Report on Copper, Chromium and Arsenic (CCA) Treated Timber

Deborah Read
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This report was prepared by Dr Deborah Read, public health physician, for ERMA New

Table of Contents

Uncertainty still remains about the transfer rate of surface residues from CCA-treated wood to skin over time, the hand-to-mouth transfer efficiency, and relative bioavailability of ingested arsenic residues and to a lesser extent soil arsenic compared to ingested arsenic in water.

Most risk assessments use toxicity values that have been developed by the US Environmental Protection Agency (EPA) for both cancer and non-cancer effects. Recently the CPSC has also used the National Research Council's (NRC) value for cancer effects. The Atkins report (WS

bioavailability of arsenic on hands from surface residues or soil from CCA-treated wood structures in New Zealand.

4

5.2

to six months after treatment (Ministry of Forestry, 1987). These deposits are absent if oxide formulations are used.

There are thought to be about 165 timber treatment plants in New Zealand, most of which use CCA. The New Zealand

Copper,

5.5 Toxicity of CCA

Acute oral LD_{50}[7] values

6.2 General population exposure

Copper is an essential element required for normal growth and development and a number of metabolic functions. The recommended dietary intake in the United States is 340 µg/day and 440 µg/day for children aged 1 - 3 years and 4 - 8 years respectively, and 900 µg/day for adults.

Food is the main source followed by water and airborne particulates. Drinking water is the primary source of excess copper. Copper concentration in drinking water varies depending on pH, hardness and leaching from the distribution system.[8] In the New Zealand drinking water guidelines the maximum acceptable value (MAV)[9] for copper is 2 mg/L (Ministry of Health, 2000).

Soil criteria are set to protect the health of site users (exposed through ingestion of soil, dermal absorption from soil, inhalation of contaminated particulates, and consumption of home-grown produce), protect public health (exposed through ingestion of produce from the site) and protect plants and livestock on the site. The residential land use criterion in New Zealand assuming 10% of produce consumed is home-grown is 130 mg/kg for copper (Ministry for the Environment and Ministry of Health, 1997).

6.3 Bioavailability

Bioavailability[10] is a critical factor in determining the magnitude of potential exposure and risks. It is influenced by factors including chemical speciation, the matrix in which the substance is present, the amount of time that the substance is in a matrix, and exposure route. Ingested copper salts are readily absorbed (24 - 60%) from the gastrointestinal tract and after nutritional requirements for copper are met several homeostatic mechanisms prevent overload.

Following absorption, most is excreted in faeces. Limited data on dermal absorption suggest it is poorly absorbed through intact skin.

The bioavailability of copper in soil is unknown.

6.4 Toxicity

At high levels toxicity can occur. The gastrointestinal tract is the most sensitive target. Gastrointestinal effects such as vomiting occur at 0.011 – 0.08 mg/kg. Hepatic and renal effects have been reported following high dose intentional ingestion.

It is unknown whether children are more susceptible to copper toxicity than adults.

There are no effects associated with dermal exposure although copper salts as $CuSO_4$ are highly irritant.

[8] Soft corrosive water has higher copper concentrations.
[9] The maximum acceptable value is the concentration of the substance in water estimated to cause one additional case of cancer in a population of 100,000 who consume 2L water/day over a lifetime.
[10] Bioavailability is the amount of the substance that is absorbed into the body.

Copper is not classifiable with respect to carcinogenicity. Human data are limited and relate to inhalation. There are no studies of carcinogenicity in humans following oral or dermal exposure.

Occupational exposure to copper dust is reported to be irritating to the respiratory tract eg cough, rhinitis, sneezing.

7 Chromium

Unless otherwise stated the following information has been sourced from a review published by ATSDR (ATSDR, 2000a).

7.1 What is chromium?

Chromium is a naturally occurring element found in rocks, soil, animals, plants, volcanic dust and gases. Chromium occurs naturally in ores in its trivalent form.

It also occurs as a result of anthropogenic activity. In general, sites with soil chromium concentrations in the range 0.5-110 mg/kg do not require further assessment (Australian and New Zea

7.3 Bioavailability

Trivalent chromium is less readily absorbed from all exposure routes than hexavalent chromium with greater bioavailability for both from inhalation than either ingestion or dermal contact. It is poorly absorbed from the gastrointestinal tract (0.5-2.8% for trivalent and 1.7-6.9% for hexavalent chromium).

Following ingestion hexavalent chromium is reduced to trivalent chromium in the stomach accounting for its relative low oral toxicity. Its toxicity is thought to result from damage to cellular components during this reduction process.

Both forms can penetrate skin to some extent, particularly if skin is damaged. Skin absorption is estimated to be 1% (FIFRA SAP, 2001).

Absorbed chromium is excreted primarily in urine, at least 90% within a day.

Bioavailability of chromium from soil requires further research. Any uptake into plants is predominantly confined to the roots.

Urinary and blood levels of chromium are poor biomarkers in assessing low level exposure.

7.4 Toxicity

There is limited information on the toxicity of chromium in children. Most of it is from case reports of children who have ingested lethal concentrations of hexavalent chromium. The effects are part of the sequelae leading to death and similar to those seen in adults.

Hepatic, gastrointestinal and renal effects are the most common effects following ingestion and have been reported in individuals who ingested from 4-29 mg/kg hexavalent chromium (ATSDR, 2000a). In all cases death resulted. The estimated lethal dose for children is 10 mg/kg (US CPSC, 2003). Trivalent chromium is significantly less toxic than hexavalent because it is less readily crosses cell membranes. It is extremely unlikely that low level exposure would cause acute health effects.

It is not known whether children differ in susceptibility to chromium toxicity compared to adults.

Hexavalent chromium is classified as a human carcinogen based on excess lung cancer found

residues (FIFRA SAP, 2001). However even if small amounts of hexavalent chromium were present in surface residues hexavalent chromium is not carcinogenic via the oral route. There are no studies of carcinogenicity following dermal exposure.

Chromium is a common skin sensitiser. Direct dermal contact with both trivalent and hexavalent chromium causes skin irritation and allergic contact dermatitis though the hexavalent form is much more potent. The main cause is occupational exposure and environmental exposure to chromium is unlikely to result in these effects. Soil concentrations up to

In 1989 the JFECFA set a Provisional Tolerable Weekly Intake (PTWI)[11] of 15 µg/kg body weight/week for oral exposure to inorganic arsenic. A provisional value was set to indicate the desirability of reducing the arsenic intake of populations with naturally elevated levels of inorganic arsenic in drinking water and the need for further research to define more clearly levels that may result in health effects. It is possible that if the PWTI was revised based on more recent research a lower PTWI would be set.

