

**Part one:**

**Toxicology Assessment**

## Table of Contents

### Part one: Toxicology Assessment

<b>1</b>	<b>INTRODUCTION.....</b>	<b>3</b>
<b>2</b>	<b>HAZARD AND RISK ASSESSMENT.....</b>	<b>3</b>
2.1	COPPER.....	3
	<i>Bioavailability</i> .....	4
	<i>Toxicity</i> .....	4
	<i>Exposure</i> .....	5
2.2	CHROMIUM.....	6
2.3	ARSENIC.....	9
<b>3</b>	<b>CONCLUSIONS.....</b>	<b>19</b>
<b>4</b>	<b>STUDIES ON BIOAVAILABILITY OF ARSENIC FROM WOOD AND SOIL.....</b>	<b>19</b>
<b>5</b>	<b>STUDIES ON DISLODGEABLE ARSENIC, CHROMIUM AND COPPER FROM CCA-TREATED WOOD.....</b>	<b>20</b>
<b>6</b>	<b>STUDIES ON ARSENIC, CHROMIUM AND COPPER INTAKE FROM CCA-CONTAMINATED SOIL.....</b>	<b>32</b>
<b>7</b>	<b>REGULATORY HISTORY AND PUBLIC HEALTH STANDARDS.....</b>	<b>34</b>
7.1	REGULATORY HISTORY OF ARSENIC IN AUSTRALIA.....	34
7.2	INTERNATIONAL TOXICOLOGY ASSESSMENTS AND REGULATORY DECISIONS.....	35
7.3	CONSIDERATION OF PUBLIC HEALTH STANDARDS.....	35
7.4	PRODUCTS.....	39
	<b>REFERENCES.....</b>	<b>40</b>
	<b>APPENDIX I.....</b>	<b>ERROR! BOOKMARK NOT DEFINED.</b>

## **1 Introduction**

The timber preservatives copper, chromium and arsenic (CCA) and arsenic trioxide are used extensively to prevent damage caused by insects (termites, borers, beetles), wood rot and wood fungus. CCA-treated pine is the most common type of treated timber in Australia and its uses include decks, garden furniture, picnic tables, playground equipment, landscaping timbers, retaining walls, fences, gazebos and patios.

In CCA formulations, copper acts as a fungicide, arsenic acts against insects and chromium fixes the chemicals in the timber to resist leaching. The strength of CCA treating solution can be varied depending on the conditions under which the timber will be used (Norton, 1998).

There has been ongoing media and public interest in the use of CCA-treated timber and the possible health risks it may present, particularly when used in settings where children may be exposed. This review considered the risk to children and makes a number of recommendations in relation to the on-going registration of CCA-treatment products.

## **2 Hazard and Risk Assessment**

Several reviews of arsenic, chromium and copper are available (IPCS, 1981, 1988, 1998, 2001; ERMA, 2003; UK Environment Agency, 2002a, 2002b; RIVM, 2001; US EPA, 2001a,b,d; US CPSC, 2003a). The Australian Department of Health has also reviewed arsenic toxicity (DHFS, 1999). The following information was taken from these reviews.

### **2.1 Copper**

Copper is a naturally occurring element found in a variety of organic compounds, mineral salts and as a solid metal. Natural sources of copper include soil and windblown dust, decaying vegetation, bushfires, volcanoes and water (seawater, surface water, ground water and drinking water). In addition, copper is released into the environment via industrial emissions and mining operations.

Elemental copper is insoluble but its salts such as copper sulfate or copper oxide are readily soluble. Copper compounds are used as bactericides, fungicides, insecticides and animal feed additives. Copper compounds are also used in pharmaceuticals and as food additives. Copper is an essential element in mammals, being incorporated into a large number of enzymes, particularly the oxidoreductases.

In Australia, a National Environmental Health Monograph (Metal Series No. 3) was published in 1997. The International Program on Chemical Safety (IPCS) released a health monograph on copper in 1998 (IPCS, 1998). The IPCS concluded that, based on human exposures worldwide, there is a greater risk of adverse health effects from copper deficiency than from excess copper intake. The IPCS set a lower limit of the acceptable range of oral intake of 0.02 mg/kg bw/d in adults and 0.05 mg/kg bw/d in infants. The upper limit in adults is uncertain, but an estimated range is 2-3 mg/kg bw/d, based on studies of gastrointestinal effects of copper-contaminated drinking water.

## Bioavailability

The level of absorption of copper compounds through the gastrointestinal tract is 20-60%, with the remainder excreted via the faeces. Intestinal absorption is influenced by the presence of other metals, such as zinc and iron, dietary proteins, fructose, ascorbic acid and fibre. A recent *in vitro* study suggested that copper may be more bioavailable from wood dust of CCA-treated timber relative to the intact wood (Gordon *et al.*, 2002).

## Toxicity

### *Laboratory animal studies*

The oral LD<sub>50</sub> of copper compounds varies between species, ranging from 15 to 1664 mg/kg bw. Toxic signs include gastric haemorrhage, tachycardia, hypotension, haemolytic crisis, convulsions and paralysis, followed by death. Reported dermal LD<sub>50</sub> values are >1124 mg/kg bw in rats and >2058 mg/kg bw in rabbits. The inhalational LC<sub>50</sub> in rabbits is >1303 mg/kg bw. According to RTECS (2003), the lethal oral dose of copper oxide in rats is 470 mg/kg bw. The oral LD<sub>50</sub> for hydrated copper sulphate is 300 mg/kg bw in rats and 43 mg/kg bw in mice. The rat dermal LD<sub>50</sub> is >2 g/kg bw. The lowest lethal oral dose in dogs is 60 mg/kg bw. For anhydrous copper sulphate, the lowest oral LD<sub>50</sub> in rats and mice is 300 and 87 mg/kg bw, respectively, while the oral LD<sub>50</sub> in dogs is 37.5 mg/kg bw.

Short-term repeat-dose oral studies with copper compounds found effects on clinical chemistry and haematology parameters and adverse effects on the liver, kidney and lungs. Subchronic and chronic dietary studies indicated effects on the liver and kidney. Laboratory animal studies have provided no indication that copper is carcinogenic. Copper does not appear to affect reproduction. High oral doses of copper reportedly cause fetotoxicity and soft tissue malformations in mice at and above 260 mg/kg bw/d, while lower concentrations had an apparently beneficial effect on development (Lecyk, 1980). Delayed ossification has been reported in rats following *in utero* exposure (Haddad *et al.*, 1991). DNA damage and adducts have been detected in patients with Indian childhood cirrhosis (a discrete clinical and histological entity in which large amounts of copper are deposited in the liver), however, there is little evidence that copper is genotoxic *in vivo*, given that it is mostly protein bound.

### *Human data*

Adverse health effects in humans relate to deficiency as well as excess exposure. Data from human poisoning cases has estimated that the acute lethal dose for adults is 4-400 mg copper<sup>2+</sup>/kg bw (IPCS, 1998). According to RTECS (2003), the lowest published oral lethal dose of hydrated copper sulphate is 1088 mg/kg bw, while the lowest oral toxic dose is 272 mg/kg bw/d. For anhydrous copper sulphate, lethal oral doses have been reported as 50 and 857 mg/kg bw. In children the lowest toxic oral dose has been reported to be 150 mg/kg bw. Acute oral exposures have resulted in the presence of a metallic taste, epigastric pain, headache, nausea, dizziness, vomiting and diarrhoea, tachycardia, respiratory difficulty, haemolytic anaemia, haematuria, gastrointestinal bleeding, liver and kidney failure and death.

Single and repeated ingestion of drinking water containing high levels of copper compounds has caused gastrointestinal effects. In a double-blinded clinical study, the NOEL for nausea and gastrointestinal symptoms following a single weekly dose of copper sulfate solution for 5 weeks was 4 mg/L, with a LOEL of 6 mg/L (Araya *et al.*, 2001). Other studies have confirmed that vomiting occurs at a concentration of 6 mg/L (Olivares *et al.*, 2001; Poirier *et al.*, 2002). Long term exposure to copper via drinking water also results in gastrointestinal disturbances. Cirrhosis and liver failure occurred in an individual following 2 years of ingesting 30 or 60

mg/d copper as a dietary supplement (O'Donohue *et al.*, 1993). Dermal exposure does not cause systemic toxicity but may induce allergic responses in sensitive individuals.

The IPCS (1998) identified a number of "at risk" groups in the population that may be particularly sensitive to either copper deficiency or overexposure: individuals with genetic conditions such as Menkes disease (a copper deficiency disorder), Wilsons disease (disorder due to excess copper), Indian childhood cirrhosis and idiopathic copper toxicosis; dialysis patients, persons with chronic liver disease, infants and persons with malabsorption syndromes (eg. coeliac disease and cystic fibrosis).

## **Exposure**

The main sources of exposure to copper are via food and drinking water. The IPCS (1998) calculated that the total intake of copper (ie. food plus drinking water) in adults is between 1-2 mg/d, while it may occasionally reach 5 mg/d. Inhalation and dermal exposure to copper are considered to be insignificant, with inhalation exposure of 0.3-2.0 µg/d (IPCS, 1998).

### *Food*

In 1996, the World Health Organisation (WHO) set a tolerable dose for copper of 0.2 mg/kg bw/d (200 µg/kg bw/day), a value that has also been adopted by Australia. The 20<sup>th</sup> Australian Total Diet Survey<sup>1</sup> (ATDS) found the highest amounts of copper in almonds, prawns, processed wheat bran, peanut butter, mushrooms, sultanas, breakfast cereal, liver pate and baked beans. It is also widely distributed in a range of plant and animal products. In the majority of foods, copper is found bound to proteins rather than as a free ion. Calculations performed by Food Standards Australia New Zealand (FSANZ) determined that the mean estimated daily dietary exposure to copper was 16 and 14 µg/kg bw/d in adult males and females, respectively. In 12-year old boys and girls it is 21 and 16 µg/kg bw/d, respectively, while in toddlers (2 years) and infants (9 months) it is 40 and 65 µg/kg bw/d, respectively. The intake as a percentage of the tolerable dose for these groups was 8.0%, 7.2%, 11%, 8.2%, 20% and 32%, respectively.

The USA and Canada have set a Recommended Dietary Allowance of 900 µg/d for adults, 340 µg/d for children up to 3-years of age, 440 µg/d for ages 4-8, 700 µg/d for ages 9-13 and 890 µg/d for ages 14-28. The US Academy of Sciences has recommended that all adults should receive a daily intake of 1-3 mg copper to satisfy physiological requirements. Australia does not have a recommended dietary intake (RDI) for copper.

### *Drinking water*

According to the National Health and Medical Research Council (NHMRC), in major Australian reticulated water supplies, total copper concentrations range up to 0.8 mg/L, with typical concentrations of approximately 0.05 mg/L. Based on health considerations, the NHMRC has set a Health Guideline Value for copper in Australian drinking water at 2 mg/L, which is the same as that set by the WHO. However, based on aesthetic considerations, the concentration of copper in drinking water should not exceed 1 mg/L.

### *Risk to humans from exposure to copper in CCA-treated timber*

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<sup>1</sup> [http://www.foodstandards.gov.au/\\_srcfiles/Final\\_20th\\_Total\\_Diet\\_Survey.pdf](http://www.foodstandards.gov.au/_srcfiles/Final_20th_Total_Diet_Survey.pdf)

Based on a consideration of the toxicology profile of copper and the high natural background exposure to copper in food and drinking water, the risk to humans from exposure to copper compounds present in dislodgeable residues from CCA-treated timber is considered to be negligible.

## **2.2 Chromium**

Elemental chromium does not occur naturally and is virtually insoluble in water. It is a hard, silvery white metal, and is a transition element with possible oxidation states from -2 to +6, but only the 0 (elemental), +2, +3 and +6 states are common. Divalent compounds are relatively unstable as they are readily oxidised to the trivalent form. Trivalent compounds are stable and most naturally occurring chromium is in the trivalent (chromic) state. Although hexavalent chromium (chromate) rarely occurs naturally, it is produced from anthropogenic sources (ie. from industries). The pentavalent and tetravalent compounds are generally unstable. This hazard assessment will only consider the trivalent, Cr(III), and hexavalent, Cr(VI), forms.

Chromium compounds have a wide range of water solubilities, but the general rule is that the trivalent chromium salts are almost insoluble and the hexavalent ones are soluble. Hexavalent compounds are reduced to the trivalent form in the presence of oxidisable organic matter such as timber and in living organisms. In living organisms the conversion back to the hexavalent state is considered not to occur (ie. under acidic conditions or by organic matter) due to the high energy required. Trivalent chromium is generally considered to be stable and immobile in soil.

### **Bioavailability**

Gastrointestinal absorption of chromium is relatively poor (0.5-3%), with hexavalent chromium being more readily absorbed than trivalent chromium. This difference is due to the fact that trivalent chromium cannot cross cell membranes. Hexavalent chromium is taken up by an anion transporter and is then reduced intracellularly, via reactive intermediates, to trivalent chromium. Dermal absorption in guinea pigs is 1-4% of the applied dose (Bagdon & Hazen, 1991).

### **Toxicity**

Reviews on the toxicology of chromium have been conducted by a variety of national and international agencies. The IPCS released a health monograph on chromium in 1988 (Environmental Health Criteria 61, IPCS, 1988). The Agency for Toxic Substances and Disease Registry within the USA published a review in 2000<sup>2</sup>. The Netherlands published a toxicological review on chromium in 2001<sup>3</sup>. The US EPA completed an assessment of hexavalent chromium in 2001<sup>4</sup> as part of the Re-registration Eligibility Decision (RED) for the non-food use of chromium in CCA products. The International Agency for Research on Cancer (IARC) evaluated the carcinogenic potential of chromium and chromium compounds in 1990 (both trivalent and hexavalent chromium)<sup>5</sup>.

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<sup>2</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp7.pdf>

<sup>3</sup> <http://www.rivm.nl/bibliotheek/rapporten/711701025.pdf>

<sup>4</sup> <http://www.epa.gov/scipoly/sap/2001/october/arsenicgraphdata.pdf>

<sup>5</sup> <http://www-cie.iarc.fr/htdocs/monographs/vol49/chromium.html>

### *Laboratory animal studies*

According to RTECS (1993), the lethal oral dose of sodium dichromate in rats is 50 mg/kg bw, while the lethal dermal dose in guinea pigs is 335 mg/kg bw. Chromic acid has an oral LD<sub>50</sub> of 52 mg/kg bw in rats and a dermal LD<sub>50</sub> of 57 mg/kg bw in rabbits (US EPA, 2001d). The dermal LD<sub>50</sub> for chromium trioxide in rabbits is 30 mg/kg bw (ATSDR, 2000). Hexavalent chromium compounds are corrosive to the eyes and skin of laboratory animals (US EPA, 2001d). Trivalent and hexavalent chromium are skin sensitisers in guinea pigs (Gross *et al.*, 1968; Jansen & Berrens, 1968). Hexavalent chromium is carcinogenic to laboratory animals and is also genotoxic in a number of *in vitro* and *in vivo* assays. There is no evidence that trivalent chromium compounds are carcinogenic or genotoxic. Hexavalent but not trivalent chromium has been found to cause developmental and reproductive effects in rodents.

