ENVIRONMENTAL RISK RED CHAPTER

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Executive Summary

This environmental risk assessment addresses hazard to birds, mammals, plants, insects, fish, and aquatic invertebrates from the components of inorganic arsenical wood preservatives. The predicted exposure to these compounds from the use of these wood preservatives is also estimated as part of the risk assessment process. The hazard and exposure information are then combined to estimate the risk inorganic arsenical wood preservatives pose to plants and animals in terrestrial and aquatic habitats.

Certain registered inorganic arsenical products are mixtures of several active ingredients. Some active ingredients in these product mixtures are not covered under this Inorganic Arsenicals RED but have been or will be assessed for reregistration eligibility under separate RED cases:

> Cupric Oxide, the form of copper used in CCA treatment solutions, is not included as an active ingredient covered under this RED. Reregistration requirements for Cupric Oxide will be addressed separately in a RED document for Copper, and oxides, Case Number 4025, to be issued at a future date.
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> Zinc Oxide, the zinc component of ACZA, has already been assessed by the Agency for the wood preservative use patterns under the Zinc Salts RED, Case Number 4099, dated August, 1992.

Inorganic Arsenical wood preservatives are most commonly formulated as Chromated Copper Arsenate (CCA). Ecological toxicity data are available for the components of CCA in the form of studies that have been submitted to the Agency and studies found in the open literature. In some cases, the toxicity studies evaluated other forms of the metals than the specific compounds found in CCA (e.g., arsenic acid and chromic acid).

The results of the terrestrial risk assessment indicate that the potential for adverse effects to birds and mammals from exposure to ***average*** concentrations of CCA components in soil is low. Average soil concentrations are considered more likely to represent the exposure level for mobile receptor species such as birds and mammals than maximum soil concentrations. It should be noted, however, that the risk assessment was only based on exposure to CCA components in soil. A quantitative assessment of the risks to birds and mammals from direct contact with CCA-treated lumber was not conducted due to the lack of exposure and toxicity data available. As a result, the potential risks from direct contact with CCA-treated wood were not evaluated. Additional uncertainties associated with the assessment are discussed in the Uncertainty section of this report.

A numerical risk assessment was not conducted for aquatic organisms. There is a lack of validated models available to estimate the water-column concentrations of arsenic and chromium as leached from CCA-treated wood structures. The open literature provides laboratory-derived

leaching study values, but these data are highly variable as leaching rates of the metals are highly dependent on the test conditions as well as the age of the wood and CCA retention level. Additionally, water-column concentrations of these metals in aquatic habitats would likely be much lower than the values obtained in leaching studies conducted in small vessels, due to dispersion in the water body and partitioning into biota and sediment. Calculating risk quotients (RQ) using the published values as "worst-case" EECs was considered, but due to the variability of the published data, as well as the high degree of uncertainty in extrapolating the results to "real-world" conditions, it was determined that this approach would not provide meaningful estimates of the risk to aquatic organisms from CCA-treated wood. There are some published studies on the effects of CCA-treated wood on aquatic organisms, which indicate that the metals released from the treated wood are taken up by biota and cause adverse effects to aquatic organisms, at both community and individual levels (Weis et al., 1991; Weis and Weis, 1995; Weis and Weis, 1996).

I. Ecological Effects Hazard Assessment

The toxicity endpoints (e.g., LC50, NOEC, etc.) used in the ecological risk assessment were obtained from several sources. Primary sources include the Pesticide Ecotoxicity Database maintained by the U.S. EPA, Office of Prevention, Pesticides and Toxic Substances/Office of Pesticide Programs (U.S. EPA, 2002a), and the Health Effects Division's Hazard Identification Assessment Review Committee's (HIARC) report on arsenic and chromium, which included several toxicity endpoints for mammalian species.

A secondary source for toxicity information is the ECOTOX database maintained by the U.S. EPA, Office of Research and Development (ORD), and the National Health and Environmental Effects Research Laboratory's (NHEERL's) Mid-Continent Ecology Division (U.S. EPA, 2002b). This database includes both aquatic and terrestrial toxicity studies from peer-reviewed literature. The information in this database is generally limited to toxicity endpoints; it does not include sufficient detail about study methodology to determine whether the studies would meet EPA Guideline requirements. Since the data are obtained from peer-reviewed literature, they are considered scientifically sound, and provide supplemental data which can be used in a risk assessment.

If a toxicity endpoint was not available in the primary or secondary sources, a review of the open literature was performed. The ecotoxicological effects literature on arsenic and chromium is fairly extensive. Toxicity data obtained from the open literature were included in the risk assessment only in cases where similar data were not found in either the primary or secondary sources. As with the data from the databases discussed above, the information in articles from the open literature is usually not detailed enough regarding study methodology to determine whether the study would meet EPA Guideline requirements. However, since they are published in peer-reviewed journals, the studies are considered scientifically sound and provide supplemental data which is appropriate for use in a risk assessment.

A. Toxicity to Terrestrial Animals

1. Birds, Acute and Subacute

a. Arsenic acid

An acute oral toxicity study using the technical grade of the active ingredient (TGAI) is required to establish the toxicity of arsenic acid to birds. The preferred test species is either mallard duck (a waterfowl) or bobwhite quail (an upland game bird). The results of two studies identified in the pesticide database are presented in the following table.

Table 1. Acute Oral Toxicity of Arsenic Acid to Birds

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
arsenic acid/ 75%	Northern bobwhite (*Colinus virginianus*)	LD50 = 28.9	- core study - organism age: 27 wks - 21 day test duration	Fletcher, 1987 (40409013)
arsenic acid/ 76.1%	Northern bobwhite (*Colinus virginianus*)	LD50 = 46 NOEL = 12.5	- core study - organism age: 18 wks - 14 day test duration - NOEL effect: weight loss	Campbell et al., 1990 (41719201)

These results indicate that arsenic acid is highly toxic to avian species on an acute oral basis. The guideline requirement (71-1/OPPTS 850.2100) is fulfilled (MRID# 40409013, 41719201).

Two subacute dietary studies using the TGAI are required to establish the toxicity of arsenic acid to birds. The preferred test species are mallard duck (a waterfowl) and bobwhite quail (an upland gamebird). The results of three avian subacute dietary tests that were included in the pesticide database are tabulated below.

Table 2. Subacute Dietary Toxicity of Arsenic Acid to Birds

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
arsenic acid/ 75%	Northern bobwhite (*Colinus virginianus*)	LC50 = 168.5	- core study - 8 day test duration	EPA 2002a (00121618)
arsenic acid/ 76%	Northern bobwhite (*Colinus virginianus*)	LC50 = 432 NOEL = 15.6	- core study - organism age: 10d - 8 day test duration - NOEL effect: pharmacotoxic signs	Long et al, 1990 (41719202)
arsenic acid/ 75%	Fulvous whistling duck (*Dendrocygna bicolor*)	LC50 = 1145 NOEC < 156	- core study - organism age: 9 days - 8 day test duration - NOEL effect: reduced weight gain	Fletcher, 1987 (40409012)

These results indicate that arsenic acid is slightly to highly toxic to avian species on a subacute dietary basis. The guideline requirement (71-2/OPPTS 850.2200) is fulfilled (MRID # 00121618, 41719201, 40409012).

b. Chromic acid

An acute oral toxicity study using the TGAI is required to establish the toxicity of a chromic acid to birds. The preferred test species is either mallard duck (a waterfowl) or bobwhite quail (an upland gamebird). The results of two studies are presented in the following table.

Table 3 . Acute Oral Toxicity of Chromic Acid to Birds

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
chromic acid/ 57%	Northern bobwhite (*Colinus virginianus*)	LD50 = 93.5	- core study	Hoxter., 1990 (41621104)
chromic acid/ 57%	Northern bobwhite (*Colinus virginianus*)	LC50 = 164 NOEL > 62	- core study - organism age: 38wks - 14 day test duration - NOEL effect: weight loss	Hobden, 2000 (T15)

These results indicate that chromic acid is moderately toxic to avian species on an acute oral basis. The guideline requirement (71-1/OPPTS 850.2100) is fulfilled (MRID# 41621104).

Two subacute dietary studies using the TGAI are required to establish the toxicity of chromic acid to birds. The preferred test species are mallard duck (a waterfowl) and bobwhite quail (an upland gamebird). The results of avian subacute dietary tests are tabulated below.

Table 4. Subacute Dietary Toxicity of Chromic Acid to Birds

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
chromic acid/ 57%	Mallard duck *(Anas platyrhynchus)*	LC50 > 5100	- core study - organism age: 10d - 8 day test duration	Hoxter, 1990 (41621102)
chromic acid/ 57%	Northern bobwhite (*Colinus virginianus*)	LC50 > 5100	- core study - organism age: 10d - 8 day test duration	Hoxter,1990 (41621101)

These results indicate that chromic acid is practically non-toxic to avian species on a subacute dietary basis. The guideline requirement (71-2/OPPTS 850.2200) is fulfilled (MRID # 41621102, 41621101).

