

Summary of Available Health Information Regarding CCA-Treated Wood Products

Dr. Helena Solo-Gabriele, University of Miami
Dr. Richard P. Maas, University of North Carolina-Asheville

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This summary provides background information concerning: 1) metals contained in CCA-treated wood, 2) general information concerning the health affects of chromium, copper and arsenic, and 3) describes a series of U.S. studies used to evaluate children's risk associated with CCA-treated playground equipment. The summary closes with a discussion and a series of recommendations.

1. Metals Contained In CCA-Treated Wood

Wood treated with chromated copper arsenate (CCA) contains high concentrations of chromium, copper, and arsenic. These concentrations typically vary by a factor of 10 depending upon the retention level of the wood. "Retention" is a term used by the industry to describe the amount of CCA chemical contained within the wood. Retentions will vary depending upon the intended use of the wood. Lower retention levels are used for wood in less aggressive environments such as for above ground applications (e.g. the pickets on a fence) whereas higher retention level wood are used for industrial applications (e.g. utility poles) and for wood submerged in marine environments (e.g. support structure for docks). The retention level of CCA-treated wood typically varies from 4 to 40 kilograms of CCA chemical on an oxide basis per cubic meter of wood where the oxides are As_2O_5 , CrO_3 , and CuO (AWPA 2003). The resultant metals concentrations within the wood product can typically vary from thousands to tens of thousands of mg of metal per kilogram of wood (Table 1). At the high end, the metals from CCA represent collectively 5% of the weight of the wood.

Metal	Low Retention Level Wood (at 4 kg/m ³)	High Retention Level Wood (40 kg/m ³)
Chromium	1900 mg/kg	19,000 mg/kg
Copper	1200 mg/kg	12,000 mg/kg
Arsenic	1700 mg/kg	17,000 mg/kg

Table 1: Concentrations of chromium, copper, and arsenic in CCA-treated wood at 4 kg/m³ and 40 kg/m³ retention levels

2. General Information Concerning Health Effects of Chromium, Copper and Arsenic

Health effects from the metals contained within CCA-treated wood are a function of the chemical form (species) of the metal, exposure dose, and exposure route. The form or chemical species of the metal impacts its toxicity. Typically acute toxicity is induced

at high doses. Low doses for extended periods of time can also induce adverse health effects including cancer. The exposure route will depict how the absorption of metals can occur.

2.a Speciation

The form of the metal or species is especially significant with respect to arsenic and chromium, due to their different toxicities. Copper is typically found in the general environment within the +2 valence state only. The toxicity of chromium is largely a function of the oxidation state of the chromium within a corresponding molecule. The two most relevant environmental oxidation states of chromium are Cr(III) and Cr(VI), each of these characterized by different toxicities (James et al., 1995; Wang and Vipulanandan, 2001). For example, Katz and Salem 1993 report that hexavalent chromium, Cr(VI), is 10 to 100 times more toxic than trivalent chromium, Cr(III), when ingested orally. Also of relevance is that Cr(VI) is more mobile in the environment than Cr(III) due to its higher solubility in water. Chromium in the CCA solution is present primarily as Cr(VI) prior to contact of the solution with the wood. Once the CCA chemical is impregnated into the wood it reacts with the wood fibers and transforms over time to Cr(III) (Cooper 1995; Dahlgren and Hartford, 1972; Greaves, 1974; Bull et al., 2000). This conversion is a function of time and temperature (Cooper et al., 1997) and the predominant form of chromium available to the consumer is Cr(III) assuming that the wood was properly treated. It is important to note, however, that under strong oxidizing conditions, Cr(III) can be converted to Cr(VI). This conversion of Cr(III) back to Cr(VI) can occur through the use of bleaches and brighteners that have been designed for use on wood products (Taylor et al. 1998; Maas et al. 2002). In soil Cr(III) can be oxidized to Cr(VI) in the presence of oxidized manganese (Kim et al., 2002, James, 1996, Rai et al, 1989, Eary and Rai, 1987).

Arsenic species are typically separated into two general categories: inorganic arsenic and organic arsenic. Inorganic arsenicals are considered to be very toxic and referenced typically by their valence state as As(V) and As(III) with As(III) more toxic and mobile than As(V). The form of arsenic within treated wood is As(V). Organic arsenicals include monomethylarsonic acid (MMAA) and dimethylarsinic acid (DMAA) which can be formed by the methylation of inorganic arsenic by microbes. MMAA and DMAA are considered to be less toxic to humans than As(III) and As(V). Other organic forms of arsenic which are characterized by very low toxicities include arsenobetaine (AsB) and its reduced form, arsenocholine (AsC) (both found in marine invertebrates, shellfish, and fish) and trimethylarsine oxide (TMAO) also found in marine samples (Francesconi and Edmonds, 1994). The forms of arsenic found in seafood, AsB and AsC, are not metabolized by humans and are therefore excreted as AsB and AsC. Interferences from seafood ingestion can thus be reduced by measuring the species of arsenic in urine samples. As a result, measurement of the inorganic arsenic species (inorganic As(III) and As(V)) and its metabolites (MAA and DAA) is recommended to evaluate non-dietary exposures to arsenic (AOEH 1991).