### *Human data*

Trivalent chromium is an essential element for the potentiation of insulin and the maintenance of normal glucose and fat metabolism.

According to RTECS (1993), the lethal oral dose of sodium dichromate is 50 mg/kg bw. The lowest toxic dose of chromic acid is 100 mg/kg bw, with nausea, vomiting and normocytic anaemia reported (RTECS 2003). Symptoms following acute oral ingestion include vertigo, abdominal pain, gastrointestinal haemorrhage, thirst, vomiting, oliguria, anuria, shock, convulsions, coma and death. Acute dermal exposure can cause systemic toxicity, with symptoms similar to oral exposure.

Hexavalent chromium compounds are strong skin irritants and sensitisers. Contact dermatitis has been reported in chromium workers, and it has been suggested that trivalent chromium-protein complexes are the allergens. Pulmonary irritation and sensitisation has also been reported in workers exposed to hexavalent chromium. Data mainly from chromium workers indicates that acute and chronic exposures via the oral, dermal or inhalational routes can lead to renal and hepatic toxicity (eg. renal tubular necrosis, hepatic necrosis). Low-dose exposure generally causes transient effects and low-level environmental exposures have not resulted in any adverse effects in the human population. Data on the possible reproductive or developmental effects of chromium in humans was not identified.

Occupational exposure to hexavalent chromium has been associated with lung cancer. The International Agency for Research on Cancer (IARC) has classified hexavalent chromium in Group 1, "sufficient evidence of carcinogenicity in humans", while trivalent chromium is classified in Group 3, "not classifiable – inadequate evidence in humans and animals for carcinogenicity". The US EPA has classified inhaled hexavalent chromium as a known human carcinogen (Group A), while carcinogenicity via the oral route cannot be determined (Group D).

## **Exposure**

### *Food*

While chromium is found naturally in a variety of commodities, FSANZ has not quantified chromium intake in the 20<sup>th</sup> ATDS. Although dietary intake of chromium is important for insulin potentiation and maintenance of normal glucose and fat metabolism there is no recommended Australian dietary intake for trivalent chromium.

A 1997 UK total dietary survey [as described in a recent European Commission (EC) evaluation of trivalent chromium<sup>6</sup>] indicated that the highest chromium levels were found in meat products, oils and fats, bread, nuts and cereals. The EC did not set an upper intake level for trivalent chromium as the available human data did not give a clear picture of the dose-response relationship. However, the UK Expert Group on Vitamins and Minerals concluded that a total dietary intake of approximately 0.15 mg trivalent chromium/kg bw would not be expected to cause adverse health effects.

The following national dietary intakes of chromium were reported by the EC (2003): Up to 170 µg/d in the UK; between 50-580 µg/d in Sweden; the average intake in Germany is 61 and 84 µg/d for males and females, respectively; the average intake in the US is approximately 30 µg/d (range 3-127 µg/d).

In 2002, the UK Department for Environment, Food and Rural Affairs and the Environment Agency<sup>7</sup> set an oral tolerable daily intake (TDI) for chromium at 3 µg/kg bw/d, with a mean daily intake (MDI) of 13 µg/d (ie. 0.2 µg/kg bw/d for adults and 0.4 µg/kg bw/d for children). A oral tolerable daily soil intake (defined as the difference between the TDI and MDI) was calculated as 2.8 µg/kg bw/d for adults and 2.6 µg/kg bw/d for 6-year old children.

The US Academy of Sciences estimates that the daily dietary intake of chromium by adults is approximately half of the safe/adequate daily intake of 50-200 µg/d. However the US Food and Nutrition Board considered that there was insufficient data to establish an upper limit for trivalent chromium.

### *Drinking water*

In major Australian reticulated water supplies, total chromium concentrations range up to 0.03 mg/L, with typical concentrations being less than 0.005 mg/L. Based on health considerations, the NHMRC has set a Health Guideline Value for chromium in Australian drinking water at 0.05 mg/L. It is recommended that if the concentration of total chromium exceeds this value then a separate analysis for hexavalent chromium should be undertaken.

### *Risk to humans from exposure to chromium in CCA-treated timber*

Although hexavalent chromium compounds are hazardous to human health by virtue of their carcinogenicity potential it has been shown that sawdust from CCA-treated timber contains between 0.3-0.4% of total chromium and less than 2% of the total chromium was present in the hexavalent form (Cruz *et al.*, 1995). Hence the chromium in dislodgeable residues from CCA-treated timber is most likely to be trivalent chromium which is not classifiable with respect to carcinogenicity due to insufficient evidence (IARC). There was no suitable data to quantify the chromium concentration in dislodgeable residues.

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<sup>6</sup> [http://europa.eu.int/comm/food/fs/sc/scf/out197\\_en.pdf](http://europa.eu.int/comm/food/fs/sc/scf/out197_en.pdf)

<sup>7</sup> <http://www.defra.gov.uk/environment/landliability/pdf/tox4.pdf>

## 2.3 Arsenic

Arsenic is a metalloid element with a complex chemistry. Inorganic arsenic occurs in many minerals and is widely distributed in rocks, soils and sediments. It can exist in several oxidation states, the most common being the pentavalent and trivalent forms. In minerals, the highest arsenic concentrations generally occur as the sulphide or oxide, or as the arsenides of copper, lead, silver or gold. The most important commercial compound, arsenic(III) oxide (also known as arsenic trioxide), is produced as a by-product in the smelting of copper and lead ores. A variety of arsenates ( $\text{AsO}_4$ , pentavalent arsenic) and arsenites ( $\text{AsO}_3$ , trivalent arsenic) are found in water, soil and food.

Arsenic can undergo an extensive range of chemical reactions to form organic and inorganic compounds. Methylated arsenic compounds, such as di- and trimethylarsines, occur naturally in the environment as a result of biological activity. In water, these may undergo oxidation to methylarsinic acids, for example monomethylarsinic acid (MMA) and dimethylarsinic acid (DMA). However, the biomethylated forms of arsenic produced are subject to bacterial demethylation back to inorganic forms.

Arsenic is released into the general environment from a variety of natural and anthropogenic sources. On a global scale, releases to the air from natural sources such as volcanic eruptions and forest fires, and releases to water from weathering or leaching of arsenic-rich rocks and soils, may be the dominant ones. On a local scale, releases as a result of human activity, such as the burning of coal, the disposal of wastes from industrial activity, or the burning of wood treated with arsenic-containing preservatives, are likely sources.

Arsenic in water and in soil can undergo a series of transformations, including oxidation–reduction reactions, ligand exchange and biotransformation.

### Bioavailability and metabolism

In humans water-soluble arsenic compounds are well absorbed from the gastrointestinal tract (55%–95%). Absorption of inorganic arsenic in inhaled airborne particles (cigarette smoke, dust and fumes) is estimated to be high (75–90% in humans). Dermal absorption of inorganic arsenic is low (<5%).

In many species arsenic metabolism occurs mainly by (1) reduction reactions of pentavalent to trivalent arsenic, and (2) oxidative methylation reactions in which trivalent forms of arsenic are sequentially methylated (in liver) to form mono-, di- and trimethylated products. Methylation of inorganic arsenic facilitates the excretion of inorganic arsenic from the body, as the end-products monomethylarsenic acid (MMA) and dimethylarsinic acid (DMA) are readily excreted in urine (IPCS, 2001). In humans and most common laboratory animals, inorganic arsenic is extensively methylated and the metabolites are excreted primarily in the urine. Following ingestion in humans, arsenic has a half-life in whole body of 2–3 days.

Analysis of tissues taken at autopsy from people who were exposed to background levels of arsenic in food and water revealed that arsenic is present in all tissues of the body. Most tissues had about the same concentration level (0.05–0.15 ppm), while levels in hair (0.65 ppm) and nails (0.36 ppm) were somewhat higher (Liescher & Smith, 1968). This suggests that there is little tendency for arsenic to accumulate preferentially in any internal organs although it is known to occur in keratin-rich tissues (eg. nails and hair).

Levels of arsenic or its metabolites in blood, hair, nails and urine are used as biomarkers of arsenic exposure. Blood arsenic is a useful biomarker only in the case of acute arsenic poisoning or stable chronic high-level exposure. Arsenic is rapidly cleared from blood, and

hence it is difficult to measure the chemical forms of arsenic in blood. Arsenic in hair and nails can be indicators of past arsenic exposure, provided care is taken to prevent external arsenic contamination of the samples. Speciated metabolites in urine expressed either as inorganic arsenic or as the sum of metabolites (inorganic arsenic + MMA + DMA) provide the best quantitative estimate of a recently absorbed dose of arsenic (IPCS, 2001).

Ingested organoarsenicals such as MMA, DMA and arsenobetaine are much less extensively metabolised and more rapidly eliminated in urine than inorganic arsenic in animals and humans.

## **Toxicity**

Inorganic arsenic is considerably more toxic than the organoarsenicals. Within these two classes, the trivalent forms are more toxic than the pentavalent forms, at least at high doses. Arsenic is known to affect skin, and respiratory, cardiovascular, immune, genitourinary, reproductive, gastrointestinal and nervous systems.

### *Laboratory animal studies*

Acute poisoning due to inorganic arsenic ingestion can lead to severe toxic effects (including death) within 30-60 min. The most prominent effect is seen on gastrointestinal system (vomiting, intestinal injury with bleeding and diarrhoea), followed by multi-organ failures (IPCS, 1981; 2001). The oral LD<sub>50</sub> for arsenic trioxide, sodium arsenite and calcium arsenate in mice and rats ranged between 15 and 293 mg (arsenic)/kg bw. Trivalent inorganic arsenic appeared to be more toxic than pentavalent inorganic arsenic. The dermal LD<sub>50</sub> was >400 mg arsenic/kg bw in rats (IPCS, 2001). Sodium arsenite and sodium arsenate were not allergenic in guinea-pigs (maximisation test; Wahlberg & Bowman, 1986).

Sodium arsenate added to the drinking water of mice at 0.025 or 2.5 mg/L caused a dose-dependent increase in hepatic toxicity after 4 weeks (Hughes & Thompson, 1996). In rats exposed to sodium arsenate in drinking water (50 µg arsenic/mL), histopathological changes were seen in kidneys (focal changes in the glomerulus and tubules) and liver (swollen hepatocytes near the centrolobular vein). In female dogs fed a diet containing sodium arsenite at 1-8 mg/kg bw/d for up to 6 months, liver enzymes (ALT and AST activity) were elevated at ≥2 mg/kg bw/d although no histopathological changes were seen in the liver.

Embryofetal developmental effects occurred only at doses that were also toxic to the maternal animals. In these studies the no observed adverse effect levels (NOAEL) for (inorganic) arsenic acid were 0.75 and 7.5 mg/kg bw/d in rabbits and mice, respectively (Nemec *et al.*, 1998). In other studies it has been reported that arsenite was 3-10 fold more toxic than arsenate in mice and hamsters (studies evaluated by IPCS, 2001: Baxley *et al.*, 1981; Willhite, 1981; Hood & Harrison, 1982; Hood & Vedel-Macrandar, 1984; Nagymajtenyi *et al.*, 1985; Carpenter, 1987; Domingo *et al.*, 1991; Wlodarczyk *et al.*, 1996; NOAELs or NOELs not reported by the IPCS).

Gene mutation studies in bacteria or in mammalian cells gave either negative results or were found to be very weakly mutagenic. There is now growing evidence to suggest that arsenic acts as a co-mutagen or a promoter for some genotoxic mutagens, such as ultraviolet radiation (US CPSC, 2003d; IPCS, 2001). It also causes chromosomal aberrations *in vitro*, affects methylation and repair of DNA, induces cell proliferation, transforms cells, and promotes tumours. Clastogenic effects are also seen in mice.

Arsenic-induced tumours are generally not observed in whole-of-life bioassays. However, in a recent study in C57B1/6J mice (only females used) given arsenic at 500 µg/L (in drinking water) over 2 years, lung, liver, gastrointestinal and skin tumours were observed (IPCS, 2001).

Lifetime studies of rodents given roxarsone (3-nitro-4-hydroxyphenylarsonic acid, an organic arsenic compound) in their feed at doses up to 1.4 mg/kg bw/d gave no evidence of carcinogenicity in mice or rats, but a slight increase in pancreatic tumours was noted in male mice (NTP, 1989). The incidence of possible precancerous lesions in the livers of rats initiated with diethylnitrosamine was increased by subsequent exposure to DMA, suggesting that this compound could act as a cancer promoter (Johansen *et al.*, 1984), at least in animals.

#### *Human data*

Humans exposed to high concentrations of inorganic arsenic in their drinking water over long periods of time have an increased incidence of various dermatological lesions and skin cancer, and cardiovascular diseases such as peripheral vascular disease and myocardial damage. There is also evidence for chromosomal damage (clastogenic effects) in humans who have been exposed to high arsenic concentrations in drinking water (IPCS, 2001). The IPCS review reports that even with some negative findings, the overall weight of evidence indicates that arsenic can cause chromosomal damage in different cell types in exposed individuals. These gross changes to the chromosomes usually result in the affected cells not being able to divide and replicate successfully.

There is clear evidence of the carcinogenic potential of ingested inorganic arsenic in humans. Epidemiological studies conducted in Taiwan, Japan and Argentina found that people exposed to high levels of arsenic in drinking water showed increased (and dose-related) risks of skin, lung, bladder, kidney and liver cancers (Chen *et al.*, 1992; Chiou *et al.*, 1995; Hseuh *et al.*, 1995; Tseng *et al.*, 1968; Tseng, 1977; Tsuda *et al.*, 1989, 1995; Hopenhayn-Rich *et al.*, 1996). The studies are reviewed in the IPCS (2001) document on arsenic.

In several epidemiological studies on populations living in areas with elevated levels of arsenic in drinking water, skin lesions (hyperkeratosis; hyper- or depigmentation) were the most sensitive indicator of chronic arsenic toxicity (Borgono & Greiber, 1972; Borgono *et al.*, 1980; Cebrian *et al.*, 1983; Grantham & Jones, 1977; Huang *et al.*, 1985; Mazumdar *et al.*, 1988; Southwick *et al.*, 1983; Tseng, 1977; Tseng *et al.*, 1968; Valentine *et al.*, 1987; Zaldivar, 1977). The lesions were seen in the dose range of between 10 and 100 µg/kg bw/d. In studies conducted in Taiwan on 17,000 people exposed to arsenic contaminated drinking water (up to 1200 µg arsenic/L) from artesian wells, there was no evidence of skin lesions in people with an estimated mean daily intake of arsenic of 0.8 µg/kg bw/d (Tseng *et al.*, 1968; Tseng, 1977) although in another study (Cebrian *et al.*, 1983), the NOAEL for skin lesions was estimated to be somewhat lower (ie. 0.4 µg/kg bw/d; US EPA, 2001c).