In the most recent total diet survey (1997/98) in New Zealand total arsenic, not inorganic arsenic was analysed. The estimated weekly dietary exposures to total arsenic for six age-sex groups, including children aged 1-3 years and 4-6 years, were all below 11 µg/kg body weight/week. Using conservative assumptions that 10% of total arsenic in seafood is inorganic and 100% of arsenic in other foods is inorganic, the New Zealand dietary exposures estimated for inorganic arsenic are less than 25% of the PTWI for inorganic arsenic (Vannoort et al. 2000).

In the New Zealand drinking water guidelines the provisional MAV for arsenic is 0.01 mg/L (10 µg/L). The value is derived from the WHO drinking water guidelines and was based on a 6×10^{-4} excess lifetime skin cancer risk which is 60 times higher than the 1×10^{-5} factor typically used to protect public health. However it equates to an estimated additional lifetime risk of mortality from arsenic-related skin cancer of one in 100,000. The WHO set the MAV at this level because of limitations of the analytical methods available (Ministry of Health, 2000).

In New Zealand potentially health significant concentrations of arsenic (greater than 50% of the MAV i.e. 0.005 mg/L) in drinking water and concentrations exceeding the MAV are found most often in the geothermal areas of the North Island (Central plateau and Waikato). A study carried out for the Ministry of Health found concentrations greater then 50% MAV in 70 distribution zones serving a population of approximately 285,000 and concentrations exceeding the MAV in 28 distribution zones serving a population of approximately 21,000 (Davies et al. 2001).

Unpublished results from ESR for arsenic in drinking water based on 1300 recent samples give a mean concentration of 0.002 mg/L and a maximum reported concentration of 0.069 mg/L. Twenty-two supplies serving a population of 11,168 exceeded the MAV. Supply counts are based on there being ‘any sample above the MAV’ and therefore do not take into account natural fluctuations that may occur and analytical variance. For the mean figure, results below the limit of detection were taken as zero assuming that supplies w149eanalytical muming ies M

frequency and duration are 350 days/year for 30 years (Ministry for the Environment and Ministry of Health, 1997).

Tobacco smoking may contribute up to about 10 µg/day in a smoker and about 1 µg/day in a non-smoker (IPCS, 2001).

8.3 Bioavailability

appears to be complexed with the wood and the chromium an copper components of CCA (Exponent, 2002a).

It is assumed that the form of arsenic in CCA-treated wood surface residues is pentavalent. The SAP (2001) recommended use of a 2-3% value for dermal absorption of arsenic but noted that absorption could be different if trivalent arsenic was present. There are no data available on dermal absorption of trivalent arsenic (FIFRA SAP, 2001).

Once absorbed it is rapidly distributed throughout the body. At low to moderate doses the half-life of inorganic arsenic is about four days and it is primarily excreted in the urine (NRC, 1999).

Arsenic in urine is the best biomarker of exposure. The concentration of inorganic arsenic and its metabolites, monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA), in the urine reflects the absorbed dose on an individual level.

8.4 Toxicity

Arsenic is the most toxic of the components of CCA products and therefore the focus of risk assessments on CCA-treated wood. Trivalent arsenic is more toxic than pentavalent arsenic, the form in CCA products (ATSDR, 2000b).

In general, there is limited information about factors that influence toxicity and metabolism of arsenic in humans. Inorganic arsenic has the potential to interact with many cellular components (ATSDR, 2000b).

Metabolism of pentavalent arsenic involves reduction to the more toxic trivalent form before undergoing methylation. Methylation of inorganic arsenic to MMA and DMA in the body has been considered a detoxification process since these organic metabolites were thought to be less toxic and more easily excreted in the urine than inorganic arsenic. The metabolites are excreted in the urine along with unmetabolised inorganic arsenic. This is now debated since methylation is not universal among mammals and some recent research on metabolites suggest they are as or more toxic than inorganic arsenic (US CPSC, 2003). Differences in the pattern of excreted metabolites between individuals have also been reported (NRC, 2001). The role of the metabolites versus inorganic arsenic or the variability of human metabolism in the toxicity of arsenic is unknown (US CPSC, 2003).

Human susceptibility to adverse health effects resulting from chronic exposure is likely to vary depending on factors such as genetics, nutrition, and exposure to other compounds (NRC, 2001). Factors that inhibit methylation such as low protein intake or exposure to other contaminants may increase arsenic toxicity.

There is little information on the toxicokinetics of arsenic and its metabolites in children. There are no reliable data that indicate increased susceptibility of children to arsenic (NRC, 2001). Available data suggest the responses of children are the same as adults but these data predominantly relate to skin effects (FIFRA SAP, 2001). Children do not appear to absorb arsenic via the gastrointestinal tract more readily than adults (ATSDR, 2000b).

There is limited evidence for differences in arsenic metabolism between children and adults, at least at high arsenic exposure levels. Concha et al. (1998) found Argentinean children with

carcinogenesis is uncertain it is thought to partly result from inhibition of DNA repair or replicating enzymes (ATSDR, 2000b). This means for a carcinogenic effect to occur arsenic exposure is necessary when there is also exposure to a DNA-damaging agent. Some DNA damage occurs as a daily event. The risk assessments that have been carried out assume by default that either a DNA-damaging agent is present during or soon after arsenic exposure or that some other mechanism of carcinogenesis also exists.

There is considerable debated regarding the most appropriate dose-response relationship to quantify the cancer risks from arsenic exposure (Beck et al. 1995; Chappell et al. 1997). Epidemiological data on the dose-response relationship for cancer are insufficient to conclude there is or is not a threshold for carcinogenicity below which arsenic will not induce cancer (CSTEE, 2001). WHO (IPCS, 2001) and CSTEE, (CSTEE, 2001) have concluded that arsenic is a genotoxic carcinogen but this is debated. This has resulted in use of linear extrapolation to predict cancer risk at low levels of exposure.