A very toxic and gaseous form of arsenic, arsine gas (AsH_3) can be formed under extreme reducing conditions. The toxicity of arsenic species therefore varies as follows: $\text{AsH}_3 > \text{As(III)} > \text{As(V)} > \text{MMAA} > \text{DMAA} > \text{AsB, AsC, TMAO}$. In order to provide a sense for the relative toxicity between the different arsenic species, data from scientific studies are quoted in terms of LD_{50} where LD_{50} represents the lethal dose at which 50% of the test population dies. The lower the LD_{50} the more toxic the compound. LD_{50} values quoted by Seiler et al. (1994) for humans are 1.43, 50 and 500 mg/kg for As(III), MMAA, and DMAA, respectively. Naqvi et al. (1994) report an LD_{50} of up to 10,000 mg/kg in mice for AsB. Although, As(III) has been found to be considerably much more toxic than As(V) in most animal studies (Yamauchi and Fowler, 1994), other studies indicate that this may not hold true for humans in all cases (Squibb and Fowler, 1983) and both inorganic species (As(III) and As(V)) should be regarded as being very toxic. Furthermore, the general supposition that the inorganic species are more toxic should be considered in light of the fact that the organic species can be degraded to the more toxic inorganic forms, depending upon the environmental conditions. Also, recent findings show that the organic species can be extremely toxic to humans, in particular organic complexes of arsenic with arsenic in the +3 valence (Del Razo et al., 2001). Arsenic in the CCA solution and in the treated wood product is predominantly as As(V).

2.b Health Effects From Metal Exposures

Possible health effects from contact with CCA and CCA-treated wood can be separated into two categories: acute health effects and health effects from chronic exposures. Copper, chromium, and arsenic are capable of acute human health effects when doses are high enough. Acute effects of copper include gastro-intestinal upset (IRIS 2003) and possible hepatic and renal affects (Read 2003). Copper is not considered a carcinogen and is considered to be much less toxic than chromium and arsenic. Short term health effects from high level chromium exposures include skin irritation. Long term health effects include damage to liver, kidney, circulatory and nerve tissues (US EPA 2003). Chromium as Cr(VI) is a carcinogen, in particular causing lung cancer when absorbed via inhalation (Read 2003). Acute arsenic exposures of 0.05 mg/kg/day by ingestion have caused vomiting, diarrhea, abdominal pain and gastrointestinal hemorrhage, changes in liver and renal function, hypotension, tachycardia, pulmonary oedema and difficulty breathing (Read 2003). Acute health effects can occur during occupational exposures in the production of CCA-treated wood if proper safety equipment is not utilized and hygienic practices are not followed (Harper et al. 1990; Gallop and Glass 1979; Garrod et al. 1999; Gilbert et al. 1990)

Typically exposures to the wood product after treatment are much lower than possible occupational exposures during treatment. The general consensus in the literature is that the low level exposures that most of the general population will experience from contact with CCA-treated wood are unlikely to result in acute health effects (Read 2003; West 2004; Bidot et al. 2002). However, some individual cases of acute health effects have been reported in the literature for individuals exposed to treated wood through burning (Geschke et al. 1996; Peters et al. 1984; Peters et al. 1986). Lawsuits have been settled in cases where individuals experienced acute health effects when they were

exposed to CCA chemicals during the building of a pier, during the construction of picnic tables, during the construction of a swimming dock, and from exposures from splinters (Sharp and Walker 2001). Furthermore, there have been reports in the media concerning the possible acute health effects from an individual in contact with CCA contaminated mulch. These cases thereby contradict the general consensus in the literature indicating a low likelihood of acute health effects.

Chronic low level doses of arsenic can cause skin, lung, liver and bladder cancers, cardiovascular disease, peripheral vascular disease, respiratory disease, skin lesions, plus gastrointestinal, hepatic, hematological, reproductive, and neurological effects (NRC 2001; Read 2003; AOEH 1991; Simeonova and Luster 2004; Yoshida et al. 2004). Recent studies also show an association between chronic inorganic arsenic exposures and diabetes (Walton et al. 2004). Dermal effects such as hyperpigmentation and hyperkeratosis are characteristic of long-term exposure (Read 2003), and can occur through ingestion of arsenic or through skin contact (Bernstam et al. 2002). The correlation with cancer is believed to be due to inorganic arsenic and its role in inhibiting DNA repair; therefore inorganic arsenic acts synergistically with other carcinogens such as UV light to result in mutagenic effects (Basu et al. 2001; Rossman et al. 2004). Some also believe that arsenic facilitates the transition from benign to malignant tumors in humans (Basu et al. 2001). Moore et al., (2002) found that bladder tumors in patients with higher levels of arsenic exposure showed higher levels of chromosomal instability, suggesting that bladder tumors from arsenic-exposed patients behave more aggressively than tumors from unexposed patients. Furthermore, evidence indicates that the arsenic may be a transplacental carcinogen, suggesting that the carcinogenetic effects may occur in the offspring of individuals exposed to arsenic (Waalkes et al. 2004).

The low level arsenic dose that would induce cancer is subject of considerable debate (Schoen et al. 2004) given that the mechanisms of arsenic carcinogenesis has not been well defined, although considerable progress has been made in defining these mechanisms (Thomas et al. 2004; Luster and Simeonova 2004; Kuroda et al. 2004; Wanibuchi et al. 2004; Yamanaka et al. 2004). The evaluation of low dose arsenic exposures is further complicated since the applicability of animal models for arsenic carcinogenesis has been questioned due to possible differences in metabolism (SAP, 2001; Lai et al. 2004). Epidemiological studies are used in many cases to define doses that would induce cancer (Read 2003).

There is some evidence to suggest that childhood exposures to some chemicals result in higher cancer risks. It is not known to what extent the susceptibility of children differs for arsenic due to a lack of relevant data and uncertainty about the mechanism of action (Read 2003).

Estimated Daily Intake

Estimated daily intake from food is 0.04 mg if the diet does not include fish and 20.2 mg if the diet includes fish. Average daily intake from water is estimated at 15 ug of arsenic. Average daily inorganic arsenic intake in the U.S. is 0.1 – 2.6 ug/kg body weight

which equates to 2 to 46 ug for a young child. The estimated daily intake from air is 0.2 ug (Read 2003). Additional sources of arsenic include smoking with 12 to 42 ug of arsenic per cigarette (Read 2003). Arsenic concentrations in the urine of the general population are usually below 10 ug/L in European countries, slightly higher in the U.S. and around 50 ug/L in Japan. (AOEH 1991). For populations drinking from contaminated wells one study of 232 people indicated that a daily arsenic intake of 126 ug resulted in urinary arsenic concentrations between 40 and 80 ug/L; intake of 265 ug resulted in concentrations between 80 and 200 ug/L; and intake of 386 ug resulted in concentrations above 200 ug/L. According to another study of 14 individuals, urinary arsenic concentrations of 270 ug/L results from a daily intake of 710 ug of arsenic (Read 2003).

