BW= body weight (kg).

Using the existing surface wipe data (Cwipe) of 89.6 and 187 ng/100 cm$^2$ found on PCP-treated logs, Cwipe converts to 8.96 x $10^{-7}$ mg/cm$^2$ (min) and 1.87 x $10^{-6}$ mg/cm$^2$ (max).[15] The Pentachlorophenol residues from the surface wipe sample are assumed to be entirely transferred onto the surface of the hand. The assumed surface area (SA) of a 3-year old child's hands is 350 cm$^2$, which is approximately 5 % of the total surface area of a 3-year old child (90th percentile for children 3 yrs old).[14] For the purposes of this assessment, children are assumed to ingest the entire amount of PCP residues on their hands. The amount or frequency (FQ) of events of hand-to-mouth activity is 1.56 events per hour and the exposure time (ET) is 4 hours/day spent outdoors.[25] The mean body weight (BW) of children age 3 is 15 kg.[14]

***Dermal Exposure to Pentachlorophenol -Treated Wood Products***

The average daily dose (ADD) for dermal exposure of PCP residues from treated wood was calculated as follows:

$$ADD = \frac{Cwipe \times SA \times TF \times ABS}{BW}$$

where:

ADD= average daily dose (mg/kg-day);
Cwipe= concentration of log surface wipe samples (mg/cm$^2$);

SA= surface area of hand ($cm^2$/day);
TF= transfer factor (%);
ABS= dermal absorption (%); and
BW= body weight (kg).

Cwipe is 8.96x $10^{-7}$ mg/$cm^2$ (min) and 1.87E-06 mg/$cm^2$ (max).[15] Adults would be exposed only on the palms of one side of the hand. The assumed surface area (SA) is 420 $cm^2$.[14] Children could possibly crawl on pentachlorophenol treated wood decks. Using this assumption, approximately 25 % of the total surface area (SA) for children (90th percentile for children 3 yrs old) or a SA of 1652.5 $cm^2$ was used.[14] A transfer factor (TF) of 25 % was assumed. Essentially, 25 % of the residues were assumed to be transferred from the treated wood to the body surface area. EPA provided a dermal absorption (ABS) of 40 %.[1] The mean body weight (BW) of children age 3 is 15 kg.[14] The short- and intermediate-term ADD was calculated using an adult body weight of 60 kg because the endpoint is specific for females (i.e., female rats were dosed). The chronic ADD was calculated using a body weight of 70 kg because the endpoint is specific to males.[1]

***Calculation of Dermal and Ingestion MOEs***

The Hazard Identification Assessment Review Committee has not yet issued toxicity information for residential risk assessments.[1] The toxicity endpoints for the occupational risk assessment were used for post-application calculations of short-term, intermediate-term, and chronic dermal MOEs. The daily dermal MOE for short- and intermediate-term exposures was calculated using a NOAEL of 30 mg/kg/day. A NOAEL of 1.5 mg/kg/day was used for calculating the daily dermal MOE for chronic (long-term) exposures.[1] The following formula describes the calculation of a dermal MOE:

$$Dermal\ MOE = \left( \frac{NOAEL\ (mg/kg/day)}{Dermal\ Dose\ (mg/kg/day)} \right)$$

The acceptable dermal MOE for short- and intermediate-term adult dermal exposure is 100. An acceptable oral MOE of 100 is also used for acute dietary exposure.[1] The short- and intermediate dermal pathways of exposure were assessed for both adults and children. The acute oral pathway was only assessed for children, since children are more likely to ingest soil from their hands than adults. Note that the acceptable MOE for chronic dermal exposure of adults and children is 300. For chronic dietary exposures to children a target MOE of 300 was also used. [1]

The following formula describes the calculation of an ingestion MOE.

$$Ingestion\ MOE = \left( \frac{NOAEL\ (mg/kg/day)}{Ingestion\ Dose\ (mg/kg/day)} \right)$$

### d. Exposure Estimates - Cancer Risks

The lifetime average daily dose (LADD) was calculated by adding the chronic average daily doses (ADDs) for both ingestion and dermal. The chronic ADDs were then amortized over a lifetime by accounting for exposure frequency, exposure duration, and lifetime. The following formula describes the calculation of the lifetime average daily dose:

$$Lifetime\ Average\ Daily\ Dose \left( \frac{mg\ ai}{kg/day} \right) = \left( \frac{[Chronic\ Dermal\ Dose\ (mg/kg/day) + Chronic\ Oral\ Dose\ (mg/kg/day)]\ x\ Exposure\ Frequency\ (days/yr)\ x\ Exposure\ Duration\ (yrs)}{365\ days/yr\ x\ Lifetime\ (yrs)} \right)$$

Exposure frequency was assumed to be 365 day/yr.[23] For the purposes of determining an exposure duration (ED), adults were assumed to reside in the same location for 30 years (95th percentile for population mobility).[14] Note that for children (average age 3), an ED of 3 years was used. People in the United States have a life span of 75 years.[12] Note that the exposure frequency used in the assessment conservatively assumes that the homeowner resides in the same location all year round.

Risk was calculated by multiplying the lifetime average daily dose times the cancer slope factor (e.g., 0.12 mg/kg/day) $^{-1}$ using the following formula:

$$Risk = LADD \left( \frac{mg\ ai}{kg/day} \right)\ x\ Cancer\ Slope\ Factor \left( \frac{1}{(mg/kg/day)} \right)$$

## Residential Post-application Risk Assessment and Characterization

### a. Post-application Non-Cancer Risks from Dermal Exposure to Pentachlorophenol

Acute/subchronic and chronic toxicity endpoints related to dermal exposures to pentachlorophenol have been identified. A dermal MOE greater than 100 for pentachlorophenol is considered to indicate no risk concern for adults and children for short-term and intermediate-

term exposures. A dermal MOE of greater than 300 for pentachlorophenol is considered to indicate no risk concern for chronic exposures for adults and for children . The results presented in Table 9 are summarized in the following bulleted categories.

***Residential Post-application Scenarios with Non-Cancer Risk Concerns (Short- and Intermediate-Term Risk)***

The calculations of short-term and intermediate-term risks indicate that dermal MOEs are more than 100 for adults and children at **baseline** for the following scenarios:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (child);
- (2) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (adult);
- (3) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (child).

***Residential Post-application Scenarios with Non-Cancer Risk Concerns (Chronic Risk)***

The calculations of chronic risks indicate that dermal MOEs are more than 300 for adults and children at baseline for the following scenarios:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (child)- **based on minimum soil concentrations**;
- (2) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (adult);
- (3) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (child).

The calculations of chronic risks indicate that dermal MOEs are less than 300 for adults and for children at baseline for the following scenarios:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (child)- **based on maximum soil concentrations.**

There were no data gaps identified for this scenario.

**b. Post-application Non-Cancer Risks from Oral Exposures to Pentachlorophenol**

Acute/subchronic, and chronic toxicity endpoints related to oral exposures to pentachlorophenol have been identified. Oral exposures were only calculated for children. Adult oral exposure was considered negligible. An oral MOE greater than 100 for pentachlorophenol is considered to indicate no risk concern for children for short-term and intermediate-term exposures. A target MOE of 300 was used for chronic concerns for children. The results of Table 9 are summarized in the following bulleted categories.

***Residential Post-application Scenarios with Non-Cancer Risk Concerns (Short- and Intermediate-Term Risk)***

The calculations of short-term and intermediate-term risks indicate that oral MOEs are more than 100 for children at **baseline** for the following scenarios:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (child);
- (3) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (child).

No data gaps were identified for these scenarios.

***Residential Post-application Scenarios with Non-Cancer Risk Concerns (Chronic-Risk)***

The calculations of chronic risks indicate that oral MOEs are more than 300 for children at **baseline** for the following scenarios:

- (1) Contact With Soil Contaminated with Pentachlorophenol From a Utility Pole (child);
- (3) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (child).

No data gaps were identified for these scenarios.

**c. Post-application Non-Cancer Risks from Inhalation Exposures to Pentachlorophenol**

No data were available to calculate risks for this scenario.

**(d) Post-application Cancer Risks from Dermal & Oral Exposures to Pentachlorophenol**

***Residential Post-application Scenarios with Cancer Risk Concerns***

Carcinogenic endpoints related to dermal and inhalation exposures to pentachlorophenol have been identified. A risk at the $10^{-6}$ to $10^{-5}$ range indicates a limited risk concern. The results of Table 8 are summarized in the following bulleted categories.

The following scenario indicates a cancer risk above $10^{-5}$:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (maximum concentrations for children).

The following scenarios are within or below the $10^{-5}$ to $10^{-6}$ range:

- (1) Contact with Soil Contaminated with Pentachlorophenol From a Utility Pole (minimum concentrations for children);
- (3) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (child).

The following scenario indicates a cancer risk less than the $10^{-6}$ range:

- (2) Outdoor Residential Contact with Industry Pressure-Treated Wood Products (adult).

### iv. Uncertainties and Limitations

#### a. Occupational Exposure Scenarios

There are several uncertainties and limitations associated with the data and assumptions used in this RED that must be taken into account. For example, the exposure scenarios were developed using information available to EPA (e.g. product labeling, and LUIS reports) and use information from industry contacts. The calculations were done using existing study data from pentachlorophenol use sites and/or surrogate PHED data. Much of the existing study data on air concentrations at pentachlorophenol pressure treatment sites were collected during the early 1980s (e.g., NIOSH, 1981) and may or may not reflect current practices. Also, some of these studies were reviewed previously by EPA and were found to be unacceptable on the basis of QA/QC deficiencies. Further, the dermal data used for some of the scenarios (e.g., joinery mill workers, and pressure treatment facility yardmen) were based on Fenske et al. (1987), which was also used in California's risk assessment for PCP.[7] These data are 10 years old and were generated for a timber mill, which may not be entirely appropriate for use in these scenarios. However, it is the only dermal study that is available. In addition, several assumptions (e.g., with regard to exposure time and amount treated) are based on limited information or entirely on EPA's best technical judgement. A specific concern is that the pentachlorophenol studies submitted to the Agency, and used in the RED, failed to meet the Agency's requirements outlined in Series 875-Occupational and Residential Test Guidelines Group B, Post-application Exposure Monitoring Test Guidelines. Specific examples of areas in which the uncertainties exist are outlined below, according to the exposure pathways of concern.