Exposure to arsenic (together with other confounding factors such as other undefined water contaminants; poor nutritional status etc.; Lu, 1990) in a region of Taiwan that formerly had high levels of arsenic in drinking water has been reported to damage the vascular system, as demonstrated by the occurrence of “blackfoot disease” (progressive loss of circulation in the hands and feet, which may eventually lead to necrosis and gangrene) (Tseng, 1977). The lowest observed adverse effect level (LOAEL) for the Tseng study was 17 µg/kg bw/d. Mortality rates from diabetes mellitus were also found to be higher in the blackfoot disease endemic area (IPCS, 2001).

Neurological effects (including tingling, numbness and peripheral neuropathy) have also been reported to be associated with elevated levels of arsenic in drinking water. Evidence of hepatic damage (enlarged liver, elevated levels of liver enzymes and portal tract fibrosis) has been reported after exposure of arsenic by the oral route, with LOAELs in the range of 20-100 µg/kg bw/d (ATSDR, 2000).

Occupational exposure to arsenic, primarily by inhalation, is causally associated with lung cancer. Increased risks have been observed at cumulative exposure levels  $\geq 0.75$  (mg/m<sup>3</sup>)· year (e.g. 15 years of exposure to a workroom air concentration of 50 µg/m<sup>3</sup>). Tobacco smoking has been investigated in two of the three main smelter cohorts and was not found to be the cause of the increased lung cancer risk attributed to arsenic (IPCS, 2001).

#### *Mechanism of carcinogenicity*

A number of *in vitro* studies suggest that arsenic can act to promote or enhance carcinogenicity of other agents by effects such as oxidative DNA damage, altered DNA methylation and gene expression, inhibition of enzymes involved in cellular energy production, DNA repair, and other stress-response pathways, altered function of the glucocorticoid receptor, and other effects concerning signal transduction, cell-cycle control, differentiation, cytotoxicity, and apoptosis. Many of these effects could be involved in arsenic-related carcinogenesis, although induction of apoptosis could act to prevent cancer (US CPSC, 2003d). Arsenic-induced apoptosis has been suggested to have an important role in the treatment of acute promyelocytic leukaemia (NRC, 2001).

#### **Health standards**

Although exposure to high concentrations of inorganic arsenic results in tumour formation and chromosomal damage (clastogenic effect), the mechanism by which these tumours develop does not appear to involve mutagenesis. Arsenic appears to act on the chromosomes and acts as a tumour promoter rather than as an initiator (Gebel, 2001; Simeonova & Luster, 2000; Wang *et al.*, 2002). Furthermore, the epidemiological evidence from occupational exposure studies indicates that arsenic acts at a later stage in the development of cancer, as noted with the increased risk of lung cancer mortality with increasing age of initial exposure, independent of time after exposure (Brown & Chu, 1983). Hence arsenic appears to behave like a carcinogen which exhibits a threshold effect. This would also be conceptually consistent with the notion that humans have ingested food and water containing arsenic over millennia and so the presence of a threshold seems likely. Nevertheless the mechanism by which tumour formation develops following arsenic exposure has been and still continues to be a source of intensive scientific investigation.

While several epidemiological studies suggest the existence of a threshold effect there is considerable debate regarding the most appropriate dose-response relationship to quantify the cancer risks from arsenic exposure (Beck *et al.*, 1995; Chappell *et al.*, 1997). Studies conducted in Taiwan showed a causal relationship (with dose-dependency) between exposures of high arsenic water content in drinking water and risks of cancers, with a threshold for cancer, especially for skin cancers. Skin cancers appear to be the most sensitive indicator of carcinogenicity of inorganic arsenic in humans, with a threshold of 2.9 µg/kg bw/d. This level, rounded-off to 3 µg/kg bw/d, has been taken to be the provisional maximum tolerable daily intake (PTDI) of arsenic in food (FSANZ, 1999). The tolerable intake is the amount that can be ingested daily without any appreciable health risk for a lifetime exposure. However, the aggregate exposure which includes all other sources apart from food may be high for some children depending on their age, geographical location, housing environment and daily activity.

Based on a number of epidemiological studies the Joint Expert Committee on Food Additives (JECFA) concluded in 1983 that arsenic toxicity (arsenicism) can be associated with water levels containing an upper arsenic concentration of 1 mg/L or greater, and a concentration of 0.1 mg/L may give rise to 'presumptive signs of toxicity'. Assuming a daily water intake of 1.5 L, JECFA concluded that intakes of 1.5 mg/d of inorganic arsenic are likely to result in chronic arsenic toxicity and daily intakes of 0.15 mg (150 µg) may also be toxic in the long term to some individuals. On the basis of available data, JECFA recommended a provisional weekly

intake of 15 µg/kg bw (~2 µg/kg bw/d), and recommended further epidemiological studies in populations exposed to elevated levels of inorganic arsenic occurring in drinking water, in order to define more clearly levels of inorganic arsenic which may cause adverse effects. In 1989, JECFA confirmed the provisional maximum tolerable weekly intake (PTWI) of 15 µg/kg bw.

### **Exposure**

Since small children aged 3 to 5 have a high food intake relative to their bodyweight and are the ones most likely to display hand-to-mouth behaviour and ingest soil the following exposure estimates are focussed on this group.

### **Estimate of daily arsenic intake in children**

Using available data, total daily intake (average intake) of arsenic is estimated in the following sections.

### **Factors and assumptions used in the exposure assessment calculation**

#### **Non-playground related exposure**

##### **Intake from food**

The total intake of arsenic (organic + inorganic) from food by toddlers (2 years of age) in Australia has been estimated to be 0.55-1.3 µg/kg bw/d (0.28-0.83 µg/kg bw/d for boys and girls aged 12 years; FSANZ, 2002). The maximum intake value for toddlers was selected for the risk assessment. The proportion of inorganic arsenic in the total arsenic content in food has been estimated to be up to a maximum of 6% (FSANZ, 1999). Hence the maximum daily intake of inorganic arsenic in children was taken as 1.3 x 6% which is 0.078 µg/kg bw/d.

##### **Intake from water**

Water intake in a child aged 3-5 years has been estimated to be 0.87 L/d (US EPA, 1997b). According to the Australian Drinking Water Guidelines (ADWG), the concentration of arsenic in drinking water should not exceed 7 µg/L (NHMRC, 2003). Hence for the purposes of this intake assessment, the maximum arsenic (mostly in the form of inorganic arsenic) intake from in drinking water was estimated to be 0.87 x 7 = 6.09 µg/d/child.

##### **Intake from air**

Arsenic concentration in the air is reported to be typically in the range of 0.2-1.5 ng/m<sup>3</sup> in rural areas and 0.5-3 ng/m<sup>3</sup> in urban areas (IPCS, 2001). The US EPA (2002) has estimated that a child's (3-5 years) intake of air is 8.7 m<sup>3</sup>/d. These values (arsenic: 3 ng/m<sup>3</sup>; air intake: 8.7 m<sup>3</sup>/d) were used in the estimating a child's intake of arsenic from the air (3 ng x 8.7 = 26.1 ng = 0.026 µg/d). Systemic absorption from the lungs was assumed to be 100%.

##### **Intake from soil (non-playground)**

##### **Oral ingestion**

According to Smith *et al.* (2003), the arsenic content of Australian soils ranges between 1 and 50 mg/kg, with a mean value of 5-6 mg/kg. This background concentration range is similar to the values reported for urban and rural soils in Queensland (see below). Based on these values the background soil level of arsenic was taken as 6 mg/kg (or 0.006 µg/mg soil).

The soil ingestion rate was taken to be 100 mg/d/child and the oral bioavailability of arsenic in soil was taken to be 25% (see below under 'Intake of arsenic from contact with soil in playgrounds').

#### **Background arsenic levels in urban and rural soils (Queensland data)**

<b>Sample</b>	<b>Arsenic level (mg/kg soil)</b>
Rural soils	<5-40
New suburb	3-31
Old suburb	3-27

Source: Smith *et al.* (2003).

#### **Dermal absorption**

The values used for 'Intake of arsenic from contact with soil in playgrounds' (see below) were used for estimating dermal absorption of arsenic from non-playground soil (surface area: 1640 cm<sup>2</sup>; soil adherence factor: 0.2 mg/cm<sup>2</sup>; bioavailability: 4.5%) except that the soil arsenic concentration was assumed to be 6 mg/kg.

#### **Playground-related exposure**

#### **Intake of arsenic from contact with wood**

##### **Oral intake**

*Handload (amount of arsenic on hands):* The studies conducted by US Consumer Product Safety Commission (US CPSC, 2003a-k) were chosen as the most appropriate of the available studies for determining the amount of arsenic transferred to a child's hand when playing on wood treated with CCA. The mean value for this parameter was 7.6 µg arsenic (US CPSC, 2003a, see section 6). This value was used in the exposure calculation.

*Hand-to-mouth transfer:* The US CPSC (2003a) estimated that an average of 43% of the arsenic residue on children's hands is transferred to their mouths during the day. This transfer to the mouth includes incidental and indirect contact (food, toys, etc.), as well as direct mouthing from the hand (US CPSC, 2003e). A hand-to-mouth transfer factor of 0.43 appropriate was used in the current exposure calculation.

*Bioavailability:* Studies on bioavailability of arsenic from CCA-treated wood are not considered adequate since only two studies (dogs and pigs) are available, and both were based on urinary excretion of arsenic. In the study in dogs, data were not normalised using data obtained after IV administration of arsenic. Insufficient details were available for the study in pigs. Based on these constraints a conservative value of 100% was assumed for the bioavailability of arsenic from CCA-treated wood.

*Frequency of playground use:* As suggested by the US CPSC (2003a, j), the frequency of children's contact with CCA-treated playground equipment is assumed to be 156 days per year (3 days a week). It is noted that some risk assessments also take into account the amount of time per day that a child plays on the playground. However, the US CPSC stated that the method used by CPSC staff for estimating the amount of arsenic residue that a child might ingest does not depend on the amount of time per day (hours/d) the child spends on the playground.

### **Dermal intake**

*Adherence to skin:* The amount of arsenic which adheres to a child's skin is assumed to be similar to that which adheres to the hands ( $7.6 \mu\text{g}/\text{cm}^2$ , see above). For a child aged 2-6.5, the mean palm side surface for both hands is  $129 \text{ cm}^2$  (Snyder *et al.*, 1997). For comparison, the area of hand palm, including fingers, for an adult was measured to be  $141 \text{ cm}^2$  (thus the palm area of two hands of a child is approximately equivalent to that of one adult palm). Based on this, adherence of arsenic to skin is calculated to be  $0.06 \mu\text{g}/\text{cm}^2$  ( $7.6 \mu\text{g}/129 \text{ cm}^2$ ).

The surface area of contact was taken as  $1640 \text{ cm}^2$ , the area (upper percentile for a 3 year old child: exposed skin surfaces of hands, legs, arms) recommended by the US EPA (2001a).

*Bioavailability:* The dermal bioavailability of arsenic from CCA-treated wood was  $<0.01\%$  in monkeys (Wester *et al.*, 2003). For the purposes of a conservative risk assessment, a value of  $0.1\%$  was used.

*Frequency of playground use:* As before (156 days per year).

### **Intake of arsenic from contact with soil in playgrounds**

#### **Oral intake**

*Amount of arsenic in soil:* Studies on soil concentrations of arsenic in playgrounds have not been conducted in Australia. Studies evaluated by the US EPA revealed that mean values of arsenic ranged from 6 to 24 mg/kg soil in 5 studies although one study (Stilwell & Gorny, 1997) reported a high value of 76 mg/kg soil. The overall mean values in all these studies (mean of all means) was 27.2 mg/kg (see US EPA, 2001a in section 7). In a laboratory study simulating weather conditions in Brisbane, the maximum estimated cumulative soil concentration of arsenic (due to leaching from CCA-treated pine deck, after a rainfall of 7300 mm; see Kennedy & Collins, 2001 in section 7) was found to be similar to this value (33.1 mg/kg soil).

Based on the above, in the absence of Australian data on soil content of arsenic in different playgrounds, a value of 30 mg arsenic/kg soil was selected for the risk assessment. The background value (6 mg/kg) was deducted from the playground soil value of 30 mg/kg to calculate the arsenic content in soil (24 mg/kg;  $0.024 \mu\text{g}/\text{mg}$  soil) due to leaching of arsenic from CCA-treated wood.

*Soil ingestion:* The soil ingestion rate of 100 mg/d/child, as recommended by the US EPA (2001a, b), was used.

*Bioavailability:* Although bioavailability studies on arsenic have been conducted in different species (absolute bioavailability up to 33%), studies conducted in monkeys are

considered the most relevant for estimating human exposure. In monkeys, absolute bioavailability was in the range 8-14% (2 studies did not report any value) while the relative bioavailability with respect to that of soluble arsenic was 11-25%. Based on this information, the value chosen for arsenic bioavailability was 25%.

### **Dermal intake**

*Surface area of contact:* As for the dermal intake of arsenic from contact with wood in playgrounds (ie. skin area of 1640 cm<sup>2</sup>).

*Soil adherence factor:* The soil adherence factor is the amount of soil which adheres to the skin. The US EPA (Superfund RAG, Part E; Supplemental Guidance for Dermal Risk Assessment, draft 2000; cf: US EPA, 2001a) estimated an activity-specific surface area weighted soil adherence factor for a child (1-6 years old) resident at a day care centre to be 0.2 mg/cm<sup>2</sup>, although for a hand contacting commercial potting soil (in lieu of playground soil), the factor is 1.45 mg/cm<sup>2</sup>. Another assessment by US EPA (see table below) also estimated a factor of 0.2 mg/cm<sup>2</sup> for children playing in dry (90<sup>th</sup> percentile) or wet soil (50<sup>th</sup> percentile). For the current assessment, the value of 0.2 mg/cm<sup>2</sup> was considered appropriate.

**Activity specific surface area weighted soil adherence factor**

Exposure scenario	Age (year)	Soil Adherence Factor (mg/cm <sup>2</sup> ) <sup>a</sup>	
		50 <sup>th</sup> percentile	90 <sup>th</sup> percentile
Children playing in dry soil	8-12	0.04	0.2
Children playing in a day care center	1-6.5	0.06	0.2
Children playing in wet soil	8-12	0.2	2.7
Kids-in-mud	9-14	22 <sup>b</sup>	123 <sup>b</sup>

Source: US EPA Superfund, 2000 (cf. US EPA, 2001a). <sup>a</sup>Weighted adherence factor based on exposure to face, forearms, hands, lower legs and feet. <sup>b</sup>According to the US EPA, these are significant overestimation and will not be used (for risk assessment).