2.c Exposure Routes of Metals From CCA-Treated Wood

There are 3 population groups at potential health risk from CCA-treated wood: workers at wood treatment plants, workers who process CCA-treated wood into various end uses, and the general population who use or come in contact with the end product. Absorption of metals by any of these three groups can possibly occur via pulmonary, dermal and gastrointestinal routes and in some cases through cuts in the skin.

Exposure is a function of many different factors and typically represents the sum of the contaminant absorbed from ingestion, inhalation, and skin absorption. In the case of CCA-treated wood exposure can occur at the wood treatment plant by workers who are not properly protected. Occupational exposures can occur from contact with the CCA solution and from contact with the treated wood product. Industrial uses of treated wood may result in exposures to personnel in contact with the industrial products and in indirect exposures to the general public from environmental contamination. Cutting and handling the wood during the construction of a treated wood structure may result in the exposure to treated wood through inhalation of sawdust. The general population can be exposed through direct contact with residues on the surface of the wood (e.g. dislodgeable metals) and from contact with the environment that has been impacted by the chemicals from treated wood. In the case of CCA the chemicals are known to leach from existing structures via runoff to surrounding soil (Stilwell and Gorny 1997; Townsend et al. 2003a) with some possible impacts to groundwater and plants (Larsen et al. 1992). Losses of the CCA chemical also occur when submerged in aquatic environments (Weis and Weis 1995; Warner and Solomon 1990; Lebow et al. 1999). Direct exposures may also occur to solid waste operators during disposal of the wood through direct contact with the wood and through the inhalation of contaminated sawdust. In some cases, CCA-treated wood is inadvertently recycled into mulch products (Townsend et al. 2003b). In this case, landscapers may be at an elevated risk of arsenic exposures through direct contact with contaminated mulch and inhalation of small wood fibers. Wood that is burned during disposal may result in possible exposure through inhalation if proper air pollution control equipment is not used. Exposure may also occur through contact with metals within the ash (Figure 1).

Many recent exposure assessments focus on evaluating the exposures to children from CCA-treated playground equipment, due to increased public awareness of the issue (Fields 2001; Gray and Houlihan 2002). Children are considered to be at elevated risk due to their increased hand-to-mouth activity, in particular between the ages of 2 and 6 years. Many studies have estimated the exposure from the ingestion of arsenic from contact with the surface residues on treated wood, dermal absorption from the surface residues, ingestion of contaminated soil, and dermal absorption of metals from contaminated soil. A few studies have evaluated exposures to chromium and inhalation routes. These studies find that the risks from chromium and inhalation are small relative to risks from arsenic via hand-to-mouth ingestion and dermal sorption. No studies however include exposures from the direct mouthing of wood and contaminated toys and indirect hand-to-mouth transfer from residues on food, toys, and clothing. Exposure from splinters has not been evaluated within the literature.

3. Evaluating Children's Health Risks to CCA-Treated Playground Equipment

A risk assessment is essentially a two-step process: an exposure estimate which quantifies the amount absorbed per unit time and a response to that exposure within a given probability. The benchmark for acceptable probabilities of health risk depends upon the health endpoint. In the case of cancer, acceptable levels of risk is typically 1 per million of exposed population. Usually if the risk is less than 1 per million the exposure is considered acceptable. Some may argue that 1 per million is too conservative and that allowable risks should be greater than 1 per million.

3.a Computation of Risk

Risk assessments first require the computation of an absorbed dose or an exposure level (e.g. the amount of contaminant absorbed from ingestion, inhalation, and skin absorption). The absorbed dose is a function of many different factors including the duration of the exposure, bioavailability of the contaminant, the exposure route, etc.. Since there are currently no direct measures of exposures by children in contact with CCA-treated playground equipment (e.g. no measurements of urine arsenic concentrations in exposed and unexposed populations), exposure assessments require the use of a model from which estimates of exposure are derived from estimates of the amount of chemical on a child's hand during play, the child's hand-to-mouth behavior, estimates for the amount of chemical transferred to the mouth during a mouthing episode, and an estimate of the bioavailability of the chemical once ingested. Exposures can be computed for average daily doses or lifetime average daily doses (LADD). The exposure equation typically utilized for measuring LADDs is as follows (US CPSC 2003):

$$LADD\left(\frac{\mu g}{kg \cdot day}\right) \equiv \frac{C\left(\frac{\mu g}{handload}\right) \times HT\left(\frac{handload}{day}\right) \times EF\left(\frac{days}{year}\right) \times ED(years) \times B}{BW(kg) \times LT(days)}$$

Equation 1

where,

LADD = lifetime average daily dose

C = Amount of chemical residue on hands during play on a CCA-treated wood playground structure.

HT = handload transfers to the mouth,

EF = exposure frequency

ED = exposure duration

B = relative bioavailability. The bioavailability of arsenic from wood surface residue divided by the bioavailability of arsenic in drinking water.

BW = body weight

LT = lifetime.

An estimation of risk or the probability of a certain health effect is then computed from the calculated exposure as per a typical equation below (US CPSC 2003):

$$R(\text{cancerrisk}) \equiv LADD \left(\frac{\mu\text{g}}{\text{kg} \cdot \text{day}} \right) \times Q \left(\frac{\mu\text{g}}{\text{kg} \cdot \text{day}} \right)^{-1}$$

Equation 2

where,

Q = unit cancer risk (also called cancer slope factor or cancer potency), which is the numerical representation of cancer risk per unit of daily exposure.