***Inhalation Studies Failed Series 875 Compliance***

- Improper quality assurance/quality control (QA/QC), such as lack of field or laboratory recovery, fortifications, sample storage information, controls, and description of sampling methods.
- Lack of adequate numbers of replicates (e.g., each sampling period should use at least 10 workers).
- Lack of limit of quantification (LOQs) data.
- Only one site is represented in the study and typical work conditions are not represented.
- High degree of variability in the data.

- Lack of method validation and concurrent laboratory recoveries to support the results.

- Exposure studies were conducted in the early 1980s and may not reflect current practices (e.g., some of the studies are based on sapstain control, which may be a diminished use for pentachlorophenol products).

***Dermal Studies Failed Series 875 Compliance***

- The authors of one of the dermal studies used to measure both handler and post-application exposures (Fenske et al., 1987), recommended that the data in the study should be considered as a preliminary step towards an accurate assessment of dermal exposure to chlorophenols in timber mills. The author (Fenske et al., 1987) also notes that the sample size was small, the study was limited to one mill, and the fluorescent technique had only been recently developed at the time of the study.

- A typical end-use product was not represented in the Fenske et al. (1987) study. The end-use product examined was a mixture of pentachlorophenol (PCP) (3 %) and 2,3,4,6-tetrachorophenol (TCP). Typical registered pentachlorophenol products contain 5 percent pentachlorophenol. The Agency has also indicated that ready-to-use (RTU) solutions of 10 percent ai are also currently registered.

- The Fenske et al. (1987) study was developed for chlorophenols, not pentachlorophenol. The study was also done in 1987. Typical worker-related activities in joinery mills may be different in 1998. Industry information indicates that pentachlorophenol use in joinery mills and for sapstain control is a diminished use.[6]

- Proper QA/QC information was lacking (lack of fortifications, high deviations in reported study, insufficient field recovery information, lack of field blanks and control populations, lack of storage stability data, and lack of proper laboratory recovery information).

- An insufficient number of replicates and data from only one lumber mill test site was used for the Fenske et al. (1987) study.

***Toxicity Information***

- Inhalation toxicity endpoint information was not reported. Therefore, inhalation MOEs could not be calculated in this assessment.

***Occupational Handler Data***

- Handler scenarios, such as applying pentachlorophenol by paint brush, airblast, and low pressure handwand, may not be typical. However, these uses are still listed on the pentachlorophenol labels and in the Label Use Information System database (LUIS) reports.

- Granular data from the Pesticide Handlers Exposure Database (PHED) were used as a surrogate for analyzing exposure to crystalline block and flake formulations for the handler scenario.

***DERMAL Model***

- Because no dermal data were identified in the available literature for a pressure treatment facility QA/QC inspector, the DERMAL model was used to provide an estimate of dermal exposure. The DERMAL model is frequently used by the U.S. EPA's Office of Pollution Prevention and Toxics (OPPT) for estimates of exposure to chemical substances during the use of consumer products. It is unclear whether the film thickness approach used by this model provides an accurate representation of exposures for QA/QC inspectors.

**b. Residential Exposure Scenarios**

***Inhalation Studies Failed Series 875 Compliance***

- Only one source of residential outdoor inhalation exposure data was identified in the available literature. This study could not be used as an estimate of outdoor residential exposure because it was not specific to any particular use scenario. Instead, it provided data on ambient levels of pentachlorophenol in the atmosphere to which the general population may be exposed.

***Dermal Studies Failed Series 875 Compliance***

- Soil samples were collected approximately 12 inches from the base of 30 pentachlorophenol-treated utility poles. The soil samples were analyzed and pentachlorophenol concentrations ranged from 3.4 to 654 ppm. These data were used to estimate residential exposures. The data were highly variable and QA/QC methods (percent recovery, storage of samples, storage stability, and field and method blanks) were not reported. The exposure assessment assumed that children would play in the confined area around the utility pole and ingest and dermally absorb pentachlorophenol from soil around the base of the pole. All of the soil that the child ingested or contacted dermally would be assumed to originate from the confined space around the utility pole. It is uncertain whether this accurately represents children's exposures.

- Because of the lack of specific data on direct dermal contact with pentachlorophenol-treated wood products (e.g., utility poles, fencing, porches, shingles, steps, and decks), residues found on wipe samples from pentachlorophenol pressurized logs used to build log homes were used to assess residential dermal exposures. It is uncertain whether this accurately represents these exposures. It is also unknown whether the amount of pentachlorophenol applied to the logs was the same as used to treat the other wood products being assessed in this report. Also, the wipe sampling was conducted on

indoor interior surfaces. It is unknown whether these results are representative of pentachlorophenol levels on the outdoor exterior surfaces of buildings.

**c. Incident Reports**

To be added as an addendum once the epidemiology report is completed by the Health Effects Division and/or the Antimicrobials Division's contractor.

## 5. Aggregate Exposure and Risk Assessment / Characterization

### a. Acute Aggregate Exposure and Risk

Under current OPP policy, acute dietary aggregate risk assessment determines the acute risk from combined dietary consumption of pesticide residues, separate from residential exposures. The Antimicrobials Division concludes with reasonable certainty that use of pentachlorophenol does not result in estimates of acute aggregate human health risk that exceed the Agency's level of concern. Acute estimates associated with exposure to pentachlorophenol in food and water do not exceed AD's level of concern. Percentages of the acute RfD occupied from food sources at the 95$^{th}$ percentile were less than 1% of the acute RfD for non-nursing infants, the population subgroup with the greatest acute exposure concern. DWLOCs calculated for surface water for pentachlorophenol were 10,465 ppb for adult males and females and 2990 ppb for children ages 1-6. Using the PRZM3-EXAMS model, available environmental fate data, and conservative assumptions, the estimated environmental concentrations calculated by AD for surface water were less than 1 ppb. DWLOCs for groundwater were not calculated since the estimated environmental concentrations calculated by AD for groundwater using SCI-GROW model were less than 1 ppb.

### b. Short and Intermediate Term Aggregate Exposure and Risk

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 uses. In both cases, high end estimates of residential exposure are used for the aggregate assessment, while for food and water exposures, average values are used in the aggregate assessment.

AD concludes with reasonable certainty that use of pentachlorophenol does not result in estimates of short and intermediate term human health risk that exceed the Agency's level of concern. Short-term risk estimates associated with exposure ot pentachlorophenol in food, water, and form residential use (including dermal, oral, and inhalation contact) do not exceed AD's level of concern. In calculating these estimates, extrapolation from the oral endpoint to dermal was made using the estimate of dermal absorption (40%) as provided from the hazard assessment, while an assumption of 100% absorption by inhalation was made in extrapolating from oral to inhalation exposure, based on the lack of acceptable inhalation hazard data. Aggregate Margins of Exposure (MOEs) for both short- and intermediate-term exposures did not exceed AD's level of concern.

### c. Chronic Aggregate Exposure and Risk

AD concludes with reasonable certainty that use of pentachlorophenol results in estimates of aggregate chronic risk that exceed the Agency's level of concern for children. Chronic risk estimates associated with exposure to pentachlorophenol in food and water do not excced AD's

level of concern. However, estimates of chronic aggregate risk through residential exposure, when aggregated with food and water exposures, exceed AD's level of concern. The estimated aggregate Margin of Exposure for chronic aggregate risk (30) exceeds AD's level of concern (300). Therefore, residential uses which result in chronic exposure to pentachlorophenol may be unacceptable under the current use pattern. To obtain an acceptable level of risk, this use may need to be removed.

## 6. Other Food Quality Protection Act (FQPA) Considerations

### a. Cumulative Risk from Exposure to Substances with a Common Mechanism of Toxicity

Section 408 of the FQPA requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency must consider "available information" concerning the cumulative effects of a particular pesticide's residues and "other substances that have a common mechanism of toxicity." While the Agency has some informtation in its files that may be helpful in determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodology to resolve the scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will enable it to develop and apply policies for evaluating the cumulative effects of chemicals having a common mechanism of toxicity. At present, however, the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments. There are pesticides to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of toxicity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

EPA does not have, at this time, available data to determine whether pentachlorophenol has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. For the purposes of this document, EPA has not assumed that pentachlorophenol has a common mechanism of toxicity with other substances.

### b. Endocrine Disruption

EPA is required to develop a screening program to determine whether certain substances (including al pesticides and inerts) "may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect..." The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. A final report of the Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) has been issued for public

comment.

# VI. SCIENCE ASSESSMENT - ECOLOGICAL EFFECTS

## A. Ecological Effects

### 1. Ecological Toxicity Data

#### a. Toxicity to Terrestrial Animals

##### i. Birds, Acute and Subacute

An acute oral toxicity study using the technical grade of the active ingredient (TGAI) is required to establish the toxicity of pentachlorophenol to birds. The preferred test species is either mallard duck (a waterfowl) or bobwhite quail (an upland gamebird). Results of this test are tabulated below.