The validity of the EPA's risk assessment model that assumes a nearly linear dose-response relationship to predict skin cancer risk for low level arsenic ingestion has been questioned. A review of epidemiological studies of arsenic exposure below that used by the EPA model suggests that it is unlikely to be able to predict risk at exposures between 170 and 270 µg/L of water. At lower levels current epidemiological data are inadequate to test the model's validity (Guo and Valberg, 1997). Arsenic levels in New Zealand drinking water and likely other forms of exposure constitute daily exposures considerably less than that from drinking water at these levels.

Since the EPA derived its unit cancer risk[13] for skin cancer, estimates for internal cancers have also been derived from epidemiological data. The NRC reviewed the toxicity of arsenic for the EPA's Office of Water in 1999 and noted that the risk in the United States at 50 µg/L of water for all cancers (i.e. skin, lung, bladder) may be as high as 7.1 in 1,000. They concluded that the choice of model for statistical analysis could have a significant effect on estimated cancer risks at low dose exposures particularly when the model accounts for age as well as concentration (NRC, 1999). Subsequently they have reported that at 3 µg/L the lifetime risk estimate for lung and bladder cancer combined is between 4 and 10 per 10,000 when the risks are estimated using the Taiwanese or United States background rates of these cancers respectively (NRC, 2001).

The EPA's Office of Water carried out a risk assessment of arsenic in drinking water in 2001 using bladder and lung cancer data and also requested the NRC to evaluate the data that had become available since their 1999 report. The CPSC subsequently calculated the EPA's unit risk estimate (1 in 10^{-6}) as about 0.00041 to 0.0037 µg/kg/day for bladder or lung cancer and NRC's unit risk estimate as 0.023 µg/kg/day (US CPSC, 2003). The lower estimates derived by the EPA are due to differences in statistical method, comparison population, background incidence rates, and assumptions for arsenic in water and food (NRC, 2001).[14] Both used linear extrapolation.

[13] The unit cancer risk (also known as the cancer slope factor or cancer potency) is the estimate of the chance of developing cancer at any time during a lifetime per unit of daily exposure to a substance. It is used in risk assessment to estimate the cancer risk from a given exposure duration and dose.

[14] The EPA used a multiplicative Poisson model, internal comparison group, and Taiwanese background incidence data. The NRC used an additive Poisson model, external comparison group, and United States background incidence data.

The CPSC concluded that although there are data limitations these quantitative assessments were reasonable and they based their assessment on the range of estimates for these two analyses for lung or bladder cancer risk (i.e. 0.00041 - 0.023 µg/kg/day) (US CPSC, 2003).

There is some uncertainty in extrapolating from epidemiological data from Taiwan to countries like the United States (as does the EPA) relating to the contribution of sources other than water (eg diet) to total inorganic arsenic exposure, and population characteristics such as poor nutritional status that may affect susceptibility to arsenic toxicity. However, Smith et al, 1992 have derived similar risk estimates in South American populations with adequate nutrition.

Epidemiological studies show no evidence of adverse health effects in United States populations with elevated arsenic drinking water or soil levels. This is in contrast to the results of studies in Taiwan, Japan, Chile, Argentina and India. The number of people exposed to a level of arsenic in drinking water associated with cancer risk and for sufficient intake and time may be too small to show an excess cancer risk. No long-term cohort study has been undertaken (Exponent, 2002b).

As all reports of human toxicity are based on exposure to arsenic in media other than soil the relevance of using toxicity factors derived from studies of ingestion of high arsenic-containing drinking water to assess toxicity of arsenic in soil has been disputed (Valberg et al. 1997). An ecological study of skin cancer incidence and environmental arsenic[15] exposure found no effect of soil arsenic on skin cancer rates. Skin cancer cases were ascertained from pathologists, hospitals and dermatologists (Wong et al. 1992).

9 Migration of CCA

9.1 Migration from soil

The copper, chromium and arsenic used in CCA are non-volatile therefore transfer from soil to air can occur only associated with dust particulates (HSE, 2001). Similarly, dislodgeable CCA residues are non-volatile. This means that if CCA-treated wood is enclosed in house wall framing by linings and claddings, then provided there is no significant movement of dust from the internal wall space to the house interior there will not be significant concentrations of CCA within the house.

Leached arsenic from a CCA-treated wood structure will be confined to the areas under or immediately adjacent to the structure as arsenic, copper and chromium adsorb strongly onto soil. For all three components adsorption is generally greatest on soils of moderate to high organic content and lowest for sandy soils with low organic content. Trivalent chromium is strongly adsorbed and essentially immobile in soil although low pH may increase mobility (HSE, 2001). Holland and Orsler (1995) suggest that high organic content of soils could be associated with the ability to adsorb all components of CCA. For six soil types in the United Kingdom (pH ranging from 3.5 to 7.1) arsenic was the most easily adsorbed component followed by copper then chromium. Amounts adsorbed tended to increase with time (experimental test over 24 hours). Most New Zealand soils are acidic with pH values ranging from 4 to 7 (Carey et al. 1996). A study of two free-draining New Zealand soils found

[15] Arsenic soil contamination from a mine and former smelter.

pentavalent arsenic adsorption was between that of copper and chromium, and less affected by changes in soil pH. Hexavalent chromium is weakly adsorbed but in the presence of organic matter and low pH is reduced to trivalent chromium (Carey et al. 1996).

Since the components of CCA bind to many soil components and given the small amounts that are leached from CCA-treated wood structures groundwater is not considered to be a source of exposure.

9.2 Migration from treated wood

There is a substantial literature on the leaching of arsenic, copper and chromium from CCA-treated wood into soil and water. Reports include field studies and controlled laboratory studies. Leaching from CCA-treated wood eg marine piles into water is not discussed here as environmental effects are outside the scope of this review.

Pentavalent arsenic is less soluble and less mobile than trivalent arsenic. Recent research shows that the predominant species of arsenic in CCA-treated wood is chromium arsenate (American Chemistry Council and American Wood Preservers Institute, 2001). Others consider that the oxidation state of inorganic arsenic in CCA-treated wood is unknown given the paucity of data (US CPSC, 2003). The FIFRA SAP assumed that the form of arsenic in CCA residues is pentavalent.

CCA-treated wood contains mainly trivalent chromium. There is no reliable evidence on the presence or absence of hexavalent chromium in residues (FIFRA SAP, 2001).