The manner in which the unit cancer risk is computed for low doses is the subject of considerable debate, since data concerning cancer risk is usually available based upon higher exposure rates. As a result linear extrapolation has been used to predict cancer risk at low levels of arsenic intake from the risks at moderate to high intakes, in the absence of information available for the health effects associated with low but long term doses of exposure.

3.b Risk Assessments for Children on CCA-treated Playgrounds Conducted in the U.S.

Many risk assessments have been completed or are in progress worldwide to evaluate impacts of CCA-treated playgrounds to children. These risk assessments are in progress within various countries outside the U.S. and include work conducted by the Pest Management Regulatory Agency (PMRA) of Health Canada, the European Commission (EC) and the Australian Pesticides and Veterinary Medicines Authority (APVMA). The section below will focus on risk assessments conducted by U.S. agencies. A set of 7 risk assessments was evaluated in detail with key factors summarized in Table 2. There is one other risk assessment completed in 2001 by a company called Exponent. Exponent was contacted in an effort to receive the risk assessment, but the risk assessment was not received. The 7 studies evaluated in detail include the following:

- California Department of Health Services 1987
- Environmental Working Group 2001
- Gradient 2001
- Maas et al. 2003
- Roberts and Ochoa 2001
- U.S. Consumer Products and Safety Commission 2003
- U.S. Environmental Protection Agency. (Zartarian et al. 2003 and Dang et. al. 2003.)

Prior risk assessments were conducted by the US CPSC (USCPSC 1990) and through the California Department of Health Services (CDHS 1984). The more recent risk assessments conducted by these agencies are assumed to supercede the older risk assessments. Additional risk assessments evaluated in less detail include the work completed by HSWMR 2000a,b. The approach taken by HSWMR 2000a,b was to compute soil and surface arsenic residue quantities that would result in an insignificant health risk. According to HSWMR, a 1 per million cancer health risk would occur for a person exposed both as a child and as an adult to wood characterized by dislodgeable arsenic residues of 40 ug/100 cm². The corresponding soil concentration for a 1 per million risk was estimated at 90 mg/kg. If the person is exposed only as a child then the 1 per million risk level according to HSWMR would occur for dislodgeable arsenic concentrations of 420 ug/100 cm² and for soil concentrations of 260 mg/kg. Due to the different manner in which computations were performed, the HSWMR 2000 study was not included in Table 3.

All of the risk assessments listed above have focused on arsenic. Some have also included risks from chromium exposure (Dang et al. 2003 and others). For these studies, risks due to the exposure to chromium were found to be lower than the risks due to the exposure of arsenic. For comparative purposes only the risks from the arsenic component from CCA-treated wood are listed in Table 3.

All of the studies listed above were deterministic models with the exception of the EWG 2001, Gradient 2001, and US EPA 2003 assessments. Deterministic implies that the best estimate for each of the factors needed for equations 1 and 2 were determined and then inserted into the equations to estimate risk. This differs from a probabilistic approach in which a distribution is assigned to each factor that is based upon available data. Therefore in a probabilistic approach there is not just 1 number assigned to each factor but rather ranges of numbers are assigned to each factor within a given probability. In order compute risk these probability distributions are considered in the computation of the LADDs and cancer risks, R. The LADDs and Rs from probabilistic models are given in terms of a probability distribution. For comparative purposes, the LADDs and Rs for the mean of the populations evaluated are summarized in Table 3 for the probabilistic models. Of interest is that a Monte Carlo simulation approach was utilized by the US EPA 2003 and EWG 2001. The Monte Carlo method is based upon a the simulation of a large number of children with factors that influence exposure chosen randomly from a probability distribution that represents the available data (CSEP 1995).

Another common feature among all of the risk assessments is that they utilized cancer as the health effect endpoint for evaluation. Some risk assessments also evaluated non-cancer end-points (e.g. Gradient 2001; Roberts and Ochoa 2001), however risk associated with the non-cancer end-points for these evaluations were less than the risks associated with the cancer end-points. Some risk assessments evaluated the impacts from decks plus playgrounds (Zartarian et al. 2003; Dang et al. 2003; Gradient 2001; Maas 2003). All evaluated the impacts of playgrounds to children and for comparative purposes only the impacts from playground equipment are presented in Table 2.

All risk assessments considered the ingestion of arsenic from hand-to-mouth contact of surface residues. Some (EWG 2001; Zartarian 2003; Gradient 2001; Roberts and Ochoa 2001) also considered dermal sorption from surface residues and ingestion from hand-to-mouth contact with contaminated soils (EWG 2001; Zartarian 2003; Gradient 2001) and dermal sorption from contaminated soils (EWG 2001; Zartarian 2003; Gradient 200). Gradient 2001 and HSWMR also considered exposure through the inhalation of arsenic-contaminated dust from CCA-treated wood. In all cases, the most significant route of children's exposure was through ingestion of surface residues from hand-to-mouth contact. Dermal sorption and exposures to contaminated soils were found to be secondary followed by inhalation routes. For comparative purposes only the factors used to evaluate ingestion from direct contact with surface residues are included in Table 3. The risks reported at the bottom of that table are based upon the risks from all exposure routes, but from childhood playground exposure only. Exposures from decks are not included.

3.c Brief Summary of Each Risk Assessment

California Department of Health Services 1987

The California Department of Health Services (1987) conducted a study in which 5 adult volunteers rubbed their hands on CCA-treated playground wood for 5 minutes. The study estimated that a child could absorb 24 to 630 ug of arsenic per visit based on adult hand wipe data. Only ingestions from direct contact with surface residues are considered in their analysis. The CDHS 1987 did not utilize cancer potency factors in their risk assessment. Rather they compared estimated doses with the amount of arsenic ingested if a child were to drink water containing 50 ug/L arsenic, which is equivalent to the Maximum Contaminant Level (MCL) established by the US EPA. The 50 ug/L arsenic MCL is not a risk based number. An additional lifetime cancer risk of 100 to 6000 per million was estimated by the CDHS 1987 for children in contact with CCA-treated playgrounds.