**TABLE VI -1: Avian Acute Oral Toxicity**

| Species | % ai | LD50 (mg/kg) | Toxicity Category | MRID No. Author/Year | Study Classification[1] |
|---|---|---|---|---|---|
| Northern bobwhite quail *(Colinus virginianus)* | 88.9 | 627 | slightly toxic | 426337-01; Campbell S.M.& M. Jaber, 1993 | Core |
| Mallard duck *(Anas platyrhynchos)* | 99.6 | 380 | moderately toxic | 00160000; Hudson et. al., 1984 | Supplemental |
| Ring-necked pheasant *(Phasianus colchicus)* | 99.6 | 504 | moderately toxic | 00160000; Hudson et. al., 1984 | Supplemental |

[1] Core (study satisfies guideline). Supplemental (study is scientifically sound, but does not satisfy guideline)

Since the LD50 falls in the range of 380-627 mg/kg, pentachlorophenol is categorized as moderately to slightly toxic to avian species on an acute oral basis. The guideline (71-1 /OPPTS 850.2100) is fulfilled (MRID 426337-01; 00160000).

Avian subacute dietary studies are required if the avian acute oral LD50 of the TGAI is less than or equal to 100mg ai/kg and residues of active ingredient and /or its principal degradation products are likely to occur in avian feed items. Pentachlorophenol does not fulfill these conditions. However, data are available from a number of avian dietary tests with pentachlorophenol. Results of these tests are tabulated below.

**TABLE VI-2: Avian Subacute Dietary Toxicity**

| Species | % ai | 5-Day LC50 (ppm) | Toxicity Category | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|
| Northern bobwhite quail *(Colinus virginianus)* | 88.9 | 5581 | practically non-toxic | 426337-02; Campbell, S.M. & M. Jaber, 1993 | Core |
| Northern bobwhite quail *(Colinus virginianus)* | 40 | 3400 | practically non-toxic | 22923; Hill et. al., 1973 | Supplemental |
| Mallard duck *(Anas platyrhynchos)* | 88.9 | 3763 | practically non-toxic | 426337-03; Campbell, S.M. & M. Jaber, 1993 | Core |
| Mallard duck *(Anas platyrhynchos)* | 40 | 4500 | practically non-toxic | 22923; Hill et. al., 1973 | Supplemental |
| Japanese quail *(Coturnix japonica)* | 40 | 5204 | practically non-toxic | 22923; Hill et.al., 1973 | Supplemental |
| Ring-necked pheasant *(Phasianus colchicus)* | 40 | 4331 | practically non-toxic | 22923; Hill et. al., 1973 | Supplemental |

Since the LC50 falls in the range of 3400-5581 ppm, pentachlorophenol is categorized as practically non-toxic to avian species on a subacute dietary basis.

The guideline (71-2/OPPTS 850.2200) is fulfilled (MRID 426337-02, 426337-03; 22923).

### ii. Birds, Chronic

Avian reproduction studies using the TGAI may be required for pentachlorophenol if the following criteria are met: (1) birds may be subject to repeated or continuous exposure to the pesticide or any of its metabolites or degradation products, especially preceding or during the breeding season; (2) the pesticide or any of its major metabolites or degradation products are stable in the environment to the extent that potentially toxic amounts may persist in avian feed items; (3) the pesticide or any of its major metabolites or degradation products is stored or accumulated in plant or animal tissues, as indicated by its octanol/water partition coefficient, accumulation studies, metabolic release and retention studies or as indicated by structural similarity to known bioaccumulative chemicals; (4) any other information, such as that derived from mammalian reproduction studies that indicates the reproduction in terrestrial vertebrates

may be adversely affected by the anticipated use of the pesticide product. The preferred test species are mallard duck and bobwhite quail.

The guideline (71-4/OPPTS 850.???) is not fulfilled. No studies were evaluated under this topic.

### iii. Mammals, Acute and Chronic

Wild mammal testing is required on a case-by-case basis, depending on the results of lower tier laboratory mammalian studies, intended use pattern and pertinent environmental fate characteristics. In most cases, rat or mouse toxicity values obtained from the Agency's Health Effects Division (HED) substitute for wild mammal testing. These toxicity values are reported below.

**TABLE VI-3: Mammalian Toxicity**

| Species | % ai | Test Type | Toxicity Value | MRID No. |
|---|---|---|---|---|
| laboratory rat *(Rattus norvegicus)* | 90.4 | Acute Oral LD50 | 155 mg/kg (M)<br>137 mg/kg (F) | 00101715 |

An analysis of the results indicate that pentachlorophenol is categorized as moderately toxic to small mammals on an acute oral basis.

### b. Toxicity to Freshwater Aquatic Animals

#### i. Freshwater Fish, Acute

Two freshwater fish toxicity studies using the TGAI are required to establish the toxicity of pentachlorophenol to fish. The preferred test species are rainbow trout (a coldwater fish) and bluegill sunfish (a warmwater fish). Results of these tests are tabulated below.

**TABLE VI-4: Freshwater Fish Acute Toxicity**

| Species | % ai | 96-hour LC50 (ppm) | Toxicity Category | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|
| Rainbow trout *(Oncorhynchus mykiss)* | 100 | 0.075 | very highly toxic | GUOPEN-02; Bionomics Labs | Core |
| Rainbow trout *(Oncorhynchus mykiss)* | 100 | 0.015 | very highly toxic | 434851-04; Scow, 1980 | Core |
| Rainbow trout *(Oncorhynchus mykiss)* | 88 | 0.086 | very highly toxic | 63572; McCarty, 1977 | Core |
| Rainbow trout *(Oncorhynchus mykiss)* | 15.7 Nalco 201 | 0.370 | very highly toxic | 90429; McCann, 1971 | Core |
| Bluegill sunfish *(Lepomis macrochirus)* | 100 | 0.060 | very highly toxic | GUOPEN-02; Bionomics Labs | Core |
| Bluegill sunfish *(Lepomis macrochirus)* | 15.7 Nalco 201 | 0.345 | very highly toxic | 33067; Mattson, 1976 | Core |
| Fathead minnow *(Pimephales promelas)* | 100 | 0.600 | highly toxic | 33067; Mattson, 1976 | Core |

Since the LC50 falls in the range of 0.015 to 0.600 ppm, pentachlorophenol is categorized as very highly toxic to freshwater fish on an acute basis.

The guideline (72-1/OPPTS 850.1075) is fulfilled (MRID 434851-04; 33067; 63572; GUOPEN02).

#### ii. Freshwater Fish, Chronic

A freshwater fish early life-stage test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and when any of the following conditions apply: (1) if any LC50 or EC50 value determined in acute toxicity testing is less than 1 mg/L, (2) if the estimated environmental concentration in water is greater than or equal to (≥) 0.01 of any EC50 or LC50 determined in acute toxicity testing, (3) if the actual or estimated environmental concentration in water is less than 0.01 of any EC50 or LC50 determined in acute toxicity testing and any of the following conditions exist: [(a)studies of other organisms indicate the reproductive physiology of fish/invertebrates may be affected, (b)physicochemical properties indicate cumulative effects may occur and/or, (c) the pesticide is persistent in water]. The preferred test species is rainbow trout. Results of this test are tabulated below.

**TABLE VI-5: Freshwater Fish Early Life-Stage Toxicity Under Flow-through Conditions**

| Species | % ai | NOEC LOEC (ppm) | MATC[1] (ppm) | Endpoints Affected | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|---|
| Rainbow trout *(Oncorhynchus mykiss)* | 99 | NOEC 11 LOEC 19 | 0.014 | increase in mortality; decrease in length and weight | GUOPEN03; Stephan, 1986 | Core |
| Fathead minnow *(Pimephales promelas)* | 99 | Not Reported | 0.055 | Not Reported | GUOPEN03; Stephan, 1986 | Core |
| Fathead minnow *(Pimephales promelas)* | 88 | Not Reported | 0.024 | Not Reported | GUOPEN03; Stephan, 1986 | Core |

[1] defined as the geometric mean of the NOEC and LOEC.

The guideline (72-4/OPPTS 850.1300) is fulfilled (MRID GUOPEN03).

A freshwater fish life-cycle test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and/or (1) if the aquatic EEC is greater than or equal to (≥) 0.1 of the no-observed-effect concentration/level (NOEC/NOEL) in the fish early life stage test; or (2) if studies for other organisms indicate that the reproductive physiology of fish may be affected. The preferred test species is fathead minnow.

The guideline (72-5/OPPTS 850.1500) is not fulfilled. No studies were evaluated under this topic.

### iii. Freshwater Invertebrates, Acute

A freshwater aquatic invertebrate toxicity test using the TGAI is required to establish the toxicity of pentachlorophenol to aquatic invertebrates. The preferred test species is *Daphnia magna*. Results of this test are tabulated below.

**TABLE VI-6: Freshwater Invertebrate Acute Toxicity**

| Species | % ai | 48-hour EC50 (ppm) | Toxicity Category | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|
| Waterflea *(Daphnia magna)* | 88 | 0.450 | highly toxic | 63572; MaCarty, 1977 | Core |

With an EC50 of 0.450 ppm, pentachlorophenol is categorized as highly toxic to aquatic invertebrates on an acute basis. The guideline (72-2/OPPTS 850.1010) is fulfilled (Reference number 63572).

### iv. Whole Sediment Acute Invertebrate, Freshwater

Whole sediment acute invertebrate, freshwater studies (1) may be required when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited; (2) may be required on a case-by-case basis depending on the results of lower tier ecological studies (e.g., active ingredient or end-use products are highly toxic to aquatic organisms) and/or pertinent environmental characteristics (e.g., Kow is greater than or equal to ($\geq$) 1,000 or hydrolysis half-life is greater than ($>$) 5 days); and (3) required for organic-based compounds with a Koc (organic carbon coefficient) greater than ($>$) 1,000 and solubility is less than, of equal to, ($\leq$) 0.1 mg/l.