Leachability may be affected by parts of the timber treatment process and the environment to which the CCA-treated wood is subsequently exposed (Hingston et al. 2001). In general, leaching of all three components is reduced if the wood is dried over a period of weeks when compared with freshly treated wood. Increasing acidity of the leaching solution increases leaching particularly of copper. Leaching of arsenic seems to be related to the amount of chromium present with a minimum occurring when the chromium to arsenic ratio is 1.0 - 1.3 (HSE, 2001).

Leaching of the various components of CCA is not proportional to their formulation concentrations. With CCA type C copper and arsenic, which are present in lower concentrations than chromium, leach the most (Hingston et al. 2001).

Leaching decreases markedly with time. Leaching studies show that there is an initial rate over the first few days of use that rapidly decreases to a barely measurable rate. Other factors include climate and the amount of CCA used. The main factor affecting leaching rate is exposure to acid waters eg acid rain.

Aceto and Fedele (1994) found using simulated rainwater that between pH 4.5 and 6.1, 21-24% of copper, 7% of chromium and 6% of arsenic were released after three days from CCA-

seemed to be more resistant to leaching than arsenic (68-32%) or copper (100-92%). Variation in wood characteristics such as the sapwood/heartwood ratios may also affect leaching. Heartwood[16] is more difficult to treat with CCA than sapwood[17] and may therefore be more likely to release CCA metals (Warner and Solomon, 1990).

Maas et al. (2002) studied leaching under simulated rain conditions. No hexavalent chromium

treated structures (Gradient Corporation, 2001). This can be prevented by adequate construction site cleanup.

9.4 Dislodgeable CCA surface residue levels

Dislodgeable arsenic levels were measured using nylon wipes from seven playground equipment wood samples from manufacturers and one comparison sample of new CCA-treated wood not specifically finished and sold for playground use from a retail store. Two samples had average dislodgeable arsenic levels in the range of 21.9 - 32.1 µg/100 cm^2 compared to 68.9 µg/100 cm^2 for the unfinished wood (US CPSC, 1990).

Using moist polyester wipes Stilwell (1999) reported an average dislodgeable arsenic level of 35 µg/100 cm^2 for CCA-treated wood boards sampled for up to two years after purchase and of 8.8 µg/100 cm^2 from horizontal surfaces of playground equipment in three parks.

A preliminary study of 10 playground structures up to 10 years old in Ontario, Canada using moist cotton gauze wipe samples found arsenic concentrations ranged from 0.1 - 64.4 µg/100 cm^2 with a mean of 8.6 µg/100 cm^2. The authors noted that cotton gauze may contain a 'background' level of arsenic but this was not determined. Chromium concentrations were similar whereas copper concentrations were higher (Riedel et al. 1990).

The EWG's report (Gray and Houlihan, 2002) also included results of 300 wipe samples from 263 CCA-treated wood structureUpubl de-13.5 as om TD -0.055669 Tc 0.2719 Tw (treated w26j 33.75 -14.

9.5 Mitigation of CCA

Limited data suggest applying coatings, particularly polyurethane, every 1 - 2 years depending on wear and weathering reduces dislodgeable and leachable arsenic from CCA-treated wood (FIFRA SAP, 2001). Stilwell (1999) found dislodgeable CCA residues from boards coated with polyurethane, latex/acrylic stain, oil-based stain, or varnish were reduced for at least a year. In contrast the EWG concludes from results from CCA-treated wood structures wipe sampled by members of the public that sealants provide no reduction in concentrations beyond six months (Gray and Houlihan, 2002).

10 Plant Uptake of CCA Components

Plant uptake of arsenic depends on the extent to which it is adsorbed to soil components and the plant itself (Ministry for the Environment and Ministry of Health, 1997).

Elevated arsenic levels were found in carrots and potatoes grown near a wood preservation factory from both soil uptake and atmospheric contamination. In contrast elevated arsenic

The Wood Preservation Task Force, comprising three manufacturers of CCA wood preservatives, and the WEIWP responded to the CSTEE report and the initial proposed EC restrictions stating that a risk assessment using current principles of risk assessment, more recently available data, and data not included in the Atkins report would conclude that there is no scientific justification for restricting use. They concluded that currently available evidence can be interpreted to demonstrate that the use of CCA-treated wood is a tolerable risk to gain the economic and environmental benefits of using preserved wood (Baines, 2002). In respect of health risks the main additional information to which they refer is the risk assessment carried out by Gradient Corporation (2001) for two major CCA manufacturers (See Risk Assessments of Children's Exposure to Arsenic from CCA-

response to environmental concerns about arsenic. In Sweden use is restricted to settings where wood has a high degradation rate or use is important to a structure's safety. In Austria, Denmark and Germany there is a voluntary agreement that CCA will not be used to treat wood because of health and environmental concerns (WS Atkins International, 1998). In Finland CCA wood preservatives were re-evaluated and re-approved in 2001 for five years subject to restrictions on the dimensions of wood to be treated to limit non-professional exposure.

In the United Kingdom the HSE initiated a full review of the health and environmental issues associated with the industrial use of CCA in 1996 (HSE, 2001). The HSE is responsible under The Control of Pesticides Regulations 1986 for the registration of non-agricultural pesticides, which include wood preservatives. Risks to human health, including during intended uses of the CCA-treated wood products, and the environment are assessed as part of the decision-making process.

In 1999 the Advisory Committee on Pesticides (ACP), a committee of independent experts established to advise Ministers, considered the available data and recommended continuing use of CCA wood preservatives subject to certain conditions and environmental data requirements. The health risk assessment was based on a comparison of arsenic and chromium biomonitoring results for users of CCA with those of a number of other groups including unexposed populations, workers in other chromium industries, and people with clinical signs following occupational or environmental arsenic exposure. The conditions proposed by the ACP relating to health were regular changing of work clothing and a biomonitoring programme for CCA users. The latter was later amended to a research project to determine urinary arsenic and chromium in CCA users and a non-exposed population which is due to be completed in 2003 (HSE, 2001).

End use of CCA-treated wood was not included in the ACP review as it is outside the scope of The Control of Pesticides Regulations.