*Bioavailability:* Based on a study in monkeys (Wester *et al.*, 1993, see section 5), the dermal bioavailability was taken as 4.5%.

*Soil concentration in playground:* As for the oral intake of arsenic from contact with soil in playgrounds (ie. 24 mg arsenic/kg soil, after deducting background arsenic value).

**Calculation of daily exposure to arsenic in children**

***Non-playground-related exposure***

Arsenic (inorganic) intake	Factors used <sup>#</sup>	Estimated daily intake for a child aged 3 years
Intake from food	0.078 µg/kg bw/d; bw: 15 kg for a 3-year old child	0.078 x 15 = 1.2 µg
Intake from drinking water	Water intake, 0.87 L/d. arsenic content, up to 7 µg/L	0.87 x 7 = 6.1 µg
Intake from air	Arsenic in air: 0.003 µg/m <sup>3</sup> ; air intake: 8.7 m <sup>3</sup> /d	0.003 x 8.7 = 0.026 µg
Intake from non-playground soil - oral ingestion	Soil content: 0.006 µg arsenic/mg soil; soil ingestion = 100 mg/child; bioavailability = 25%	0.006 x 100 x 25% = 0.15 µg
Intake from soil - dermal	Skin contact area = 1640 cm <sup>2</sup> ; soil adherence = 0.2 mg/cm <sup>2</sup> ; soil content of arsenic: 0.006 µg arsenic/mg soil; bioavailability = 4.5%	1640 x 0.2 x 0.006 x 4.5% = 0.09 µg
Total daily arsenic intake = 1.2+6.1+0.026+0.15+0.09 = 7.57 µg/child (0.50 µg/kg bw/d, for a 15 kg child)		

<sup>#</sup>See above for factors used in the calculation.

**Playground-related exposure**

<b>ARSENIC (INORGANIC) INTAKE</b>	<b>Factors used<sup>#</sup></b>	<b>ESTIMATED DAILY INTAKE FOR A CHILD AGED 3 YEARS</b>
<b>Intake from contact with CCA-treated timber in playgrounds</b>		
Arsenic – oral intake	7.6 µg/handload; hand-to mouth transfer: 0.43; bioavailability: 100%; contact days: 156/year	$7.6 \times 0.43 \times 100\% \times 156/365 = 1.4 \mu\text{g}$
Arsenic – dermal intake	0.06 µg arsenic/cm <sup>2</sup> skin area; contact area: 1640 cm <sup>2</sup> (for a 3-year child); bioavailability = 0.1%; contact days: 156/year	$0.06 \times 1640 \times 0.1\% \times 156/365 = 0.04 \mu\text{g}$
<b>Intake from contact with soil in playgrounds (containing CCA-treated timber structures)</b>		
Arsenic – oral ingestion	Soil ingestion = 100 mg/child; 0.024 µg arsenic/mg soil; bioavailability = 25%; contact days: 156/year	$100 \times 0.024 \times 25\% \times 156/365 = 0.26 \mu\text{g}$
<b>ARSENIC – DERMAL INTAKE</b>	Soil adherence to skin = 0.2 mg soil/cm <sup>2</sup> ; 0.024 µg arsenic/mg soil; skin area of contact = 1640 cm <sup>2</sup> ; bioavailability = 4.5%; contact days: 156/year	$0.2 \times 0.024 \times 1640 \times 4.5\% \times 156/365 = 0.15 \mu\text{g}$
<b>Total:</b> $1.4 + 0.04 + 0.26 + 0.15 = 1.85 \mu\text{g/child}$ (0.12 µg/kg bw/d, for a 15 kg child)		

BW for a 3-year child: ~15 kg. <sup>#</sup>See above for factors used in the calculation.

**Total daily intake of arsenic from all sources**

Non-Playground	0.50 µg/kg bw
Playground	0.12 µg/kg bw <sup>#</sup>
<b>Total</b>	<b>0.62 µg/kg bw</b>

The above estimate indicated that the total intake of a child is ~0.6 µg/kg/d. Of this, ~20% intake is from playgrounds. In the estimate, lifetime average daily intake (based on 75 years or 27400 days of living; US CPSC, 2003) will be much lower (~7% of the value, for playground-related exposures only).

**Risk to humans from exposure to inorganic arsenic in CCA-treated timber**

There are no suitable studies conducted in Australia which could be used to estimate a child's exposure to the components of CCA leached from CCA-treated wood. Available studies (mostly overseas) indicated that CCA residues transferred from wood surface to a child's hand (on contact) or to a surrogate (such as a polyester wipe) were quite variable. Thus, in exposure studies conducted by the US CPSC which were used in health risk assessments for children (US CPSC, 2003a; see section 6), the amount of arsenic picked up by dry polyester wipes was in the range of 1.6 to 168.5 µg, ie. there was an approximately 100-fold variability. Such a high variability in arsenic transfer was also noted in several other studies.

The estimated aggregate background inorganic arsenic intake for an average 3-5 year old child from air, food, drinking water and soil was approximately 7.57 µg/child/d (or 0.50 µg/kg bw/d for a 15 kg child). For child playing on or near a CCA-treated timber structure the increase in exposure to arsenic was 0.13 µg/kg bw/d. The combined amount, 0.63 µg/kg bw/d, was below the tolerable daily intake of approximately 2 µg/kg bw/d set by the Joint Food and Agriculture Organisation/World Health Organisation Expert Committee on Food Additives (JECFA) and about 3 µg/kg bw/d set by Food Standards Australia New Zealand (IPCS, 1989; FSANZ, 1999). The tolerable intake is the amount which can be ingested daily per kilogram of body weight and represents a level of no appreciable health risk for a lifetime exposure.

### 3 Conclusions

A very limited amount of Australian data was available to quantify the amount of arsenic that can transfer or leach from 'in-service' CCA-treated timber structures. It is acknowledged that the use of overseas data to determine the extent of release of dislodgeable residues from treated timber has limitations, in view of the fact that the amount and quality of this data is also limited and further, that it is not known whether timber treated with CCA in Australia and exposed to local climatic conditions would have a similar degree of variability in amounts of arsenic released from ostensibly similar treated timber structures. However, based on a consideration of the exposure to CCA-treatment timber products, in particular children's play equipment, there was no compelling evidence from the available data to conclude that there was likely to be an unacceptable risk to public health from exposure to arsenic from CCA-treated timber. Based on this finding, there would not seem to be any good justification for taking immediate action to remove existing CCA-treated playground structures.

There was considerable variability in the quantity of dislodgeable arsenic residues on CCA-treated timber play equipment. The reasons for this variability were not readily apparent from the limited nature of the studies available for evaluation. Further studies would be required to provide a better understanding of this variability. Additionally measurements of CCA that has leached from treated timber into surrounding soil would also aid in refining the risk assessment. In the absence of these data, ongoing registration of CCA timber treatment products could no be supported from the toxicological assessment.

### 4 Studies on bioavailability of arsenic from wood and soil

#### *Oral bioavailability of arsenic from CCA-treated wood*

Assay	Source of material	BIOAVAILABILITY OF ARSENIC (%)		Reference
		Absolute	Relative <sup>a</sup>	
Pigs (juvenile)	Several wood boards	23	29	Casteel <i>et al.</i> (2003)
Dogs	Sawdust	26-40	-	Peoples, 1976; Peoples & Parker, 1979

<sup>a</sup>Relative to orally administered water-soluble arsenic (sodium arsenate). '-' Not known.

**Selected value for risk assessment:** Studies on bioavailability of arsenic from CCA-treated wood are not considered adequate since only two studies (dogs and pigs) are available, and both were based on urinary excretion of arsenic. In the study in dogs, data were not normalised using data obtained after IV administration of arsenic. Insufficient details were available for the study in pigs. Based on these constraints a conservative value of 100% was assumed for the bioavailability of arsenic from CCA-treated wood.

#### *Dermal bioavailability of arsenic from CCA-treated wood*

Species	Source of material	BIOAVAILABILITY OF ARSENIC (ABSOLUTE, %)	Reference
Monkeys	Wood residue	<0.01 <sup>a</sup>	Wester <i>et al.</i> (2003)
Dogs	Saw dust	n.d. <sup>b</sup>	Peoples (1979)

<sup>a</sup>Bioavailability of soluble arsenic is 2.8%. <sup>b</sup> n.d., not detected, urinary levels were below limit of detection (0.07 ppm).

*Selected value for risk assessment:* 0.1% bioavailability (0.01% conservatively taken to be 0.1%).

**Oral bioavailability of arsenic from soil (arsenic or CCA-contaminated)**

Species	Source of soil	BIOAVAILABILITY OF ARSENIC (%)		Reference
		Absolute	Relative <sup>a</sup>	
Rats	Cattle dip soil	<10	60 <sup>b</sup>	Ng <i>et al.</i> , 2003
Rabbits	Smelter soil	~20	~50	Freeman <i>et al.</i> , 1993
Dogs	Mine site	8	-	Groen <i>et al.</i> , 1994
Monkeys	Electrical substation; cattle dip site; pesticide site; wood treatment site	-	11-25	Roberts <i>et al.</i> , 2002
Monkeys	Soil from a former CCA wood treatment facility	-	16	Roberts & Bergeron., 2003
Monkeys	Smelter soil	11-14	12-20	Freeman <i>et al.</i> , 1995
Pigs <sup>c</sup>	Utility pole soil	33	49	Casteel <i>et al.</i> , 2003
Pigs <sup>c</sup>	Soil or slag	-	78	US EPA, 1996
Pigs <sup>c</sup>	Soil; smelter slag; mine tailings	-	35-45	US EPA, 1997a

<sup>a</sup>Relative to orally administered water-soluble arsenic (sodium arsenate). <sup>b</sup>Relative bioavailability compared to sodium arsenite and calcium arsenite is 8% and 14%, respectively. <sup>c</sup>Juvenile or immature pigs. '-' Data not available or reported.

*Selected value for risk assessment:* 25% bioavailability (monkeys).

**Dermal bioavailability of arsenic from soil (arsenic or CCA-contaminated)**

Species	Source of soil	BIOAVAILABILITY OF ARSENIC (%)		Reference
		Absolute	Relative <sup>a</sup>	
Monkeys	Soil mixed with radioactive arsenic-73 (arsenic acid)	3.2-4.5	70-160	Wester <i>et al.</i> , 1993

<sup>a</sup>Relative to dermally applied arsenic acid in water (bioavailability of arsenic acid in water was 2.0-6.4%).

*Selected value for risk assessment:* 4.5% bioavailability.

**5 Studies on dislodgeable arsenic, chromium and copper from cca-treated wood**

*Deukmejian G & Kizer KW (1987) Report to the Legislature. Evaluation of hazards posed by the use of wood preservatives on playground equipment. Office of Environmental Health Hazard Assessment. Department of Health Services, Health and Welfare Agency, State of California. February 1987. Submitted by Osmose, Unpublished Report No. 3.10.7 (Part 3, vol 2/7, p319-359), NRA Study No. 6923. [OCS Submission No. 12275, vol 2 of 13]*

Based on a study conducted by the Californian Department of Health in 5 volunteers (age or sex not stated) by rubbing hands on playground wood for 5 min, the estimated dose per visit from playground structure treated with CCA are shown in the table.

***Estimated exposure of arsenic in children***

	Amount child gets on hands (µg)		Estimated dose per visit (µg) <sup>#</sup>		
	Mean	Maximum	Low	Moderate	High
Arsenic	236	1260	24	60	630
Chromium	351	575	35	90	288
Copper	171	362	17	40	181

<sup>#</sup>Assuming 50% oral ingestion (of the amount on hands) and 20%, 50% and 100% oral bioavailability for the low, moderate and high estimates, respectively. For the low and moderate estimate doses, mean amount of the chemical on the child's hands was taken into account; for the high estimate, the maximum amount on the child's hands was taken into account.

Sealing is reported to reduce surface residue levels of arsenic. Thus, at Cedar Rose Park in Berkeley, surface arsenic residues collected on gauze-wipe samples were reduced from a range of 31-314 µg/100 cm<sup>2</sup> before sealing to 1-13 µg/100 cm<sup>2</sup> after sealing with an oil-base stain.

*Note:* The report does not contain individual data or details about methodology (information regarding how the samples were collected from hand, the number of times the wood surface is rubbed, the type of CCA-treated structure tested).

***Lee BC (1987) Estimate of risk of skin cancer from dislodgeable arsenic on pressure treated wood playground equipment. Memorandum from Lee BC to Tyrrell EA. US Consumer Product Safety Commission, Washington DC. 26 January 1990. Submitted by Osmose, Unpublished Report No. 3.10.8 (Part 3, vol 2/7, p360-429), NRA Study No. 6924. [OCS Submission No. 12275, vol 2 of 13]***

Seven pieces of CCA-treated (pressure treated and 'finished' with stain and/or stain, see table) lumber (pine wood) and one piece of unfinished (CCA-treated) wood were purchased from local lumbar yards. The woods had a CCA retention of 0.4 lb/ft<sup>3</sup> (6.4 kg/m<sup>3</sup>). Five sub samples from each of the samples were randomly selected for dislodgeable arsenic measurement. A rectangular shaped area (8 x 50 cm) was marked on the flat surface of each sub-sample. A wood block (surface area stated, but not legible: 8 x .. sq cm) was cut from arsenic-free wood. A nylon cloth was attached to one side of the block, with a backing paper between the fabric and carrier block, to prevent cross contamination of the samples. The weight of the block was adjusted to 1 kg with a water filled bottle on top of the block. The wood block was dragged 10 times backward and forward over the marked surface, with the nylon cloth wipe touching it. The wiping cloth was transferred to 50 mL conical flasks and 25 mL of HCL (0.01 N) was added to each flask. Extractions were performed with intermittent mixing for 18-24 h. The extracts were analysed for arsenic content using an ICP spectrometer. Detection limit was 6.25 µg/100 cm<sup>2</sup>.

The study also compared dislodgeable arsenic using chamois leather (animal skin) and nylon cloth as the wiping medium, using two playground equipment samples (with 3 sub samples for each).

The effect of coating wood samples with oil-based stain and a water repellent/sealant was also studied. The coated wood samples were allowed to cure for one week at room temperature in a ventilated room before testing for dislodgeable arsenic.

*Results:* There was a high degree of variation in dislodgeable arsenic in CCA-treated wood (finished with stain or sanded). The highest average dislodgeable arsenic level of 68 µg/100 cm<sup>2</sup> was found in a sample of unfinished pressure treated timber (not playground equipment wood). The results are shown in the following table.