The guideline (73-1/OPPTS 850.1735) is not fulfilled. No studies were evaluated under this topic.

### v. Freshwater Invertebrate, Chronic

A freshwater invertebrate life-cycle test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and when any of the following conditions apply: (1) if any LC50 or EC50 value determined in acute toxicity testing is less than 1 mg/L; (2) if the estimated environmental concentration in water is greater than or equal to ($\geq$) 0.01 of any EC50 or LC50 determined in acute toxicity testing; (3) if the actual or estimated environmental concentration in water is less than 0.01 of any EC50 or LC50 determined in acute toxicity testing and any of the following conditions exist: [(a)studies of other organisms indicate the reproductive physiology of

fish/invertebrates may be affected; (b)physicochemical properties indicate cumulative effects may occur and/or; (c)the pesticide is persistent in water]. The preferred test species is *Daphnia magna*. Results of this test are tabulated below.

**TABLE VI-7: Freshwater Aquatic Invertebrate Life-Cycle Toxicity**

| Species | % ai | 21-day NOEC or LOEC (ppm) | MATC[1] (ppm) | Endpoints Affected | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|---|
| Waterflea *(Daphnia magna)* | 100 | Not Reported | 0.240 | mortality | GUOPEN03; Stephan, 1986 | Core |

[1] defined as the geometric mean of the NOEC and LOEC.

The guideline (72-4/OPPTS 850.1300) is fulfilled (MRID GUOPEN03).

### vi. Acute Pore Water, Fish and Invertebrates

Acute pore water, fish and invertebrates study may be required when (1) treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited; (2) may be required on a case-by-case basis depending on the results of lower tier ecological studies (e.g., active ingredient or end-use products are highly toxic to aquatic organisms) and/or pertinent environmental characteristics (e.g., Kow is greater than or equal to ($\geq$) 1,000 or hydrolysis half-life is greater than ($>$) 5 days); and (3) required for organic-based compounds with a Koc (organic carbon coefficient) greater than ($>$) 1,000 and solubility is less than, or equal to, ($\leq$) 0.1 mg/l.

The guideline (73-3) is not fulfilled. No studies were evaluated under this topic.

### vii. Freshwater Field Studies

Simulated or actual field testing may be required on a case-by-case basis depending on the results of lower tier ecological studies (e.g., active ingredient or end-use products are highly toxic to aquatic organisms) and/or pertinent environmental characteristics (e.g., Kow is greater than or equal to ($\geq$) 1,000 or hydrolysis half-life is greater than ($>$) 5 days). Field testing for aquatic organisms is required when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited. The guideline (72-7) is not fulfilled. No studies were evaluated under this topic.

### c. Toxicity to Estuarine and Marine Animals

#### i. Estuarine and Marine Fish, Acute

Acute toxicity testing with estuarine/marine fish using the TGAI is required for pentachlorophenol because the end-use product is intended for direct application to the marine/estuarine environment or the active ingredient is expected to reach this environment because of its use in coastal counties. The preferred test species is sheepshead minnow. Results of these tests are tabulated below.

**TABLE VI-8: Estuarine/Marine Fish Acute Toxicity**

| Species | % ai | 96-hour LC50 (ppm) | Toxicity Category | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|
| Sheepshead minnow *(Cyprinodon variegatus)* | 100 | 0.240 | highly toxic | 434851-04; Scow, 1980 | Core |

With an LC50 of 0.240 ppm, pentachlorophenol is categorized as highly toxic to estuarine/marine fish on an acute basis. The guideline (72-3a/OPPTS 850.1025) is fulfilled (MRID 434851-04).

#### ii. Estuarine and Marine Fish, Chronic

An estuarine/marine fish early life-stage toxicity test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and when any of the following conditions apply: (1) if any LC50 or EC50 value determined in acute toxicity testing is less than 1 mg/L, (2) if the estimated environmental concentration in water is greater than or equal ($\geq$) to 0.01 of any LC50 or EC50 value determined in acute toxicity testing, (3) if the actual or estimated environmental concentration in water is less than 0.01 of any acute LC50 or EC50 value determined in acute toxicity testing and any of the following conditions exist:

(A) studies of other organisms indicate the reproductive physiology of fish/ invertebrates may be affected

(B) physicochemical properties indicate cumulative effects may occur.

(C) the pesticide is persistent in water.

The preferred test species is sheepshead minnow. Results of this test are tabulated below.

**TABLE VI-9: Estuarine/Marine Fish Early Life-Stage Toxicity Under Flow-through Conditions**

| Species | % ai | NOEC or LOEC(ppm) | MATC[1] (ppm) | Endpoints Affected | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|---|
| Sheepshead Minnow *(Cyprinodon variegatus)* | 100 | Not reported | 0.064 | decreased long-term survival of the first generation; reduced survival of second generation embryos and juveniles | GUOPEN-03; Stephan, 1986 | Core |

[1] defined as the geometric mean of the NOEC and LOEC.

The guideline (72-4/OPPTS 850.1300) is fulfilled (MRID GUOPEN-03).

An estuarine/marine fish life-cycle test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and/or when the following conditions exist: (1) if the aquatic EEC is greater than or equal to ($\geq$) 0.1 of the no-observed-effect concentration/level (NOEC/NOEL) in the fish early life stage test, (2) if studies of other organisms indicate that the reproductive physiology of fish may be affected. The preferred test species is sheepshead minnow. The guideline (72-5/OPPTS 850.1500) is not fulfilled. No studies were evaluated under this topic.

### iii. Estuarine and Marine Invertebrates, Acute

Acute toxicity testing with estuarine/marine invertebrates using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited. The preferred test species are mysid shrimp and eastern oyster. Results of these tests are tabulated below.

**TABLE VI-10: Estuarine/Marine Invertebrate Acute Toxicity**

| Species | % ai | 96-hour LC50 or EC50 (ppm) | Toxicity Category | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|---|
| Pacific oyster (shell deposition) *(Crassostrea gigas)* | 100 | EC50 0.048 | very highly toxic | 434851-04; Scow, 1980 | Core |
| Shrimp *(Palaemon elegans)* | 100 | LC50 0.084 | very highly toxic | 434851-04; Scow, 1980 | Core |

Since the LC50/EC50 falls in the range of 0.048 ppm for the Pacific oyster (shell deposition and 0.084 ppm for shrimp, pentachlorophenol is categorized as very highly toxic to estuarine/marine invertebrates on an acute basis.

The guidelines (72-3b and 72-3c/OPPTS 850.1035 and 850.1045) are fulfilled (MRID 434851-04).

### iv. Whole Sediment Acute Invertebrate, Marine

Whole sediment acute invertebrate, marine studies are required only for uses in estuarine/marine environments and may be required when (1) treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited; (2) may be required on a case-by-case basis depending on the results of lower tier ecological studies (e.g., active ingredient or end-use products are highly toxic to aquatic organisms) and/or pertinent environmental characteristics (e.g., Kow is greater than or equal to ($\geq$) 1,000 or hydrolysis half-life is greater than ($>$) 5 days); and (3) required for organic-based compounds with a Koc (organic carbon coefficient) greater than ($>$) 1,000 and solubility is less than, or equal to, ($\leq$) 0.1 mg/l.

The guideline (73-1/OPPTS 850.1740) is not fulfilled. No studies were evaluated under this topic.

### v. Estuarine and Marine Invertebrate, Chronic

An estuarine/marine invertebrate life-cycle toxicity test using the TGAI is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited and when any of the following conditions apply: (1) if any LC50 or EC50 value determined in acute toxicity testing is less than 1 mg/L, (2) if the estimated environmental concentration in water is greater than or equal ($\geq$) to 0.01 of any LC50 or EC50 value determined in acute toxicity testing, (3) if the actual or estimated environmental concentration in water is less than 0.01 of any acute LC50 or EC50 value determined in acute toxicity testing and any of the following conditions exist:

(A) Studies of other organisms indicate the reproductive physiology of fish/ invertebrates may be affected.

(B) Physicochemical properties indicate cumulative effects may occur.

(C)The pesticide is persistent in water.

The preferred test species is Mysid shrimp. The guideline (72-4/OPPTS 850.1300) is not fulfilled. No studies were evaluated under this topic.

### vi. Whole Sediment Chronic, Invertebrates

Whole sediment chronic, invertebrate testing is required when mortality exceeds 20% in any concentration level used in acute sediment testing.

### d. Toxicity to Plants

#### i. Terrestrial Plants

Seedling emergence testing (terrestrial plant testing) is required when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited. Only one plant species, rice (*Oryza sativa*), must be tested. Vegetative vigor testing is not required for any wood preservative (123-1/OPPTS 850.4250).

The guideline (123-1/OPPTS 850.4225) is not fulfilled. No studies were evaluated under this topic.

#### ii. Aquatic Plants

Aquatic plant testing is required for pentachlorophenol when treated wood will be used in the aquatic environment or use in aquatic sites is not prohibited. The following species should be tested at Tier II: *Kirchneria subcapitata*, *Lemna gibba*, *Skeletonema costatum*, *Anabaena flos-aquae*, and a freshwater diatom. Results of these tests are tabulated below.