11.2.2 United States

1. US Environmental Protection Agency

The EPA has regulated CCA as a restricted use pesticide since 1986. Under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) the EPA is required to periodically re-evaluate older pesticides to ensure they continue to meet current safety standards. Since 2001 it has been evaluating the human and environmental risks of CCA as part of the re-registration process for wood preservatives. This includes evaluation of all available data to determine the most appropriate for use in the risk assessment (personal communication, US EPA, 23 January 2003). Although FIFRA regulates the sale, distribution and use of CCA and not CCA-treated wood, potential risks from use of CCA-treated wood are included in the EPA's evaluation.

An evaluation of available exposure and hazard data associated with the use of CCA-treated wood in playground equipment was presented to the SAP, an external scientific review panel, in October 2001. The FIFRA SAP made recommendations about the best methodologies to evaluate potential risks to children

amount of arsenic that could be absorbed from playground soil and CCA-treated wood was considered insignificant compared to natural background levels in the United States (Bidot et al. 2002). It is not possible to assess the validity of these conclusions as no detailed report was found in the public domain. It is also not clear whether the report was independently peer reviewed.

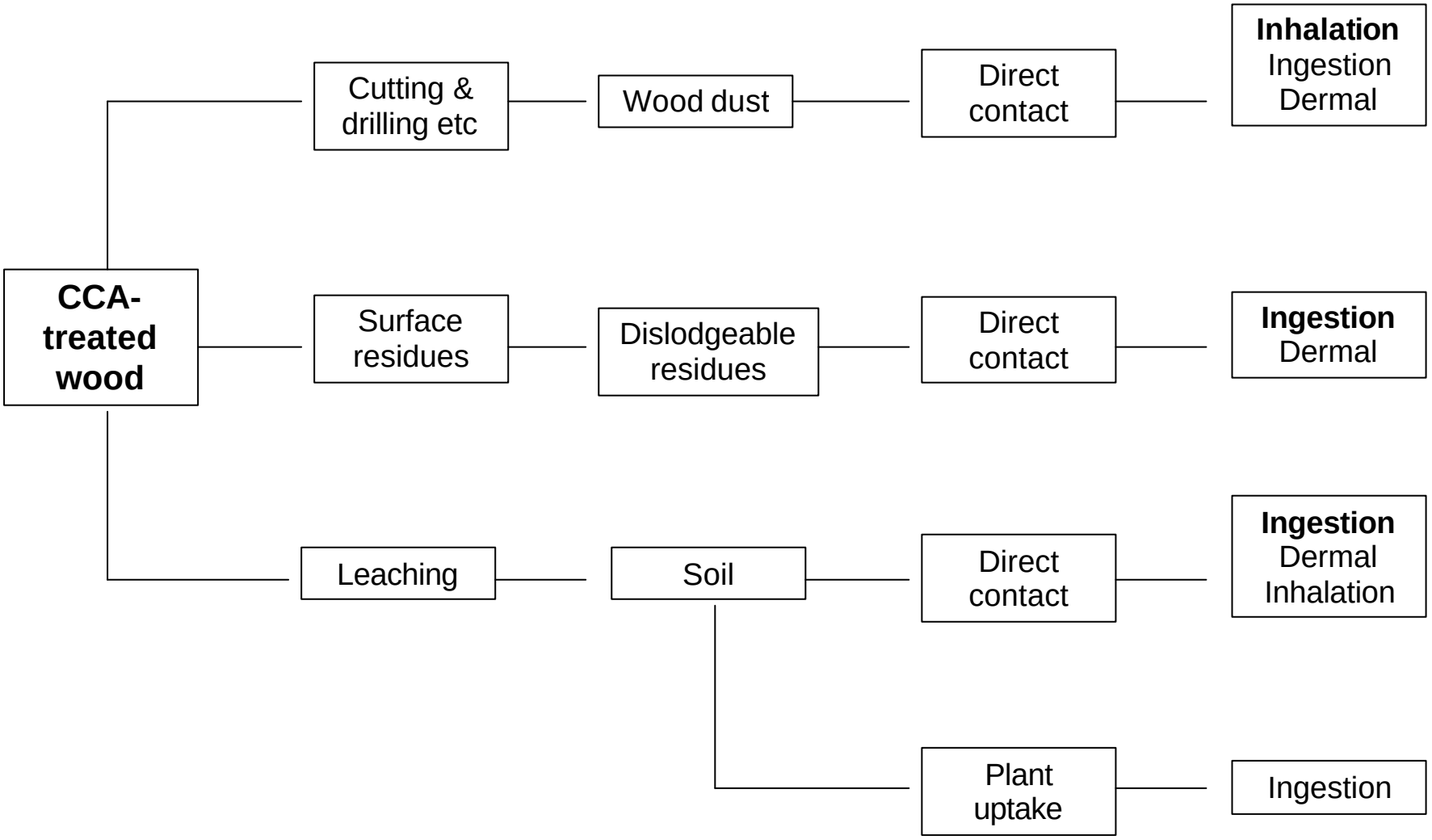
In December 2001 and February 2002 environmental and union groups and a victim family with a national advocacy group, Beyond Pesticides, as the lead petitioner, petitioned the EPA to ban three wood preservatives including CCA. Beyond Pesticides have criticised the EPA for not fully protecting the public and identify a number of outstanding issues such as public awareness about how to test for and prevent leaching, and safe disposal methods.

On 12 February 2002 the EPA announced a voluntary decision by the registrants[21] of CCA products to cancel the use of CCA-treated wood in most residential settings in favour of new alternative wood preservatives. This will take effect from 1 January 2004 and includes playground equipment, decks, picnic tables, landscaping timber, residential fencing, patios and walkways. During the transition period labelling is required specifying that no use of CCA will be permitted by the CCA industry for the affected residential uses after 31 December 2003. CCA-treated wood already in use and CCA-treated wood available for sale during the transition period are not affected. Specifically the EPA has not recommended that existing CCA-treated wood structures or surrounding soils are removed or replaced (US EPA, 2002a). Use of CCA-treated wood will continue for industrial (including farms), highway, marine, and utility uses (US EPA, 2002b). The EPA finalised its action on 17 March 2003 and

(See Risk Assessments of Children's Exposure to Arsenic from CCA-treated Wood Structures section).

In May 2001 the EWG and the HBN peti7 T1tD9CPSCure banT1tD9usents oStructu5c -0.252 Tw ( ) T75 0

Figure 1: Exposure pathways



Bold: principal exposure routes

Children may be more or less susceptible to toxicity depending on the chemical and the child's age. The impact of immaturity on biochemical and physiological processes that determine toxicity is difficult to predict and increased child susceptibility has only been shown for a few specific chemicals eg lead, mercury (Juberg, 2003).