***Mean dislodgeable arsenic in CCA-treated pine wood samples***

Sample	Treatment (in addition to CCA)	Dislodgeable arsenic ( $\mu\text{g}/100\text{ cm}^2$ )
Southern pine wood	Surface treated with a semi-transparent stain	<LOQ
Southern yellow pine wood	Lumber was sanded and molded	<LOQ
Southern pine wood	No stain, sanding or molding	<LOQ
Southern yellow pine wood	Lumber was sanded and molded	<LOQ
Southern yellow pine wood	Surface treated with an oil-base stain	<LOQ
Lodge Pole Pine construction log	Surface treated with stain	32.1
Pine wood	Surface was sanded	21.9
Southern yellow pine wood	Unfinished (not playground equipment)	68.8

<LOQ: limit of quantitation,  $6.25\ \mu\text{g}/100\text{ cm}^2$ . Nylon cloth was used as a wiping cloth.

In the two CCA treated timber (finished) samples which showed detectable levels of dislodgeable arsenic (see table above), dislodgeable arsenic was comparable when chamois leather or nylon cloth was used ( $24.0$  and  $36.3\ \mu\text{g}/100\text{ cm}^2$ , respectively). Coating of CCA-treated wood with oil stain or water repellent/sealant did not decrease the amount of dislodgeable arsenic from the wood (before coating:  $27.0\ \mu\text{g}/100\text{ cm}^2$ ;  $29.9$  and  $33.3\ \mu\text{g}/100\text{ cm}^2$  after coating with oil stain or repellent/sealant, respectively).

*Note:* Mean arsenic content in the wood samples varied from  $3.06$ - $3.89\ \mu\text{g}/\text{mg}$ , copper was  $1.98$ - $2.43\ \mu\text{g}/\text{mg}$  whilst chromium was not measured. In the study, non-treated wood samples were not used as controls since no dislodgeable arsenic was detected in samples of non-treated wood during methodology development.

Gradient Corporation (2001) Evaluation of Human health risks from exposure to arsenic associated with CCA-treated wood. Gradient Corporation, 238 Main Street, Cambridge, MA 02142. Submission by Osmose Inc. & Koppers Arch (Australia) Pty Ltd. Unpublished Report No. 3.10.9 (201068/reports) dated 5 October 2001. Part 3, vol 3/7, p 430-692; NRA Study Nos. 6925 & 6748, [OCS Submission No. 12275, vol 3 of 13; 9 of 13]

*The following information is from the evaluation report prepared by Gradient Corporation, on studies related to dislodgment of arsenic from CCA treated wood. The original studies were not available to the OCS for evaluation.*

The Maine Department of Human Services (MEDHS, 1998) conducted a 'hand loading' (transfer of arsenic from the surface of CCA-treated wood to hand) study conducted in 1997 and 1998 using a single 3-year old CCA-treated residential deck located in the central portion of the state of Maine. An adult male volunteer gently rubbed (using a single hand, wet or dry) a  $1350\text{ cm}^2$  and a  $12600\text{ cm}^2$  section of railing for different time periods (3 s to 1 min). Arsenic was removed from the hand for laboratory analysis using a wipe from a lead dust sampling kit. The study also measured the amount of arsenic on the hands of a 20-month old child allowed to play freely on the same CCA-treated deck for a period of less than 10 min.

The results revealed that a wet hand removed more arsenic than a dry hand. Repeated rubbing (4 times) of the same section of wood did not appear to 'deplete' arsenic from the wood surface. There was a slight increase in the amount of hand arsenic levels with longer rubbing periods, but there was 'little difference between the amount of arsenic loaded onto the child's hands compared to the adult volunteer'.

***Arsenic levels on hands (in different trials)***

Adult/child	Dry/wet hand	Arsenic level	
		Per hand (µg)	µg/cm <sup>2</sup> hand area <sup>#</sup>
Adult	Dry hand	6-88	0.02-0.33
	Wet hand	37-110	0.12-0.39
Child (20 months)	(presumably dry hand)	11-28 (22-55 µg for both hands)	0.21-0.53

<sup>#</sup>Assuming the adult hand surface area available for contact with a treated surface area is 267 cm<sup>2</sup>, which is ~1/3 of the total surface area of both hands as reported in US EPA (Exposure Factors Handbook, 1997). For a child, the hand surface area available for contact with a treated wood surface is assumed to be 104 cm<sup>2</sup> (52 cm<sup>2</sup>/hand), which is ~1/3 of the total surface area of both hands for a 2-year old child. A 5 µg correction was applied based on data from blank studies.

In a hand loading and wipe study conducted by Scientific Certification Systems (SCS, 1998), seven different samples of CCA type C (from Osiose)-treated wood and one control sample of untreated wood were used. In addition to these wood samples which were Southern Pine, a treated Hemlock/Fir was used. One of the pine wood samples was treated with CCA as well as a proprietary water repellent (Osiose). Further treatment of some of the wood samples was performed by SCS prior to testing, and included staining, sealing, and cleaning with two different types of brightening agents (to remove the grey colouring of wood when exposed to outdoor conditions). One of the wood samples were aged 5 years.

Hand sizes (left and right) for 5 adult male volunteers (18-40 years) were measured by tracing the outline of each hand. The volunteers rubbed each wood sample 10 times with each hand. Each hand was subsequently rinsed 3 times with reagent grade water and the rinsate was collected and analysed for arsenic, chromium and copper.

Details about the wipe study (including area wiped) were not provided.

The results are shown in the table below:

***Arsenic content on hand surface and in wipe samples***

Type of wood	Mean arsenic content per hand surface area (maximum; 95% upper confidence limit on the mean) µg/cm <sup>2</sup>	Mean arsenic content per wipe (µg)
Untreated Southern Pine	0.003 (0.005; 0.012)	0.91
CCA-treated Southern Pine	0.039 (0.061; 0.107)	15.5
CCA-treated Southern Pine, stained	0.025 (0.030; 0.040)	22.9
CCA-treated Southern Pine, sealed	0.006 (0.012; 0.016)	6.5
CCA- and water repellent-treated Southern Pine	0.100 (0.130; 0.174)	27.4
CCA-treated Southern Pine, brightener treatment	0.020 (0.044; 0.059)	12.9
CCA-treated Southern Pine, aged	0.051 (0.066; 0.093)	3.2
CCA-treated Southern Pine, brightener treatment	0.020 (0.030; 0.033)	4.4
CCA-treated Hemlock/Fir	0.095 (0.120; 0.159)	57.4

n = 10 for hand loading data (from 5 volunteers) and 5 for wipe data, for each wood; mean hand (single) surface area: 147.8 cm<sup>2</sup>. Data source: Scientific Certification Systems (SCS, 1998). Metal removal from CCA-treated lumber under simulated normal use conditions. Report to Osiose. November 23.

*Note:* Individual data were provided for this study.

The Gradient report also lists the following wipe studies (the study reports, except that of Riedel *et al.*, 1990, were not submitted to OCS for evaluation):

*Wipe studies conducted with CCA-treated wood*

Study No.#.	Methods and timber samples	Results (arsenic content)	Comments
1	Wet Kimwipe tissues rubbed over 100 cm <sup>2</sup> of CCA-treated wood. '2 x 4 Southern Pine boards' (stained) and 1" dowels (bare wood).	Mean wipe loading: 7.5 µg/100 cm <sup>2</sup> . (range: 0.6-23.8 µg/cm <sup>2</sup> ). n = 20	Background arsenic in the 'controls': 1.6 µg/100 cm <sup>2</sup> .
2	Wet Kimwipe tissues rubbed over 100 cm <sup>2</sup> of CCA-treated and weathered wood. 2 x 4 Southern Pine boards (stained) and 1" dowels (bare wood) that had been weathered for 1 year.	Mean wipe loading of weathered wood: 6.3 µg/100 cm <sup>2</sup> . (range: 3.7-8.2 µg/cm <sup>2</sup> ). n = 10	Background arsenic in the 'controls': 0.34 µg/100 cm <sup>2</sup> .
3	Wood-rubbing sampler with a cotton gauze pad (dry or wet) with an acidic wash moving one cycle back and forth each second (time rubbed is not stated). CCA-treated jack-pine wood, new or weathered for 1 year.	New wood yielded more arsenic on wipe loads, and yields were highest with pH 1.0 solution applied. 350 µg arsenic/100 cm <sup>2</sup> for new wood; 150 µg/100 cm <sup>2</sup> for weathered wood. (Mean for new and weathered wood: 250 µg/100 cm <sup>2</sup> ).	Mechanised rubbing of wood is likely to produce higher arsenic loads than more casual contact, particularly in the wet contact measurements.
4	Nylon covered 1 kg block wiped over a 400 cm <sup>2</sup> area of wood; nylon to leather chamois comparison; sealed wood to unsealed wood comparison. 7 Southern Pine wood samples retaining CCA at 0.40 lb/ft <sup>3</sup>	Average of 27 µg/100 cm <sup>2</sup> in 2 of 7 samples (other 5 'ND's); nylon dislodge equalled or exceeded that of leather; oil stain or water sealant did not significantly reduce arsenic dislodgeability.	
5	Wet cotton gauze pad (10 cm x 10 cm, folded into 5 cm x 10 cm; moistened with 3 mL distilled water) rubbed on 2 m (length; area: 0.025 m <sup>2</sup> ) of CCA-treated wood; unknown playground wood with unknown stains and finishes at 10 playgrounds aged 10+ years or 2 years in Eastern Ontario, Canada.	Mean wipe loading: 8.6 µg/100 cm <sup>2</sup> arsenic (range: 0.1-64.4 µg/100 cm <sup>2</sup> ). n = 40	Older playground wood yielded less dislodgeable arsenic.
6	Nylon covered 1 kg block wiped over a 400 cm <sup>2</sup> area of wood in accordance with CPSC (1990) protocol. 24 Southern Pine boards and dimension lumber purchased from different lumberyards, and treated with CCA from different chemical suppliers.	16.8 µg arsenic/100 cm <sup>2</sup> (average). Concentration in wood: ~0.4/ft <sup>3</sup> .	
7	Wet gauze pad dragged under a 575 g block across 2 metres of CCA-treated wood aged 3 months with a second sample taken at 5 months post-construction. Research site with test structure built with treated white pine and red pine wood having either a varnish, sealer, stain or no finish coat.	Mean wipe loading: 233.5 µg arsenic/100 cm <sup>2</sup> (range: 10-960 µg/100 cm <sup>2</sup> ). n = 28.	Wipe samples taken from the treated wood aged 3 months were consistently higher than the samples taken at 5 months, regardless of finish coats and sealant treatments.

8	5-Stroke dampened wipe samples using a weighted apparatus on playset; n = 57 (12 vertical poles and 45 flat decks). Unknown playground wood of unknown age with unknown stains and/or sealants.	35 µg arsenic/100 cm <sup>2</sup> (average) on vertical poles and 8.8 µg/100 cm <sup>2</sup> on flat decks. (Mean for vertical poles and flat decks: 21.9 µg/100 cm <sup>2</sup> ).	Estimates were also made of ingestion using a 50 cm <sup>2</sup> child's hand, however, the authors stated that this wet wipe sampling technique was not designed to mimic hand to mouth exposure (values not provided)
Mean values: 6.3, 7.5, 8.6, 16.8, 21.9, 27, 233.5, 250 (mean of all these means: 571.6/8 = 71.45 µg/100 cm <sup>2</sup> ). Median: 16.8+21.9/2 = 19.4 µg/100 cm <sup>2</sup>			

#Studies:

<sup>1</sup>Osmose (1983) Surface sampling and analysis of CCA wood by the wipe test method.

<sup>2</sup>Osmose (1983) Surface sampling and analysis of weathered and aged CCA wood by the wipe test method.

<sup>3</sup>Solomon & Warner (1989) Ontario Ministry of the Environment report on leaching of CCA.

<sup>4</sup>CPSC (1990) Estimate of skin cancer risk from dislodgeable arsenic on pressure treated wood playground equipment.

<sup>5</sup>Riedel *et al.* (1990) Residues of arsenic, chromium and copper on and near outdoor structures built of wood treated with CCA type preservatives.

<sup>6</sup>Williams & McNamara (1991) Measurement of dislodgeable arsenic on commercially treated CCA dimension lumber.

<sup>7</sup>'Doyle' for Health and Welfare, Canada (1992) Field study to investigate the leaching and dislodgeability of copper, chromium, and arsenic residues from CCA-C treated lumber and to evaluate the means for reducing environmental contamination and user exposure.

<sup>8</sup>Stilwell (1998). Arsenic dislodged from CCA-treated wood surfaces.

*Note:* In the study conducted by Solomon & Warner (1989; see study no. 3 in the Table above), old or weathered timber released less arsenic than new CCA-treated timber. A similar result was seen in the study conducted by Maas *et al.* (2002; see below).

***Maas RP, Patch SC, Stork AM, Berkowitz JF & Stork GA (2002) Release of total chromium, chromium VI and total arsenic from new and aged pressure treated lumber. University of North Carolina – Asheville Environmental Quality Institute. Technical Report No. 02-093. February 2002.***

CCA pressure-treated lumber obtained at various in-service ages and from two different locations was tested for exposure potential of arsenic and chromium using several different methods. To determine the arsenic and chromium present on the wood surfaces, laboratory wipe sampling was conducted on 16 newly purchased CCA boards from two separate sources, and on five 6-month weathered CCA boards, using US CPSC (2001) 'horizontal sampling methodology'. Laboratory assistants conducting these sampling techniques used fresh, sterile poly examination gloves with each wipe sample. After sampling, all wipes were placed immediately in 50 mL polypropylene vials. The wipes were then digested and the concentrations of arsenic and total chromium were determined by atomic absorption spectroscopy.

The results are given below.

**Total arsenic and total chromium in wiped samples (445 cm<sup>2</sup> surface area) of new CCA-treated wood**

	No. of wipe samples (no. of wood pieces used)	Total arsenic µg/445 cm <sup>2</sup> (µg/cm <sup>2</sup> )	Total chromium µg/445 cm <sup>2</sup> (µg/cm <sup>2</sup> )
New lumber (1 <sup>st</sup> source) <sup>#</sup>	16 (8)	269.6 (0.61)	346.3 (0.78)
New lumber (2 <sup>nd</sup> source) <sup>#</sup>	16 (8)	203.1 (0.46)	230.8 (0.52)
Mean of 1 <sup>st</sup> and 2 <sup>nd</sup> sources		236.4 (0.53)	288.6 (0.65)
Old lumber (1 <sup>st</sup> source; approximately 6-month-weathered)	10 (5)	119.3 (0.27)	143.7 (0.32)

1<sup>st</sup> source: Home Depot; 2<sup>nd</sup> source: Lowes.

When wipe samples were collected in CCA-treated lumber exposed outdoor for about 6 months, decreased levels of total chromium and arsenic were seen.