2. Birds, Chronic

Avian reproduction studies using the TGAI may be required for a pesticide when any of the following conditions are met: (1) birds may be subject to repeated or continuous exposure to the pesticide, especially preceding or during the breeding season, (2) the pesticide is stable in the environment to the extent that potentially toxic amounts may persist in animal feed, (3) the pesticide is stored or accumulated in plant or animal tissues, and/or, (4) information derived from mammalian reproduction studies indicates reproduction in terrestrial vertebrates may be adversely affected by the anticipated use of the product.

Avian chronic toxicity tests are required for the components of CCA because birds may be exposed to the compound on treated outdoor structures (e.g., docks, utility poles, etc.) or to residues from these structures during daily activities such as feeding, nesting and grooming. There were no avian chronic toxicity studies for arsenic acid or chromic acid in the pesticide database; the data requirement (850.2300/old 71-4) are required as confirmatory data.

A study found in the open literature (Sample et al.,1996) identified a chronic avian NOAEL of 5.1 mg/kg-day for sodium arsenite (As 3+). This value was based on the results of a chronic dietary study that evaluated the effects of sodium arsenite (As 3+) to mallard ducks over a 128-day study period. Sample et al. (1996) also identified a chromium avian NOAEL of 1 mg/kg-day. This value was based on the results of a chronic dietary study that evaluated the effects of chromium (as Cr III) to black ducks during a ten month study duration.

3. Mammals, Acute and Subacute

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, toxicity values for laboratory animals (e.g., rat, mouse) obtained from the Agency's Health Effects Division (HED) substitute for wild mammal testing.

a. Arsenic acid

No studies on the acute effects of arsenic acid to wild mammals were found in the primary or secondary data sources considered for this assessment; therefore, laboratory mammal acute toxicity endpoints are used. Acute toxicity values identified for arsenic acid in Chen et al. (2001) are summarized in the table below.

Table 5. Mammalian Acute Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
Arsenic acid/ 75%	Laboratory mouse (*Mus musculus*)	LD50 = 150	LD50 is the mean value for male (141mg/kg) and female mice (160mg/kg)	#40409001 as cited in Chen et al,. 2001
Arsenic acid/ 75%	Laboratory rat *(Rattus norvegicus)*	LD50 = 52	LD50 is the mean value for male (76mg/kg) and female rats (37mg/kg)	# 26356 as cited in Chen et al,. 2001

These results indicate that arsenic acid is moderately to highly toxic to mammalian species on an acute oral basis. The guideline requirement (81-1/OPPTS 870.1100) is fulfilled (MRID# 40409001, 26356).

b. Chromic acid

No studies on the acute effects of chromic acid to wild mammals were found in the primary or secondary data sources considered for this assessment; therefore, laboratory mammal acute toxicity endpoints are used. The results of one acute toxicity study identified in McMahon and Chen (2001) for chromic acid are provided in the table below.

Table 6. Mammalian Acute Toxicity of Chromic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg)	Comments	Reference
Chromic acid/ 100%	Laboratory rat *(Rattus norvegicus)*	LD50 = 52	LD50 is the mean value for male (56 mg/kg) and female mice (48mg/kg)	MRID# 434294-01 as cited in McMahon and Chen, 2001

This study indicates that chromic acid is highly toxic to mammalian species on an acute oral basis. The guideline requirement (81-1/OPPTS 870.1100) is fulfilled (MRID# 434294-01).

4. Mammals, Chronic

Mammalian reproduction studies using the TGAI may be required for a pesticide; this requirement is considered in the human toxicology assessment portion of this RED document. Chronic hazard to wild mammals from CCA is of concern due to the potential for repeated or continuous mammalian exposure to CCA components from either direct contact with treated structures or exposure to residues in soil and/or water from treated structures.

a. Arsenic acid

The Health Effects Division's Hazard Identification Assessment Review Committee's (HIARC) report on arsenic (McMahon and Chen, 2001) included several toxicity endpoints for mammalian species. These endpoints have been provided in the following table.

Table 7 . Mammalian Chronic Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg/day)	Comments	Reference
arsenic acid/ 75%	Laboratory mouse (*Mus musculus*)	LOAEL = 64 NOAEL = 32	- gavage administration to pregnant females during day 6-15 of gestation - effects were reduced body weight, increased mortality and increased total litter resorption	Nemac 1968b, cited in McMahon and Chen, 2001
arsenic acid/ 75%	New Zealand White Rabbit	LOAEL = 4 NOAEL = 1	- gavage administration to pregnant females during day 6-18 of gestation - effects were reduced body weight, increased mortality and histological effect to the liver/kidneys	Nemac 1988a, cited in McMahon and Chen, 2001
arsenic V/ AI not available	Dog – Beagle	LOAEL = 2.4 NOAEL = 1	- study duration – 2 yrs - endpoint was histology effects to the liver and anemia	Byron et al., 1967, cited in ATSDR, 1997

These studies indicate that arsenic acid is moderately toxic to mammalian species on a chronic basis.

b. Chromic acid

The Health Effects Division's Hazard Identification Assessment Review Committee's (HIARC) review of chromium (McMahon and Chen, 2001) included several toxicity endpoints for mammalian species. These endpoints have been provided in the following table.

Table 8. Mammalian Chronic Toxicity of Chromic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/kg/day)	Comments	Reference
Chromic acid/ 55%	Laboratory rabbit	LOAEL = 0.48 NOAEL = 0.12	- effects included reduced body weight, increased mortality	Tyl, 1991, cited in McMahon and Chen, 2001
Potassium dichromate/ AI not available	Swiss albino mice	LOAEL = 42.1 NOAEL = 22.3	- administered through drinking water during days 6-14 of gestation - effects were reduced maternal body weight, retarded fetal development and increased fetal resorption - NOTE: The endpoints were based on maternal effects, the fetal effects were seen at every dose tested so a developmental NOAEL could not be determined	Junaid et al., 1996, cited in McMahon and Chen, 2001

These studies indicate that chromic acid is moderately to highly toxic to mammalian species on a chronic basis.

5. Insects

A honey bee acute contact study using the TGAI is required for the active components of CCA because its use as a wood preservative may result in honey bee exposure. Results of this test for arsenic acid are tabulated below.

Table 9. Nontarget Insect Acute Contact Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (ug/bee)	Comments	Reference
arsenic acid/ 75.1%	Honey bee (*Apis mellifera*)	LD50 = 7.7	- core study	Hoxter, 1987 (40351301)

The results indicate that arsenic acid is highly toxic to bees on an acute contact basis. The guideline requirement (141-1/OPPTS 850.3020) is fulfilled (MRID #40351301).

There were no honey bee studies identified for chromic acid in the pesticide database; the guideline requirement (141-1/OPPTS 850.3020) is not fulfilled for chromic acid, and is now required as confirmatory data.

6. Terrestrial Field Testing

Terrestrial field testing is not required for the wood preservative use of CCA.

B. Toxicity to Freshwater Aquatic Animals

1. Freshwater Fish, Acute

a. Arsenic acid

Two freshwater fish toxicity studies using the TGAI are required to establish the toxicity of arsenic acid to fish. The preferred test species are rainbow trout (a coldwater fish) and bluegill sunfish (a warmwater fish). Several acute toxicity studies were identified for each compound of interest in the pesticide database. The results of these tests are summarized below.

Table 10. Freshwater Fish Acute Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
arsenic acid/ 76.1%	Rainbow trout *(Oncorhynchus mykiss)*	LC50 = 72 NOEL = 3.4	- core study - static system - 96-hr test duration	LeLievre, 1990 (41620003)
arsenic acid/ 75%	Rainbow trout *(Oncorhynchus mykiss)*	LC50 = 53	- supplemental study - static system - 96-hr test duration - test organism weight 0.57g	EPA 2002 a (MRID # not reported)
arsenic acid/ 75.8%	Bluegill sunfish *(Lepomis macrochirus)*	LC50 = 50 NOEL = 10	- core study - flow-through system - 96-hr test duration - test organism weight 1.1g	Machado, 1991 (41950601)
arsenic acid/ 75%	Bluegill sunfish *(Lepomis macrochirus)*	LC50 = 54 NOEL < 12	- supplemental study - static system - 96-hr test duration - test organism weight 1.8g	Suprenant, 1987 (40409014)

These results indicate that arsenic acid is slightly toxic to freshwater fish on an acute basis. The guideline requirement (72-/OPPTS 850.1075) is fulfilled (MRID #41620003, 41950601).

b. Chromic acid

Two freshwater fish toxicity studies using the TGAI are required to establish the toxicity of chromic acid to fish. The preferred test species are rainbow trout (a coldwater fish) and bluegill sunfish (a warmwater fish). Two acute toxicity studies were identified for this compound in the pesticide database. The results of these tests are summarized below.