It is also difficult to generalise about the effect of age on susceptibility to carcinogens in terms of dose-response relationship (Charnley and Putzrath, 2001). Recently the EPA has released draft revised guidelines for carcinogen risk assessment stating that since there is some animal evidence of higher cancer risks following early life exposure, particularly for mutagenic chemicals, it is reasonable to expect that children can be more susceptible to many carcinogens (US EPA, 2003a; US EPA, 2003b). To address the impact of early life exposure they propose a ten-fold adjustment to risk estimates for mutagenic chemicals relating to exposure before 2 years of age and three-fold adjustment for 2-15 year old children if tumour data specific to early life exposure do not exist. No adjustment to risk estimates is recommended for chemicals acting through a non-mutagenic mechanism due to the need for further research (US EPA, 2003b). It is not known to what extent susceptibility of children differs for arsenic due to a lack of relevant data and uncertainty about its mechanism of action.

Indirect exposure assessments are usually used to carry out risk assessments because of difficulties undertaking direct exposure assessments (Cohen Hubal et al. 2000).

The principal sources of potential exposure are contact with CCA-contaminated soil as a result of leaching of CCA from treated wood structures and contact with dislodgeable residues that may form on the surface of CCA-treated wood structures. There is the potential for exposure by dermal contact and ingestion for each source. Ingestion is the main exposure route as arsenic is poorly absorbed through the skin. Arsenic may be transferred to the mouth by mouthing the hands or eating with unwashed hands. Potential exposure scenarios for children in relation to use of CCA-treated wood are given in Table 4.

Table 4: Potential exposure scenarios for children

Exposure route-medium
oral/wood

outdoors playing (1.32 hours/day) as children from other states (1.27 hours/day). Age and gender also have an effect on time use (Silvers et al. 1994; Freeman et al. 2001).

The EPA's Consolidated Human Activity Database contains data from nine studies on human activity patterns. Children under two years, the group with the highest mouthing behaviour, spend the least amount of time outdoors at home, and outdoors at a park or playground. From 46% to 52% of children aged 2-6 years spend time outdoors at home and 17% to 32% spend time outdoors at a park or playground (Cohen Hubal et al. 2000).

Frequency and duration of playing outside at a playground will depend on climate as well as proximity to a playground and behavioural differences.

Information about children's micro-activity such as hand-to-mouth activity and contact with soil and CCA-treated wood is required to understand how exposure occurs. Micro-activities may influence dermal contact and ingestion through transfer from the environment to food through contaminated hands or directly from putting contaminated fingers or objects in the mouth. One study of activity patterns of children in Minnesota aged 3-12 years in summer included questionnaire-based micro-activity data that was validated using videotaped observations of a small sub-sample. Although hand contact events were frequent they were typically no more than five seconds each (Freeman et al. 2001).

Mouthing behaviour occurs most frequently in preschool children and declines with age. Since children under seven years are most likely to exhibit mouthing behaviour children aged 2-6 years are considered the most at risk group in risk assessments of CCA-treated wood structures. There is considerable year-to-year variability in exposure among children aged 2-6 years and understanding of relationships between behaviour and exposure is limited.

It is assumed that residues adhere to an area equivalent to the palmar side of the hand. Assuming the same hand-to-mouth activity that leads to soil ingestion results in parallel exposure to dislodgeable residues, the hand loads[24] per day estimate can be used with the dislodgeable levels on the wood to estimate exposure by ingestion.

Data on residues are predominantly from studies using wipe sampling. Only two studies were identified that have compared hand loading and wipe data (US CPSC, 2003 and a study carried out for a CCA manufacturer by Scientific Certification Systems in 1998). The results suggest that hands are less efficient than wipes at removing arsenic and therefore hand data should be used in exposure assessment.

However there is no standardised validated method of determining dislodgeable arsenic on hands. The best available is that recently developed by CPSC (2003). In this study deck boards from eight decks up to 18 years old were rubbed with adults' hands and dry polyester wipes to establish a correlation between the results of the two methods (and therefore a conversion factor[25]). The maximum amount of arsenic that can be loaded onto a hand was reached after rubbing hands just a few times over wood. This finding suggests that the amount e assessment.

ranging from 1 µg to 20.9 µg (mean 7.7 µg). Wipes picked up about five times the amount of arsenic that the hand did.

Surface-to-skin transfer is influenced by factors including the nature of CCA treatment, type and condition of wood, nature of the surface residues, skin condition, and nature of the contact (FIFRA SAP, 2001). For example, higher CCA solution concentrations and poor penetration may result in higher concentrations near the wood surface. This is typical of wood species used in Canada (where the Riedel et al. (1990) study on dislodgeable arsenic residues was carried out) but not the United States where southern pines are the predominant species treated with CCA (Exponent, 2002b). The presence of surface treatments may also be a factor. One-to-one transfer from the surface to the skin has been assumed but the SAP concluded that this is not justified.

The approach to estimating wood residue ingestion rate is based on the data available for soil ingestion. The amount of soil children ingest is a major area of uncertainty. The mean soil ingestion rate for children recommended by the EPA for risk assessment is 100 mg/day or 200 mg/day as a conservative estimate. The EPA does not recommend upper percentile estimates, as there are insufficient data (US EPA, 1997). Using a more methodologically sophisticated approach than earlier studies Stanek and Calabrese (1995) found a mean of 149 mg/day for children aged 1 - 4 years. Their findings also suggest that most children in this age group will periodically display soil pica[26] during a year. However children have not been studied long enough to fully characterise day-to-day variability.

Available data on arsenic concentrations in soil in general do not characterise potential playground exposure as they do not represent concentrations across an entire play area. The most appropriate value to include in risk assessment is the long-term average concentration to which a child might be exposed (Exponent, 2002b).

14 Epidemiological Studies

14.1 At risk population groups

There are three population groups at potential health risk from CCA-treated wood that can be studied: workers in timber treatment plants, workers who process CCA-treated wood into various end uses and the general population who use or come into contact with the end product.

1) Workers in timber treatment plants

Historically workers in timber treatment plants were the most exposed group as they were potentially exposed to CCA itself. As a result of improvements in the treatment process and greater attention to occupational health and safety it is uncertain whether these workers are currently more or less exposed than workers who process CCA-treated wood into various end uses.