*Note:* Two apparent errors were noted in the Maas *et al* study, for the data reported for arsenic and chromium in wipe samples. For converting arsenic and chromium content/445 cm<sup>2</sup> to content/ft<sup>2</sup>, a conversion factor of 2.08 (1 ft<sup>2</sup> = 929 cm<sup>2</sup>) was used (ie. 929/445) in Table 2 in the study report, but the conversion factor used in Table 3 was only 1.69. The second error in Table 3 was that the same values (individual as well as mean) reported for total chromium/ft<sup>2</sup> were reported for arsenic. Thus, the mean value reported for chromium and arsenic was 422.5 µg/ft<sup>2</sup> although different individual values were reported for chromium and arsenic. Based on these discrepancies, the validity of the data/calculations reported in the Maas *et al* study is questionable.

***Exponent (2002) Technical issues associated with the risk assessment of children's exposure to arsenic at playgrounds with structures constructed from CCA-treated wood. Exponent, 4940 Pearl East Circle, Boulder, CO 80301. Unpublished Report No. 3.10.11. ACC Reference No. Exponent – Risk Assessment 1.0. May 2002. Part 3, vol 3/7, p 708-802; NRA Study No. 6927. [OCS Submission No. 12275, vol 3 of 13]***

*The following information is from the evaluation report of Exponent (prepared for American Chemistry Council, Arsenical Wood Preservatives Task Force).*

This evaluation report acknowledges that a considerable variation exists in the database on arsenic release from playground wood and recommended a concentration of 21 µg arsenic/100 cm<sup>2</sup> wood surface (dislodgeable residue) for use in risk assessment. The value was the 95% upper confidence limit on the mean calculated from the data of the Riedel *et al.* (1990) study (wipe study; see Table above). The Riedel *et al.* study was selected (by Exponent) as best data set available currently, based on the internal consistency of the data and the quality of the experimental design. The study sampled 10 aged playground structures (with 4 replicate samples from each site) and selected areas of the structure where it was estimated that maximum hand contact would occur (railings, etc.). The average arsenic level (dislodgeable arsenic) was 8.6 µg/100 cm<sup>2</sup> while the range was 0.1-64.4 µg/100 cm<sup>2</sup>.

Koppers Arch (Australia) Pty Ltd (year not stated) An occupational hygiene exercise designed to assess the effect of handling timber treated with wood preservative. Unpublished Report No. T/1/1 & 0/73. Study year not stated. NRA Study No. 6734, OCS Submission No. 12275, vol 10 of 13

*The following information is from the submission by Koppers. Only limited information has been provided.*

Timber (type and number of samples not stated) was vacuum-pressure impregnated with CCA wood preservative. Wipe tests with water-wet cellulose gauze swabs (details of sampling not given) were carried out on treated timber (both planed and rough sawn; 6" x 4"; 15 cm x 10 cm) 'over a period 48 hours to 192 hours'.

The results are shown in the Table. Extraction (details not provided) of the swabs showed the presence of soluble arsenic as a proportion of total arsenic at 3.5% in planed timber and 4.5% in rough sawn timber.

***Arsenic levels in swabs***

	Hours after treatment			
	48 h	96 h	144 h	192 h
<b>Planed timber</b>				
Arsenic ( $\mu\text{g}/\text{cm}^2$ )	2.07	1.18	1.46	1.17
Chromium ( $\mu\text{g}/\text{cm}^2$ )	1.89	1.91	1.24	1.03
Copper ( $\mu\text{g}/\text{cm}^2$ )	2.33	2.53	1.95	1.95
<b>Rough, sawn timber</b>				
Arsenic ( $\mu\text{g}/\text{cm}^2$ )	1.35	1.61	1.27	1.28
Chromium ( $\mu\text{g}/\text{cm}^2$ )	1.54	1.64	1.26	0.95
Copper ( $\mu\text{g}/\text{cm}^2$ )	2.96	2.63	1.90	1.92

***Johanson R & Dale FA (1973) Arsenic on the surface of round pine treated with Cu-Cr-As preservative. Division of Building Research, Forest Products Laboratory, CSIRO, PO Box 310, South Melbourne, Vic. 3205.***

*Pinus radiata* rounds (3; all from Australia) treated with Cu-Cr-As preservatives (arsenic content: 10-12%) were examined for amounts of arsenic and copper remaining on the surface of unweathered material. The surfaces (round surfaces and intact end areas) were washed and gently scrubbed for 10 min and washings (n = 30 each on round surfaces and end areas; 3 pairs of rounds) analysed for arsenic (soluble and insoluble) and copper (total).

Up to 4 mg of total arsenic/100 cm<sup>2</sup> (mean/washing: 0.49 mg/100 cm<sup>2</sup>) was found on the round surfaces and 6-16 mg/100 cm<sup>2</sup> (mean: 2.1 mg/100 cm<sup>2</sup>) on the intact ends. The soluble fraction represented only a small fraction of the total arsenic removed by washing (~3%; 0.017 mg/100 cm<sup>2</sup> on the round surface, 0.050 mg/100 cm<sup>2</sup> on intact end area). Mean copper levels were 0.41 and 1.80 mg/100 cm<sup>2</sup> on the round surface and intact end area, respectively.

***Loveridge K (2003) High levels of arsenic, chromium and copper found on the surface of CCA treated timber used to build a children's playground. City of Maroondah, Australia - poisoned playgrounds. Croydon Conservation Society.***

According to this report, high levels of arsenic, chromium and copper were found in a wipe study using CCA treated pine playground equipment located at a school in the city of Maroondah. The highest levels wiped from the surface of the timber were 710  $\mu\text{g}$  of arsenic, 630  $\mu\text{g}$  of chromium and 670  $\mu\text{g}$  of copper. This initial report did not give any details about how the wipe study was conducted (area wiped, number of samples, etc.).

In a subsequent submission to the APVMA (27 June 2003), the Croydon Society Inc. provided more details regarding the methodology used. The sample collection procedure used was based on that used by the 'Connecticut Agricultural Experiment Station' (CAES; the reference cited was Stilwell D *et al.*, in press, 'Dislodgeable copper, chromium and arsenic from CCA-treated wood surfaces, Journal of the Science of the Total Environment; reference not submitted to the OCS) and that documented in Attachment A of the US CPSC 2003 report. The sample collecting equipment consisted of an 8 cm diameter-rubbing disk that applies a weight of 1 kg to the horizontal rubbing surface; a wiper guides with an 8 x 50 cm slot, giving a wiped area of 386.3 cm<sup>2</sup> and a moistened polyester wipe material (Alphawipe, TX 1009; size used: 9" x 9"; 22.9 x 22.9 cm). Each sample was taken using 5 strokes (one stroke = one forward and one backward movement) followed by a further 5 strokes after rotating the rubbing disk through 90 degrees, for a total of 10 strokes. For vertical sampling an estimate of a 1 kg force was applied to the disk. In order to ascertain reproducibility, a second sample was taken adjacent to the first as one vertical sampling point. Sample and field blanks were obtained using the sample procedure but without contact with the sample disk or a timber surface, respectively. Analysis was as per CAES procedure (extraction/digestion in 100 mL of 10% nitric acid for 2 h at 60 °C. 'Determination was by ICP MS').

The results are given below:

**Total arsenic, chromium and copper in 'AlphaWipes'**

Sample	Total arsenic (µg/100 cm <sup>2</sup> )	Total chromium (µg/100 cm <sup>2</sup> )	Total copper (µg/100 cm <sup>2</sup> )
Reagent blank	<0.1	<0.6	<0.2
Field blank	<0.1	1.0	3.0
North Horizontal	21	22	20
South Horizontal	24	23	22
SW Vertical	91	190	88
NE Vertical 1	140	630	140
NE Vertical 2	710	310	670

'Soil below NE corner' has been reported to contain 16 mg total arsenic/kg ('field background', presumably non-playground soil: 10 mg total arsenic/kg).

The number of samples collected in the study was low. The report has not estimated the amount of inorganic arsenic (or of copper and chromium) that is expected to be transferred to human hands. It is to be noted that in the US CPSC studies (2003 a, i), the conversion factors for translating surrogate measurements to hands were 0.08 for wet polyester and 0.20 for dry polyester (ie. a bare adult human hand will remove ~8% of what the wet polyester will remove from rubbing an equivalent area of wood). In the absence of information on conversion factor to be used, the submitted data were found to be of limited value. However, they do provide an indication that arsenic can be wiped or leached from treated timber. This confirms results of other studies in the variability of amounts which can be wiped from CCA treated surfaces.

***United States Consumer Product Safety Commission (US CPSC, 2003a). Briefing Package. Petition to ban chromated copper arsenate (CCA)-treated wood in***

*playground equipment (Petition HP-01-3). February 2003.*  
<http://www.cpsc.gov/library/foia/foia03/brief/cca0.pdf>

*The following information is from the US CPSC report which contained the following studies: Cobb, 2003 (US CPSC 2003b); Cobb & Davis, 2003 (US CPSC 2003c); Levenson 2003a,b,c (statistical analysis; US CPSC 2003f,g,h); Midgett 2003a (US CPSC 2003i); Thomas 2003 (US CPSC 2003k)*

Based on preliminary studies (Cobb, 2003) conducted in the CPSC laboratory to characterise the properties of CCA-treated wood and to explore parameters that might have an impact on arsenic migration to hands and surrogate materials (parameters such as the surface area of wood rubbed; the pressure of the hand or surrogate material that is applied during rubbing; the number of strokes; the rinsing procedure for efficient removal of arsenic from the volunteers' hands), CPSC conducted two field investigations that involved sampling board on decks and playgrounds with bare hands and/or surrogate materials (Cobb & Davis, 2003).

*Field studies:* Decks made of CCA-treated wood (type of wood not stated) were used in the first field study, to examine the amount of arsenic that might be reasonably expected to be picked up by the hand on horizontal surfaces, and to establish a correlation between the amount of arsenic picked up by a hand and the amount picked up by a suitable surrogate material for the hand. Test structures for the first study included a sample of 8 residential decks at homes in the Washington DC metropolitan area. The structures ranged in age from 0-18 years and had a variety of use patterns and surface treatments.

Deck boards were rubbed with adult volunteers' hands and a surrogate material (dry polyester cloth) in order to establish a correlation between the results of the two methods. Deck wood was used to establish this correlation because decks provided large flat surfaces for 'paired sampling' of cloth and volunteers' hands and enough surface area to enable replicates to be run.

Eight adult rubbed a hand over a predefined area of wood for a predetermined number of times (see below). The rubbing procedure was specified so that the volunteer would have sufficient contact with the wood to approach an equilibrium level or 'maximum hand load' after which point the hand would not continue to pick up significantly greater amounts of arsenic (2 volunteers/deck; the selected boards were rubbed in two separate 700 cm<sup>2</sup> sections, one section was rubbed 10 times, and the other was rubbed 20 times; Thomas, 2003). The surface area of the wood that needed to be rubbed to approach this equilibrium was established in previous CPSC laboratory studies (Cobb, 2003). According to the CPSC report, the surface area touched by the volunteers was within the limits of what a child playing on a playground structure might reasonably be expected to touch (Midgett, 2003a; Thomas, 2003). The hand was then repeatedly rinsed and wiped to ensure that most of the arsenic picked up the hand was measured, and the rinses and cloth wipes were chemically analysed to determine the amount of arsenic present.

A weighted surrogate cloth (dry polyester) was rubbed over a predefined area of a sample of wood for a predefined number of times (~400 cm<sup>2</sup>; 10 back-and-forth strokes;

Thomas, 2003). The surrogate cloth was then chemically analysed for the amount of arsenic.

A playground study was then performed that tested 12 CCA-treated wood and 3 non-CCA wood home playground structures in the Washington DC metropolitan area. These playgrounds represented a variety of ages of in-situ timber (0.5-18 years), wood treatments, and manufacturers.

The laboratory and field deck and playground studies led to the following conclusions:

- The amount of arsenic that can be 'loaded' onto a hand appeared to approach equilibrium ('maximum hand load' values) when rubbing CCA-treated boards. This 'maximum' hand load is reached relatively quickly, ie. after rubbing the hands just a few times over the test area.
- In the first field study (8 CCA-treated decks), there was a significant difference in arsenic levels picked up by the hand among the various decks tested. The levels of arsenic transferred to a human hand ranged from 1.0-20.9 µg among the 8 decks sampled, with a mean value of 7.7 µg (see Table below).
- There was a significant correlation between the results from the rubs performed with dry polyester cloth surrogate and results from rubs performed with the hand. The results from the hand and the cloth rubbings were highly correlated (r = 0.91).

The dry polyester cloth picked up ~5 times the amount of arsenic that the hand picked up, when tested according to the described protocol. Thus, a conversion factor of 0.2 can be used to estimate the amount of arsenic on the hand from the dry polyester cloth data collected under conditions of this study. It was noted that the wet cloth picked up more arsenic than the dry one.

- In the second field study (12 CCA-treated playgrounds) using the dry polyester cloth to rub the wood, the mean dislodgeable arsenic level was 7.6 µg (expressed as converted 'hand' values, not dry cloth values).
- The average amount of arsenic removed from the 8 deck samples (mean: 7.7 µg; actual value from bare test hand) or using surrogate materials (extrapolated to bare hands; mean: 8.3 µg) was comparable to the amount removed from playsets (when the surrogate results were extrapolated to bare hands; mean = 7.6 µg).

The results are summarised in the following table.

***Removal of arsenic by hands (adult) and surrogates***

CCA-treated wood, surface contact	Mean (µg)	Median (µg)	Range (µg)	Converted 'hand' (adult) value (µg) <sup>#</sup>	n
Decks, bare hands	7.7	4.8	1.0-20.9	-	8
Decks, dry polyester	41.7	38.1	2.3-114.1	8.3	8
All playsets, dry polyester	37.9	17.4	1.6-168.5	7.6	12
Playgrounds and decks, dry polyester	39.4	26.9	1.6-168.5	7.9	20

<sup>#</sup>The hand values were derived from the mean value for dry polyester, by applying a factor of 0.2.