Environmental Working Group 2001

The EWG 2001 study was based upon a probabilistic approach that simulated 1 million children. The risk end-point for this assessment was lung and bladder cancer with a cancer potency factor of 0.0015 (ug/(kg*day))-1. Exposure routes considered included exposures through direct contact with surface residues via ingestion and dermal contact

and exposures through contact with contaminated soil via ingestion and dermal contact. The duration of exposure was 3 years and the exposure frequency was one hour each day for 156 days per year. The model utilizes a rate of hand-to-mouth activity of 9.5 per hour in accordance with EPA 2001 and each hand to mouth event is represented on average by 1/3 of the hand in contact with the mouth and a 50% removal efficiency for a resultant 0.8 hand loads per day ingested (Houlihan et al. 2001). Relative bioavailability was assumed at 100% in accordance with EPA 2001. The study utilized wipe data to estimate the amount of dislodgeable arsenic that is transferred to skin. The wipe data was obtained from several studies (CDHS 1987; MEDHS 1998 (not in reference list); US CPSC 1990; Stilwell 1998 (not in reference list); Riedel et al. 1991; SCS 1998; Sharp et al. 2001) and ranged from 0 to 1,020 ug/100 cm². Body weight and skin surface area estimates were based upon NHANES measurements and the work of Gehan and George (1970). Dermal sorption rate was assumed at 6.4% (Houlihan et al. 2001). The soil model was based on literature data compiled by EPA 2001 to obtain the arsenic concentration below the decks. A soil ingestion rate was selected from a chosen distribution and the fraction of the total arsenic that is bioavailable in soil was estimated at 25%. The excess lifetime cancer risk (bladder plus lung cancer) were obtained from a graph prepared by Houlihan et al. 2001.

Gradient 2001

The Gradient 2001 risk assessment considers three scenarios: 1) a child who is in contact with a CCA-treated playground between the age of 2 to 6 years, 2) a child who is in contact with a CCA-treated playground between the age of 7 and 12 years, and 3) a person who contacted a playground during age 2 to 6 years and who then contacted a deck from age 7 to 31 years. Additional scenarios focused on the impacts of sealants to reduce risks. Gradient 2001 also evaluated non-cancer risks. For comparative purposes only the playground scenario for a child 2 to 6 years of age along with a cancer risk endpoint are included in Table 2. Soil arsenic concentrations for playgrounds (3.7 mg/kg mean) were based upon the work of Malcolm Pirnie 2001. The fraction of soil on a child's hand from contaminated soil was assumed to be 100% for the playground scenario. The soil ingestion rate was estimated at 36 mg/day and was based upon the work of Stanek and Calabrese, 1995a and an EPA model which accounts for the variations in soil ingestion rates with age (USEPA 1994). The relative bioavailability for arsenic in soil of 16.3% was based upon the work of Roberts et al. 2001.

Mean hand loading of arsenic from surface contact (6.1 ug/100 cm²) was obtained from SCS 1998. The child hand surface area was estimated at 132 cm² resulting in an average handload of 8.1 ug/handload. The hand transfer factor of 0.25 was based upon an estimated 36 mg/day of soil ingested as described above and upon a total daily soil ingestion of 145 mg as estimated by Roels et al. 1980. An exposure frequency of 31 days per year was based upon the US EPA 1997b. The average body weight for children 2 to 6 years of age and the average lifetime was obtained from the USEPA 1997b.

Maas et al., 2003

The Maas et al. 2003 considered exposures from ingestion due to direct contact with surface residues. The cancer end point was bladder and lung cancers and the unit cancer risk value used, $0.023 \text{ (ug/(kg*day))}^{-1}$ was based upon the NRC 2001 study. Experimental data from wipes collected from 800 sites from throughout the U.S. found mean arsenic wipe transfer of 63.6 ug/100 cm^2 and a median value of 12.5 ug/100 cm^2 . A value of 63.6 ug/100 cm^2 was used in risk computations for exposures to decks. For playgrounds a value of 49.6 ug/100 cm^2 was used due to data which indicated that playgrounds have lower amounts of dislodgeable arsenic.

Four scenarios were evaluated: a toddler from the ages of 6 to 18 months who plays on a deck, a young child from the age of 3 to 6 years who plays on a CCA playset and on a deck, an older child from the age of 7 to 13 years who plays on a CCA-treated playset and who comes in contact with a CCA-treated deck, and an adult who is exposed to a CCA-treated deck and picnic table for a period of 20 years. The risks associated with these scenarios were estimated at 6.6, 720, 160, 1000 per million for the toddler, young child, older child, and adult, respectively.

Roberts and Ochoa 2001

From the information available it appears as though Roberts and Ochoa evaluated both cancer and non-cancer risks. These risks were evaluated for a range of dislodgeable arsenic residue values ($1 \text{ to } 632 \text{ ug/100 cm}^2$) which was based upon data contained within CDHS 1987; US CPSC 1990; SCS 1998; and data published on the internet by David Stilwell. The hand transfer value of 0.31 handloads per day (based upon the amount present on both palms of surface area 228 cm^2) was based upon soil adherence values and gender-specific palm areas as obtained from the US EPA and as evaluated through the US CPSC 1990. Computations were performed for exposures for 365 days per year and for an exposure duration of 5 years. A relative bioavailability value of 1 was used in computations and the US EPA oral slope factor was used for computations of risk.

A dermal absorption rate of 1% was used. This value was based upon measurements using human cadaver skin (Wester 1993). The dermal surface area exposed (528 cm^2) was assumed to be both palms and the soles of both feet for a female in the 2 to 6 year age range.