Table 11. Freshwater Fish Acute Toxicity of Chromic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
Chromic acid/ 57%	Rainbow trout *(Oncorhynchus mykiss)*	LC50 = 28 NOEL = 8.6	- core study - static system - 96-hr test duration - test organism weight – 0.5g	LeLievre, 1990 (41621105)
Chromic acid/ 57%	Bluegill sunfish *(Lepomis macrochirus)*	LC50 = 44 NOEL = 13	- core study - static system - 96-hr test duration - test organism weight – 0.5g	LeLievre, 1990 (41658401)

These results indicate that chromic acid is slightly to moderately toxic to freshwater fish on an acute basis. The guideline requirement (72-/OPPTS 850.1075) is fulfilled (MRID #41621105, 41658401).

2. Freshwater Fish, Chronic

Fish early life stage tests are required if the product is applied directly to water or expected to be transported to water from the intended use site, and when any one or more of the following conditions apply: (1) if the pesticide is intended for use such that its presence in water is likely to be continuous or recurrent regardless of toxicity; (2) if any LC_{50} or EC_{50} value determined in acute toxicity testing is less than 1 mg/L; or (3) if the estimated concentration in water is equal to or greater that 0.01 of any EC_{50} or LC_{50} determined in acute toxicity testing; (4) if the actual or estimated environmental concentration in water resulting from use is less than 0.01 of any EC_{50} or LC_{50} determined in acute toxicity testing and any of the following conditions exist: (a) studies of other organisms indicate the reproductive physiology of fish and /or invertebrates may be affected; (b) physicochemical properties indicate cumulative effects; (c) the pesticide is persistent in water (e.g. half-life in water greater than 4 days). The preferred test species is fathead minnow (*Pimephales promelas*), but other species may be used. Fish early life-stage testing is required due to the likelihood of continuous exposure from CCA-treated structures in aquatic habitats. Freshwater and marine/estuarine fish are comparably sensitive to arsenic acid and chromic acid on an acute basis, so early life-stage testing on either type of fish is sufficient to satisfy guideline requirements. Early life-stage testing was submitted to the agency for arsenic acid and chromic acid using freshwater species; that data is summarized below.

a. Arsenic acid

Table 12. Freshwater Fish Early Life Stage Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
Arsenic acid/ 76.1%	Fathead minnow *(Pimphales promelas)*	LOAEC = 1.9 NOAEC = 0.97	- core study - flow-through system - 35 day test duration - endpoint is larval survival	Machado, 1991 (41802201)

The results of this study indicate that arsenic acid affects fish larval survival at levels greater than 0.97 ppm. The guideline requirement (72-4/OPPTS 850.1400) is fulfilled (#41802201).

b. Chromic acid

Table 13. Freshwater Fish Early Life Stage Toxicity of Chromic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
chromic acid/ 57%	Fathead minnow *(Pimphales promelas)*	LOAEC = 8.2 NOAEC = 4.0	- core study - flow-through system - 35 day test duration - endpoint is larval growth	Machado, 1991 (41974901)

This result indicates that chromic acid causes larval growth effects at levels above 4.0 ppm. The guideline requirement (72-4/OPPTS 850.1400) is fulfilled (#41974901).

3. Freshwater Invertebrates, Acute

A freshwater aquatic invertebrate toxicity test using the TGAI is required to establish the toxicity of the components of CCA to aquatic invertebrates. The preferred test species is *Daphnia magna*. Results of this test for arsenic acid and chromic acid are provided in the tables below.

Table 14. Acute Toxicity of Arsenic Acid to Freshwater Invertebrates

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
arsenic acid/ 76.1%	Water flea *(Daphnia magna)*	EC50 = 15 NOEL = 2.6	- core study - static system - 48-hour test duration - endpoint is immobilization	LeLievre, 1990 (41620001)

This study indicates that arsenic acid is slightly toxic to aquatic invertebrates on an acute basis. The guideline requirement (72-2) is fulfilled (41620001).

Table 15. Acute Toxicity of Chromic Acid to Freshwater Invertebrates

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
chromic acid/ 57%	Water flea *(Daphnia magna)*	EC50 = 0.76 NOEL < 0.35	- core study - static system - 48-hour test duration - endpoint is immobilization	LeLievre, 1990 (41621103)

This study indicates that chromic acid is highly toxic to aquatic invertebrates on an acute basis. The guideline requirement (72-2) is fulfilled (MRID# 416211043).

4. 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, or equal to, ($\leq$) 0.1 mg/ml. The components of CCA meet the above environmental fate criteria for whole sediment toxicity testing since CCA-treated wood is used in aquatic environments and available data regarding the acute toxicity of the components of CCA indicates high toxicity to freshwater invertebrates from chromic acid. Whole sediment acute testing is therefore required as confirmatory data. Because no data on this topic could be located, the risk to invertebrates from exposure to CCA- contaminated sediment cannot be addressed at this time.

5. Freshwater Invertebrate, Chronic

A freshwater invertebrate life-cycle test using the TGAI is required for a pesticide when it is used in the aquatic environment or use in aquatic sites is not prohibited and when any of the following conditions apply: (1) if any LC_{50} or EC_{50} value determined in acute toxicity testing is less than 1 mg/L; (2) if the estimated environmental concentration is water is greater than or equal to ($\geq$) 0.01 of any EC_{50} or LC_{50} determined in acute toxicity testing; (3) if the actual or estimated environmental concentration in water is less than 0.01 of any EC_{50} or LC_{50} 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*.

Aquatic invertebrate life-cycle testing is required for the components of CCA since CCA-treated lumber will be used to construct structures in aquatic environments (e.g., pier, piling, or dock uses). The results of these tests for arsenic acid and chromic acid are provided below.

Table 16. Freshwater Aquatic Invertebrate Life-Cycle Toxicity of Arsenic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (ug/L)	Comments	Reference
arsenic acid/ 76.1%	Water flea *(Daphnia magna)*	LOEC = 38 NOAEC = 20	- supplemental study - flow-through system - 21day test duration - NOAEC effect - growth	McNamara, 1991 (42001601)

These results indicate that arsenic acid impacts the growth of aquatic invertebrates at levels greater than 20 ppb. The guideline requirement (72-4b) is fulfilled (MRID #42001601).

Table 17. Freshwater Aquatic Invertebrate Life-Cycle Toxicity of Chromic Acid

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (ug/L)	Comments	Reference
Chromic acid/ 57%	Water flea *(Daphnia magna)*	LOEC < 8.6 NOAEC < 2.6	- core study - flow-through system - 21day test duration - NOAEC effect - reproduction	McNamara, 1991 (41881501)

These results indicate that chromic acid impacts the reproduction of aquatic invertebrates at levels greater than 2.6 ppb. The guideline requirement (72-4b) is fulfilled (MRID#41881501).

6. Acute Pore Water, Fish and Invertebrates

An 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) 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 components of CCA meet the above environmental fate criteria for acute pore water toxicity testing since CCA-treated wood is used in aquatic environments and available data indicate a high level of toxicity to invertebrates from chromic acid. Acute pore water testing is therefore required as confirmatory data. Because this data was not readily available in the primary sources, the risk to fish and invertebrates from exposure to CCA-contaminated pore water cannot be addressed at this time.

7. 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). Certain components of CCA meet these environmental fate criteria, therefore, confirmatory aquatic field data are needed to fully characterize the risk CCA poses to aquatic organisms. This data was unavailable at the time of this risk assessment, so the potential impact of CCA-treated wood to organisms in aquatic habitats cannot be fully assessed at this time.

8. Freshwater Organism Toxicity Data from Published Scientific Literature

Several studies in the open literature have evaluated the effects of CCA components to aquatic organisms. Scientific literature indicates that aquatic organisms may be adversely affected by the components of CCA if they are exposed to very high concentrations of the metals. For example, benthic organisms that occur near CCA-treated structures such as docks and bulkheads are likely to be exposed to very high metal concentrations (Weis et al., 1991). Actual concentrations of arsenic and chromium in surface water and sediment as the result of leaching from CCA-structures are dependent on many factors such as size of the waterbody, flow patterns (e.g., current, tides), physiochemical properties of the surface water and sediment, and characteristics of the treated structure (e.g., size, wood type, structural age).