US EPA (2001a) Final Exposure Document: Office of Science Coordination and Policy.  
[http://www.epa.gov/scipoly/sap/2001/october/final\\_expo\\_doc\\_927.pdf](http://www.epa.gov/scipoly/sap/2001/october/final_expo_doc_927.pdf)

*The following information has been taken from the US EPA's exposure assessment report on the arsenic and chromium components of CCA.*

**Wipe studies for dislodgeable arsenic in CCA-treated wood**

Studies	Arsenic ( $\mu\text{g}/\text{cm}^2$ )		n
	Mean	Range	
<b>Wet wipes</b>			
Cedar Rose Park (CDHS, 1987)	0.89	0.03-3.14	28
Joggers Exercise Station (CDHS, 1987)	1.37	1.20-1.70	3
Initial survey (CDHS, 1987)	2.77	0.001-33.27	21
Wet Wipes from Five Parks	0.13	0.01-1.14	15
MTU (CDHS, 1987)	0.70	0.17-1.62	8
Monterey Fishing Pier (CDHS, 1987)	9.60	0.10-21.30	11
Lumberyard Cotton Gauze (CDHS, 1987)	0.89	0.27-2.54	16
Stilwell (1998)	0.40	0.06-1.22	52
Stilwell (1998)	0.09	0.02-0.45	45
Stilwell (1998)	1.05	0.05-6.32	12
Riedel (Nov. 1991)	0.09	0-0.64	40
Osmose (1980)	1.20	0.12-5.11	9
Osmose (Sept. 1983)	0.06	0.03-0.08	10
Osmose (Oct. 1983)	0.06	0.001-0.22	20
Woolson & Gjovik (1981, unweathered)	1.14	0.50-2.49	5
Woolson & Gjovik (1981, weathered)	0.28	0.10-0.51	7
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993)	0.07	0.004-0.19	66
<b>Dry wipes</b>			
Monterey Fishing Pier (CDHS, 1987)	10.98	0.04-25.67	11
Hiroziroglu, 1985 (cited within CDHS, 1987)	1.78	NA	21
US CPSC (1990)	0.69	NA	NA
US CPSC (1990)	0.22	NA	NA
US CPSC (1990)	0.32	NA	NA
US CPSC (1990)	<0.062	NA	NA
Osmose (1998)	0.18	0.019-0.96	40
<b>Vacuum brush</b>			
MTU (CDHS, 1987)	6.18	2.84-10.81	16
MTU Field Test Parks (CDHS, 1987)	2.43	0.41-16.40	20
Hiroziroglu, 1985 (cited within CDHS, 1987)	0.63	NA	21
MTU – Clean (cited within CDHS, 1987)	1.60	NA	NA
MTU – Light Residue (cited within CDHS, 1987)	4.11	NA	NA
MTU – Heavy Residue (cited within CDHS, 1987)	8.07	NA	NA
MTU – Visibly Dirty (cited within CDHS, 1987)	23.18	NA	NA
MTU – Lumberyard (CDHS, 1987)	13.76	0.43-75.05	14
Joggers Exercise Station (CDHS, 1987)	1.22	0.57-1.60	3
Saur <i>et al.</i> , 1983 (cited within CDHS, 1987)	0.18	0.002-1.27	27
<b>Dry hand wipes</b>			
Arsenault, 1975	0.008	0.001-0.026	8
MTU – Clean (cited within CDHS, 1987)	0.23	NA	NA
MTU – Light Residue (cited within CDHS, 1987)	0.40	NA	NA
MTU – Heavy Residue (cited within CDHS, 1987)	1.03	NA	NA
MTU – Visibly Dirty (cited within CDHS, 1987)	1.80	NA	NA
5 Volunteers (CDHS, 1987)	0.26	0.13-0.43	5
Urine Arsenic Study (CDHS, 1987)	1.40	-	1
Osmose (1998)	0.045	0.003-0.17	78

<b>Wet hand wipes</b>			
Arsenault, 1975	0.22	0.08-0.57	8

NA Not available.

### Wipe studies for dislodgeable chromium in CCA-treated wood

Studies	Chromium ( $\mu\text{g}/\text{cm}^2$ )		n
	Mean	Range	
<b>Wet wipes</b>			
Cedar Rose Park (CDHS, 1987)	1.04	0.07-3.88	28
Riedel (Nov. 1991)	0.06	0.002-0.51	40
Osmose (Sept. 1983)	0.052	0.032-0.10	10
Osmose (Oct. 1983)	0.092	0.035-0.222	20
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993)	0.08	0.003-0.25	66
<b>Dry wipes</b>			
Osmose (1998)	0.19	0.037-0.75	40
<b>Dry hand wipes</b>			
Arsenault, 1975	0.044	0.043-0.045	2
5 Volunteers (CDHS, 1987)	0.390	0.192-0.640	5
Osmose (1998)	0.044	0.002-0.15	79
<b>Wet hand wipes</b>			
Arsenault, 1975	0.49	0.31-0.67	2

NA Not available.

## 6 Studies on arsenic, chromium and copper intake from cca-contaminated soil

US EPA (2001a) Final Exposure Document: Office of Science Coordination and Policy.  
[http://www.epa.gov/scipoly/sap/2001/october/final\\_expo\\_doc\\_927.pdf](http://www.epa.gov/scipoly/sap/2001/october/final_expo_doc_927.pdf)

*The following information has been taken from the US EPA's exposure assessment report on the arsenic and chromium components of CCA. The original studies have not been evaluated by the OCS.*

In the following studies, arsenic and chromium levels in soil which comes into contact with CCA-treated wood have been monitored.

### Arsenic content in CCA-contaminated soil

Studies	Arsenic (mg/kg)		n
	Mean	Range	
Riedel (Nov. 1991)	5.51	0.04-113.1	40
Osmose (2000)	23.97	5.14-85.0	73
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993) – soil	16.73	0.37-54.32	58
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993) – sand	12.71	0.24-27.21	56
Stilwell & Gorny (1997)	76	3-350	85
Townsend & Solo-Gabriele (2001)	28.3	0.25-217	65

Mean of all means (arsenic content) in the above studies: 27.2 mg arsenic/kg soil.

**Chromium content in CCA-contaminated soil**

Studies	Chromium (mg/kg)		n
	Mean	Range	
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993) – soil	11.33	0.27-45.58	58
Doyle (1992), Malaiyandi (no date), Malaiyandi (1993) – sand	8.83	1.34-21.03	56
Stilwell & Gorny (1997)	43	16-154	85
Townsend & Solo-Gabriele (2001)	30.5	0.71-198.5	65

*Comfort M (1993) Environmental and occupational health aspects of using CCA treated timber for walking track construction in the Tasmanian Wilderness World Heritage Area. Scientific Rep. 93/1. Hobart, Tasmania: Tasmanian Parks and Wildlife Service, 1993 [cf. Lebow S (1996) Leaching of Wood Preservative components and their mobility in the environment. Summary of pertinent literature. US Dept. of Agriculture. General Technical Report FPL-GTR-93]*

*The following information is from the US Dept. of Agriculture report (1996).*

In a study conducted by the Tasmanian Parks and Wildlife Service, arsenic, chromium and copper levels were measured adjacent to CCA-treated boardwalks at several sites in southern Tasmania. At each site, 3 soil samples were taken within 15 cm of the boardwalk, and 3 reference samples were removed several metres from the boardwalk. The boardwalks at the sites varied from 1-14 years in age; the preservative retention and treating solution formulation were not reported.

Levels of copper and chromium adjacent to the track were significantly elevated in comparison with the control samples, but not extreme levels. Arsenic levels were not found to be significantly elevated above that of the controls. The highest copper level detected was 49 ppm; the control level was between 1-3 ppm for that site; the highest chromium level detected was 88 ppm, ~60 ppm more than the reference sample. There did not appear to be any relationship between the age of the boardwalk and the CCA levels detected.

*Kennedy MJ & Collins PA (2001) Leaching of preservative components from pine decking treated with CCA and copper azole, and interactions of leachates with soils. Prepared for 32<sup>nd</sup> Annual Meeting of the International Research Group on Wood Preservation. Queensland Forestry Research Institute, Queensland, Australia, May, 2001. IRG/WP 01-50171.*

*The following information is from the evaluation report of Environment Australia on CCA (2003; see section 'Leaching from simulated deck units in Queensland and interactions of leachate with soils').*

The study used simulated deck units to monitor leaching of CCA components from CCA-treated radiata pine decking stock. The CCA used had a copper, chromium and arsenic content of 86, 147.9 and 132.7 g/kg, respectively. This was followed by a one week fixation and 4-6 week air drying period. Sections were removed from the boards and cut into 19 mm cubes for accelerated laboratory leaching tests, which were extended from the standard 14 days to 50 days, as the leaching rate was still rising sharply at 14 days. Boards 300 mm long were then end-sealed, leaving a few ends unsealed so that an average 2.4 m deck length could be simulated when 4 short boards were placed side by side over a collection tray. Decks were exposed to the weather in

Brisbane for ~300 days, during which cumulative rainfall reached ~600 mm. Run-off water was collected after each of 48 rain events for analysis, determination of volume, and collection of an aggregate sample for soil column leaching studies with 3 different soils (using the OECD Test Guideline, presumably No. 107).

The rate of loss of copper declined over time, from 1.54-1.88 mg/m<sup>2</sup>/day over the first 21 days, to 0.51-0.64 mg/m<sup>2</sup>/day over days 0-90, and 0.34-0.52 mg/m<sup>2</sup>/day over days 0-300. The rate of chromium loss was relatively high initially (1.05-1.112 mg/m<sup>2</sup>/day), but similar over 0-90 days and 0-300 days (0.41-0.58 mg/m<sup>2</sup>/day). That for arsenic was similar for 0-21 days and 0-300 days (1.47-3.14 and 1.40-2.10 mg/m<sup>2</sup>/day), and lower at 0-90 days (0.75-1.64 mg/m<sup>2</sup>/day).

The study estimated that leaching from an initial rainfall of 600 mm (10 months) would give soil concentrations of 0.54 ppm (mg/kg) copper, 0.75 ppm chromium and 3.1 ppm arsenic. For a rainfall of 7300 mm (~10 years in Brisbane), the estimated cumulative soil concentrations due to the leaching would be 2.15 ppm (mg/kg) Cu, 5.99 ppm Cr and 33.1 ppm As.

## **7 Regulatory history and public health standards**

### **7.1 Regulatory history of arsenic in Australia**

November 1980	PACC: The MRL applying to cattle and/or sheep was extended to goats.
March 1981	DPSC: It was noted that Fowler's Solution (1% arsenic trioxide) was still manufactured and sold. The arsenic entry in Schedule 4 was considered; no decision on the deletion of 'organic' from this entry was taken.
May 1981	DPSC: Advised that demand for Fowler's Solution stemmed for use in veterinary medicine. Consideration of the amendment to the S4 entry was deferred.
February 1982	PACC: The MRL entry for arsenic was amended from Arsenic 1.15 mg/kg (as As) entry to read 1.0 mg/kg (as As).
May 1982	DPSC agreed to amendment entries in S6 and S7, while entries in S4 and S5 remained unchanged. S7 amended to: Arsenic except (a) when included in S4, 5 or 6; (b) in animal feedstuffs containing 75 mg/kg or less of arsenic. S6 amended to: Arsenic, (a) in ant poison containing 0.5% or less arsenic trioxide; (b) organic compounds of arsenic prepared for use as herbicides or defoliant except when included in S5; (c) in animal feedstuff premixes containing 4% or less of arsenic
Aug 1982	DPSC amended entries to discourage domestic use
November 1983	DPSC: the arsenic entry cross referencing to thiacetarsamide included in S4
March 1985	PACC considered the Department of Health's review of arsenic in kelp, and passed it to the FST Subcommittee
November 1987	PACC noted US EPA decision to cancel registrations for inorganic arsenicals for non-wood preservative use. PACC agreed to remove all MRLs for heavy metals, and place them in a separate appendix (covered under Food Standards Code A12)
November 1990	DPSC exempted a silicone sealant containing 736 ppm OBPA from scheduling
June 1991	DPSC deleted all Schedule 5 arsenicals and changed to Schedule 6
January 1999	The Australian Department of Health conducted a review on inorganic arsenic and

	recommended that veterinary products containing arsenic should not be used in food producing animals. Inorganic arsenic herbicides removed from the market.
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## 7.2 International toxicology assessments and regulatory decisions

In February 2002, US EPA announced a voluntary decision by industry to move away from timber treatments containing arsenic by December 31 2003, in favour of new alternatives. This transition affects virtually all residential uses of wood treated with CCA, including wood used in play structures, decks, picnic tables, landscaping timbers, residential fencing, patios and walkways/boardwalks. US EPA will not allow CCA products to be used to treat wood intended for any of these residential uses from 1 January 2004.

The US EPA has not concluded that there is unreasonable risk to the public from these products, but is of the view that any reduction in exposure to arsenic is desirable. This action comes ahead of the US EPA completing its regulatory and scientific assessment of CCA (expected in December 2003).

Canadian regulatory authorities (PMRA) are working in collaboration with the US EPA to effect similar actions in Canada.

A risk assessment conducted by the EC Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) noted that the main risks associated with CCA were those to human health from the disposal of timber treated with CCA and in particular risks to children's health from the use of CCA-treated timber in playground equipment. The CSTEE raised further concerns regarding the potential for children to be exposed to CCA through ingestion and/or inhalation of sand particles in playground equipment. They concluded that arsenic is both carcinogenic and genotoxic.

The CSTEE considered that there are serious knowledge gaps in relation to arsenic-treated timber in landfills. It concluded that it would be advisable to exercise caution by limiting the use of arsenic-based timber treatment to those situations where it is "absolutely necessary" (such as railway sleepers, electric power transmission and telecommunication poles and in cooling towers). Member states are to adopt and publish the provisions necessary to comply with the EC directive (2003/02/EC) by 30 June 2003 and apply the provisions by 30 June 2004.

The EC will review timber treatment products. Full dossiers for evaluation must be presented by 28 March 2004.

New Zealand recently reviewed (ERMA, April 2003) the use of CCA products in timber and stated that the mechanism of carcinogenesis of arsenic is not well established and that despite uncertainty and potential overestimation of cancer risk it would be prudent public health policy to reduce human exposure to arsenic from all sources wherever feasible.

## 7.3 Consideration of Public Health Standards

### Standards for arsenic

JECFA has set a tolerable limit of 15 µg/kg/week (2 µg/kg/day) while the Australian standard for arsenic as set by FSANZ is a maximum of 3 µg/kg/day. Since arsenic products will not be used in food producing animals, no ADI (acceptable daily intake) or ARfD (acute reference dose) has been set for arsenic.

### Poisons scheduling

Copper oxides in preparations containing 25% or less of copper oxides are in schedule 5 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). Exemptions from schedule 6 are in preparations for internal use, in marine paints or in other preparations containing 5 per cent or less of copper oxides. Copper sulfate is included in S6, or S4 for preparations for internal use, with cut-offs to S5 for preparations containing 15% or less, and a cut-off to unclassified for preparations containing 5% or less.

Chromates (including dichromates) except in paints or tints containing 5 per cent or less of chromium as the ammonium, barium, potassium, sodium, strontium, or zinc chromate calculated on the non-volatile content of the paint or tint are in schedule 6 of the SUSDP. Similarly, chromium trioxide (excluding its salts and derivatives) is also in schedule 6 of the SUSDP.