**TABLE VI-11: Nontarget Aquatic Plant Toxicity (Tier II)**

| Species | % ai | EC50 (ppm) | MRID No. Author/Year | Study Classification |
|---|---|---|---|---|
| **Vascular Plants** | | | | |
| Duckweed *Lemna gibba* | 88.9 | 0.250 | 426337-08; Hoberg, 1993 | Core |
| **Nonvascular Plants** | | | | |
| Green algae *Selenastrum capricornutum* | 88.9 | 0.050 | 426337-06; Hoberg, 1993 | Core |
| Marine diatom *Skeletonema costatum* | 88.9 | 0.027 | 426337-04; Hoberg, 1993 | Core |
| Freshwater diatom *Navicula pelliculosa* | 88.9 | 0.124 | 426337-05; Hoberg, 1993 | Core |
| Blue-green algae *Anabaena flos-aquae* | 88.9 | 0.050 | 426337-07; Hoberg, 1993 | Core |

The Tier II results indicate that the marine diatom, *Skeletonema costatum* is the most sensitive nonvascular aquatic plant. The guideline (123-2/840.4400 & 840.5400) is fulfilled (MRID 426337-04; 426337-05; 426337-06; 426337-07; 426337-08).

## 2. Exposure and Risk Characterization

Risk characterization integrates the results of the exposure and ecotoxicity data to evaluate the likelihood of adverse ecological effects. The means of this integration is called the quotient method. Risk quotients (RQs) are calculated by dividing exposure estimates by acute and chronic ecotoxicity values.

RQ = EXPOSURE/TOXICITY

RQs are then compared to OPP's levels of concern (LOCs). These LOCs are used by OPP to analyze potential risk to nontarget organisms and the need to consider regulatory action. The criteria indicate that a pesticide used as directed has the potential to cause adverse effects on nontarget organisms. LOCs currently address the following risk presumption categories: (1) **acute high** -- potential for acute risk is high; regulatory action may be warranted in addition to restricted use classification, (2) **acute restricted use** -- the potential for acute risk is high, but may be mitigated through restricted use classification, (3) **acute endangered species** - endangered species may be adversely affected, and (4) **chronic risk** - the potential for chronic risk is high regulatory action may be warranted. Currently, EFED does not perform assessments for chronic risk to plants, acute or chronic risks to nontarget insects, or chronic risk from granular/bait formulations to birds or mammals.

The ecotoxicity test values (measurement endpoints) used in the acute and chronic risk quotients are derived from required studies. Examples of ecotoxicity values derived from short-term laboratory studies that assess acute effects are: (1) LC50 (fish and birds), (2) LD50 (birds and mammals), (3) EC50 (aquatic plants and aquatic invertebrates) and (4) EC25 (terrestrial plants). Examples of toxicity test effect levels derived from the results of long-term laboratory studies that assess chronic effects are: (1) LOEC (birds, fish, and aquatic invertebrates), (2) NOEC (birds, fish and aquatic invertebrates), and (3) MATC (fish and aquatic invertebrates). For birds and mammals, the NOEC generally is used as the ecotoxicity test value in assessing chronic effects, although other values may be used when justified. Generally, the MATC (defined as the geometric mean of the NOEC and LOEC) is used as the ecotoxicity test value in assessing chronic effects to fish and aquatic invertebrates. However, the NOEC is used if the measurement end point is production of offspring or survival.

Risk presumptions and the corresponding RQs and LOCs, are tabulated below.

**TABLE VI-12: Risk Presumptions for Terrestrial Animals**

| Risk Presumption | RQ | LOC |
|---|---|---|
| Birds | | |
| Acute High Risk | EEC[1]/LC50 or LD50/sqft[2] or LD50/day[3] | 0.5 |

**TABLE VI-12: Risk Presumptions for Terrestrial Animals**

| Risk Presumption | RQ | LOC |
|---|---|---|
| Acute Restricted Use | EEC/LC50 or LD50/sqft or LD50/day (or LD50 < 50 mg/kg) | 0.2 |
| Acute Endangered Species | EEC/LC50 or LD50/sqft or LD50/day | 0.1 |
| Chronic Risk | EEC/NOEC | 1 |
| Wild Mammals | | |
| Acute High Risk | EEC/LC50 or LD50/sqft or LD50/day | 0.5 |
| Acute Restricted Use | EEC/LC50 or LD50/sqft or LD50/day (or LD50 < 50 mg/kg) | 0.2 |
| Acute Endangered Species | EEC/LC50 or LD50/sqft or LD50/day | 0.1 |
| Chronic Risk | EEC/NOEC | 1 |

[1] abbreviation for Estimated Environmental Concentration (ppm) on avian/mammalian food items
[2] $mg/ft^2$
[3] mg of toxicant consumed/day (LD50 * wt. of bird)

**TABLE VI-13: Risk Presumptions for Aquatic Animals**

| Risk Presumption | RQ | LOC |
|---|---|---|
| Acute High Risk | EEC[1]/LC50 or EC50 | 0.5 |
| Acute Restricted Use | EEC/LC50 or EC50 | 0.1 |
| Acute Endangered Species | EEC/LC50 or EC50 | 0.05 |
| Chronic Risk | EEC/MATC or NOEC | 1 |

[1] EEC = (ppm or ppb) in water

**TABLE: VI-14: Rish Presumptions for Plants**

| Risk Presumption | RQ | LOC |
|---|---|---|
| | Terrestrial and Semi-Aquatic Plants | |
| Acute High Risk | EEC[1]/EC25 | 1 |
| Acute Endangered Species | EEC/EC05 or NOEC | 1 |
| | Aquatic Plants | |
| Acute High Risk | EEC[2]/EC50 | 1 |
| Acute Endangered Species | EEC/EC05 or NOEC | 1 |

[1] EEC = lbs ai/A
[2] EEC = (ppb/ppm) in water

### a. Exposure and Risk to Nontarget Terrestrial Animals

Birds and mammals may be exposed to pentachlorophenol via residues leached from treated wood into the aquatic environment or into the soil near treated objects.

#### i. Acute Effects

The Agency is unable to conduct a typical risk assessment for pentachlorophenol and terrestrial organisms because information is insufficient to characterize exposure for these organisms. However, two factors indicate that the use of pentachlorophenol as a wood preservative is not likely to present a significant acute risk to birds and mammals:

- The highest aquatic PEC (predicted environmental concentration) from wood preservative use of pentachlorophenol is 0.176 ppb, while avian dietary LC50's range from 3400 to 5581 ppm. These figures do not indicate a significant potential for acute risk.

- Data from the toxicology database provide a rat LD50 of 137-155 mg/kg. Although this indicates moderate toxicity, it is not likely that wood preservative use will provide for significant potential for acute exposure via contaminated food items.

Based on the above information, acute risk to birds and mammals is not expected from the wood preservative use of pentachlorophenol.

### ii. Chronic Effects

The pentachlorophenol database is insufficient for the Agency to conduct a typical chronic risk assessment for birds and mammals. The Agency is unable to characterize long-term exposure, and there are no data from avian reproduction studies. However, the Agency determined in 1987 that avian reproduction data would not be required to support the use of pentachlorophenol as a wood preservative, due to the low potential for terrestrial exposure from this use. Thus, chronic risk to birds and mammals is not expected.

## b. Exposure and Risk to Nontarget Freshwater and Marine/Estuarine Aquatic Animals

Nontarget freshwater and marine/estuarine aquatic animals and plants may be exposed to pentachlorophenol via residues leached from treated wood into the aquatic environment or into the soil near treated objects.

### i. Freshwater Fish

Acute and chronic risk quotients are tabulated below.

**TABLE VI-15: Risk Quotients for Freshwater Fish Based On Rainbow Trout (*Oncorhynchus mykiss*) LC50 of 15 ppb and Rainbow Trout (*Oncorhynchus mykiss*) MATC of 14 ppb.**

| Site | LC50 (ppb) | MATC (ppb) | EEC Initial/Peak (ppb) | EEC 60-Day Ave. (ppb) | Acute RQ (EEC/LC50) | Chronic RQ (EEC/MATC) |
|---|---|---|---|---|---|---|
| Treated utility poles 0.004 | 15 | 14 | 0.176 | | 0.055 | 0.012 |

An analysis of the results indicates that no aquatic acute or chronic levels of concern are exceeded for freshwater fish from application to utility poles.

### ii. Freshwater Invertebrates

Acute and chronic risk quotients are tabulated below.

**TABLE VI-16: Risk Quotients for Freshwater Invertebrates Based On *Daphnia magn* EC50 of 450 ppb and *Daphnia magna* MATC of 240 ppb.**

| Site | EC50 (ppb) | MATC (ppb) | EEC Initial/Peak (ppb) | EEC 21-Day Average | Acute RQ (EEC/EC50) | Chronic RQ (EEC/MATC) |
|---|---|---|---|---|---|---|
| Treated utlity poles | 450 | 240 | 0.176 | 0.075 | 0.0004 | 0.0003 |

An analysis of the results indicates that no aquatic acute or chronic levels of concern are exceeded for freshwater invertebrates from application to utility poles.

### iii. Estuarine/Marine Fish

Acute and chronic risk quotients are tabulated below.

**TABLE VI-17: Risk Quotients for Estuarine/Marine Fish Based on Sheepshead minnow (*Cyprinodon variegatus*) LC50 of 240 ppb and Sheepshead minnow (*Cyprinodon variegatus*) MATC of 64 ppb.**

| Site | LC50 (ppb) | MATC (ppb) | EEC Initial/Peak (ppb) | EEC (60 day) (ppb) | Acute RQ (EEC/LC50) | Chronic RQ (EEC/MATC) |
|---|---|---|---|---|---|---|
| Utlity poles | 240 | 64 | 0.176 | 0.055 | 0.0007 | 0.0009 |

An analysis of the results indicates that no aquatic acute or chronic levels of concern are exceeded for estuarine/marine fish from application to utility poles.

### iv. Estuarine/Marine Invertebrates

Acute and chronic risk quotients are tabulated below.