U.S. Consumer Products and Safety Commission 2003

The U.S. CPSC risk assessment evaluated the ingestion of arsenic from surface residues only. The cancer end point was lung and bladder cancer. Unit cancer risks utilized in the assessment were based upon the assessments of the NRC 2001 ($0.023 \text{ (ug/(kg*day))}^{-1}$) and the EPA 2001 ($0.00041 \text{ ug/(kg*day)}^{-1}$). Both of these assessments were based upon the association of lung and bladder cancer and drinking water arsenic in southwest Taiwan (Chen et al. 1985; Chen et al 1986; Chen et al. 1988; Wu et al 1989; Chen and Wang 1990; and Chen et al. 1992).

This study was extensive with respect to the wipe data collected. Results suggest that a dry polyester cloth can be used to estimate arsenic transfer from wood surfaces that would occur with bare hands with a reasonable degree of confidence. Hands extracted approximately 20% of the dislodgeable arsenic removed by dry polyester cloths using the U.S. CPSC methodology. The study found that for a typical playset, a reasonable estimation of the amount of arsenic removed by a human hand is 7.6 ug of arsenic after rubbing 1.4 m² (10 rub cycles, 700 cm²) of the same section of board. These values are a function of extensive studies involving adult volunteers using 8 residential decks in the Washington D.C. metropolitan area. The decks ranged in age from new construction to approximately 18 years. A correlation was also established between the amount of arsenic found on human hands after contact with the wood and the amount found on polyester wipes using a standardized methodology developed through the U.S. CPSC. Polyester wipe samples were collected from 12 CCA-treated playground structures ranging in age from about 6 months to 18 years.

The hand transfer factor utilized in the computation of risk was established by multiplying two factors: the soil adherence to skin (mg soil per centimeter square for a child with a 129 square centimeter palm hand area) and soil ingestion (mg soil ingested per day). Soil adherence to skin was compiled from 4 studies (Roels et al. 1980; Charney et al. 1980; Gallacher et al. 1984; Duggan et al 1984) which were based upon children between the ages of 1 and 14 years of age. Statistical analysis of this set of data was performed by Finley et al. 1994 indicating that the average soil handload for both hands for 2 to 6 year old children is 84 mg assuming a palm surface area of 129 cm². Soil ingestion rates were based upon data collected by Stanek and Calabrese (1995) for children between the ages of 2 and 6 years of age. This data was modified to account for the increase in children soil ingestions rates with age in a fashion consistent with a USEPA model used to evaluate children's exposure to lead (US EPA 1994) for an average soil ingestion rate of 36 mg/day. The HF factor was therefore computed as the ratio of the soil adherence and soil ingestion rate ($0.43 = 36/84$). The exposure frequency utilized was 156 days per year. The mean body weight for children 2 – 6 years of age was estimated at 17.7 kg and the lifetime was assumed to be 75 years based on the EPA 1997. In estimating the relative bioavailability the US CPSC indicated that it knew of no human or animal studies of bioavailability of arsenic from surface residue of CCA-treated wood. Issues were raised about some animal studies concerning the manner in which the animals were dosed. Given the limitations in the data, the US CPSC assumed a value of 100% relative bioavailability.

U.S. Environmental Protection Agency. (Zartarian et al. 2003 and Dang et al. 2003.)

The most recent exposure and risk assessment conducted through the US EPA has been published in a sequence of two reports by Zartarian et al. 2003 and Dang et al. 2003. Zartarian et al. 2003 provides estimates for average daily doses (ADDs) and lifetime average daily doses (LADDs). Chen et al. 2003 utilizes the ADDs and the LADDs from Zartarian et al. 2003 to compute risk. Various scenarios were evaluated by Zartarian et al. 2003. These included a child exposed to CCA-treated wood surface residues and CCA contaminated soil from public playsets. A subset of these children were assumed to

also contact CCA-treated wood residues and contaminated soils from residential playsets and/or decks. Special cases were also considered in the case of a child with pica behavior and increased gastro-intestinal sorption. The impacts of reduced arsenic exposures due to use of sealants and hand-washing were also considered. Two climate scenarios were considered, a warm climate and a cold climate scenario. Simulations considered the ingestion of arsenic and chromium from the surface of wood and from contaminated soil through hand-to-mouth behavior and dermal absorption. As mentioned earlier the most recent US EPA exposure assessment utilized a probabilistic approach. This probabilistic approach was based upon the use of the Stochastic Human Exposure and Dose Simulation (SHEDS) model. The SHEDS model was refined by Zartarian et al. 2003 specifically for the wood preservative scenario in a version called SHEDS-Wood. Simulation of children's activity patterns were based upon time-location-activity diaries from EPA's Consolidated Human Activity Database (CHAD).

Zartarian et al. 2003 found that the most significant exposure route from the simulations was ingestion of residues from the wood surface, dermal sorption of the surface residues, followed by ingestion of contaminated soil and dermal contact with contaminated soil.

For direct exposures through contact with wood, the dislodgeable amount of arsenic from the surface of the wood was estimated at 316/100 cm² for the warm climate scenario and 325/100 cm² for the cold climate scenario. These values were based upon results from the US CPSC 2003 and ACC 2003. Hand to mouth contact frequency averaged 8.6/hour and was based upon the work of Leckie et al 2000, Zartarian et al. 1998; Reed et al. 1999; Tulve et al. 2002. The exposure frequency was estimated at 123 days per year for the warm climate scenario and 55 days per year for the cold climate scenario. The total exposure time was estimated at 1 hour on the wood and a second 1 hour on the soil. The mean value of relative bioavailability (GI route) was estimated at 0.27 for wood residues and 0.47 for soil and were based upon ACC 2003c,d. The exposure time was assumed at 6 years, the average child body weight was 17.5 kg, and the average lifetime was 75 years. For soil exposures, the mean soil concentration for the warm climate scenario was assumed at 34 mg/kg whereas for the cold climate scenario the mean soil concentration was estimated at 3.8 mg/kg. Dermal absorption was estimated at 3%. Mean lifetime average daily arsenic doses (LADD) for the populations considered were estimated at 0.0064 (ug/(kg*day))⁻¹ for the warm climate scenario and 0.0032 (ug/(kg*day))⁻¹ for the cold climate scenarios for children exposed to a playset only and 0.011 (ug/(kg*day))⁻¹ and 0.006 (ug/(kg*day))⁻¹ for children exposed to a playset and a deck, for warm and cold climates respectively.