[26] Soil pica is the eating of soil.

2) Workers who process CCA-treated wood into various end uses

Workers who process CCA-treated wood into various end uses include builders and garden furniture manufacturers who are potentially exposed through handling, drilling, sawing and sanding. Within this group there are likely to be subgroups who do not use protective equipment.

3) The general population who use or come into contact with the end product

Within the general population who use or come in contact with CCA-treated wood the group at greatest risk is children aged 2 - 6 years because of their behaviour.

Some indirect exposure of children and other household members may occur from residues on workers' or children's clothing that are subsequently transferred onto other surfaces eg furniture and then to hands, or to hands during home laundering.

14.2 Occupational studies

For workers exposure is through inhalation, dermal contact with dislodgeable residues, and ingestion through inadequate personal hygiene before eating or smoking.

Workers using CCA or CCA-treated wood are typically exposed at much higher levels than the general population and also through inhalation. They can be seen as sentinels of risk if it is present. Data from occupational studies can then be extrapolated to determine whether any risk is likely to exist for low level general population exposures.

Although the occupational health risks associated with timber treatment are outside the scope of this review, studies on timber treatment workers have been included here given the limited number of relevant occupational epidemiological studies found.

Industry frequently cites the results from a case control study of timber treatment workers in

for a latency[27] of at least 20 years. Timber treatment companies identified workers and cancer cases were identified from cancer registries (Ohlson et al. 1995). Although it is likely that the older workers in the study would have been exposed to much higher concentrations than currently would be the case,

Urinary arsenic in workers handling CCA-treated wood all day in the manufacture of garden fences was four times higher than controls (Jensen et al. 1991).

Occupational exposure to untreated wood dust has been well studied and unprotected workers in sawmills, furniture factories and similar settings have a higher risk of cancers of the nasal cavities and sinuses. If wood being sawed, sanded or drilled has been treated with CCA then the dust will contain a proportion of that CCA leading to potential inhalation exposure. Inhalable particles are likely to deposit predominantly in the nasal cavity and are eventually cleared and swallowed contributing to oral exposure. Only limited occupational exposure data to arsenic, copper and chromium associated with inhalable dust from CCA-treated wood are

After standardising for socio-economic level as well as age, carpenters were found to have significantly increased incidence of lung cancer (SIR^{30} = 165; 95% CI: 141 - 193). Previous census data have found smoking prevalence among carpenters to be lower than for the total labour force. Increased incidence was also found for the buccal cavity, stomach, bladder (SIR = 184; 95% CI: 127 - 257) and multiple myeloma (Firth et al. 1996). Lung cancer in carpenters could be related to asbestos exposure as Firth et al. (1993) reported increased mortality risk for pleural mesothelioma. Exposure to chemicals other than CCA may also play -

14.3 Non-occupational studies

Among the non-occupationally exposed general population it is likely that children using CCA-treated wood structures will have the highest exposure because the main route of

Table 6: Exposure routes evaluated for children's exposure to CCA-treated wood structures

Risk assessor(s)	Exposure route
CDHS (1987)	ingestion (residues only)
CPSC (1990)	ingestion (residues only)
Roberts and Ochoa (2001)	ingestion (residues only) dermal (residues only)
HSWMR (2000, 2001)	ingestion (residues and soil) dermal (residues and soil) inhalation (soil only)
Gradient Corporation (2001)	ingestion (residues and soil) dermal (residues and soil) inhalation (soil only)
CPSC (2003)	ingestion (residues only)

This study evaluated skin cancer risk only. CPSC developed a unit cancer risk for this risk assessment that was one-third that developed by the EPA using the same epidemiological data but a different methodology. This is not known to have been peer reviewed or used elsewhere. In addition they used the daily hand loading rate of boys rather than the more conservative approach of using the higher rate of girls or an average for both sexes (Roberts and Ochoa, 2001; Sharp and Walker, 2001). The study has also been criticised for not taking into account routes of exposure other than ingestion and exposure from CCA-treated wood structures other than playground equipment (Sharp and Walker, 2001). The CPSC have subsequently u995 Tc 0.5ee4m4m4m

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Assumptions were that all of the time spent outdoors at either a residence or a playground was spent exposed simultaneously to both dislodgeable and soil arsenic, that the structure was made of new CCA-treated wood, and the amount of dislodgeable residues does not decrease with age (Gradient Corporation, 2001). Risk estimates were also calculated for adults exposed in a residential setting (based on 30 years of exposure from 2 - 31 years) and children aged 7 - 12 years exposed in a playground setting.

Results for mean exposure of children aged 2 - 6 years are given in Tables 8 and 9.

Table 8: Estimated lifetime cancer risk for a child aged 2-6 years (based on mean exposure)

Medium	Residential	Playground
Soil	1.7×10^{-7}	2.5×10^{-8}
Residues	9.6×10^{-7}	5.4×10^{-7}

Exposure frequency and duration were assumed to be 1.8 hours/day outside at home for 350 days/year and 1 hour/day outside at a playground for 365 days/year.

Exposure parameters for soil included soil arsenic concentration of 28.7 mg/kg for decks and 4.1 mg/kg for playgrounds, 16.3% bioavailability, soil ingestion of 36 mg/day, and dermal absorption of 0.5%.

Exposure parameters for dislodgeable residues included a hand arsenic concentration of 6.1 µg/100 cm^2, 47% bioavailability, 25% hand-to-mouth transfer efficiency and 1.4% dermal absorption.

Table 9: Estimated non-cancer risk for a child aged 2-6 years (based on mean exposure)

Medium	Residential	Playground
Soil	9.3×10^{-5}	1.5×10^{-5}
residues	6.0×10^{-4}	3.4×10^{-4}

If the unit risks for lung or bladder cancer instead of skin cancer are used in this assessment the lifetime risk is up to 2×10^{-5} (US CPSC, 2003).

Estimated lifetime cancer risk for average exposure for adults in a residential setting were 3.9×10^{-7} for soil and 2.3×10^{-6} for dislodgeable arsenic.

The rinsing procedure used in the hand loading study from which the arsenic concentration was used for this assessment is considered to have underestimated the amount of arsenic on hands (US CPSC, 2003).