C. Toxicity to Estuarine and Marine Animals

1. Estuarine and Marine Fish, Acute

Acute toxicity testing with estuarine/marine fish using the TGAI is required for a pesticide when 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. Marine/estuarine acute testing was required for the components of CCA due to its use as a wood preservative for lumber used to construct docks, piers and pilings. Results of these tests are tabulated below for arsenic acid and chromic acid.

Table 18. Acute Toxicity of Arsenic Acid to Estuarine/Marine Fish

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
arsenic acid/ 76.1%	Sheepshead minnow *(Cyprinodon variegatus)*	LC50 = 28 NOEL = 13	- core study - static system - 96 hour test duration	41620004 LeLievre, 1990

This study indicates that arsenic acid is moderately toxic to estuarine/marine fish on an acute basis. The guideline requirement (72-3a) is fulfilled (MRID #41620004).

Table 19. Acute Toxicity of Chromic Acid to Estuarine/Marine Fish

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
chromic acid/ 57%	Sheepshead minnow *(Cyprinodon variegatus)*	LC50 = 39 NOEL = 20	- core study - static system - 96 hour test duration	41703601 LeLievre/1990

The results indicate that chromic acid is moderately toxic to estuarine/marine fish on an acute basis. The guideline requirement (72-3a) is fulfilled (MRID #41703601).

2. Estuarine and Marine Fish, Chronic

Fish early life-stage testing using the TGAI is required for wood preservatives if the product will be used to treat wood used in an aquatic habitat, such as piers, pilings, and docks. Early life-stage toxicity data are required on the more sensitive type of fish, freshwater or estuarine/marine. Based on review of the acute toxicity data, freshwater and marine/estuarine fish appear to be comparably sensitive to arsenic acid and chromic acid. Since acceptable fish early life-stage data were available for the freshwater species fathead minnow, fish early life-stage testing with a marine/estuarine fish is not required for either arsenic acid or chromic acid.

3. Estuarine and Marine Invertebrates, Acute

Acute toxicity testing with estuarine/marine invertebrates using the TGAI is required for a pesticide when 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 are mysid shrimp and eastern oyster. Estuarine/marine invertebrate testing was required for the components of CCA due to its use as a wood preservative for lumber used to construct docks, piers and pilings in coastal waters. Results of these tests are tabulated below.

Table 20. Acute Toxicity of Arsenic Acid to Estuarine/Marine Invertebrates*

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
arsenic acid/ 76.1%	Mysid (*Mysidopsis bahia*)	LC50 = 2 NOEL < 0.32	- core study - static system - 96 hour test duration	41620002 LeLievre/1990

* Note: Oyster testing was waived for arsenic acid

The results indicate that arsenic acid is moderately to highly toxic to estuarine/marine invertebrates on an acute basis. The guideline requirement 72-3b is waived, and the guideline requirement 72-3c is fulfilled (MRID #4160002).

Table 21. Acute Toxicity of Chromic Acid to Estuarine/Marine Invertebrates*

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
chromic acid/ 57%	Mysid (*Mysidopsis bahia*)	LC50 = 5.9 NOEL = 2.3	- core study - static system - 96 hour test duration	41703602 LeLievre, 1990

* Note: Oyster testing was waived for chromic acid

The results indicate that chromic acid is moderately to highly toxic to estuarine/marine invertebrates on an acute basis. The guideline requirement 72-3b is waived, and the guideline requirement 72-3c is fulfilled (MRID #41703602).

4. Whole Sediment Acute Invertebrate, Marine

Whole sediment acute invertebrate, marine studies are required 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 (≥) 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 that (>) 1,000 and solubility is less than or equal to (≤) 0.1 mg/l.

Several published studies have evaluated the effects of CCA to marine and estuarine sediment-dwelling organisms. Weis et al. (1998) studied five CCA-treated wood bulkheads of different ages in estuaries from New York to South Carolina. These authors concluded that metals leached from the treated wood and accumulated in the fine-grain fractions of nearby sediments. Benthic community species richness, diversity and biomass were reduced at sample stations one meter from the bulkheads, generally returning to background characteristics at a distance of 10 meters from the bulkheads.

5. Estuarine and Marine Invertebrate, Chronic

Aquatic invertebrate life-cycle testing using the TGAI is required for wood preservatives if the product will be used to treat wood used in an aquatic habitat, such as piers, pilings, and docks. Data are required on the more sensitive type of invertebrate, freshwater or estuarine/marine. Acute data show that freshwater and marine/estuarine invertebrates are comparably sensitive to arsenic acid, and that freshwater invertebrates are more sensitive than marine/estuarine invertebrates to chromic acid. Since acceptable aquatic invertebrate life-cycle data was submitted for the freshwater species *Daphnia magna*, testing with a marine/estuarine invertebrate is not required for arsenic acid or chromic acid.

6. Estuarine and Marine 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 (≥) 1,000 or hydrolysis half-life is greater than (>) 5 days). Certain components of CCA meet these environmental fate criteria, therefore, confirmatory aquatic field data are needed to fully characterize the risk CCA poses to aquatic organisms. This data was unavailable at the time of this risk assessment, so the potential impact of CCA-treated wood to organisms in aquatic habitats cannot be fully assessed at this time.

D. Toxicity to Plants

1. Terrestrial / Semi-aquatic

Terrestrial plant testing, including seedling emergence (123-1/OPPTS 850.4100) and vegetative vigor testing (123-1/OPPTS 850.4150), is required for wood preservatives in cases where the treated wood will be used in the aquatic environment (e.g., pier, piling or dock uses). Only one plant species, rice (*Oryza sativa*), must be tested. CCA meets the criteria for testing, since CCA-treated wood can be used in the aquatic environment. These tests (123-1a/850.4100 and 123-1b/850.4150) are now required as confirmatory data. Several studies in the open literature have evaluated the effects of CCA components to various terrestrial plant species. The results of these studies are summarized below.

a. Arsenic

Arsenic is taken up actively by plant roots, with arsenate being more easily absorbed than arsenite. Because arsenic is chemically similar to phosphorus, it is translocated in the plant in a similar manner and is able to replace phosphorus in many cell reactions. Mechanisms of arsenic (V) toxicity to terrestrial plants include interrupted phosphorylation and adverse effects to enzyme systems (Efroymson et al. 1997).

In review of the literature, one study evaluated the toxicity of arsenic (V) to corn grown from seed for 4 weeks in a loamy sand with a pH of 7.1. Results of the study indicated that corn fresh weight reductions rose from less than 10% with the addition of 10 ppm arsenic to more than 65% with the addition of 100 ppm arsenic (Efroymson et al. 1997). Another study assessed the toxicity of arsenic (III) and arsenic (V) added to soil on the yield of ryegrass and barley grown from seed for 1 year in a greenhouse. According to the study, there was a significant reduction in the yield of ryegrass (63%) following the addition of 250 ppm arsenic (V) to the soil (Efroymson et al., 1997). It should be noted that rice and legumes appear to be more sensitive to arsenic than other plants. Symptoms include wilting of new-cycle leaves, followed by retardation of root and top growth, and leaf necrosis.

b. Chromium

Chromium VI is more soluble and available to plants than chromium III and is therefore considered to be the more toxic form. Chromium that is taken up by plants generally remains in the roots because of the many binding sites in the cell wall capable of binding chromium ions. Symptoms of chromium toxicity in plants include stunted growth, poorly developed roots and leaf curling. Chromium may interfere with carbon (C), nitrogen (N), phosphorus (P) and iron (Fe) metabolism and enzyme reactions (Efroymson et al., 1997).

One study investigated the effect of chromium (VI) on soybean seedlings grown three days in a loam soil. The study found that fresh shoot weight was reduced 30% following the addition of 30ppm chromium, while 10 ppm chromium had no observed effect. Another study calculated EC50 concentrations for effects of chromium (VI) on lettuce, tomato and oats grown in a growth chamber from seed for 14 days. Test plants grown in a loam soil had EC50 values ranging from 1.8 ppm (in lettuce) to 7.4 ppm (in oats). Test plants grown in a humic sand soil appeared to be relatively less sensitive to chromium (VI) exposure with EC50 values ranging from > 11 ppm (in lettuce) to 31 ppm (in oats), respectively (Efroymson et al.,1997).

2. Aquatic Plants

Aquatic plant testing is required for wood preservatives if they are used to treat wood intended for use in aquatic environments (e.g., pier, piling, or dock uses). Testing is conducted with one species of aquatic vascular plant (usually *Lemna gibba*) and four species of algae (*Skeletonema costatum*, *Anabaena flos-aquae*, *Selenastrum capricornutum* and a freshwater diatom). CCA meets the criteria for testing, since CCA-treated wood can be used in the aquatic environment. The results of the submitted data for this requirement are listed below.