Dang et al., 2003 utilized a cancer slope factor of 0.00367 (ug/(kg*day))⁻¹ in the computation of cancer risk (bladder and lung) due to arsenic exposure. This value was based on the US EPA's risk assessment associated with inorganic arsenic in drinking water (US EPA 2001e). It is consistent with the slope factor used by the Office of Water for the arsenic drinking water standard. Mean cancer risk for warm climate scenario was 23 per million for exposure to a playset only and 42 per million for exposure to a playset

and an deck. The mean cancer risk for the cold climate scenario was 12 per million for exposure to a playset only and 22 per million for exposure to a playset and a deck.

Reduction of residue concentrations by 90 to 99.5% through the use of sealants considerably decreased the risk by 2 to 3 orders of magnitude. Currently an on-going study sponsored by the U.S. EPA and U.S. CPSC is evaluating the effects of sealants at reducing the risks due to surface residues of arsenic on treated wood.

4. Comparison of Each Risk Assessments and Recommendations

A comparison of the different risk assessments indicate that there is considerable uncertainty in the possible health effects due to contact with CCA-treated playgrounds. Cancer risks vary from a low of 0.5 per million (Gradient 2001) to a high of 8,000 per million (EWG 2001). Risk assessments by government regulatory agencies appear to lie within the middle-range with values of 2 to 1,000 per million by the US CPSC 2003 and 12 to 23 per million as estimated by the US EPA 2003. The majority of the risk assessments indicate that the risks from children playing on CCA-treated wooden structures is in excess of the 1 per million acceptable risk level and therefore children's exposures to CCA-treated wood should be minimized.

As of January 1, 2004 the production of CCA-treated wood for use in most residential applications has been banned, thereby restricting future wooden playgrounds to be made of durable untreated wood or with wood treated with alternative wood treatment preservatives. None of the regulatory decisions to ban the use of CCA-treated wood for many residential uses apply to CCA-treated wood already in use. Given the uncertainty associated with the cancer risk, exposure to arsenic from all sources should be reduced wherever feasible. Exposures from existing CCA-treated structures can be possibly reduced through appropriate hygiene (hand-washing) and through the sealing of existing structures (Stilwell 1998; Feist and Ross 1995) or use of water repellents (Cooper and MacVicar 1995; Cooper and Ung 1997). Lebow et al. 2003 found that water repellants reduce the amount of leachable metals from CCA-treated wood. Maas et al. 2004 found that water sealants and water-proofing materials appear to last for only about 6 months, while stains and paints exhibit As reduction properties through about 2 years of outdoor exposure. They recommend that treatments should include both a penetrant/water repellent material as well as a surface crack sealant. The US EPA recommends the application of penetrating coatings (e.g. oil-based semi-transparent stains) on a yearly or bi-yearly basis (US EPA 2004). In selecting a coating, the US EPA warns that, in some cases, "film forming" or non-penetrating stains (latex semitransparent, latex opaque, and oil-based opaque stains) on outdoor surfaces such as decks and fences may increase exposures due to accumulation of metals on the paint and subsequent peeling and flaking.

Given the large range in uncertainty with the models involved to estimate risk and with the input parameters used for these models, there is a need to directly measure children's exposures to CCA-treated playground equipment through measurements of absorbed arsenic via urine analyses (SAP 2001). Such a study has been initiated at the pilot scale through Rutgers University (Stuart Shalat, PI). This study includes an

environmental assessment of the playground and analysis of children hand-rinse and urine samples after playing on wooden playgrounds. The results from this study were pending as of August 2004.

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Summary of Available Health Information Regarding CCA-Treated Wood Products

Appendix

	CDHS 1987	EWG 2001	Gradient 2001	Maas et al. 2003	Roberts & Ochoa 2001	USCPSC 2003	US EPA (Zartarian et al. 2003; Dang et al. 2003)
Exposure Routes Evaluated							
Ingest Surface Residues via Hand to Mouth	X	X	X	X	X	X	X
Dermal Sorption of Surface Residues		X	X		X		X
Ingest Contaminated Soil via Hand-to-Mouth		X	X				X
Dermal Sorption via Soil		X	X				X
Other			Evaluated Non-Cancer endpoints. Evaluated inhalation route				Evaluated Chromium Exposures and Special Case Studies

Table 2: Scenarios Considered in Risk Assessments of Children's Exposure to Arsenic from CCA-Treated Playsets.