**TABLE VI-18: Risk Quotients for Estuarine/Marine Aquatic Invertebrates Based on Pacific oyster (*Crassostrea gigas*) shell deposition EC50 of 48 ppb.**

| Site | EC50 (ppb) | MATC (ppb) | EEC Initial/Peak (ppb) | EEC 60-Day Ave. (ppb) | Acute RQ (EEC/LC50) | Chronic RQ (EEC/NOEC) |
|---|---|---|---|---|---|---|
| Utlity poles | 48 | 240 | 0.176 | 0.075 | 0.004 | 0.003[1] |

[1]Based on chronic toxicity values from freshwater study (Daphnid chronic study).

An analysis of the results indicates that no aquatic acute levels of concern are exceeded for estuarine invertebrates from application to utility poles. No chronic estuarine/marine aquatic invertebrate studies were available for PCP. However, based on data from a freshwater invertebrate chronic study, no chronic aquatic levels of concern are exceeded for estuarine invertebrates from application to utility poles.

### c. Exposure and Risk to Nontarget Plants

The use of pentachlorophenol as a wood preservative is unlikely to result in significant exposure for terrestrial plants. Thus, data from testing with terrestrial nontarget plants is not required, and risk to these organisms is expected to be minimal.

Aquatic plants may be exposed to pentachlorophenol via residues leached from treated wood into the aquatic environment.

An aquatic plant risk assessment for acute high risk is usually made for aquatic vascular plants from the surrogate duckweed *Lemna gibba*. Non-vascular acute high aquatic plant risk assessments are performed using either algae or a diatom, whichever is the most sensitive species. An aquatic plant risk assessment for acute- endangered species is usually made for aquatic vascular plants from the surrogate duckweed *Lemna gibba*. To date there are no known non-vascular plant species on the endangered species list. The risk quotient is determined by dividing the pesticide's initial or peak concentration in water by the plant EC50 value. Acute risk quotients for vascular and non-vascular plants are tabulated below.

**TABLE VI-19: Acute Risk Quotients for Aquatic Plants based upon a duckweed (*Lemna gibba*) EC50 of 250 ppb ai and a nonvascular plant (*Skeletonema costatum*) EC50 of 27 ppb ai.**

| Site | Species | EC50 (ppb) | EEC (ppb) | RQ (EEC/EC50) |
|---|---|---|---|---|
| Treated utility poles | Duckweed | 250 | 0.176 | 0.0007 |
| Treated utility poles | Algae or diatom | 27 | 0.176 | 0.007 |

An analysis of the results indicates that plant acute levels of concern are not exceeded for vascular and non-vascular aquatic plants from application to utility poles.

## 3. Endangered Species

Endangered species LOCs are not exceeded for any nontarget organisms. The Endangered Species Protection Program is expected to become final in the future. Limitations in the use of pentachlorophenol may be required to protect endangered and threatened species, but these limitations have not been defined and may be formulation specific. EPA anticipates that a consultation with the U.S. Fish and Wildlife Service will be conducted in accordance with the species-based priority approach described in the Program. After completion of consultation, registrants will be informed if any required label modifications are necessary. Such modifications would most likely consist of the generic label statement referring pesticide users to use limitations contained in county Bulletins.

## 4. Value of Information

In a 1995 decision, the Agency determined that the only remaining data requirements for pentachlorophenol, in the area of ecotoxicology, were 5 aquatic acute studies using a typical pentachlorophenol end use product. The requirement for these studies was reserved, pending receipt of additional information on the degradates of pentachlorophenol. In developing the new reregistration document, the Antimicrobial Division (AD) has reexamined the pentachlorophenol database and reviewed additional data. Also, the database has been evaluated under the revised requirements of the proposed 40 CFR Part 158, Subpart W.

Considering the above factors, following is a discussion of the deficiencies in the ecotoxicity database for pentachlorophenol, with an evaluation of the relative importance of the missing data.

Important data gaps for pentachlorophenol are in the areas of chronic testing with aquatic invertebrates and sediment acute toxicity testing. The actual requirements which have not been fulfilled are the estuarine/marine invertebrate life cycle test and the whole sediment acute tests for freshwater and estuarine/marine invertebrates.

Due to the lack of data from a life cycle study with an estuarine/marine invertebrate, the chronic assessment was conducted using data from a freshwater study. The use of freshwater values in an estuarine assessment introduces uncertainty into the chronic assessment. Data from an estuarine invertebrate life cycle study would reduce this uncertainty.

Data from sediment toxicity testing is important to this assessment because of the nature of pentachlorophenol. Fate characteristics indicate that this chemical may persist in the environment and will have a tendency to bind to sediment. Because of these factors, testing which is confined to the water column does not provide a complete picture of the potential risk to aquatic organisms. Data from sediment toxicity testing would reduce the uncertainty in the aquatic invertebrate risk assessment.

In addition to the above, it should be noted that there are a number of requirements in Part 158 that have been designated as reserved. In other words, studies may be required in addition to those discussed above. The 5 TEP studies mentioned in the first paragraph will remain reserved.

## VII. RISK REDUCTION/LABELING/PRODUCT TOXICOLOGY (BATCHING)

A. Human Risks- Mitgation Measures

To be completed after risk mitigation discussions with the registrants.

B. Environmental Risks - Mitigation Measures

To be completed after risk mitgation discussions with the registrants.

C. Product Toxicology (Batching)

See the attached memorandum dated January 13, 1999 from Ian Blackwell/Efficacy Science Support Branch/Antimicrobials Division.

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

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

January 13, 1999

MEMORANDUM:

Subject: Batching for Products for the PentaChloroPhenol RED
Barcode: D252144

To: Laura Morris, Team Leader
Team 2
RASSB
Antimicrobials Division (7510C)

From: Ian Blackwell, Biologist
Efficacy Evaluation Team
Efficacy and Science Support Branch
Antimicrobials Division (7510C)

Through: Michele Wingfield, Branch Chief
Efficacy and Science Support Branch
Antimicrobials Division (7510C)

I am attaching the batching appendix for the PentaChloroPhenol (PCP) RED. [1] I feel that this will be adequate for the PCP RED. Should you have any problems with this appendix, please let me know so that they can be rectified.

[1]This appendix was completed on 12/11/98.

**ANTIMICROBIAL DIVISION'S BATCHING OF PRODUCTS CONTAINING PENTACHLOROPHENOL AS THE ACTIVE INGREDIENT FOR MEETING ACUTE TOXICITY DATA REQUIREMENTS FOR REREGISTRATION**

In an effort to reduce the time, resources and number of animals needed to fulfill the acute toxicity data requirements for reregistration of products containing the active ingredient Pentachlorophenol, the Agency has batched products which can be considered similar in terms of acute toxicity. Factors considered in the sorting process include each product's active and inert ingredients (identity, percent composition and biological activity), product form (liquid, paste, solid, etc.), and labeling (e.g., signal word, precautionary labeling, etc.).

Using available information, batching has been accomplished by the process described in the preceding paragraph. Notwithstanding the batching process, the Agency reserves the right to require, at any time, acute toxicity data for an individual product should the need arise.

Registrants of products within a batch may choose to cooperatively generate, submit or cite a single battery of six acute toxicological studies to represent all the products within that batch. Registrants have the option of participating with all or some other registrants of products in their product's batch, to deal only their own products within a batch, or to generate all the required acute toxicological studies for each of their own products. If a registrant chooses to generate the data for a batch, he or she must use one of the products within the batch as the test material. If a registrant chooses to rely upon previously submitted acute toxicity data, he or she may do so provided that the data base is complete and valid by today's standards (see the attached acceptance criteria), the formulation tested is considered by EPA to be similar for acute toxicity, and the formulation has not been significantly altered since submission and acceptance of the acute toxicity data. Registrants may not support their product using data conducted on a product from a different batch. AD must approve any new or canceled formulations (that were presented to the Agency after the publication of the RED) before data derived from them can be used to cover other products in a batch. Regardless of whether new data is generated or existing data is referenced, registrants must clearly identify the test material by EPA Registration Number. If more than one confidential statement of formula (CSF) exists for a product, the registrant must indicate the formulation actually tested by identifying the corresponding CSF.

In deciding how to meet the product specific data requirements, registrants must follow the directions given in the Data Call-In Notice and its attachments appended to the RED. The DCI Notice contains two response forms which are to be completed and submitted to the Agency within 90 days of receipt. The first form, "Data Call-In Response," asks whether the registrant will meet the data requirements for each product. The second form, "Requirements Status and Registrant's Response," lists the product specific data required for each product, including the standard six acute toxicity tests. A registrant who wishes to participate in a batch must decide whether he or she will provide the data or depend on someone else to do so. If a registrant supplies the data to support a batch of products, he or she must select one of the following options: Developing Data (Option 1), Submitting an Existing Study (Option 4), Upgrading an Existing Study (Option 5) or Citing an Existing Study (Option 6). If a registrant depends on another's data, he or she must choose among: Cost Sharing (Option 2), Offers to Cost Share (Option 3) or Citing an Existing Study (Option 6). If a registrant

does not want to participate in a batch, the choices are Options 1, 4, 5 or 6. However, a registrant should know that choosing not to participate in a batch does not preclude other registrants in the batch from citing his or her studies and offering to cost share (Option 3) those studies.