Table 22. Acute Toxicity of Arsenic Acid to Aquatic Plants

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
arsenic acid / 75.8%	Duckweed (Lemna gibba)	EC50 > 9.8 ppm NOEL < 9.8 ppm	- core study - static system - 14 day test duration	EPA 2002a (42290901)
arsenic acid / 75.8%	Green alga (Selenastrum capricornutum)	EC50 = 0.038 ppm NOEL =0.005 ppm	- core study - static system - 5 day test duration	EPA2002a (42290902)
arsenic acid / 75.8%	Freshwater diatom (*Navicula pelliculosa*)	EC50 = 1.4 ppm NOEL < 0.66 ppm	- supplemental study - static system - 5 day test duration	EPA2002a (42290903)
arsenic acid / 75.8%	Marine diatom (*Skeletonema costatum*)	EC50 = 0.0092 ppm NOEL < 0.007 ppm	- supplemental study - static system - 5 day test duration	EPA 2002a (42278801)
arsenic acid / 75.8%	Bluegreen alga (*Anabaena flos-aquae*)	EC50 > 2.4 ppm NOEL = 0.28 ppm	- supplemental study - static system - 5 day test duration	EPA2002a (42278802)

The results indicate that arsenic acid is moderately toxic to very highly toxic to aquatic plants. The guideline (123-2) is partially fulfilled. Studies which were classified as supplemental should be repeated as confirmatory data, particularly with the marine diatom, *Skeletonema costatum*, since it appears to be substantially more sensitive to arsenic acid than the other species tested.

Aquatic plant toxicity data have not been submitted for chromic acid. Two studies were identified in the ECOTOX database that evaluated the effects of chromic acid to aquatic plants. Results of these studies are summarized in the following table:

Table 23. Acute Toxicity of Chromic Acid to Aquatic Plants

Substance/ % Active Ingredient (AI)	Organism	Endpoints/Results (mg/L)	Comments	Reference
chromic acid / AI not available	Green alga (Selenastrum capricornutum)	EC50 = 0.2 ppm	- static system - 120 hour test duration - endpoint is population growth	EPA, 2002b, (ref #3690)
chromic acid, dipotassium salt / AI not available	Marine diatom (*Skeletonema costatum*)	EC50 = 14.7 ppm	- static system - 5 day test duration - endpoint is population growth	EPA, 2002b (ref #2233)

The results indicate that chromic acid is moderately to highly toxic to aquatic plants. The guideline (123-2) is not fulfilled since the studies were not submitted to the Agency and only two species of aquatic plants were evaluated. The full set of aquatic plant toxicity studies for chromic acid are now required as confirmatory data.

V. Ecological Risk Assessment

Risk assessment integrates the results of the exposure and ecotoxicity data to evaluate the likelihood of adverse ecological effects. One method of integrating the results of exposure and ecotoxicity data is called the quotient method. For this method, risk quotients (RQs) are calculated by dividing exposure estimates by ecotoxicity values, both acute and chronic:

RQ = EXPOSURE/TOXICITY

RQs are then compared to AD's levels of concern (LOCs). These LOCs are criteria used by OPP to indicate 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 this may be mitigated through restricted use classification; (3) **acute endangered species** - the potential for acute risk to endangered species is high, and regulatory action may be warranted, and (4) **chronic risk** - the potential for chronic risk is high, and regulatory action may be warranted. Currently, AD does not perform assessments for chronic risk to plants, acute or chronic risks to nontarget insects, or chronic risk from granular/bait formulations to mammalian or avian species.

The ecotoxicity test values (i.e., measurement endpoints) used in the acute and chronic risk quotients are derived from the results of required studies. Examples of ecotoxicity values derived from the results of 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 value is used as the ecotoxicity test value in assessing chronic effects. 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 endpoint is production of offspring or survival.

Risk presumptions, along with the corresponding RQs and LOCs are tabulated below.

Risk Presumptions for Terrestrial Animals

Risk Presumption	RQ	LOC
Birds and Wild Mammals		
Acute High Risk	EEC[1]/LC50 or LD50/sqft[2] or LD50/day[3]	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] $\frac{mg/ft^2}{LD50 * wt.\ of\ bird}$ [3] $\frac{mg\ of\ toxicant\ consumed/day}{LD50 * wt.\ of\ bird}$

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

Risk 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

Exposure to avian and mammalian species from the application of pesticides is usually estimated using the ELL-Fate Model (version 1.2) (EPA, 1999) or other similar terrestrial model. The ELL-Fate model was developed for agricultural pesticide uses, and calculates the decay of a chemical applied to foliar surfaces for single and multiple applications. The model takes into account the weight (lbs.) of active ingredient applied per acre, the number of applications per year and the half-life of the chemical in soil in order to estimate the chemical concentration in the treated foliage.

Based on review of the model parameters, the ELL-Fate model was determined to be inappropriate for the evaluation of risks to terrestrial species from exposure to CCA, since this compound is applied to processed lumber rather than agricultural crops. Birds, insects, mammals, and plants may be exposed to CCA through direct contact with treated lumber and contact with metals that have leached from treated lumber into the surrounding soil. Uptake of these metals into plants and insects, which are subsequently consumed by birds or mammals, can also result in exposure. Exposure and toxicity data for direct contact with treated lumber were not readily available; therefore, the terrestrial assessment was based on exposure of ecological receptors to CCA components in soil.

Risks to birds and mammals were evaluated using a simple terrestrial food web model. To estimate exposure for receptor species, it was assumed that metal leaching occurs from in-service CCA-treated wood into the surrounding soil. The leached metals in soil were then assumed to be taken up by terrestrial vegetation. Based on these conditions, the model assumed that avian and mammalian receptor species would be exposed to components of CCA through ingestion of vegetation and incidental ingestion of soil.

The EECs used in this model were based on metal concentrations observed in soils below seven CCA-treated decks ranging in age from 4 months to 15 years (Stilwell and Gorney, 1997). These data, summarized in the table below, are assumed to be representative of metal concentrations in soil that ecological receptors may be exposed to as the result of metal leaching from CCA-treated structures.

Table 24. Summary of Metal Concentrations (mg/kg) in Soil Under CCA-Treated Decks*

	Maximum Concentration	Mean Concentration
Arsenic	350	76
Chromium	154	43

*data from Stilwell and Gorny (1997).

The methods used to estimate the potential dose of CCA components to avian and mammalian species through the ingestion of vegetation and soil are provided in Appendix A. The results of the exposure assessment for avian and mammalian species have been provided in Tables A1 through A40. These tables also contain the estimated risk quotients (RQs) for avian and mammalian species.

When more than one toxicity reference value was available for a compound, toxicity data were selected for the risk assessment using the following hierarchy of sources: 1) core studies from the pesticide database, 2) supplemental studies from the pesticide database, 3) Health Effects Division's Hazard Identification Assessment Review Committee (HIRAC) reviews, 4) ECOTOX studies, and 5) studies from the open literature. In cases where there was more than one toxicity value for the same source, the lowest toxicity value was selected in order to be conservative.

1. Birds

As shown in Table A1, the risks to avian species from exposure to arsenic and chromium in soil as a result of leaching from CCA-treated structures do not appear to be significant. The acute RQs based on *maximum* soil concentrations were all less than the acute high risk level of concern of 0.5. The only exceedence for acute exposure was the **RQ for arsenic of 0.16 which was greater than the acute endangered species level of concern of 0.1**. The chronic RQs based on maximum soil concentrations only **exceeded the chronic risk level of concern for chromium VI with an RQ of 1.5**. Notably, the acute and chronic RQs that were based on *mean* soil concentrations did not exceed any of the respective levels of concern.

2. Mammals

The risk to mammalian species exposed to leached metals in soil from CCA-treated structures is low. The acute RQs based on *maximum* soil concentrations (Table A2) did not exceed the acute high risk level of concern of 0.5. However, **the RQ for arsenic exceeded the acute restricted use levels of concern**. The chronic RQs based on maximum soil concentrations **exceeded the chronic risk level of concern for both arsenic and chromium**, although only chromium had a significantly elevated RQ of 15.7. As shown in Table A2, the RQs based on *mean* soil concentrations did not exceed any of the acute levels of concern. **Chronic RQs based on mean soil concentrations exceeded the chronic risk level of concern for arsenic with an RQ of 1.2 and chromium VI with an RQ of 4.4**. The actual risk levels for mammalian species may be lower than the estimated RQs due to the conservative assumptions used in the model.