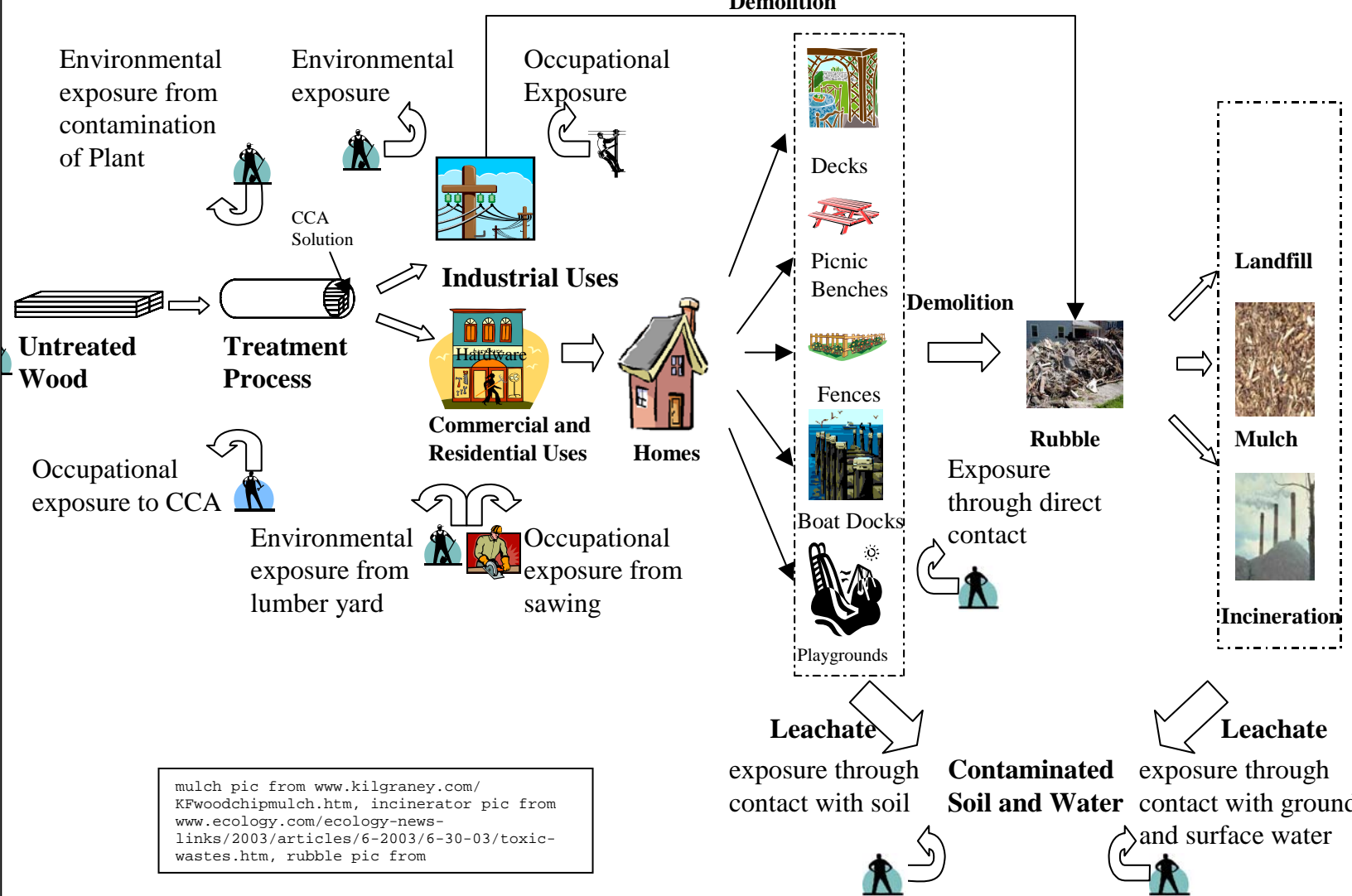
	CDHS 1987	EWG 2001	Gradient 2001	Maas et al. 2003	Roberts & Ochoa 2001	USCPSC 2003	US EPA (Zartarian et al. 2003; Dang et al. 2003)
C ug/handload		0 to 1330 assuming hand area of 130 cm ²	8.1	62	2-1440 (1 to 632 ug/100cm ² and 228 cm ² hand area)	7.6	37.8 (warm) 39.1 (cold) assuming 120 cm ² hand area
HT handloads per day	236 ug for all handloads per visit plus	0.8	0.25	0.43	0.31	0.43	0.8 (8.6 hand to mouth frequency per hour, 0.205 (w) & 0.305 (c) hand transfer eff, fraction of hand area mouthed 54/120)
EF, Exposure frequency, days/year	183	156	31	120	365	156	123 (warm) 55 (cold)
ED, Exposure duration (years)	Not found	3	5	3	5	5	6
B, Relative bioavailability, wood	1	1	0.47	1	1	1	0.27
BW, Body weight(kg)	Not found	Variable	17.8	16	18	17.7	17.5
LT, Lifetime (years)	Not found	Not Found	70	70	Not Found	75	75
LADD (ug/(kg*day))	Not found	Not Found	0.00032	0.031	Not Found	0.0053	0.0064 (warm), 0.0032 (cold)
Cancer end point	Skin	Lung & bladder	Skin	Lung & bladder	Cancer? (& non- cancer)	Lung & bladder	Lung & bladder
Q, Unit cancer risk (ug/(kg*day))-1	Not found	0.0015	0.0015	0.023	0.0015?	0.00041- 0.023	0.00367
R, Cancer risk, per million	100 to 6,000	8,000 ^c	0.5 per million ^b	720 ^a	4.2 to 2,700 non-cancer	2 to 100	23 (warm), 12 (cold)

^a Values provided correspond to the young child scenario with exposures to a playset and deck.

^b Estimated lifetime cancer risk for central tendency exposure. Includes risk from all exposure routes considered.

^c Risk value estimated from a graph in Houlihan et al 2001.

Table 3: Mean values used in Various Studies on Exposure and Risk Assessment Models. A Child Exposed to a CCA-Treated Playground was the Scenario in All Assessments. Numbers Provided Correspond to the Mean or Central Value.



mulch pic from www.kilgraney.com/KFwoodchipmulch.htm, incinerator pic from www.ecology.com/ecology-news-links/2003/articles/6-2003/6-30-03/toxic-wastes.htm, rubble pic from

Figure 1: Exposure Routes for Metals Contained Within CCA-Treated Wood. Exposure Routes Associated with the Manufacture of the CCA chemical are not included.