Table 1 displays the batches for the active ingredient Pentachlorophenol

Table 1.

| Batch | Registration Number | Percent Active Ingredient | Form |
|---|---|---|---|
| 1 | 1022-438 | pentachlorophenol ... 86%<br>other chlorinated phenols ... 10% | solid |
| | 5382-16 | pentachlorophenol ... 86%<br>other chlorinated phenols ... 10% | solid |
| | 61483-3 | pentachlorophenol ... 86%<br>other chorinated phenols ... 10% | solid |
| | | | |
| 2 | 1022-120 | pentachlorophenol ... 35.3% | liquid |
| | 7234-57 | pentachlorophenol ... 43.0% | liquid |
| | | | |
| 3 | 1022-46 | pentachlorophenol ... 10.21% | liquid |
| | 1022-240 | pentachlorophenol ... 9.14%<br>other chlorinated phenols ... 1.06% | liquid |
| | 1022-446 | pentachlorophenol ... 7.00% | liquid |
| | | | |
| 4 | 7234-7 | pentachlorophenol ... 4.48%<br>other chlorophenols and related compounds ... 0.52% | liquid |
| | 7234-60 | pentachlorophenol ... 4.48%<br>other chlorophenols and related compounds ... 0.52% | liquid |
| | 7234-61 | pentachlorophenol ... 6.27%<br>other chlorophenols and related compounds ... 0.73% | liquid |

There are products in the "No Batch" Group that may be supported by citing data conducted on another product or other products; however, those other products will not be allowed to cited data derived from the first product. The following is a list of such situations:

1. Reg. nos. 1022-537 and 1022-16 may be supported by citing data conducted on batch #3 products; however, batch #3 products may not cite data conducted on reg. nos. 1022-537 or 1022-16. Reg. no. 1022-16 may cite data conducted on 1022-537, but 1022-537 may not cite data conducted on 1022-16.
2. Reg. no. 7234-16 may cite data conducted on 7234-11. Reg. no. 7234-11 may not cite data derived from 7234-16.

Table 2 lists the products in the "No Batch" group. These products can not be batched because they were not considered to be similar to other the products in terms of acute toxicity or because there was insufficient information available to assist in making the decision.

Table 2.

| Registration Number | Percent Active Ingredient | | Product Type |
|---|---|---|---|
| 1022-15 | pentachlorophenol | ... 41.28% | liquid |
| 1022-68 | pentachlorophenol | ... 24.29% | liquid |
| 1022-356 | pentachlorophenol | ... 4.47% | liquid |
| 1022-408 | pentachlorophenol<br>other chlorinated phenols<br>creosote | ... 12.90%<br>... 1.50%<br>... 15.50% | liquid |
| 1022-469 | pentachlorophenol<br>other chlorinated phenols | ... 8.60%<br>... 1.00% | liquid |
| 3008-62 | pentachlorophenol<br>other chlorinated phenols | ... 35.86%<br>... 4.17% | liquid |
| 5382-36 | pentachlorophenol<br>other chlorophenol and related compounds | ... 35.30%<br>... 4.10% | liquid |
| 61483-1 | pentachlorophenol | ... 4.30% | liquid |
| 61483-2 | pentachlorophenol | ... 34.0% | liquid |

If a registrant would like to have the batching status of a product reconsidered, they need to have submit detailed information on their product. An MSDS for each "inert" ingredient should be included where possible.

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b) Mr. Gene Meyer, Vulcan Chemicals, Phone (205) 877-3543
c) Mr. Martin Rollins, H.M. Rollins Co., Inc. Phone (601) 832-1738
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## APPENDIX : Product Chemistry Data Requirements for Reregistration of Pentachlorophenol

Group A: Series 830-Product Identity, Composition and Analysis Test Guidelines ( 40CFR §158.155, §158.160, § 158.162, §158.170, § 158.178, § 158.180) And
Group B: Series 830- Physical & Chemical Properties ( 40PCFR §158.190)

PRODUCT CHEMISTRY DATA SUMMARY

| Guideline Number | Requirement | Are Data Requirements Fulfilled | MRID Number |
|---|---|---|---|
| 830.1550 | Product Identity & Disclosure of Ingredients | Y | 41002701/ 40999401 |
| 830.1600 | Starting Materials & Manufacturing Process | Y | 40999401/ 41002701 |
| 830.1620 | Description of Production Process | Y | 41002701 |
| 830.1650 | Description of Formulation Process | N/A | |
| 830.1670 | Discussion of Formation of Impurities | Y | 41002702 |
| 830.1700 | Preliminary Analysis | Y | 40999402/ 41002702 |
| 830.1750 | Certified Limits | Y | 40999402 |
| 830.1800 | Enforcement Analysis Method | N/A | |
| 830.1900 | Submittal of Samples | N/A | |
| 830.6302 | Color | Y | 40999403 |
| 830.6303 | Physical State | Y | 40999403 |
| 830.6304 | Odor | Y | 40999403 |
| 830.6313 | Stability to Sunlight, Normal & Elevated Temp./ Metal/Metal ions | Y | 41002703 |
| 830.6314 | Oxidation/Reduction: Chemical Compatibility | Y | 41002703 |
| 830.6315 | Flammability/ Flame Extension | N/A | |
| 830.6316 | Explodibility | N/A | |
| 830.6317 | Storage Stability | N/A | |
| 830.6319 | Miscibility | N/A | |
| 830.6320 | Corrosion Characteristics | N/A | |
| 830.6321 | Dielectric Breakdown Voltage | N/A | |

| Guideline Number | Requirement | Are Data Requirements Fulfilled | MRID Number |
|---|---|---|---|
| 830.7000 | pH of Water Solutions or Suspensions | Y | 40999403 |
| 830.7050 | UV/VIS Absorption | Y[1] | 41002703 |
| 830.7100 | Viscosity | N/A | |
| 830.7200 | Melting Point/ Melting Range | Y | 40999403 |
| 830.7220 | Boiling Point/ Boiling Range | Y | 40999403 |
| 830.7300 | Density/Relative Density/Bulk Density | Y | 40999401 |
| 830.7370 | Dissociation Constant in Water | Y | 40999403 |
| 830.7520 | Particle Size, Fiber Length & Diameter Distribution | N/A | |
| 830.7550 | Partition Coefficient($K_{O/W}$), Shake Flask Method | Y | 41002703 |
| 830.7560 | Partition Coefficient ($K_{O/W}$), Generator Column Method | N/A | |
| 830.7570 | Partition Coefficient, ($K_{O/W}$), Liquid Chromatography Method | N/A | |
| 830.7840 | Water Solubility, Column Elution Method & Shake Flask Method | Y | 40999403 |
| 830.7860 | Water Solubility, Generator Column Method | N/A | |
| 830.7950 | Vapor Pressure | Y | 41002703 |

Note: 1. Wave length at which PCP decomposes was not provided by the registrants

## APPENDIX II: RESIDUE CHEMISTRY DATA REQUIREMENTS

Series 860- Residue Chemistry Test Guidelines

Table A

| Guideline No. | Requirement | Are the Requirements Fulfilled? | MRID / Reference |
|---|---|---|---|
| 860-1000 | Background | n/r | n/a[3] |
| 860.1100 | Chemical Identity | n/r[1] | n/a |
| 860.1200 | Directions for Use | n/r | n/a |
| 860.1300 | Nature of the Residue-Plants, Live Stocks | n/r | n/a |
| 860.1340 | Residue Analytical Method | n/r | n/a |
| 860.1360 | Multi Residue Method | n/r | n/a |
| 860.1380 | Storage Stability Data | n/r[2] | n/a |
| 860.1400 | Water, Fish and Irrigated Crops | n/r | n/a |
| 860.1460 | Food Handling | n/r | n/a |
| 860.1480 | Meat/Milk/Poultry/Eggs | n/r | n/a |
| 860.1500 | Crop field Trials | n/r | n/a |
| 860.1520 | Processed Feed/ Food | n/r | n/a |
| 860.1550 | Proposed Tolerances | n/r | n/a |
| 860.1560 | Reasonable Grounds in Support of the Petition | n/r | n/a |
| 860.1650 | Submittal of Analytical Reference Standards | n/r | n/a |
| 860.1850 | Confined Accumulation in Rotational Crops | n/r | n/a |
| 860.1900 | Field Accumulation in Rotational Crops | n/r | n/a |

Notes: 1. n/r means not required 2. n/a stands for not applicable.
3. It is required for Product Chemistry Data and has been fulfilled ( see chapter on Science Assessment).

## Appendix III. Toxicology Data Base

REREGISTRATION DATA REQUIREMENTS FOR PENTACHLOROPHENOL

TABLE 1 **TOXICOLOGY DATA SUMMARY**

| Guideline Number | Data Requirement | Are Data Requirements Fulfilled? [1] | MRID Number |
|---|---|---|---|
| 870.1100 | Acute Oral Toxicity | Y [1] | 0010715 |
| 870.1200 | Acute Dermal Toxicity | Y | 0010715 |
| 870.1300 | Acute Inhalation Toxicity | N | no study available |
| 870.2400 | Primary Eye Irritation | Y | 0010715 |
| 870.2500 | Primary Dermal Irritation | Y | 0010715 |
| 870.2600 | Dermal Sensitization | Y | 42594301 |
| 870.3250 | Subchronic Dermal Toxicity | Y | 43182301 |
| 870.4100 | Chronic Toxicity | Y | 43982701 |
| 870.4200 | Carcinogenicity in Mice | Y | NTP study |
| 870.4300 | Combined Chronic Toxicity / Carcinogenicity in Rats | Y | NTP study |
| 870.3700 | Developmental Toxicity in Rats | Y | 43091702 |
| 870.3700 | Developmental Toxicity in Rabbits | Y | 43091701 |
| 870.3800 | 2-Generation Reproduction Toxicity in Rats | Y [4] | 44464101 |
| 870.5575 | Mitotic gene conversion in Saccharomyces cerevisiae | Y | NTP study |
| 870.5265 | Salmonella thyphimurium reverse mutation assay | Y | NTP study |
| 870.5395 | Erythrocyte micronucleus assay | Y | 43911301 |
| 870.6200 | Neurotoxicity screening battery | N [2] | literature studies |
| 870.8700 | Immunotoxicity | N [3] | literature studies |

[1] Y = Yes; N = No; N/A = Not Applicable.

[2] the cited studies were not conducted according to the OPPTS guideline. The available literature data indicate the need for conduct of a Neurotoxicity Screening Battery for pentachlorophenol.