The SUSDP lists arsenic in schedule 7 with an Appendix J entry to restrict its availability and use to authorised or licensed persons. Exemptions from schedule 7 are permissible when present as selenium arsenide in photocopier drums; as 10,10'-oxydiphenoxarsine in silicone rubber mastic containing 120 mg/kg or less of arsenic; or in animal feeds containing 75 g/tonne or less, or in paints containing 0.1 per cent or less of arsenic calculated on the non-volatile content of the paint. Arsenic is included in schedule 6 when present in ant poisons containing 0.4 per cent, in animal premixes containing 4 per cent or less, or in preparations for the treatment of animals except thiacetarsamide when included in schedule 4.

### First Aid Instructions and Safety Directions

Based on the evaluation of hazards associated with the actives and non-actives (see Appendix II) in the products, the current statements for the First Aid Instructions and Safety Directions are considered adequate and no further changes are required.

The following First Aid Instructions, General Statements and General Safety Precautions have been established for arsenic, chromium and copper compounds present in CCA formulations.

Arsenic compounds	a	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; NewZealand 03 4747000.</i>
Arsenic trioxide	a,s	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; New Zealand 03 4747000.</i> If in eyes, hold eyes open, flood with water for at least 15 minutes and see a doctor.
Chromates	a,f,s	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; NewZealand 03 4747000.</i> If skin contact occurs, remove contaminated clothing and wash skin thoroughly. If in eyes, hold eyes open, flood with water for at least 15 minutes and see a doctor.
Chromium trioxide	a,c,f,s, 02, 14, 15, 22, 101, 104, 108, 113	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; NewZealand 03 4747000.</i> If swallowed, do NOT induce vomiting. Give a glass of water. If skin contact occurs, remove contaminated clothing and wash skin thoroughly. If in eyes, hold eyes open, flood with water for at least 15 minutes and see a doctor. Corrosive. Dust will irritate and burn eyes, nose and skin.

**Australian Pesticides and Veterinary Medicines Authority (APVMA)**

		Liquid will cause burns. May cause fire or explosion. Avoid contact with eyes. Avoid contact with skin. Avoid breathing dust (or) vapour (or) spray mist. Do not allow product to come into contact with combustible materials such as paper, fabric, sawdust or kerosene.
Copper oxides	a	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; New Zealand 03 4747000.</i>
Copper sulfate	a,f,s	If poisoning occurs, contact a doctor or Poisons Information Centre. <i>Phone Australia 131126; New Zealand 03 4747000.</i> If skin contact occurs, remove contaminated clothing and wash skin thoroughly. If in eyes, hold eyes open, flood with water for at least 15 minutes and see a doctor.

The following Safety directions<sup>#</sup> exist for the components present in CCA formulations.

<b>Copper-chrome-arsenic wood preserving solutions</b> LD (note: special systems are required for this treatment)	120 130 132 133 210 211 212 220 223 279 280 281 286 290 292 294 297 330 332 340 342 350 360 361 363 366
<b>Arsenic compounds (arsenite, sulphide, trioxide, pentoxide)</b> PA WP EC LC DU all strengths	120 121 130 132 133 161 162 163 164 190 210 211 220 (221 DU WP) 223 279 280 281 282 290 292 293 294 298 299 (300 302 312 DU) 350 360 361 362 363 364 366
<b>Arsenic trioxide</b> DU for termiticide use	130 132 133 161 162 163 164 190 210 211 220 221 279 280 282 290 292 312 298 299 300 302 350 360 361 362 365 366
<b>Sodium dichromate</b>	See Copper, chrome, arsenic wood preserving solutions
<b>Chromium trioxide</b>	No separate entry (See Copper, chrome, arsenic wood preserving solutions)
<b>Copper oxides</b>  LD 530 g/L or less in hydrocarbon solvents 450 g/L or less   Paint all strengths	129 133 161 162 163 164 180 181 210 211 220 222 279 280 283 290 292 295 297 298 300 340 342 340 343 351 360 361 363 364 366  129 133 160 162 164 210 211 279 280 290 292a 294b 279 287 282 290 292a 294b 297 351 360 361 363 366
<b>Copper sulfate</b> All forms except where otherwise specified  HG LD 10 g/L or less  PD 50 g/kg or less	129 133 161 162 163 164 210 211 279 283 290 294 340 342 340 343 351 360 361 366  160 162 164 210 211 351  161 162 164 210 211 279 280 283 290 292b 294 297 306 340 343 351 360 361 363 366

#Codes:

120	product (and) (is)
121	spray (dip) (is) (are)
129	harmful (if)
130	poisonous (if)
132	inhaled (or)
133	swallowed (or)
160	may irritate the
161	will irritate the
162	eyes (and)
163	nose and throat (and)
164	SKIN
180	repeated exposure may cause allergic disorders
190	repeated minor exposure may have a cumulative poisoning effect
181	sensitive workers should use protective clothing
210	avoid contact with
211	eyes and skin (and)
212	clothing
220	do not inhale
221	dust (or)
222	vapour (or)
223	spray mist
279	when
280	opening the container (and)
281	preparing (spray) (dip) (other form) (and)
282	using the prepared (spray) (dip) (other form)
283	using the product (and)
285	preparing product for use (and)
287	mixing (and)
290	wear
292	cotton overalls buttoned to the neck and wrist and a washable hat (and)
292a	cotton overalls buttoned to the neck and wrist (or equivalent clothing) and a washable hat (and)
292b	cotton overalls buttoned to the neck and wrist (or equivalent clothing) (and)
293	pvc or rubber apron (and)
294	elbow-length pvc gloves (and)
294b	elbow-length rubber gloves (and)
295	elbow-length (nominate other specific material) gloves (and)
297	goggles (and)
298	impervious footwear (and)
299	face shield or goggles
300	half facepiece respirator
302	with dust cartridge or canister
306	disposable (dust), (mist) or (fume) mask (specify)
312	rubber gloves
330	if clothing becomes contaminated with product
332	remove clothing immediately
340	if product (or)
342	on skin, immediately wash area with soap and water
343	in eyes, wash it out immediately with water
350	after use and before eating, drinking or smoking, wash hands, arms and face thoroughly with soap and water
351	wash hands after use
360	after each day's use, wash
361	gloves (and)
362	face shield (and)
363	goggles (and)
364	respirator and if rubber wash with detergent and warm water
365	face shield or goggles (and)

366 contaminated clothing

## ADI

CCA and arsenic products are not used in food producing animals and hence there is no acceptable daily intake set for these products.

## 7.4 Products

There are 9 registered timber preservative products containing CCA and 3 termite treatment products containing arsenic trioxide. Termite treatment products are formulated as dusts, with timber preservatives available as aqueous concentrates, blending concentrates, liquids, liquid concentrates and pastes.

Timber is treated with CCA chemicals in specially designed treatment plants using vacuum pressure impregnation processes to protect sapwood from insect and fungal decay.

As termite treatments, arsenic trioxide dusts are applied to timber both inside and outside the home as well as to living trees where necessary. Within buildings, small amounts of product (1-2 g) are dusted into the termite workings with a hand blower. Once applied the treated areas are sealed (with tape) and left undisturbed for 10-20 days. For termite control outside buildings and in tress, only small amounts of the powder are applied, via drilled holes. Application is by hand blower.

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#### **Further submissions examined (\*)**

*(\*) The following submissions were received and reviewed but they either provided no data (ie comment or opinion only) or the data submitted was insufficiently detailed to enable an independent assessment to be made.*

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## **Public submissions submitted July 2003; in response to the review scope**

### ***Submission 1***

The submission stated that 'there are inherent risks in all aspects of CCA industry' and that recent research indicated that arsenic can leach from CCA treated timber. The submission recommended that 'CCA impregnated timber must immediately be banned from production throughout Australia'.

### ***Submission 2***

The submission is reported to be from a victim of CCA wood poisoning. The submission stated that the true risks of CCA have been kept from regulators and public alike by not understanding the nature of the contamination that is caused by arsenic to the human body. The submission also pointed out that arsenic and chromium free substitutes and alternatives have been available for a number of years and several are awaiting registration by the US EPA. The submission added that there was 'no test for low level long term arsenic poisoning'. According to the submission, 'doctors and other medical personal failed to document CCA poisoning cases', and they were 'completely unaware of how to test and treat victims'.

The submission included a CCA report (Copper chromium arsenate (CCA) in the environment) and a medical report prepared by the submitter.

### ***Submission 3***

The submission included a few papers relating to the burning of CCA-treated wood and potential health impacts. According to a study conducted by Golec & Wilson of The State Chemistry Laboratory (Werribee, Victoria), burning of CCA-treated timber volatilised significant amounts of arsenic, resulting in the contamination of meat cooked in the flame and of the air in the breathing zone. The study stated that the inhalation of arsenic contaminated smoke and vapour from CCA-treated timber fires represented a significant potential exposure route, which could contribute to acute systemic effects. The study concluded that the combination of ingestion of contaminated meat and inhalation of arsenic smoke and vapour increased the potential intake of arsenic and hence the potential for poisoning.

### ***Submission 4***

The submission (the submitter has been involved in research and development in the urban pest management industry for more than 20 years) does not recommend continuation of registration of arsenic and stated that there was 'no data held on the product' ('there are no scientific studies to show the efficacy of arsenic in the manner in which it is most commonly applied ie. remote dusting into termite leads in the hope it will eliminate the colony'); arsenic is a 'deadly poison and carcinogen'; arsenic is 'not safe for people, animals and the environment'; and arsenic 'does not meet contemporary standards'.

### ***Submission 5***

The submitter is of the view that CCA treated wood is relatively safe to use, provided safety precautions are followed.

### ***Submission 6***

The submitter has expressed interest to know the outcome of the review, especially in relation to the extent to which timber in the construction of buildings is to be affected and whether there was any role for the Building Code of Australia in respect to compliance in the future.

### ***Submission 7***

According to the submission, arsenic trioxide is an inexpensive and very effective glueline termiticide. The submission stated that 'it is believed that the arsenic molecules are fully encapsulated within the phenolic resin matrix and cannot be released to the surrounding environment, making products treated with glueline addition of arsenic trioxide very safe'.

### ***Submission 8***

The submission included a leaflet 'Public Health Danger. Ash from CCA treated timber' (Department of Human Services) which contained information about ways of collection and disposal of CCA ash; personal protection when collecting ash; health symptoms and actions to be taken.

### ***Submission 9***

The report is about the effectiveness of a dust toxicant (supplied by the manager of W. Holloway and Co.; active ingredient not stated) in eliminating colonies of *Coptotermes acinaciformis* at three sites in Queensland.

### ***Submission 10***

The submission stated that 'the APVMA scope paper on CCA is an inadequate vehicle to properly address this serious issue with a degree of urgency' and suggested that 'a proper review document of CCA was required similar to a National Environment Protection Measure (NEPM) with reviews similar to the USA EPA'.

### ***Submission 11***

According to the submission, 'a mixture of experimental data, animal tests, epidemiological surveys, expert opinion, and varying lengths of experience - suggests that CCA treatment is not a threat to human life'.

### ***Submission 12***

The submission stated that arsenic dust must be made available for the control of *Mastotermes darwiniensis* - RAPID Solutions (a licensed Insurance Broker) is not aware of any other termite dust or termite bait that is registered to control *Mastotermes*. The submission has suggested some changes and conditions regarding the sale and product packing.

### ***Submission 13***

The submission requested that APVMA consider potential exposures and risks associated with the use of CCA treated products for purposes such as constructing vegetable or compost enclosures.

*Note:* There are no relevant Australian data submitted to OCS to determine human exposures arising from the use of CCA-treated wood in horticulture. However, intake of arsenic from food

as estimated by FSANZ (The 20<sup>th</sup> Australian Total Diet Survey; FSANZ, 2002) included inorganic arsenic present in vegetables and fruits as a result of environmental arsenic.

#### ***Submission 14***

Based on the results of a survey conducted by the New Zealand timber Council with 71 CCA treaters, the submitter recommended that serious consideration should be given to the conclusions reached by ERMA in its review of CCA treated timber (April 2003). In the survey, the submitter noted that only five cases of adverse health effects (over all the years of operation at the 71 sites; 4 cases of adverse health effects from raised arsenic levels and 1 from raised chromium levels) were detected from regular medical testing. All the five cases were presumably due to failure to wear required protective clothing.

#### ***Submission 15***

Members of the Timber Preservers Association of Australia were surveyed (29 responses, 60% return; about 800 staff). The results revealed that 'CCA is an effective, safe preservative for the treatment of timber, particularly sustainably managed plantation softwoods which supply the bulk of our building and agricultural timbers used today'. According to the submitter, 400,000 m<sup>3</sup> of CCA-treated timber is produced annually in Australia. Based on the survey, the submitter concluded that CCA-treated product has not caused any adverse health effects either in production or when used properly.

#### ***Submission 16***

The submission enclosed the evaluation of report of ERMA, New Zealand on CCA treated timber. One of the recommendations of the submission was that the APVMA review process should take into account the literature review and conclusions outlined in the ERMA review.

#### ***Submission 17***

The submission is of the view that arsenic trioxide dust can be a very effective agent for the control of termites at a relatively low cost and is of the view that this product should remain accessible until a suitable alternative becomes available.

#### ***Submission 18***

The submission recommended that CCA-treated pine structures should be replaced with acceptable alternatives and all residential uses and some aquatic uses of CCA treated pine should be banned.

#### ***Submission 19***

The submitter is of the opinion that banning of arsenic trioxide for the treatment of termites would lead to a far greater cost to the consumer (damage to houses and cost of repairs). Handled correctly, the chemical would cause 'no more damage than many other chemicals used in the world at this time'.

#### ***Submission 20***

The submitter recommended that arsenic trioxide be retained for use by licensed pest controllers and that sale and use be restricted to those who have demonstrated competency in the relevant Units of the National Pest Management Competency Standards.

***Submission 21***

A study conducted in Florida, USA, revealed that arsenic concentrations for surface soils collected from underneath 9 pre-existing decks ranged from 1.2-217 mg/kg with an average of 28.5 mg/kg. The average arsenic concentration of the control samples was 1.5 mg/kg.

***Submission 22***

A study is currently under way at the University of Sydney to investigate the combustion end of service life of CCA treated timbers with the primary objective of determining whether there is a trade-off between energy recovery from the combustion process and the stability and/or recyclability of the metals contained in the timber. The final report for the initial experimental work is due at the end of 2003.

***Submission 23***

The email describes a case of poisoning reportedly caused by arsenic treated timber.

***Submission 24***

The submission pointed out that CCA preservative has been used around the world since the mid-1930s and used commercially in Australia for more than 40 years. CCA-treated wood has been reported to be used without discernible and reported adverse health effects suggesting that if there is a true increased risk it is very small (NZ ERMA). The submission also pointed out that adherence to Codes of Practice and standards for safe use and handling contribute to product safety.

***Submission 25***

The submitter stated that the CCA review by APVMA needs to address the following: the need for these products and the availability of safe and effective alternatives; current industry standards and practices; and the life cycle impacts for CCA and arsenic trioxide products during application, use and disposal on the environment, health, occupational health and safety and agriculture.