Conclusions of the Risk Assessment for Birds and Mammals:

The results indicate that the potential for adverse effects to birds and mammals from exposure to ***average*** concentrations of CCA components in soil is low. Average soil concentrations are considered more likely to represent the exposure level for mobile receptor species such as birds and mammals than maximum soil concentrations. It should be noted, however, that the risk assessment was only based on exposure to CCA components in soil. A quantitative assessment of the risks to birds and mammals from direct contact with CCA-treated lumber was not conducted due to the lack of exposure and toxicity data available. As a result, the potential risks from direct contact with CCA-treated wood were not evaluated. Additional uncertainties associated with the assessment are discussed in the Uncertainty section of the report.

3. Insects

Exposure to CCA components from treated lumber and the surrounding soil may result in adverse effects to insects such as honey bees. The potential risks to insects could not be quantitatively evaluated, however, since exposure data for insects were not readily available. Bee hives constructed from treated wood have been shown to cause toxicity to bees and result in residues of wood preservatives in honey (Kalnins and Detroy, 1984).

B. Exposure and Risk to Nontarget Freshwater and Marine/Estuarine Aquatic Organisms

Freshwater and marine/estuarine aquatic organisms could potentially be exposed to the components of CCA via residues leached from treated wood into the aquatic environment, either as runoff from land-based structures, such as utility poles, or from CCA-treated structures which stand directly in aquatic habitats, such as docks, piers, pilings, and bulkheads.

A risk assessment for copper as a component of inorganic arsenical wood preservatives is not included in this RED, as explained in the Executive Summary, above. However, copper is known to be highly toxic to aquatic organisms. While CCA-treated wood does leach copper into aquatic environments (Hobson, 2000), other copper-containing wood preservatives have been shown to leach greater amounts of copper (Townsend et al., 2001), and the copper-containing wood preservatives that do not contain arsenic are likely to be more toxic to marine organisms than CCA (Weis and Weis, 1996). Additionally, copper is found in aquatic habitats from a variety of other pesticidal uses, such as marine antifoulants and agricultural applications. The forthcoming copper RED will address all of the pesticidal uses of copper, and will provide a complete assessment of the risks they pose to aquatic organisms.

There are two major ways in which the components of CCA can reach aquatic habitats:

1. The metals can leach directly from CCA-treated structures installed in or above the water, such as docks, piers, pilings or bulkheads. There are numerous published studies which provide data on the leaching of CCA components from these types of structures [Brooks, 1997; Hobden, 2000; Townsend et al, 2001]; however the data are extremely variable because leaching rates of the metals depend on many factors, such as the salinity and pH of the water or leaching solution, age of the wood, type of wood, and retention time. Additionally, much of the data are reported as losses of metals from the wood as opposed to metal concentrations of the leachate. There is also a lack of validated methods for estimating the dispersion of the metals into the larger aquatic environment surrounding the treated structures, so developing a realistic estimation of risk from these structures is difficult.

Lee et al (1993) examined freshwater leaching of CCA components in a laboratory test, involving exposing 2.5 cm2 yellow pine cubes to a water bath for 14 days and measuring the leachate concentrations of the metals. The range of values for chromium and arsenic from these studies are summarized in the table below. Townsend et al (2001) performed leaching tests with CCA-treated wood using a variety of methods: deionized water, salt water, Synthetic Precipitation Leaching Procedure (SPLP),and Toxicity Characteristic Leaching Procedure (TCLP). The range of values from these tests is also reported in the table below.

Table 25. Maximum and Minimum Leaching Values from Lee et al, 1993 (as cited in Hobden, 2000 and Brooks, 1997) and Townsend et al, 2001.

	Lee et al., min (mg/L)	Lee et al., max (mg/L)	Townsend et al, min (mg/L)	Townsend et al, max (mg/L)
As	19	28	3.6	8.9
Cr	3	28	1.8	3.4

2. The metals can leach from CCA-treated structures into soil, and reach aquatic habitats via surface runoff.. Since the model typically used by OPP to determine EECs from surface runoff [e.g., the Generic Expected Environmental Concentrations (GENEEC) model (USEPA, 2001)] was developed for organic compounds, it is not appropriate to use it to model the runoff of metals leached from CCA-treated structures. Cooper (1990, as cited in Brooks, 1997) examined the concentration of metals in run-off water from CCA-treated poles and lumber exposed to natural rain; these values are provided in the table below.

Table 26. Maximum and Minimum Runoff Values from Cooper, 1990 (as cited in Brooks, 1997).

	Cooper, poles (mg/L)	Cooper, lumber (mg/L)
As	0.300	1.1 - 7.3
Cr	0.400	0.080 - 1.0

Attempting to estimate realistic EECs for arsenic and chromium leaching from CCA-treated wood is extremely difficult. Water-column concentrations of these metals in aquatic habitats would likely be much lower than the values obtained in the leaching studies discussed above, due to dispersion in the water body and partitioning into biota and sediment; however, there are no methods currently available to OPP to reliably quantify these effects and estimate realistic water-column EECs. Calculating risk quotients (RQ) using the published values as "worst-case" EECs is not useful, given the variability of the values and the high degree of uncertainty in extrapolating the results to "real-world" conditions. Therefore, a numerical risk assessment for aquatic organisms will not be performed at this time.

Published literature on effects to aquatic organisms from the use of CCA-treated wood has shown that the metals leached from it are taken up by biota and can have adverse effects (Weis and Weis, 1995). The most affected aquatic community appears to be the epibiotic , or "fouling," community, which grows in direct contact with treated structures (Weis and Weis, 1996), but effects on benthic community diversity were also observed, and were correlated with elevated levels of metals in sediments adjacent to treated wood (Weis and Weis, 1996). Copper leaches the most of the three metals (Weis and Weis, 2002), and it is known to be the most toxic of the three metals to aquatic organisms. The leaching of metals and the corresponding toxic effects are greatest when the wood is new (Weis et al, 1991).

C. Endangered Species

The results of the terrestrial risk assessment indicate that threatened and endangered birds may be adversely affected by arsenic residues leached from CCA-treated structures. Birds may also be adversely affected on a chronic basis by chromium leached from those structures. These estimates are based on maximum exposure estimates, however. Average estimated levels of arsenic and chromium are not likely to impact sensitive avian species. Maximum exposure estimates indicate that arsenic may cause adverse acute effects to endangered and threatened mammal species, and both arsenic and chromium may adversely affect mammals from chronic exposure. Average predicted exposure levels for both metals are still high enough to cause concern for chronic adverse effects to mammals.

Since it is not possible to quantify the risk to aquatic organisms from arsenic and chromium leached from treated wood, it is not known whether Endangered Species Levels of Concern are exceeded. Literature data do suggest that the components of CCA do cause adverse effects to aquatic organisms, particularly epibiotic and benthic organisms, including impacts on species richness and diversity as well as pathological and genotoxic effects and reduced growth in individual organisms (Weis and Weis, 2002). Based on these observed effects, and considering the known toxicity of the component metals, particularly copper, to aquatic organisms, it is possible that the component metals of CCA could adversely impact endangered or threatened aquatic organisms, particularly those in epibiotic or benthic communities which cannot move away from treated wood leachate.

The Agency has developed a program (the Endangered Species Protection Program) to identify pesticides whose use may result in adverse impacts to endangered and threatened species, and to implement mitigation measures that will eliminate the adverse impacts. At present, the program is being implemented on an interim basis as described in a Federal Register notice (54 FR 27984-28008, July 3, 1989), and is providing information to pesticide users to help them protect these species on a voluntary basis. As currently planned, the final program will call for label modifications referring to required limitations on pesticide uses, typically as depicted in county-specific bulletins or by other site-specific mechanisms as specified by state partners. A final program, which may be altered from the interim program, will be described in a future Federal Register notice. The Agency is not imposing label modifications through the RED process at this time. Rather, any requirements for product use modifications will occur in the future under the Endangered Species Protection Program.

D. Uncertainties in the Risk Assessment

Many of the toxicity values used in the assessment were from the ECOTOX database or the open literature rather than submitted studies that meet the pesticide guideline requirements. Toxicity studies conducted with components of CCA according to guideline requirements would remove some of the uncertainty in this risk assessment. Toxicity data were not identified for terrestrial plants, as a result the risks to terrestrial plants could not be evaluated

Also, many of the toxicity values used in the assessment were based on the evaluation of different metal compounds than the forms used to manufacture CCA (e.g., arsenic acid and chromic acid). These compounds may have different levels of toxicity for the receptor species, which would increase the level of uncertainty for those specific endpoints. For example, chromium III toxicity data were used to evaluate risks to avian receptors since chromium VI toxicity data were not available. Because chromium VI and chromium III often have different levels of toxicity, the risk levels estimated based on chromium III toxicity data are uncertain relative to exposure to chromium VI.

The toxic effects of the combined components of CCA are unknown. The toxicity of the three metals when received as a combined dose may be greater than any of their individual toxicities. Toxicity testing with CCA formulations would address the potential for additive or synergistic toxic effects from arsenic, chromium and copper in combination.