[3] the cited studies were not conducted according to the OPPTS guideline. A study with the purified test material is required to properly assess immunotoxic potential of pentachlorophenol.

[4] the study received was unacceptable, but may be upgraded upon receipt and review of the range-finding toxicity study.

## APPENDIX IV: Ecological Effects Data Requirements fo Pentachlorophenol

| Data Requirements | Composition | Does EPA Have Data To Satisfy This Requirement? | MRID | Must Additional Data Be Submitted Under FIFRA3(c)(2)(B)? |
|---|---|---|---|---|
| 71-1(a)/850.2100 Acute Avian Oral, Quail/Mallard | TGAI | Yes | 426337-01 | No |
| 71-2(a)/850.2200 Acute Avian Diet, Quail/Mallard | TGAI | Yes | 426337-03 | No |
| 71-4/850.???? Avian Reproduction (Preferably Mallard and/or Quail) | TGAI | No | _____ | No |
| 72-1(a)/850.1075 Acute Fish Toxicity, Rainbow Trout | TGAI | Yes | 434851-04 | No |
| 72-1(a)/850.1075 Acute Fish Toxicity, Rainbow Trout | TEP | No | _____ | Reserved*[1] |
| 72-1(a)/850.1075 Acute Fish Toxicity, Bluegill | TGAI | Yes | GUOPE N-02 | No |
| 72-1(a)/850.1075 Acute Fish Toxicity, Bluegill | TEP | No | _____ | Reserved*[1] |
| 72-2/850.1010 Acute EC50 Freshwater Invertebrate Toxicity, (Daphnia) | TGAI | Yes | 63572 | No |
| 72-3/850.1025; 850.1035; 850.1045;850.1055; 850.1075 Acute Estuarine/Marine Organisms Toxicity | TGAI | Yes | 434851-04 | No |
| 72-3/850.1025; 850.1035; 850.1045;850.1055; 850.1075 Acute Estuarine/Marine Organisms Toxicity | TEP | No | _____ | Reserved*[1] |
| 72-4(a)/850.1300 Fish Early Life Stage | TGAI | Yes | GUOPE N-03 | No |
| 72-4(b)/850.1300 Aquatic Invertebrate Life Cycle (Daphnia) | TGAI | Yes | GUOPE N-03 | No |
| 72-5/850.1500 Fish Life Cycle | TGAI | No | _____ | No |

| | | | | |
|---|---|---|---|---|
| 72-6/850.1710; 850.1730; 850.1850 Aquatic Organism Bioavailability/Biomagnification/ Toxicity Tests | TGAI | No | _____ | Reserved*[2] |
| 72-7/850.1950 Simulated or Actual Field Testing for Aquatic Organisms | TGAI | No | _____ | No |
| 73-1/850.1735 Whole Sediment, Acute Freshwater Invertebrates | TGAI | No | _____ | Reserved*[2] |
| 73-2/850.1740 Whole Sediment, Acute Marine Invertebrates | TGAI | No | _____ | Reserved*[2] |
| 73-3/???? Acute Pore Water, Fish and Invertebrates | TGAI | No | _____ | Reserved*[2] |
| 74-1/???? Whole Sediment, Chronic Invertebrates | TGAI | No | _____ | Reserved*[2] |
| 123-1/8504225 Seedling Emergence, Dose Response | TGAI | No | _____ | No |
| 123-1/850.4250 Vegetative Vigor, Dose Response | TGAI | No | _____ | No |
| 123-2/840.4400; 840.5400 Aquatic Plant Growth, Algal and Aquatic Plant Toxicity (Tier II) | TGAI | Yes | 426337-04 426337-08 | No |

*[1]Deferred pending review of special leaching study.

*[2]These studies might be required under the new guidelines established by the Agency in 1998. However, because they were not required prior to the review of the PCP RED, the Agency has placed a reserve on these data until further notice.

Appendix V . Human exposure Data base

## Reregistration data requirements for Pentacholrophenol

| Guideline Number | Data Requirements | Are Data requirements Fulfilled? |
|---|---|---|
| 875.1700 | Product use information | N |
| 875.1100, 875.1600 | Dermal exposure | N |
| 875.1300, 875.1600 | Inhalation exposure | N |
| 875.1500, 875.1600 | Biological monitoring | N |
| | Post-Application | N |
| 875.2800, 875.2900 | Description of human activity | N |
| 875.2300, 875.2900 | Indoor surface residue dissipation | N |
| 875.2400, 875.2900 | Dermal exposure | N |
| 875.2500, 875.2900 | Inhalation exposure | N |
| 875.2600, 875.2900 | Biological monitoring | N |

**APPENDIX : PRZM3/EXAMS INPUT DATA FILES:**

**Table 1. The Input Cotton Scenario Used in PRZM3 Model.**

---

```
PRZM3 Input File, COTTON.inp
Location: MS Crop: cotton  MLRA 134
   0.76    0.15       0   17.00       1       1
      4
   0.49    0.40    0.75   10.00              4    6.00   354.0
      1
      1    0.20  125.00   98.00       3  99  93  92              0.00  120.00
      1       3
0101 2109 2209
0.63 0.16 0.18
0.02 0.02 0.02
      1
  01 547  07 947  220984       1
Application: 1 soil appl. 0.103 kg/ha @100% eff, w/o drift
      1       1       0       0
Pentachlorophenol: KOC=3420;  AESM t1/2= 63.0 days
  010148  0 1 0.00 .103 1.00 0.00
     0.       1
Soil Series: Loring silt loam; Hydrogic Group C, koc = 191
  155.00    0.00   0   0   0   0   0   0   0   0   0
   0.00    0.00    0.00
      6
      1   13.00   1.400   0.385   0.000   0.000   0.000
          .0110   .0110   0.000
          0.100   0.385   0.151   2.180    74.6
      2   23.00   1.400   0.370   0.000   0.000   0.000
          .0110   .0110   0.000
          1.000   0.370   0.146   0.490    16.8
      3   33.00   1.400   0.370   0.000   0.000   0.000
          .0110   .0110   0.000
          1.000   0.370   0.146   0.160    5.47
      4   30.00   1.450   0.340   0.000   0.000   0.000
          .0110   .0110   0.000
          1.000   0.340   0.125   0.124    4.24
      5   23.00   1.490   0.335   0.000   0.000   0.000
          .0110   .0110   0.000
          1.000   0.335   0.137   0.070    2.39
      6   33.00   1.510   0.343   0.000   0.000   0.000
          .0110   .0110   0.000
          1.000   0.343   0.147   0.060    2.05
```

```
  0
     YEAR     5          YEAR     5          YEAR     5  1
  6
 11 -----
  5  DAY
RFLX   TSER  0  0  1.E5
EFLX   TSER  0  0  1.E5
ESLS   TSER  0  0  1.E0
RUNF   TSER  0  0  1.E0
PRCP   TSER  0  0  1.E0
```

---

The chemical file for pentachlorophenol used to run EXAMS is shown in Table 2. The data used in this file are documented in the EPA Pentachlorophenol files.

**Table 2. Chemical input data used in EXAMS for Pentachlorophenol.**

---

```
        *** Chemical-specific data: SET via "entry( 1)"
MWT:   2.66E+02 VAPR:  1.00E-04 HENRY:         KOW:   1.12E+05
KVO:           EVPR:          EHEN:          KOC:   3.42E+03
*** Ion-specific data: "entry(1, 1)"
SOL:   1.40E+01 KPB:           KPS:
ESOL:          KPDOC:
*** Reactivity of dissolved species: SET via "entry(1, 1, 1)"
KAH:           EAH:           KNH:           ENH:
KBH:           EBH:           KRED:          ERED:
KBACW: 5.80E-03 QTBAW:         KBACS: 8.00E-04 QTBAS:
*** Reactivity of solids-sorbed species:     "entry(2, 1, 1)"
KAH:           EAH:           KNH:           ENH:
KBH:           EBH:           KRED:          ERED:
KBACW: 5.80E-03 QTBAW:         KBACS: 8.00E-04 QTBAS:
*** Reactivity of "DOC"-complexed species:   "entry(3, 1, 1)"
KAH:           EAH:           KNH:           ENH:
KBH:           EBH:           KRED:          ERED:
KBACW: 5.80E-03 QTBAW:         KBACS: 8.00E-04 QTBAS:
*** Reactivity of biosorbed species:         "entry(4, 1, 1)"
KBACW: 5.80E-03 QTBAW:         KBACS: 8.00E-04 QTBAS:

Photochemical process data;  Ion-specific data: "entry(1, 1)"
KDP(1, 1): 1.98E-01 RFLAT(1, 1):   0.0    LAMAX(1, 1):   0.0
*** Reactivity of dissolved species: SET via "entry(1, 1, 1)"
K1O2:          EK1O2:         KOX:           EOX:
*** Reactivity of solids-sorbed species:     "entry(2, 1, 1)"
```

K1O2: EK1O2: KOX: EOX:
*** Reactivity of "DOC"-complexed species: "entry(3, 1, 1)"
K1O2: EK1O2: KOX: EOX:
QUA(1,1, 1) QUA(2,1, 1) QUA(3,1, 1)
Light ABSORption (n,1, 1): (1) (2)
(3) (4) (5) (6)
(7) (8) (9) (10)
(11) (12) (13) (14)
(15) (16) (17) (18)
(19) (20) (21) (22)
(23) (24) (25) (26)
(27) (28) (29) (30)
(31) (32) (33) (34)
(35) (36) (37) (38)
(39) (40) (41) (42)
(43) (44) (45) (46)
1Exposure Analysis Modeling System -- EXAMS Version 2.97, Mode 3
Chemical: 1) penta

---