15.7 Environmental Working Group (2001)

In 2001 the EWG released a risk assessment based on a mean arsenic concentration of 247 µg/100 cm^2 from new CCA-treated wood using moist polyester wipes that estimated a lifetime risk of lung or bladder cancer of 2×10^{-3} (Sharp et al. 2001). Public interest groups in 13 cities carried out sampling on wood purchased from two retail chains using an EWG test kit. The assumptions and input parameters used for the risk assessment were not fully

described and there is no discussion in the report a[illegible]y and relative bioavailability.

15.8 Maas et al. (2002)

Maas et al. (2002) tested CCA-treated wood for di[illegible]ble arsenic using wipe [illegible] based on the method developed by the CPSC.

The authors estimated 8.5 x 10^5 µg/year arsenic ing[illegible]r a baby crawling on [illegible] (assuming 200 hand touches/week, hands are 40 c[illegible]/ hand-to-mouth t[illegible] efficiency). They then calculated the cancer risk fo[illegible]ing on a de[illegible]0 based on a lifetime exposure of 1.53 x 10^5-µg (6 µg[illegible]D /F0 12 Tf 0a2[illegible]

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15.9 US Consumer Product Safety Commission (2003)

From the results of a field study arsenic exposure from CCA-treated wood playground equipment was estimated to be about 3.5 µg each day that includes a playground visit.[38] This is based on the estimated amount of arsenic that would be removed from the wood surface onto a child's hands during a typical play episode and subsequently ingested and absorbed.

The CPSC carried out a deterministic risk assessment for arsenic with uncertainty analysis in which several input parameters (arsenic concentration on hands, hand-to-mouth transfer efficiency, exposure frequency and bioavailability) were individually changed to its upper and lower bounds to approximate best and worst case risk estimates. A probabilistic approach was rejected due to the limited data available for some of the important input parameters. The assessment did not include other potential exposures such as direct dermal uptake of dislodgeable arsenic or exposure to arsenic-contaminated soil. The CPSC concluded that a child who plays on CCA-treated wood playground equipment during early childhood (from 2 to 6 years) has an increased lifetime risk of 2×10^{-6} to 1×10^{-4} of developing lung or bladder cancer. The range of risk estimates from sensitivity analysis was from 2×10^{-7} to 5×10^{-3}.

Assumptions included that the bioavailability of dislodgeable arsenic is 100%, a child aged 2-6 years visits a playground 3 times/week (i.e.156 times/year), a child spends enough time in contact with CCA-treated wood to load their hands, and hand-to-mouth transfer efficiency is 43% and occurs irrespective of where the child is, and there is no effect of wood age on the amount of dislodgeable arsenic.

Prior to the field study laboratory experiments were carried out to develop the study protocol. The factors that had the most impact on dislodgeable arsenic levels were type of hand contact, t Tc 0e of hand unsk207189 Tw (01c7cs oi the cl2e bsureev and occ 3ntact,) Tk14 TcA2TD f Tfect ous Tl

dermal absorption from CCA-treated wood structures other than playground equipment eg decks, sand-pits. In addition some children will be exposed to arsenic from environmental tobacco smoke. For a 3 year old child if this additional intake was greater than about 10 µg/day the tolerable intake would be exceeded.

17 Information Gaps and Uncertainties

Information gaps include the transfer rate of surface residues from CCA-treated wood to skin over time, the relative bioavailability of arsenic from CCA-treated wood in soil and from wood surface residues, arsenic dermal absorption, chromium speciation in residues and soil, New Zealand data on the prevalence of CCA-treated wood decks or playground equipment and their age, activity pattern data for New Zealand children and the number of children likely to be exposed, and wood surface residue data from CCA-treated radiata pine structures in New Zealand.

Assessment of human health risk from exposure to environmental media involves many steps. If the uncertainty inherent in each step is high, the probability of significantly overestimating exposures increases. The product of several such overestimated parameters can result in risk estimates that are implausible.

Since the mechanism of carcinogenesis of arsenic is not well established there is uncertainty associated with the cancer toxicity values that have been derived and used in the risk assessments. These may overestimate risk at low levels of exposure.

Urinary biomonitoring would overcome the uncertainty that currently exists concerning the hand-to-mouth transfer efficiency and bioavailability of arsenic from surface residues and bioavailability of soil arsenic from CCA-treated wood structures.

Given the difficulties in getting urine samples from preschool children and the likelihood that exposure among builders is higher, as they are exposed for longer periods of time and also through inhalation, builders would constitute an ideal study group for biomonitoring. Within the building industry there are also some who are mainly involved in deck construction.

The fraction of total urinary arsenic derived from inorganic arsenic (inorganic arsenic and its metabolites, MMA and DMA) needs to be determined for such a group and compared with the results from a control group not exposed to CCA-treated wood.

Results from such a study, if appropriately controlled for other sources of arsenic, could be used as an indicator of likely urinary inorganic arsenic in children exposed to CCA-treated wood structures. For example, if the urinary inorganic arsenic (including metabolites) levels among builders with high levels of exposure to CCA-treated wood who do not wear dust masks or gloves are not elevated compared to controls then those of children exposed to CCA-treated wood structures are unlikely to be either. Such a study would be limited by the fact that the main exposure route is different for builders and children. However if urinary levels were found to be significantly elevated then a biomonitoring study of children would be indicated.

18 Precautionary Health Advice

The EC labelling requirement includes advice to wear gloves when handling and wear a dust mask and eye protection when cutting or crafting CCA-treated wood (EC, 2002).

In addition the EPA advice includes advice to saw, sand and machine CCA-treated wood outside, to wash exposed parts of the body particularly hands before eating, drinking or smoking and to wash work clothes separately from other clothing (US EPA, 2002c).

Similar recommendations for handling and recommendations for use are given by the PMRA, in manufacturers' product information and by the New Zealand TPC.

Construction debris should be removed from the site and local authority advice sought about the appropriate means of disposal.

General public health advice such as washing hands before eating and not placing food directly on outside surfaces applies irrespective of whether there is contact with CCA-treated wood or not.

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19 Conclusion

Whilst there is international and local concern over the potential adverse health effects from CCA-treated wood, research to date is inconclusive as to whether exposure to arsenic from this source poses a significant health risk to children.

Few well-designed epidemiological studies have been carried out of timber treatment workers using CCA or workers using CCA-treated wood. Results from studies of urinary arsenic levels have been mixed with studies by Jensen

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