In general, the toxicity of metals to terrestrial receptors will depend on many site-specific factors such as the soil type (e.g., sand, loam, clay, etc.), soil pH, organic content of the soil, and soil moisture which will affect the availability of the metals to the receptors. Similarly, the toxicity of metals to aquatic receptors will depend on site-specific conditions such as surface water pH, sediment characteristics, and surface water flow patterns (e.g., tidal or river current) that will affect the metal concentrations and availability of the metals to aquatic receptors.

Another source of uncertainty regarding the toxicity data used in the assessment is that most of the toxicity studies were conducted in a laboratory environment and therefore may not accurately reflect field conditions experienced by ecological receptors. Toxicity studies also frequently use different terrestrial or aquatic species than those expected to occur in the natural environment. As a result, the toxicity endpoints may be inaccurate with regard to the species of interest.

There are several uncertainties associated with the exposure assessment for CCA in terrestrial and aquatic environments. The terrestrial exposure assessment assumed that birds and mammals were only exposed to CCA in soil since toxicity and exposure data were not readily available for direct contact with CCA-treated wood. The model also included many assumptions regarding the indicator species such as diet, food ingestion rate and home range size that may not accurately reflect actual conditions in the environment.

Finally, there are numerous uncertainties regarding the estimated environmental concentrations (EECs) used in the risk assessment. Concentrations of CCA components in soil and surface water may vary extensively depending on the type of exposure. The leaching of metals from treated wood is influenced by a variety of factors, such as the age and type of wood, the pH of the soil or water contacting the wood, the retention time used in the treatment process, and environmental conditions. EECs used in the assessment were based on monitoring and modeled data based on the leachate of metals from CCA-treated structures such as decks and telephone poles. The level of uncertainty associated with the EECs directly affects the uncertainty regarding the estimated risk levels.

REFERENCES
CCA Ecological Effects Bibliography

Albuquerque, R.M., and S. M. Cragg. 1995. Fouling Organisms as Indicators of the Environmental Impact of Marine Preservative-Treated Wood. Presented at The International Research Group on Wood Preservation 26th Annual Meeting, Helsingør, Denmark, 12-16 June, 1995.

Agency for Toxic Substances and Disease Registry (ATSDR). U.S. Public Health Service. 1997. Toxicological Profiles. CRC Press, Inc.

Baes, C.; Sharp, R.; Sjoreen, A. and R. Shor. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Oak Ridge National Laboratory, Doc. No. 5786.

Baldwin, W. J., E. A. Pasek, and P. D. Osborne. 1996. Sediment Toxicity Study of CCA-C-Treated Marine Piles. Forest Prod. J. 46(3): 42-50.

Besser, J., Dwyer, F., Ingersoll, C. and N. Wang. 2001. Early Life-Stage Toxicity of Copper to Endangered and Surrogate Fish Species. U.S. Environmental Protection Agency, Office of Water. EPA 600/R-01/051.

Beyer, W.; Conner, E. and S. Gerould. 1994. Estimates of Soil Ingestion by Wildlife. Journal of Wildlife Management, 58: 375-382.

Breslin, V.T., and L. Adler-Ivanbrook. 1998. Release of Copper, Chromium and Arsenic from CCA-C Treated Lumber in Estuaries. Estuarine, Coastal and Shelf Science 46(1): 111-125.

Brooks, K.M. 1996. Evaluating the Environmental Risks Associated with the Use of Chromated Copper Arsenate-Treated Wood Products in Aquatic Environments. Estuaries 19 (2A): 296-305.

Brooks, K.M. 1997. Literature Review and Assessment of the Environmental Risks Associated with the Use of CCA Treated Wood Products in Aquatic Environments. Prepared for the Western Wood Preservers Institute, Vancouver, WA. (www.wwpinstitute.org/mainpages/thesciencewoodinaquat.shtml)

Brooks, K. M. 2000. Assessment of the Environmental Effects Associated with Wooden Bridges Preserved with Creosote, Pentachlorophenol, or Chromated Copper Arsenate. U.S. Department of Agriculture, Forest Service, Forest Products Laboratory, Research Paper FPL-RP-587.

Brown, P.M., S. Lakhani, J. Moffat and F. Rodrigues. 1994. Summary of Results and Recommendations from the 1991-1992 Site Auditing Program of the Ontario CCA Wood Preservation Industry. (unpublished)

Campbell, S., K.A. Hoxter, and G.J. Smith. 1990. Arsenic Acid (76.1%) Encapsulated: An Acute Oral Toxicity Study with the Northern Bobwhite (Colinus virginianus). MRID 41719201. Conducted by Wildlife International for Chemical Manufacturers Association.

Chen, J., Malish, S., and McMathon, T. 2001. Inorganic arsenic – Report of the Hazard Identification Assessment Review Committee. Memorandum to Cook, N., Risk Assessment and Science Support Branch, Antimicrobial Division, USEPA.

Cox, C. 1991. Chromated Copper Arsenate. J. Pesticide Reform Spring 1991.

Efroymson, R.; Will, M.; Suter II, G. and A. Wooten. 1997. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1997 Revision. ES/ER/TM-85/R2.

Eisler, Ronald. 1994. Review of Arsenic Hazards to Plants and Animals with Emphasis on Fishery and Wildlife Resources. in Arsenic in the Environment, Part II: Human Health and Ecosystem Effects, edited by Jerome O. Nriagu. John Wiley, New York, NY.

Fletcher, D.W. 1987. 8-Day Dietary Study with Arsenic Acid as Desiccant L-10 in Mallard (*Anas platyrhynchos*) Ducklings. MRID 40409012. Conducted by Bio-Life Associates for Pennwalt Corporation.

Fletcher, D.W. 1987. 21-Day Acute Oral Toxicity Study with Arsenic Acid as Desiccant L-10 in Bobwhite Quail. MRID 40409013. Conducted by Bio-Life Associates for Pennwalt Corporation.

Hobden, B. R. 2000. Proposed Acceptability for Continued Registration Document (PACR) for Chromated Copper Arsenate. PMRA Environmental Assessment Component of the NAFTA Joint Re-Evaluation of Chromated Copper Arsenate. April 28, revision.

Hoxter, K.A., and M. Jaber. 1987. Arsenic Acid: An Acute Contact Toxicity Study with the Honey Bee (*Apis mellifera*). MRID 40351301. Conducted by Wildlife International for Pennwalt Corporation.

Hoxter, K.A. 1990. Eight-Day Dietary Toxicity LC50 Test with Mallard Duck, *Anas platyrhynchos.* MRID 41621102. Conducted by Wildlife International for Chemical Manufacturers Association.

Hoxter, K.A. 1990. Chromic Acid: Avian Acute Oral Toxicity Using *Colinus virginianus*. MRID 41621104. Conducted by Wildlife International for Chemical Manufacturers Association..

Hoxter, K.A. 1990. Eight Day Dietary Toxicity LC50 Test with Bobwhite Quail (*Colinus virginianus*). MRID 41621101. Conducted by Wildlife International for Chemical Manufacturers Association.

Imlay, Mark J., and Parley V. Winger. 1983. Toxicity of Copper to Gastropoda with Notes on the Relation to the Apple Snail: A Review. Malacol. Rev. 16: 11-15.

Kalnins, M. A., and B. F. Detroy. 1984. Effect of Wood Preservative Treatment of Beehives on Honey Bees and Hive Products. J. Agric. Food Chem. 32: 1176-1180.

LeLievre, M. 1990. Arsenic Acid: 96-hour Static Acute Toxicity Test with Rainbow Trout, *Oncorhynchus mykiss*. MRID 41620003. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Arsenic Acid: 96-hour Static Acute Toxicity Test with Rainbow Trout, *Oncorhynchus mykiss*. MRID 41620003. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Arsenic Acid: 96-hour Static Acute Toxicity Test with Sheepshead Minnow, *Cyprinodon variegatus*. MRID 41620004. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Arsenic Acid: Static Acute Toxicity Test Using *Daphnia magna*. MRID 41620001. Conducted by Springborn Laboratories, Inc. for Chemical Manufacturers Association.

LeLievre, M. 1990. Arsenic Acid: Static Acute Toxicity Test with Mysid Shrimp, *Mysidopsis bahia*. MRID 41620002. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Chromic Acid: 96-hour Static Acute Toxicity Using Bluegill Sunfish (*Lepomis macrochirus*). MRID 41658401. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Chromic Acid: 96-hour Static Acute Toxicity Test Using Rainbow Trout, *Oncorhynchus mykiss*. MRID 41621105. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Chromic Acid: Static 48-hour Acute Toxicity Using *Daphnia magna*. MRID 41621103. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Chromic Acid: 96-hour Static Acute Toxicity Test Using Sheepshead Minnow (*Cyprinodon variegatus*). MRID 41703601. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

LeLievre, M. 1990. Chromic Acid: Static Acute Toxicity Test Using Mysid shrimp (*Mysidopsis bahia)*. MRID 41703602. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

Long, R.D., J. Foster, K.A. Hoxter and G.J. Smith. 1990. Arsenic Acid: A Dietary LC50 Study with the Northern Bobwhite (*Colinus virginianus*). MRID 41719202. Conducted by Wildlife International for Chemical Manufacturers Association.

Machado, M.W. 1991. Arsenic Acid: Acute Toxicity to Bluegill Sunfish (*Lepomis macrochirus*) Under Flow-Through Conditions. MRID 41950601. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

Machado, M.W. 1991. Arsenic Acid: Toxicity Test with Fathead Minnow (*Pimephales promelas*) Embryos and Larvae. MRID 41802201. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

Machado, M.W. 1991. Chromic Acid: Toxicity Test with Fathead Minnow (*Pimephales promelas*) Embryos and Larvae. MRID 41974901. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association

McMahon, T. and Chen J., 2001. FIFRA Scientific Advisory Panel Background Document – Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium. US Environmental Protection Agency, Office of Pesticide Programs, Antimicrobials Division.

McNamara, P.C. 1991. Arsenic Acid: Chronic Toxicity to Daphnids (*Daphnia magna*) Under Flow-Through Conditions. MRID 42001601. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

McNamara, P.C. 1991. Chronic Acid: Chronic Toxicity to Daphnids (*Daphnia magna*) Under Flow-Through Conditions. MRID 41881501. Conducted by Springborn Laboratories, Inc., for Chemical Manufacturers Association.

Playle, R.C., R.W. Gensemer, and D. G. Dixon. 1992. Copper Accumulation on Gills of Fathead Minnows: Influence of Water Hardness, Complexation and pH of the Gill Micro-Environment. Env. Toxicol. Chem. 11: 381-391.

R. Faust. 1992. Toxicity Summary for Copper. Oak Ridge National Laboratory, Health and Safety Research Division. Oak Ridge, TN.

Salanki, J., and K. V.-Balogh. 1989. Physiological Background for Using Freshwater Mussels in Monitoring Copper and Lead Pollution. Hydrobiol. 188/189: 445-454.

Sample, B.E., Opresko, D.M. and G.W. Suter II. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. U.S. Department of Energy, Office of Environmental Management, Oak Ridge. ES/ER/TM-86/R3.

Sheppard, S.C. 1992. Summary of Phytotoxic Levels of Soil Arsenic. Water, Air and Soil Pollution 64: 539-550.

Solo-Gabriele, H., and T. Townsend. 1999. Disposal Practices and Management Alternatives for CCA-Treated Wood Waste. Waste Management Res. 17: 378-389.

Stilwell, D.E. and Gorny, K.D. 1997. Contamination of Soil with Copper, Chromium, and Arsenic Under Decks Built from Pressure Treated Wood. Bulletin of Environmental Contamination and Toxicology, 58: 22-29.

Sunila, I., and R. Lindstom. 1985. Survival, Growth and Shell Deformities of Copper- and Cadmium-Exposed Mussels (Mytilus edulis L.) in Brackish Water. Estuarine, Coastal and Shelf Science 21: 555-565.

Suprenant, D.C. 1987. Acute Toxicity of Arsenic Acid (Desiccant L-10) to Bluegill (Lepomis macrochirus). MRID 40409014. Conducted by Springborn Life Sciences, Inc., for Pennwalt Corporation.

Tolaymat, T. M., T.G. Townsend, and H. Solo-Gabriele. 2000. Chromated Copper Aresnate-Treated Wood in Recovered Wood. Env. Eng. Sci 17(1): 19-28.

Townsend, T., K. Stook, M. Ward, and H. Solo-Gabriele. 2001. Leaching and Toxicity of CCA-Treated and Alternative-Treated Wood Products (DRAFT). Florida Center for Solid and Hazardous Waste Management, State University System of Florida, Gainesville, FL.

U.S. Environmental Protection Agency, ELL-Fate Model (Version 1.2) 1999

U.S. Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund. Volume II: Environmental Evaluation Manual. Interim Final. EPA/540/1-89/001. March, 1989.

U.S. Environmental Protection Agency (USEPA). 1993. Wildlife Exposure Factors Handbook. Volume I. Office of Research and Development, Washington, D.C. EPA/630/R-93/187a.

U.S. Environmental Protection Agency (USEPA). 2001. Generic Estimated Environmental Concentration Model (GENEEC) Version 2.0. Environmental Fate and Effects Division, Office of Pesticide Programs

U.S. Environmental Protection Agency. 2002a. Office of Prevention, Pesticides and Toxic Substances/Office of Pesticide Programs. Pesticide Ecotoxicity Database.

U.S. Environmental Protection Agency. 2002b. ECOTOX User Guide: ECOTOXicology Database System. Version 2.0. Available: http:/www.epa.gov/ecotox, 8/1/2002, 8/14,2002

Weis, J., and P. Weis. 1995. Effects of Chromated Copper Arsenate (CCA) Pressure-Treated Wood in the Aquatic Environment. Ambio 24(5): 269-274.

Weis, J.S., and P. Weis. 1996. The Effects of Using Wood Treated with Chromated Copper Arsenate in Shallow-Water Environments: A Review. Estuaries 19(2A): 306-310.

Weis, J.S. and P. Weis. 2002. Contamination of Saltmarsh Sediments and Biota by CCA Treated Wood Walkways. Mar. Pol. Bull 44: 504-10.

Weis, J., P. Weis, and T. Proctor. 1998. The Extent of Benthic Impacts of CCA-Treated Wood Structures in Atlantic Coast Estuaries. Arch. Environ. Contam. Toxicol. 34: 313-322.

*Weis, P., J.S. Weis, and L.M. Coohill. 1989. Biological Impact of Wood Treated with Chromated Copper Arsenate on Selected Estuarin
e Organisms. In Pesticides in Terrestrial and Aquatic Environments: Proceedings of a National Research Conference May 11-12, 1989, edited by Diana L. Weigmann. Virginia Polytechnic Institute and State University, Blacksburg, VA.*

Weis, P., J.S. Weis, and L.M. Coohill. 1991. Toxicity to Estuarine Organisms of Leachates from Chromated Copper Arsenate Treated Wood. Arch. Environ. Contam. Toxicol. 20: 188-124.

Weis, R., J.S. Weis, A. Greenberg, and T.J. Nosker. 1992. Toxicity of Construction Materials in the Marine Environment: A Comparison of Chromated-Copper-Arsenate-Treated Wood and Recycled Plastic. Arch. Environ. Contam. Toxicol. 22(1): 99-106.

Weis, P., J.S. Weis, and J. Couch. 1993. Histopathology and Bioaccumulation in Oysters Crassostrea virginica Living on Wood Prserved with Chromated Copper Arsenate. Dis. Aquat. Org. 17: 41-46.

Wendt, P.H., R.F. Van Dolah, M.Y. Bobo, T.D. Mathews, and M.V. Levisen. 1996. Wood Preservative Leachates from Docks in and Estuarine Environment. Arch Environ Contam Toxicol 31(1): 24-37.

Whiticar, D. and D.E. Konasewich. 1992. Lower Mainland Region Antisapstain Facilities: Assessment of Operational Practices and Environmental Discharge Volume Two. Prepared for B.C. Ministry of Environment, Lands and Parks, Lower Mainland Regional Office.

APPENDIX A

CCA: Arsenic, Chromium and Copper Exposure Assessment for Avian and Mammalian Species

The meadow vole (Microtus pennsylvanicus) was selected as the mammalian receptor species for evaluating potential effects of the components of CCA to mammals. The meadow vole is primarily herbivorous and is widely distributed in the United States (EPA, 1993). The northern bobwhite (Colinus virginianus) was selected as the avian receptor species for evaluating potential effects of the components of CCA to avian species. The northern bobwhite feeds mainly on seeds and low-lying vegetation. The bobwhite range includes the eastern and central U.S. as well as portions of the Rocky Mountains and the southwest. The mouse and bobwhite were selected because a relatively large proportion of the diet of both species is comprised of vegetation and there is an extensive amount of toxicity data available for these species, particularly for the bobwhite.

The following discussion presents the methods used to calculate the potential ingestion of chemicals by the mouse and bobwhite via the ingestion of food (i.e., terrestrial plants) and surface soil. The equations presented below were derived based on equations presented by EPA (1989). The following equation was used to calculate the dose of chemicals that a mouse or bobwhite would be expected to obtain from the ingestion of terrestrial plants: