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NGĀ KAIWHAKATŪPATO WHAKARARU TAIAO

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

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1 Terms of Reference

The objective is to produce a report that:

(1) reviews the report *Release of Total Chromium, Chromium VI and Total Arsenic from New and Aged Pressure Treated Lumber* by Dr R Maas et al. (2002) to provide guidance as to its scientific credibility, the extent to which it constitutes new information and the relevance of this information to New Zealand circumstances.

(2) reviews other relevant information about the public health (including occupational health) risks related to the use of CCA-treated timber particularly around homes and in children's playgrounds, on a comparable basis to the review of the Maas report.

(3) identifies data and information gaps and uncertainties (including matters of dispute).

(4) provides advice on whether the levels of public and occupational health risk indicated may justify further regulatory action, taking account of the matters set out above.

The scope of the project is limited to undertaking a literature review and interpreting the findings in a New Zealand context that focuses on current public and occupational health risk. Occupational risks related to the manufacture of copper, chromium and arsenic (CCA) treated timber, risks to the environment, and alternatives to CCA-treated timber are excluded.

2 Method

A literature search of on-line bibliographic databases was undertaken using DIALOG for publications in English about the public and occupational health risks from use of CCA-treated wood in residential and public settings. Key words used in the search included synonyms and related terms for CCA. The search period focused on publications from 1990 to 2002. Some publications outside this period have been included. Since many of the reports, in particular the risk assessments, are not identifiable from bibliographic databases the bibliographies of identified reports and papers were also examined and organisational websites were searched. Not all articles that were viewed have been cited.

Although the Maas report has its own term of reference the author considered it more appropriate to review it in the context of all other risk assessments of CCA-treated wood, and therefore merged Terms of Reference 1 and 2. The Institute of Environmental Science and Research (ESR) reviewed the Maas report for the Ministry of Health from which a separate report is available.

This review focuses on the forms of the components of CCA that pertain to CCA-treated wood and gives more detail on arsenic than copper and chromium since it is the most toxic component and also appears to leach more from CCA-treated wood than chromium, the second most toxic component. For this reason the sections on copper and chromium are largely drawn from review documents rather than source references.

3 Executive Summary

The wood preservative copper, chromium and arsenic (CCA) has been used in New Zealand since the 1950s but widespread exposure of the general population to CCA-treated wood did not occur until the late 1960s. Arsenic is the most toxic of the three components and has therefore been the focus of the health risk assessments that have been undertaken to date.

CCA-treated radiata pine is commonly used in outdoor settings. Uses in residential and public settings include decks, garden furniture, picnic tables, playground equipment, landscaping timbers, retaining walls, fences, gazebos and patios. However
CCA is only one source of potential human exposure to inorganic arsenic. New Zealanders are exposed to low levels of arsenic present in food, water, air and soil, particularly in the central North Island where background levels are naturally high in the volcanic soil.

Decreasing amounts of copper, arsenic and chromium migrate from CCA-treated wood over time. All three components of CCA adsorb strongly onto soil so will be confined to the areas under the deck or immediately adjacent to the playground equipment.

Public exposure particularly that of children is most likely to occur through the ingestion of dislodgeable residues from the surface of, and contaminated soil adjacent to, CCA-treated wood structures such as playground equipment and decks. In contrast the main exposure for builders is inhalation of CCA dust.

It is assumed that the form of arsenic in CCA-treated wood surface residues and soil is pentavalent and the chromium is trivalent. Pentavalent arsenic is less toxic than trivalent arsenic and trivalent chromium is significantly less toxic than hexavalent chromium. Conversion of trivalent chromium to hexavalent chromium has been reported following use of commercial deck wash treatments containing oxidising agents and in some soil conditions.

Chronic exposure to arsenic through ingestion is associated with skin, lung and bladder cancer and through inhalation with lung cancer. Hexavalent chromium is also carcinogenic but only through inhalation.

There are no epidemiological studies or human case reports involving disease related to direct contact with CCA-treated wood and the low level exposures that most of the general population will experience from contact with CCA-treated wood are extremely unlikely to result in acute health effects. CCA-treated wood has also been in use for many years without discernible adverse health effects suggesting that if there is a true increased risk it is very small.

The studies and risk assessments that have been carried out overseas are relevant to New Zealand. The CCA formulations used in New Zealand are similar to CCA Type C used in the United States. Radiata pine is also similar to southern pine species which are the predominant wood treated with CCA in the United States. The main limitation in extrapolating American results for residues and soil from CCA-treated wood to New Zealand relates to climate, particularly rainfall. In addition in the United States almost all CCA-treated decking and playground equipment is treated to a higher CCA retention specification than in New Zealand which means the dislodgeable residue concentrations reported there may be higher than what would be found on such structures in New Zealand.

Since children under seven years are most likely to exhibit mouthing behaviour children aged 2-6 years are considered the most at risk group in risk assessments of CCA-treated wood structures. The risk assessments cover a variety of exposure scenarios. In the absence of data on New Zealand children's activity patterns it is not possible to accurately assess how applicable these are for New Zealand. However several playground visits a week for children aged 2-6 years in towns and cities may be plausible and some children are likely to have more frequent exposure from a sand-pit, deck and/or play equipment at home.

It is difficult to compare assessments with one another as the values used for some exposure parameters vary widely and hence there is a large variation in the risk estimates. No standardised study protocol has been used for assessing exposure to dislodgeable CCA residues and prior to the recent US Consumer Product Safety Commission (US CPSC) study (2003) sampling methodology appears not to have been validated. Assessments based on wipe data are likely to have overestimated the amount of dislodgeable arsenic residue that would be transferred to hands and there is high variability among the reported results. Until the CPSC study (2003) little attempt had been made to correlate wipe and hand loading data. Wipe arsenic concentrations were found to overestimate hand arsenic concentrations approximately five-fold. Future use of this conversion factor is likely to give more realistic measures.

For those risk assessments that are well described in terms of assumptions made and exposure parameters used, the risk estimates for lung and bladder cancer range from about one additional case in a million people (1×10^{-6}) exposed above the background lifetime risk of developing lung or bladder cancer due to other factors to one in ten thousand (1×10^{-4}) people exposed. In other words the additional lifetime cancer risk ranges from less than the risk level of one in one hundred thousand (1×10^{-5}) regarded as tolerable for carcinogens by New Zealand regulatory agencies to an order of magnitude higher. Any increased risk for skin cancer is in addition to the risk for lung and bladder cancer.

Available New Zealand data on inorganic arsenic intake are insufficient for a risk assessment to be carried out with reasonable certainty. On the basis of limited data the estimated aggregate inorganic arsenic intake for an average New Zealand 2-6 year old child from food, drinking water and a daily playground visit (assuming the CPSC value of 3.5 µg ingested arsenic from CCA-treated wood surface residues applies in New Zealand) is below the tolerable intake of about 2 µg/kg body weight/day set by the Joint Food and Agriculture Organisation / World Health Organisation Expert Committee on Food Additives (JFECFA) (FAO/WHO, 1989). The tolerable intake is the amount that can be ingested daily per kilogram of body weight that represents a level of no appreciable health risk for a lifetime exposure. However aggregate exposure may be high for some children depending on their age, geographical location, home characteristics and daily activity.

Much of the information identified during this review was available only in the form of technical reports and was unpublished in the peer reviewed scientific literature. It was only possible to confirm that two risk assessments (WS Atkins International, 1998; US CPSC, 2003) had undergone independent scientific peer review.

There are no New Zealand data on the prevalence of CCA-treated wood decks or playground equipment and their age, the number of children likely to be exposed, activity patterns of New Zealand children involving these structures, and dislodgeable residue results from CCA-treated radiata pine structures.

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

Most risk assessments use toxicity values that have been developed by the US Environmental Protection Agency (EPA) for both cancer and non-cancer effects. Recently the CPSC has also used the National Research Council's (NRC) value for cancer effects. The Atkins report (WS Atkins International, 1998) used the World Health Organisation's (WHO) value for lung cancer from inhalation. Acceptance of these values varies depending on the perspective of the risk assessor with some emphasising their limitations more than others. The main uncertainty for the cancer toxicity values is that the mechanism of carcinogenesis of arsenic is not well established. In the absence of certainty linear extrapolation has been used to predict cancer risk at low levels of arsenic intake from the risks at moderate to high intakes. There is also uncertainty associated with averaging low dose arsenic short-term exposure over a lifetime.

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. In relation to reducing potential exposure from CCA recommended regulatory action includes ensuring "best practice" during timber treatment, ready identification of CCA-treated wood, improved public information including occupational health advice, sealing recently constructed CCA-treated wood structures particularly in public settings, and use of alternative materials in certain circumstances.

Since risk assessments necessarily assume that "best practice" has been followed during timber treatment and treated wood is not released onto the market before fixation is completed, monitoring of compliance with current standards needs to demonstrate that this in fact does occur.

Treated timber is already branded with the registered number of the treatment plant, the hazard class to which it has been treated, and in some instances the preservative. Branding or run-on printing on each board (and/or labelling) to identify the preservative could become a requirement.

Consumer information about safe handling and use needs to be widely available at the point of sale and precautionary health advice more widely disseminated to the general public and builders.

New playground equipment in schools, early childhood centres, and public parks could be built of alternative materials to restrict public 'involuntary' contact with CCA-treated wood.

Increased lung and bladder cancer risks have previously been reported among New Zealand builders. Although there are many exposures other than arsenic exposure eg asbestos, tobacco that may contribute to or be responsible for one or both of these increased risks, occupational cancer incidence and mortality data for the last 15 years need to be analysed to see if this trend has persisted, and if there is a need for further action.

A urinary biomonitoring study of children or builders, given the likelihood that their exposure is higher than that of children as they are regularly exposed for longer periods of time and also through inhalation, would overcome the uncertainty that currently exists concerning the

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

In the United States, Canada and the European Union a ban on the use of CCA-treated wood in residential and recreational settings will take effect in 2004. The regulatory decisions in the United States and Canada resulted from decisions made by the CCA industry. In the European Union the decision followed an assessment of health and environmental risks and took the precautionary principle into account. A number of member states already have restrictions on the use of CCA in place though in the United Kingdom a review by the Health and Safety Executive (HSE) resulted in a recommendation to government in 1999 for continuing use subject to certain environmental data and occupational health requirements. None of these regulatory decisions to ban the use of CCA-treated wood apply to CCA-treated wood already in use.

Risk assessments and/or evaluations of CCA are in progress by the US EPA and Pest Management Regulatory Agency (PMRA) of Health Canada, the European Commission (EC), and the Australian Pesticides and Veterinary Medicines Authority (APVMA).

4 Introduction

Since November 2002 there has been ongoing media and public interest in New Zealand in the use of CCA-treated wood and the health risks it may present particularly when used in settings where children may be exposed. This was initiated by a study of soil arsenic concentrations adjacent to CCA-treated wood playground equipment carried out for *The Dominion Post* newspaper and the findings of a group of American researchers (Maas et al. 2002). In November 2002 ERMA New Zealand established an inter-agency co-ordinating group[1] and commissioned this review of CCA-treated wood to determine whether further action may be required.

5 Copper, chromium and arsenic (CCA)

5.1 What is CCA?

CCA[2] wood preservatives are mixtures of the salts or oxides of the elements arsenic, chromium and copper. They are used for vacuum-pressure treatment (or variants of this such as alternating pressure treatment) of timber sold to commercial users and the general public. CCA is intended to protect wood against pests such as decay fungi, wood boring insects or marine borers that can threaten the integrity of wood products. Copper is used to control fungi and marine borers, arsenic to control insects and some copper-resistant fungi, and chromium to fix the copper and arsenic in the wood.

CCA treatment for wood preservation was invented in 1933 and subsequently became the most widely used waterborne preservative globally in the 1960s. Industry estimates that the global market reached a peak of about 100,000 tonnes (sufficient to treat 15 million m^3 of wood) in the early 1980s, declining to about 44,000 tonnes in 1996. In the 1990s the market declined due to the world economy and environmental concerns about CCA (WS Atkins International, 1998). Global use of CCA-treated wood was initially industrial but since the 1980s residential use has become widespread.

A number of CCA formulations are in use worldwide. Different formulations have been developed depending on the relative importance attached to control of insects or fungi, or, as in New Zealand, where changes in relative proportions of the active ingredients have been shown to improve resistance to leaching and improve efficacy (Hedley, 1984). In the United States and most of Europe CCA formulations are one of three types that conform to American Wood Preservers Association (AWPA) standards. Type C is the main formulation used worldwide and the formulation most commonly used to treat timber for above ground residential applications. Its composition (34% arsenic pentoxide, 47.5% chromium trioxide and 18.5% copper oxide) has similar ratios of active ingredients (copper, chromium and arsenic) to the CCA used in New Zealand and broadly equivalent to CCA Type 2 in Britain.

[1] Agencies represented on the Inter-agency Co-ordinating Group are ERMA New Zealand, Ministry of Health, Occupational Safety and Health, Ministry for the Environment, Ministry of Agriculture and Forestry, Ministry of Economic Development, Ministry of Education, Building Industry Authority and representatives of local government and the regional public health service.

[2] CCA is also known as chromated copper arsenate particularly in the United States.

5.2 CCA in New Zealand

CCA is the most common wood preservative used in New Zealand and has been in widespread use since the 1950s. In New Zealand the arsenic in CCA solutions used to treat timber is in its soluble pentavalent form as arsenic pentoxide (arsenic acid). The chromium used is in its soluble hexavalent form as chromic acid, sodium dichromate, or chromic oxide. Divalent copper is used in the form of copper sulphate or copper oxide (Ministry for the Environment and Ministry of Health, 1997).

In New Zealand CCA formulations are specified as proportions of the active elements. This is copper 23-25%, chromium 38-45% and arsenic 30-37%. Originally CCA formulations in New Zealand were categorised as either Class I or Class II (Table 1). There are 13 formulations (14 trade name products) of CCA registered for use in New Zealand, but only three are commonly used, which are oxide formulations conforming to the above specification.

Table 1: Former categorisation of CCA formulations

Element	Class I	Class II[3]
Copper	23-25%	20-30%
Chromium	43-47%	25-47%
arsenic	30-32%	30-50%

(Standards Association of New Zealand, 1992)

For salt formulations working strength solutions vary from less than 1% weight/volume to about 6% weight/volume depending on the treatment process and the level of protection for the timber (Occupational Safety and Health, 1994). Most treatment plants in New Zealand now use oxide formulations and solution strengths are around 60% of these values (personal communication, Forest Research, 28 February 2003).

5.3 Timber treatment in New Zealand

Timber is treated to a hazard class,[4] which is one of six environments (H1-6) where timber is at risk of biodegradation (Table 2). In New Zealand H2 is not used. CCA preservatives are approved for use in all hazard classes. The timber used most commonly for residential purposes is in classes H3 and H4. Timber for decks and garden furniture would typically be H3 with H4 for deck support posts in the ground and timber for playground equipment in the ground a mixture of H3, H4 and H5, depending on the local soil conditions and the equipment being supported on piles. H3 and H4 treated timber contain 0.11% and 0.22% arsenic per oven dry wood weight respectively.

In a typical CCA treatment process schedule, untreated wood is placed into a cylindrical treatment vessel and a vacuum applied to remove air in the dry wood. The vessel is then filled with CCA solution and pressure applied which forces the preservative into the wood cells. Typical time on pressure is 45-60 minutes to achieve the required preservative solution uptake. Following pressure release and draining, a further vacuum is applied to extract excess solution and minimise post-treatment drip. Steam may also be used at the end of the process

[3] Other formulations not complying with Class I but with these proportions of the active elements.
[4] The hazard class system of categorising timber treatment was introduced in New Zealand in 1986 replacing commodity specifications.

to increase the rate of fixation, though this is only practised by some of the large treatment plants in New Zealand and for certain hazard classes eg H4. The wood is then removed and stored for 7-14 days before release onto the market.

Table 2 - Hazard Classification

Hazard class	Exposure	Service Conditions	Biological Hazard
H1	Protected from the weather, above ground	Protected from weather, dry, exposed to ground atmosphere where well ventilated.	Anobiid or lyctid (ie, insects other than termites)
H2	Protected from the weather, above ground	Protected from weather, dry, exposed to ground atmosphere where well ventilated but not in contact with ground.	Borers and termites
H3	Exposed to the weather, above ground	Periodic wetting, not in contact with the ground	Decay fungi, insects including termites
H4	Exposed to the weather, in ground or in fresh water	Severe or continuous wetting, continuous ground contact	Decay fungi, insects including termites
H5	Exposed to the weather in ground or in fresh water	Severe or continuous wetting, continuous ground contact where uses are critical and a high level of protection is expected	Decay fungi, insects including termites
H6	Marine, water or estuarine ground	Immersion in seawater or esturine ground.	Decay fungi and marine wood borers

The fixation mechanism is complex and the reactions involved depend on time, temperature, wood species, preservative type, pH and drying conditions. Treatment performance is often better in softwood species such as radiata pine, which are high in lignin, than hardwoods (Hingston et al. 2001). Fixation begins during the treatment process and generally takes about 14 days for completion. This would only apply to surface fixation (outer 5-10 mm) in parts of New Zealand in winter (personal communication, Forest Research, 28 February 2003). Arsenic is fixed as insoluble pentavalent arsenic and chromium ends up as its insoluble trivalent form. The ratio of arsenic to chromium influences the amount of arsenic that is fixed. A proportion of the arsenic will be available for leaching if there is insufficient excess chromium. Chromium is the slowest fixing component. It is possible to undertake on-site testing to confirm efficacy of fixation. Fixation is deemed to be complete when all hexavalent chromium has been reduced to trivalent chromium.

CCA present on the surface of the wood is essentially completely fixed within 2-3 days at ambient temperatures. After that time the timber may be safely handled wearing gloves and overalls (Occupational Safety and Health, 1994).

Freshly treated CCA wood is green and over time changes to grey with weathering, making it indistinguishable from untreated wood. If salt formulations are used, sodium sulphate which is non-toxic may appear on the wood surface as white crystals or a powdery substance for up

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

There are thought to be about 165 timber treatment plants in New Zealand, most of which use CCA. The New Zealand Timber Preservation Council (TPC) estimate 5,000 tonnes of CCA salt equivalent are used annually to treat an estimated 650,000 m^3 of timber to various treatment grades. Non-TPC processors would add several thousand cubic metres to this total. For the year ended March 2002 annual production of sawn timber was 3,845,000 m^3 and "small log" production about 400,000 m^3. CCA-treated posts and poles would be drawn from the latter category.

Timber treatment was regulated in New Zealand from 1955 to 1987. The Timber Preservation Regulations 1955 led to the establishment of the Timber Preservation Authority (TPA). The main issue at that time was the treatment of framing timber with boron-based wood preservatives. Following deregulation the regulatory role of the TPA was taken over in 1987 by an industry-based body, the TPC. The Timber Industry Federation (TIF) promotes New Zealand treated pine locally and overseas and administers the TPC.

The TPC's primary function is to monitor a quality assurance scheme for 148 treatment plants that represent 98% of New Zealand production. The TPC manages the WOODmark® scheme through its Timber Preservation Quality Manual and quarterly sampling and analysis of treated timber at licensees' plants. The WOODmark® brand indicates that wood has been treated to the requirements of NZS MP3640 and the treater follows the quality assurance procedures in the Timber Preservation Quality Manual. The sampling programme includes provisions to suspend or delicense plants where required treatment standards are not being met. It also publishes health and safety information and holds training seminars for point of sale staff at retail yards (Hawkins, 2003). Non-TPC treatment plants fall outside this scheme, but are technically supported by chemical suppliers. An independent assessment of the extent to which the timber industry as a whole adheres to "best practice" is not available.

Standards include methods for sampling timber and analysing timber preservatives and preservative-treated wood (AS/NZS 1605:2000), standards to improve occupational health and safety and minimise environmental hazard (AS/NZS 2843: 2000 Parts 1 and 2) and standards for penetration and retention requirements and element proportions for the composition of CCA (NZS MP3640: 1992, amended in 2003 as NZS 3640).

An approved Code of Practice under the Health and Safety in Employment Act 1992 for the safe use of timber preservatives has been published by the Occupational Safety and Health Service (OSH) (Occupational Safety and Health, 1994). Compliance was mandatory immediately for all new preservation plants and from June 1995 for existing plants. The Code specifies initial holding periods (for 48 hours on a roofed holding pad or until the wood is drip-free whichever is the lesser) but not requirements for storage of treated wood which may not yet be fixed.

A small study of selected timber treatment plants and treated timber storage sites in Auckland found only one of nine operating plants fully complied with the environmental provisions of the Code. Examples of non-compliance included ineffective or absence of drip pads and uncovered treatment and drip pad areas. The results of targeted sampling including soil and stormwater runoff indicated leaching from CCA-treated wood at timber storage sites (White,

1998). Reasons for non-compliance were not mentioned. This study raises the possibility that occupational exposure may be greater than generally thought particularly at storage sites.

Following the US EPA announcement in February 2002 to phase out most residential uses of CCA-treated wood, the New Zealand industry sought clarification about health and environmental concerns from Australasia's major CCA suppliers (Osmose and Koppers Arch Wood Protection) and various government agencies. Subsequently the TIF issued a statement that there is no scientific evidence that the continued use of CCA-treated wood poses any significant risk to public health and safety.

5.4 CCA-treated wood

New Zealand and Australia are the largest per capita users of CCA-treated wood (frequently referred to as tanalised® timber) in the world (Christmas, 2002). In New Zealand the predominant wood type treated is *Pinus radiata* (Connell et al. 1995). Radiata pine is similar to southern pine[5] (personal communication, Forest Research, 28 February 2003) which is the wood generally used in the United States studies of leaching and dislodgeable wood surface residues of CCA. Southern pine is a generic name used to refer principally to *Pinus palustris, Pinus echinata, Pinus taeda, Pinus elliottii, Pinus rigida* and *Pinus serotina* (personal communication, Ministry of Agriculture and Forestry, 18 March 2003). These are the predominant species treated with CCA in the United States.

In New Zealand treated timber, irrespective of the type of wood preservative, must be branded with the registered number of the treatment plant and the hazard class to which it has been treated. Packet branding is allowed for small dimension timber eg fence palings (Ministry of Forestry, 1987). Treatment plants which supply the Australian market also identify the preservative in the brand (personal communication, Forest Research, 28 February 2003). The main limitation in terms of informing the end user is that the brand can be cut off, although this limitation does not apply if strip branding along one face of the timber is undertaken.

There is no legislation in New Zealand that restricts use of CCA treated wood to particular end-uses. General recommendations on treated timber use in New Zealand include that it should not be used for food receptacles, toys, barbecues, smoking meat or fish, or domestic fires. Treated timber sawdust should not be used for areas under playground equipment (Ministry of Forestry, 1987).

CCA-treated wood is most commonly used in outdoor settings. Domestic uses include decks, garden furniture, picnic tables, playground equipment, landscaping timbers, retaining walls, fences, gazebos and patios. Other common uses include docks, signposts, utility poles, and agricultural or horticultural posts.

Except for decking, residential use of CCA-treated wood is thought to have begun earlier in New Zealand than the 1980s. CCA treatment started in New Zealand in the late 1950s but was initially mainly for fencing materials and poles. Use for building timbers started to expand in the late 1960s but available statistics do not distinguish the specific use. Production of all preservative treated wood in New Zealand has declined since the mid-1990s but not to a large extent. Although there are no national trend data available that indicate size and timing of the peaks for CCA-treated wood these coincided with expansion of kiwifruit orchards and viticulture (personal communication, Forest Research, 28 February 2003).

[5] Southern pine is also referred to as southern yellow pine.

There are no data on the prevalence of playground equipment or decks constructed from CCA-treated wood in New Zealand.

In response to the current weather-tightness problems associated with unsuitable housing design and certain building practices, the Building Industry Authority is proposing amendments to the Building Code (NZBC B2/AS1 dealing with the durability of timber) which will require a move away from the use of untreated timber in exterior framing. The proposed amendments will shortly be released for public consultation with a final decision likely in June 2003. This may mean an increase in use of CCA-treated wood.

The types of CCA-treated wood also differ in their retention level of the preservative elements. With the exception of H1 timber, retentions in New Zealand and Australia are expressed in terms of the weight of the total active elements (for H6 in New Zealand it is percentage of copper only). Other countries express retentions as kilograms of CCA salt or oxide per cubic metre or pound per cubic foot (United States). In New Zealand retentions are those in specified retention zones which differ with hazard class. Lower levels are suitable for above ground uses eg for H3 timber the minimum retention is 0.37 % total active elements within the outer 10 mm. This approximates to a CCA salt retention of 6 kg/m^3 (oxide retention of 3.7 kg/m^3). In the United States almost all CCA-treated decking and playground equipment is treated to the ground contact retention (6.4 kg oxides/m^3) rather than above ground retention specification. This means that the dislodgeable residue concentrations reported there may be higher than what would be found on such structures in New Zealand.

In the ground untreated radiata pine sapwood will completely fail from decay within 2-3 years depending on its cross-section dimensions. In above ground situations the average life varies (personal communication, Forest Research, 28 February 2003). Expected service life of CCA-treated wood is 20-50 years, depending on end-use and exposure conditions. Manufacturers guarantee CCA-treated wood from insect attack and fungal decay for 50 years when prepared, treated and used in accordance with New Zealand standards.

Product standards NZS 5828:1986 Parts 2 and 3 give the specification for playground equipment. Timber must comply with NZS 3602:1975[6] *Code of practice for specifying timber and wood-based products for use in building* as appropriate to species, grades and preservative treatment. Less durable timbers may be used if preservative-treated to a level appropriate to the extent of exposure. Wood preservatives are required to comply with the relevant requirements contained in MP 3640:1992 *Minimum requirements of the NZ Timber Preservation Council (Inc)*. There must be at least three weeks between the time the timber was treated with CCA and put into service. As freshly treated timber may initiate corrosion of metal fastenings, six weeks is suggested unless measures are taken to protect fastenings. Off-cuts of CCA-treated timber should be removed from the site and the wood should not be used for cooking or burned in confined places (Standards Association of New Zealand, 1986). New and existing equipment are required to meet these product standards (Standards Australia and Standards New Zealand, 1997). A new playground equipment standard is currently being developed.

[6] NZS 3602 was amended in 1990 and 1995.

5.5 Toxicity of CCA

Acute oral LD_{50}[7] values for CCA formulations are in the range < 150 - < 400 mg/kg. Acute dermal LD_{50} values are in the range 200 - 1188 mg/kg. The lethal dose for humans is much lower and as low as 1-2 mg/kg for some formulations (personal communication, National Poisons Centre, 20 March 2003). They are classified as corrosive based on their pH of less than 2 and as a skin sensitiser (Health and Safety Executive (HSE) 2001). The proposed Hazardous Substances and New Organisms (HSNO) classifications and default controls are contained in a consultation document (ERMA New Zealand, 2002).

Exposure to CCA-treated wood is a combined exposure to chromium, copper and arsenic. Effects associated with such exposure may differ from effects caused by exposure to each component in isolation.

Extensive data exists for the components of CCA but only a few reports on the toxicology of the CCA mixture (HSE, 2001).

Six animal studies involving short-term oral or dermal exposures to sawdust from CCA-treated wood and arsenic levels ranging from 0.4-130 mg/kg body weight found no evidence of adverse health effects (Gradient Corporation, 2001).

A few animal studies involving relatively high doses indicate interactions between copper, chromium and arsenic such as changes in toxicity and toxicokinetics (US CPSC, 2003). The relevance of these findings to humans exposed to CCA-treated wood is unknown although it is noted that the form of chromium used was hexavalent rather than trivalent chromium which is the predominant form that humans would be exposed to from CCA-treated wood.

No carcinogenicity studies on CCA per se have been identified (Huff, 2001).

6 Copper

Copper is also toxic but less so than arsenic or chromium. Unless otherwise stated the following information has been sourced from a review published by (ATSDR, 2002).

6.1 What is copper?

Copper is a naturally occurring element that is found in rocks, soil, water, sediment, plants and animals. It also occurs as a result of anthropogenic activity. In general, sites with soil copper concentrations in the range 1 - 190 mg/kg do not require further assessment (Australian and New Zealand Environment and Conservation Council, National Health and Medical Research Council, 1992).

Divalent copper, which is the form of copper in CCA, binds to soil components and has limited mobility. Mobility is greater in sandy soils.

[7] The dose which has been calculated to cause death in 50% of a defined experimental animal population.

6.2 General population exposure

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

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

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

6.3 Bioavailability

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

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

The bioavailability of copper in soil is unknown.

6.4 Toxicity

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

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

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

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

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

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

7 Chromium

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

7.1 What is chromium?

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

It also occurs as a result of anthropogenic activity. In general, sites with soil chromium concentrations in the range 0.5-110 mg/kg do not require further assessment (Australian and New Zealand Environment and Conservation Council, National Health and Medical Research Council, 1992).

CCA products contain hexavalent chromium which is reduced to trivalent chromium by organic compounds once inside wood. Sawdust from CCA-treated wood has been found to contain 0.3-0.4% total chromium, less than 2% of which was its hexavalent form (Cruz et al. 1995).

Trivalent chromium is generally considered to be stable and immobile in soil. However trivalent chromium in soil has been shown to oxidise to the more soluble and mobile hexavalent chromium in the presence of oxidised manganese (Bartlett and James, 1979). Reduction of hexavalent to trivalent chromium is facilitated by low soil pH.

7.2 General population exposure

Trivalent chromium is an essential element in humans involved in glucose, fat and protein metabolism. The recommended dietary intake in the United States is 10 - 80 µg/day and 30 - 120 µg/day for children aged 1 - 3 and 4 - 6 years respectively, and 50 - 200 µg/day for those aged seven or more years of age.

Food, followed by drinking water and air, is the main source of exposure of the general population. Content in food varies considerably and depends on processing and preparation eg total chromium levels are higher in acidic foods cooked in stainless steel utensils.

In the New Zealand drinking water guidelines the provisional MAV for chromium is 0.05 mg/L (Ministry of Health, 2000) and the residential land use soil criteria assuming 10% of produce consumed is home-grown are 600 mg/kg for trivalent chromium and 25 mg/kg for hexavalent chromium (Ministry for the Environment and Ministry of Health, 1997).

7.3 Bioavailability

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

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

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

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

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

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

7.4 Toxicity

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

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

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

Hexavalent chromium is classified as a human carcinogen based on excess lung cancer found in heavily exposed workers through inhalation in chromium plating and chromate and chromate pigment production. Epidemiological investigation of cancer mortality in communities with environmental hexavalent chromium exposure through drinking water has found no evidence of cancer hazard (Fryzek et al. 2001). This study, although an ecological study, is the best epidemiological data currently available on potential carcinogenicity of environmental hexavalent chromium exposure. The sample size was large and there was a long latency period. Animal data are not definitive because of methodological limitations (Flegal et al. 2001).

The chromium in dislodgeable residues from CCA-treated wood is most likely to be trivalent chromium which is not classifiable with respect to carcinogenicity due to insufficient evidence. There is no reliable evidence on the presence or absence of hexavalent chromium in

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

Chromium is a common skin sensitiser. Direct dermal contact with both trivalent and hexavalent chromium causes skin irritation and allergic contact dermatitis though the hexavalent form is much more potent. The main cause is occupational exposure and environmental exposure to chromium is unlikely to result in these effects. Soil concentrations up to 450 mg/kg for hexavalent chromium and 165,000 mg/kg for trivalent chromium have been reported as not posing a hazard of allergic contact dermatitis to almost all people who might be exposed through dermal contact.

8 Arsenic

8.1 What is arsenic?

Arsenic is a naturally occurring element found in rocks, soil, water, air, plants and animals. Both inorganic and organic compounds of arsenic are widely distributed in the environment. Inorganic forms predominate in soil and water. Organic forms are found mainly in plants and animals, and are considered relatively non-toxic to mammals (Agency for Toxic Substances and Disease Registry (ATSDR), 2000b).

In New Zealand arsenic occurs naturally in soils and from geothermal activity, and from anthropogenic activity such as the use of pesticides. The average arsenic concentration of New Zealand soils is 6-7 mg/kg (Ministry for the Environment and Ministry of Health, 1997). In general, sites with soil arsenic concentrations in the range 0.2-30 mg/kg do not require further assessment (Australian and New Zealand Environment and Conservation Council, National Health and Medical Research Council, 1992). Background levels are naturally high in the volcanic soils of the central North Island.

The arsenic used in the manufacture of CCA is a by-product of copper refining. It is estimated that 70% of global arsenic production is used in timber treatment as CCA (IPCS, 2001).

Inorganic arsenic occurs in both trivalent (arsenite) and pentavalent (arsenate) states. Pentavalent arsenic is the form of arsenic in CCA.

8.2 General population exposure

Arsenic has not been shown to be an essential element in humans.

Food followed by drinking water is the main source of exposure of the general population to inorganic arsenic. Seafood is the main dietary source of arsenic but the arsenic present is organic and relatively non-toxic. In the United States diet accounts for about 70% and soil for less than 1% of estimated daily inorganic arsenic exposure (Valberg et al. 1997).

Average daily inorganic arsenic intake in the United States is 0.1-2.6 µg/kg body weight which equates to about 2-46 µg for a young child (ATSDR, 2000b). Approximately 25% of arsenic present in food is inorganic, although this is highly dependent on the type of food eaten (IPCS, 2001).

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

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

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

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

Unpublished results from ESR for arsenic in drinking water based on 1300 recent samples give a mean concentration of 0.002 mg/L and a maximum reported concentration of 0.069 mg/L. Twenty-two supplies serving a population of 11,168 exceeded the MAV. Supply counts are based on there being 'any sample above the MAV' and therefore do not take into account natural fluctuations that may occur and analytical variance. For the mean figure, results below the limit of detection were taken as zero assuming that supplies with arsenic detected are over-represented in the sampling and this balances counting non-detects as zero (personal communication to the Ministry of Health, ESR, 6 August 2002).

The interim soil criterion recommended for arsenic in agricultural and residential land use in New Zealand is 30 mg/kg. The value is interim because of uncertainty about the bioavailability of arsenic in soil and the need for further research. The criterion corresponds to an additional lifetime risk of mortality from skin cancer of 0.5 to 7 in 100,000 for residential land use assuming 10% of produce consumed is home-grown. Assumptions in this risk assessment are that bioavailability is 100%, soil ingestion is 100 mg/day, and exposure

[11] The PTWI is the amount of a substance that can be ingested over a week per kilogram of body weight that represents a level of no appreciable health risk for a lifetime exposure. Average intake over a week rather than each day is important which means exposure on a particular day may exceed its proportionate weekly share.

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

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

8.3 Bioavailability

Soluble arsenic compounds are well absorbed when inhaled or ingested (55-95%) by humans, and to some extent also through the skin (2-6%) (US CPSC, 2003).

Factors that can affect bioavailability of ingested soil arsenic include the form of arsenic, soil particle size and soil chemistry. Solubility of the form of arsenic in the soil is critical with increased water solubility correlated with increased absorption. Biological factors such as the presence of food in the gastrointestinal tract and health status may also influence absorption from soil.

Bioavailability estimates for arsenic in soil based on animal studies range from almost zero to about 98% (ATSDR, 2000b). The relative bioavailability of arsenic resulting from CCA-treated wood in a range of soil types is unknown (Federal Insecticide, Fungicide, and Rodenticide Act Scientific Advisory Panel (FIFRA SAP), 2001). The EPA's Scientific Advisory Panel (SAP) did not agree to use the value of 25% based on data from primates that was proposed by the EPA for use in its risk assessment, and recommended 100% until further research has been carried out (FIFRA SAP, 2001).

Bioavailability of arsenic in soil from a wood treatment site was 16% in a study which involved monkeys (Roberts et al. 2002). To give the monkeys sufficient arsenic in a reasonable volume that allowed analytical detection in urine the soil had an arsenic concentration of 101 mg/kg. Animal studies usually involve soil arsenic concentrations of 100 mg/kg or more. It is not known whether bioavailability at these concentrations predicts bioavailability at lower arsenic concentrations.

Urinary arsenic comprises organic and inorganic arsenic. In contrast to epidemiological evidence showing a positive correlation between urinary arsenic and water arsenic concentrations, the evidence correlating urinary arsenic in potentially affected populations and soil ingestion is inconsistent. This may relate to lack of or variable exposure to soil arsenic, or low soil arsenic bioavailability (Valberg et al. 1997).

Dermal absorption from soil is about 1-5%. Soap and water readily removes arsenic from skin in both *in vivo* and *in vitro* experiments (US CPSC, 2003).

There are no human studies measuring arsenic bioavailability from ingestion of CCA-treated wood surface residues. The bioavailability estimate for dislodgeable arsenic used by Gradient Corporation (2001) was 47% based on two studies in dogs fed sawdust from CCA-treated wood.

Since the bioavailability of ingested dislodgeable CCA residues has not been characterised it is assumed to be 100% (FIFRA SAP, 2001). The assumption that it is equivalent to the bioavailability of soluble arsenic in water has been disputed because arsenic in CCA residues

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

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

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

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

8.4 Toxicity

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

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

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

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

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

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

chronic inorganic arsenic exposure from drinking water and food had a significantly higher proportion of inorganic arsenic in urine than women, indicating low methylation efficiency. This could be related to dietary deficiency as the authors noted that some malnutrition existed in one of the study villages and did not comment on the general health status of the study participants, or to a genetic polymorphism for the methylation enzymes. The health significance of this is unknown.

Acute exposures = 0.05 mg/kg/day by ingestion have caused vomiting, diarrhoea, abdominal pain and gastrointestinal haemorrhage, changes in liver and renal function, hypotension, tachycardia, pulmonary oedema and difficulty breathing. Death may occur (US CPSC, 2003). The low level exposures that most of the general population will experience from contact with CCA-treated wood are extremely unlikely to result in these acute health effects. For example, a 15 kg child (3 year old) would need to ingest 0.75 mg arsenic/day which is about 200 times more than the amount of arsenic that the CPSC estimates is ingested from dislodgeable residues on CCA-treated playground equipment per playground visit (US CPSC, 2003).

Arsenic dust is an irritant to mucous membranes of the nose, throat and upper respiratory tract from around 0.1-1 mg/m^3 (European Commission's Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), 2001).

Inorganic arsenic is classified as a human carcinogen. A wide range of adverse health effects including skin, lung and bladder cancers, cardiovascular disease, diabetes, gastrointestinal, hepatic, haematological and neurological effects have been attributed to chronic arsenic exposure primarily from drinking water. Dermal effects such as hyperpigmentation and hyperkeratosis are characteristic of long-term exposure. They often but do not always appear before skin or internal cancers. Dermal and cardiovascular effects occur with chronic exposure from about 0.002 mg/kg/day (US CPSC, 2003). One large study did not detect any effects at 0.0008 mg/kg/day and is the basis for the EPA's oral reference dose[12] of 0.0003 mg/kg/day.

The most sensitive toxic endpoints on which human health risk assessment is based are lung cancer by inhalation, skin, lung and bladder cancers by ingestion and non-cancerous skin lesions (hyperkeratosis, hyperpigmentation) by ingestion. Arsenic carcinogenicity is considered the most significant endpoint for use in risk assessments of CCA-treated wood.

Non-cancer effects from chronic ingestion have been detected at doses as low as 0.01 mg/kg (NRC, 1999). Most of the epidemiological studies for cancer are of populations eg Taiwanese exposed to arsenic concentrations in drinking water of at least several hundred milligrams per litre. Few data address cancer risk at lower concentrations though a recent study in Taiwan suggests a significant risk of bladder cancer exists at levels within the range 10.1 –50.0 µg/L (Chiou et al. 2001).

Ingestion of arsenic has also been associated with increased risks of liver and kidney cancers (Smith et al. 1992) and possibly other sites (Bates et al. 1992). The Taiwanese studies show dose-response relationships that are strongest for bladder cancer followed by cancers of the kidney, lung and liver (Bates et al. 1992). More recent data from epidemiological studies in

[12] The EPA's oral reference dose is an estimate of daily exposure to a substance that a sensitive population can experience over a lifetime with a negligible risk of adverse systemic health effects. For arsenic the value is derived from the No Observed Adverse Effect Level using an uncertainty factor of 3.

Taiwan and Chile have increased the weight of evidence for an association between lung and bladder cancers and arsenic in drinking water (NRC, 2001).

Although trivalent arsenic is more toxic than pentavalent arsenic, in the drinking water studies levels are generally reported as total inorganic arsenic so that the arsenic may be present as trivalent and/or pentavalent arsenic.

Inhalation of inorganic arsenic is associated with lung cancer. Although lung cancer is the critical effect following chronic inhalation, total arsenic exposure rather than airborne exposure alone seems to determine the incidence of lung cancer (CSTEE, 2001). Tobacco smoking increases the lung cancer risk from arsenic inhalation (IPCS, 2001).

Arsenic is the only established human carcinogen for which there is mostly negative animal evidence of carcinogenicity when given alone (ATSDR, 2000b). Some animal studies have reported arsenic carcinogenesis when there was coexposure to other carcinogenic treatments suggesting copromotional or cocarcinogenic effects. In contrast, findings of a recent animal study suggest that arsenic may act as an initiator. In this study tumours were found at multiple sites in adult offspring of mice which were briefly exposed to inorganic arsenic in drinking water during pregnancy (Waalkes et al. 2003). Several of the sites eg lung correspond to human sites associated with oral arsenic exposure. Epidemiological studies are therefore more important for arsenic than for some other chemicals because of this lack of an established animal model of arsenic carcinogenesis and provide the only quantitative data for guiding risk assessment.

Although animal data indicate that arsenic is a probable developmental toxicant (US CPSC, 2003) exposure to arsenic from CCA-treated wood is unlikely to affect unborn children. A review of toxicological studies on developmental toxicity of arsenic concluded that the studies that indicated adverse developmental effects involved non-oral doses (eg intravenous) and doses in excess of those relevant to human exposure (DeSesso et al. 1998).

A few toxicological studies have shown some endocrine-related effects of trivalent arsenic (Gradient Corporation, 2001). The data, however, are insufficient to conclude that arsenic is an endocrine disruptor (US CPSC, 2003).

Acute arsenic poisoning has been reported following burning of CCA-treated wood in a barbecue (Geschke et al. 1996).

There have been about 30 successful lawsuits in the United States for acute poisoning associated with construction using CCA-treated wood. The Gradient Corporation report (2001) discusses three of these injury claims and the diagnosis of arsenic poisoning is disputed. During this review no reports of health effects arising from ingestion of arsenic in the absence of building or sanding activity were identified. There are no known reports of health effects associated with CCA-treated wood in New Zealand.

The mechanisms by which inorganic arsenic causes toxicity including cancer are not well established, but probably occur through multiple independent and interdependent mechanisms (NRC, 2001).

Results of genotoxicity studies are mixed. In general arsenic is an inactive or weak mutagen but able to produce chromosomal effects in most test systems. Although the mechanism of

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

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

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

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

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

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

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

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

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

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

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

9 Migration of CCA

9.1 Migration from soil

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

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

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

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

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

9.2 Migration from treated wood

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

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

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

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

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

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

Aceto and Fedele (1994) found using simulated rainwater that between pH 4.5 and 6.1, 21-24% of copper, 7% of chromium and 6% of arsenic were released after three days from CCA-treated wood. At pH 3 the proportions were 100%, 14% and 18% respectively. The experiment was carried out under conditions to optimise leaching i.e. coarsely powdered wood rather than wood blocks.

Leaching from new and weathered CCA-treated wood was shown with continual immersion particularly under acidic conditions. Chromium (53 - 12% leached when pH 3.5 - 5.5)

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

Maas et al. (2002) studied leaching under simulated rain conditions. No hexavalent chromium was detected and arsenic results for 6 month old weathered wood (mean 806.4 µg/L) were 73% and 47% of those for new CCA-treated wood from two different retailers. As expected due to the larger exposed surface area per unit mass, much higher leachate arsenic concentrations were found for sawdust from new wood and detectable levels (10 µg/L) of hexavalent chromium were found in two samples.

Standard and extended laboratory leaching tests (American Wood Preservers Association procedure E11 for 14 days and extended for 50 days) were found to significantly overestimate the amount of CCA leached from radiata pine decking during 300 days of in-service weather exposure in Brisbane, Australia. During this period, leaching rates ranged from 0.34 - 0.52 mg/m^2/day for copper, 0.50 - 0.58 mg/m^2/day for chromium, to 1.4 - 2.10 mg/m^2/day for arsenic. One percent of copper, 1% of chromium, and 4% percent of arsenic were leached (Kennedy and Collins, 2001).

Taylor et al. (2001) evaluated the effect of commercial deck wash/brightener treatments applied to new decks on leaching. With two exceptions the amount of CCA components leached by treatments was comparable to leaching from natural rain events prior to application of the treatment. The exceptions were high copper levels from citric acid treatment and hexavalent chromium levels (following conversion of trivalent chromium) from strong oxidising treatments eg sodium hypochlorite. Amounts were reduced by about half when treatments were repeated after one year of natural exposure.

Hexavalent chromium was also reported by Maas et al. (2002) in rinsate from two commercial deck wash treatments containing oxidising agents on 6 month and 8½-year-old decks.

9.3 Levels of CCA components in soil/sand from treated wood structures

Copper, chromium and arsenic levels in soil beneath CCA-treated wood decks have been found to be significantly elevated compared to control soil samples taken at least five metres away from the deck (Stilwell and Gorny, 1997). Average soil levels beneath decks were 75 mg/kg for copper, 43 mg/kg for chromium and 76 mg/kg for arsenic. Concentrations tended to increase with deck age. Relative chromium and to a lesser extent arsenic soil levels were less than the levels in new CCA-treated wood indicating that they are bound more effectively in wood than is copper.

In a Canadian study most of the sand samples taken below and near playground structures had 2-10 times the arsenic concentrations as control soil samples taken at the same playgrounds (Riedel et al. 1990).

Stilwell and Gorny (1997) found a marked decrease in copper, arsenic and chromium levels a short distance from the deck. The concentration of arsenic in soil has been reported to be near

[16] Heartwood is the non-living central part of a tree trunk.
[17] Sapwood is the outer part of a tree trunk which contains living cells and water conducting and storage tissue.

background levels within 0.5-1 metre of a CCA-treated wood structure (Baines, 2002). Soil arsenic concentrations in raised garden beds made from CCA-treated wood were highest within a few centimetres of the wood with decreases in concentration at greater distances (Alamgir et al. undated).

The Environmental Working Group (EWG), an environmental research organisation in the United States, has operated a home sampling programme with the Healthy Building Network (HBN) and the Environmental Quality Institute, University of North Carolina-Asheville since November 2001. In August 2002 it released a report that included results of 121 soil samples near 109 structures collected by members of the public using a home sampling kit.[18] Thirty-eight percent of the backyards or parks tested were reported to have soil contaminated with arsenic at the EPA's recommended cleanup level of 20 mg/kg or greater (Gray and Houlihan, 2002). The results and the data on which they were based are to be evaluated by the CPSC (US CPSC, 2003). There is no discussion in the report about methodological issues such as adherence to sampling instructions, or reliability and sensitivity of the test kits.

A study of three Wellington public playgrounds and one school playground carried out for *The Dominion Post* found arsenic concentrations in the soil around public playground structures in excess of New Zealand human health guidelines for residential land of 30 mg/kg (Mirams, 2002a). The exposures considered in deriving this health-based criterion are not fully applicable to a park setting where exposure frequency and duration are much less and there is no consumption of produce grown on the land. What was not reported in the media was the finding that in two instances the background soil levels also exceeded the New Zealand criterion (Table 3). It is plausible that the values reflect the presence of construction debris rather than leaching of arsenic from CCA-treated wood. This is supported by a media report quoting the scientist who undertook the analysis as saying the highest concentrations were found in samples that included wood shavings as well as soil (Mirams, 2002b).

Only three samples, one of which was a background sample taken about six metres from the playground equipment, were taken from each site. Both other samples were taken near to CCA-treated wood – one at about 2 cm and the other 15-18 cm from the surface.

Table 3: Soil arsenic results (mg/kg)

Site	2cm	18cm	Background
Karori	290	300	110
Khandallah	270	54	< 10
Island Bay	52	65	44
school	< 10	20	< 10
Mean	155.5	109.8	43.5

(Data derived from Mirams (2002a) and anonymous data (A1-A3, B1-B3 etc) received from *The Dominion Post* by the New Zealand Timber Industry Federation (Hawkins, 2003)).

A review of studies of soil arsenic from CCA-treated decks indicates that construction debris eg sawdust, wood shavings can be an important source of arsenic for soils surrounding CCA-

[18] Availability of this home test kit for wipe and soil sampling from www.leadtesting.org at a cost of US$17 was promoted by Dr Maas in *The Dominion Post*. Expert wants to assess NZ risk levels. 4 December 2002.

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

9.4 Dislodgeable CCA surface residue levels

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

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

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

The EWG's report (Gray and Houlihan, 2002) also included results of 300 wipe samples from 263 CCA-treated wood structures carried out by members of the public using a home sampling kit. Apart from high end results, dislodgeable arsenic results are presented in the report graphically and not numerically so it is difficult to determine the mean accurately. The report concludes that dislodgeable residue arsenic concentrations are not affected by the age of the structure.

Maas et al. (2002) tested CCA-treated wood for dislodgeable arsenic using surface wipes and chromium using glove wipes. Wipe sampling was based on the method developed by the CPSC and varied slightly. Mean arsenic was 60.5 µg/100 cm^2 and 45.6 µg/100 cm^2 for new CCA-treated wood from two retail locations and 26.8 µg/100 cm^2 for 6 month old weathered wood. These values are within the wide range of those previously reported. No hexavalent chromium was detected.

Some studies indicate that the amount of dislodgeable arsenic residues on CCA-treated wood surfaces decreases within a year as a result of sunlight and rain (Baines, 2002).

In a laboratory experiment carried out by the CPSC (2003) the amount of dislodgeable arsenic decreased as the number of rubs applied to a given area of new wood increased, although measurable amounts remained. This may be the result of a saturation point and/or removal of easily dislodgeable arsenic. Further dislodgeable residue is expected to return to the surface but the time it takes to do so from subsurface arsenic in the wood once the initially available arsenic is removed is unknown (US CPSC, 2003).

The mean dislodgeable arsenic concentration was 7.7 µg for decks (n = 8) and 7.6 µg for playground equipment (n = 12) in the field study carried out in metropolitan Washington, DC by the CPSC (US CPSC, 2003). Arsenic was found on surfaces ranging from a few days old to 18 years old. Effects of age and maintenance on arsenic migration could not be determined from the CPSC studies.

9.5 Mitigation of CCA

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

10 Plant Uptake of CCA Components

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

Elevated arsenic levels were found in carrots and potatoes grown near a wood preservation factory from both soil uptake and atmospheric contamination. In contrast elevated arsenic levels found in leafy vegetables was from atmospheric deposition not soil uptake (Larsen et al. 1992). Levels of arsenic in carrots, spinach and beans grown in raised garden beds made with CCA-treated wood were significantly higher than those grown in control soils but below recommended public health limits for consumption (Alamgir et al. undated). Lettuce grown alongside CCA-treated wood blocks contained 1.7 mg/kg arsenic compared to less than 0.4 mg/kg for lettuce grown in control conditions (Stilwell, 1999).

Levi et al. (1974) reported that no chromium or arsenic was detected and levels of copper similar to controls were found in grapes grown near CCA-treated posts.

11 Regulatory Approaches

11.1 New Zealand

In New Zealand CCA is regulated under the Hazardous Substances and New Organisms (HSNO) Act 1996 by the Environmental Risk Management Authority.

The CCA formulations currently present in New Zealand were registered under the Pesticides Act 1979 and are in the process of being considered for transfer from the transitional parts of the HSNO Act to the HSNO regulatory framework (ERMA New Zealand, 2002). However since CCA-treated wood is a product and not a hazardous substance its direct regulation falls outside of the HSNO Act. In other words the HSNO controls able to be set for CCA cannot be directly use-related.

Other regulatory mechanisms that relate to the use of CCA and/or CCA-treated wood include the Resource Management Act 1991, Health and Safety in Employment Act 1992, Building Act 1991 and Building Regulations 1992, and local authority bylaws.

11.2 International

11.2.1 European Union

The EC published a Marketing and Use Directive on 6 January 2003 (Commission of the European Communities, 2003) stating labelling requirements for CCA-treated wood, and banning the sale of CCA-treated wood unless structural integrity of the wood is needed for human or livestock safety and skin contact by the public is unlikely. The directive is to take effect by 30 June 2004 and applies only to CCA Type C preservatives. Situations in which CCA preservatives may not be used include residential or domestic constructions, where there is a risk of repeated skin contact, and where the wood may come into contact with intermediate or finished products intended for human consumption. The directive does not apply to CCA-treated wood already in use.

This decision originated from a health and environmental risk assessment that considered each stage of the life cycle of arsenic and analysis of further restrictions on use of arsenic in wood preservatives (WS Atkins International, 1998). The main risks identified included those to human health from the disposal of CCA-treated wood, particularly household burning, and risks to children's health from the use of CCA-treated wood in playground equipment. The health component of the risk assessment concluded that in a worst case scenario children's ingestion of dislodgeable residues on playground equipment can result in arsenic intake that exceeds the TDI. The authors stated that the risk to children should be kept in perspective by comparison with the higher arsenic intake from playing in soil with normal background levels (WS Atkins International, 1998). This view was based on background soil arsenic exposure estimates derived from a study carried out by the California Department of Health Services in 1984. However it does not appear that the authors independently evaluated this study but rather cited it from information from the Western European Institute for Wood Preservation (WEIWP).

The CSTEE reviewed the assessment (CSTEE, 1998) and concluded that the methodological approach and conclusions were valid, although discussion of arsenic toxicology was limited and cancer by ingestion was not included as an endpoint.

Initially the review by the CSTEE led to discussions between the Commission and CCA manufacturers on a voluntary agreement to address the concerns raised. Key issues in the agreement included recommendations for handling and disposal of treated wood to be given to users of CCA and CCA-treated wood (HSE, 2001).

Subsequently the CSTEE considered human health issues in relation to arsenic in ambient air. They concluded that it may be appropriate to assume that no threshold, below which adverse effects do not occur exists (CSTEE, 2001).

The initial draft directive proposed restricting CCA to three end uses - sleepers, poles and cooling tower fill. Consultation resulted in 156 submissions, 86% from industry, and suggested broadening the scope of permitted uses based on low human exposure and professional control consistent with the risks considered unacceptable. Significant usage of CCA-treated wood in areas not identified in the risk assessment such as outdoor furniture and decking was also identified (European Commission, 2002).

The Wood Preservation Task Force, comprising three manufacturers of CCA wood preservatives, and the WEIWP responded to the CSTEE report and the initial proposed EC restrictions stating that a risk assessment using current principles of risk assessment, more recently available data, and data not included in the Atkins report would conclude that there is no scientific justification for restricting use. They concluded that currently available evidence can be interpreted to demonstrate that the use of CCA-treated wood is a tolerable risk to gain the economic and environmental benefits of using preserved wood (Baines, 2002). In respect of health risks the main additional information to which they refer is the risk assessment carried out by Gradient Corporation (2001) for two major CCA manufacturers (See Risk Assessments of Children's Exposure to Arsenic from CCA-treated Wood Structures section). They concluded it is likely that the Atkins report overestimates arsenic release from CCA-treated wood to soil and arsenic exposure from ingestion of dislodgeable residues. These views were based on a review of five United States studies published from 1997 in which the mean arsenic concentration in soil below CCA-treated decks ranged from 3.8 to 76 mg/kg, below the range in the Atkins report, and the use of wipe sampling to determine the amount of arsenic from dermal contact with CCA-treated wood in the study cited in the Atkins report rather than hand loading methodology (Baines, 2002).

Further drafts were discussed with member states and stakeholders and a compromise position was approved by member states in November 2002 (Western European Institute for Wood Preservation, 2002; Arch Timber Protection, 2002). Debarked round conifer livestock fence posts were added to the list of permitted end uses to allow this use which occurs in lowly populated hilly regions of Ireland. Statements excluding CCA-treated wood already in place and prohibiting placement of CCA-treated wood on the market before completion of preservative fixation were added.

The CSTEE emphasised that a major source of concern relates to high uncertainty regarding the behaviour of CCA-treated wood in landfills[19] and therefore advised limiting use to situations where it is absolutely necessary. The Wood Preservation Task Force also dispute this referring to information that has become available since the Atkins report or was available but not included in the report. It is unclear as to what extent this uncertainty concerning disposal influenced the Commission's decision-making. The Commission decided based on the risk assessment and taking the precautionary principle into account (Commission of the European Communities, 2003). As part of this review it has not been possible to ascertain whether health and environmental concerns were of equivalent weight or not in the decision-making.

The Commission is also evaluating wood preservatives as a priority in a review of biocidal products and information for evaluation of arsenic as an existing substance has to be presented by 28 March 2004. The measures in the directive are interim until this full evaluation of CCA under the Biocidal Products Directive occurs. Only the uses permitted in the Marketing and Use Directive can be considered under the Biocidal Products Directive so if any further changes are made they will be more restrictive.

Restrictions on use of CCA already exist in a number of member states. Germany, Sweden, Austria, Finland, the Netherlands and Denmark had already initiated voluntary agreements or regulations restricting the use and marketing of CCA and CCA-treated wood primarily in

[19] Disposal of large quantities of CCA-treated wood will become an issue for New Zealand, peaking round 2030 (based on peak use in the 1980s and a service life of 50 years). This is not discussed further here as environmental effects are outside the scope of this review.

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

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

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

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

11.2.2 United States

1. US Environmental Protection Agency

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

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

In August 2002 the FIFRA SAP met to discuss a probabilistic method of assessing children's exposure to CCA-treated wood play equipment and residential decks. The model simulates the 365-day longitudinal activity patterns of children aged 1 - 6 years to predict the variability of absorbed doses of arsenic from CCA-treated wood. The FIFRA SAP identified the need to aggregate exposures and made research recommendations to develop data to reduce uncertainty associated with some of the models' inputs such as frequency and duration of child contact with wood and contaminated soil, skin transfer rate, and rate of hand-to-mouth activity (FIFRA SAP, 2002). Dr Maas made a public comment that included his research findings to the August SAP meeting on behalf of the EWG (Maas et al. 2002).[20]

Studies of soil and surface residue bioavailability are in progress with results expected in March and April 2003 respectively. A hand wipe study is also underway with results expected in May 2003. The data from these three studies will contribute to the children's risk assessment which will be available for public consultation and review by the SAP in December 2003.

In the United States debate about CCA and possible health and environmental risks is highly charged. Litigation against CCA manufacturers and building supply stores alleging consumers were inadequately informed of the risks, increasing public awareness about the toxicity of arsenic with the debate about the lowering of the drinking water standard, and environmental group lobbying has contributed to public opposition to CCA-treated wood.

Since the mid-1990s playgrounds have been closed in areas of Florida due to high soil arsenic levels and several states and municipalities have introduced legislation ranging from a ban on CCA-treated wood to restricted use in specific settings (Fields, 2001). New York State passed legislation in 2002 prohibiting use of CCA-treated wood for any new public or school playground and requiring public education that existing structures need to be maintained to minimise leaching.

An EPA-approved consumer awareness programme in the form of consumer information sheets was implemented by industry in 1985 following an EPA review of CCA, and enhanced in 2001 to also include safe handling end-tag labels and retail signs (Shields, 2002). The labels and signs advised caution as arsenic is in the pesticide applied to the wood, never to burn treated wood, to wear a dust mask and goggles when cutting or sanding wood, to wear gloves when working with wood, and to ask for the consumer safety information sheet, look at the web site, or call the toll-free number (US EPA, 2001a). The original awareness programme has been criticised for rarely reaching consumers (Sharp and Walker, 2001).

In 2001 the Florida Department of Health appointed a panel of physicians (Florida Physicians Arsenic Work Group) to evaluate the health risk from arsenic associated with the use of CCA-treated wood for playground equipment and recreational facilities. The brief report that was released in 2002 (Bidot et al. 2002) states that available data do not show any disease associated with arsenic exposure from this use of CCA-treated wood or from surrounding soil. They did not recommend sampling of playgrounds due to the low bioavailability of arsenic from wood and soil and supported the EPA's recommendation not to replace or remove existing structures made with CCA-treated wood or the soil around those structures. The

[20] This is contradictory to a report in *The Dominion Post* 25 November 2002 and a Radio New Zealand interview with Dr Maas on 27 November 2002 which claimed that this research was influential in the decision to phase out residential use of CCA-treated wood.

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

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

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

Environmental groups such as Beyond Pesticides have called for an immediate ban that covers all uses (Feldman, 2002).

In addition to the risk assessment considering children's exposure to CCA-treated wood structures the EPA is carrying out another risk assessment that focuses on the uses of CCA that are not being phased out. This risk assessment is expected to be available for public consultation in the American spring 2003 (personal communication, US EPA, 23 January 2003).

2. US Consumer Product Safety Commission

Playground equipment made with CCA-treated wood is the jurisdictional responsibility of the CPSC. The Federal Hazardous Substances Act requires that consumer products that meet the statutory definition of hazardous substance are labelled with advice about safe use and handling. Children's products that are hazardous substances are automatically banned.

The CPSC carried out a study in 1990 to estimate skin cancer risk from dislodgeable arsenic on CCA-treated wood playground equipment since the EPA and California Department of Health Services had indicated inadequate data and assessment procedures existed for estimating cancer risk to children from playing on CCA-treated wood playground equipment

[21] Arch Wood Protection, Chemical Specialties, Phibro-Tech, and Osmose requested to cancel two CCA products and end certain uses of other CCA products (US EPA, 2002b).

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

In May 2001 the EWG and the HBN petitioned the CPSC to ban the use of CCA-treated wood for playground equipment and review the safety of CCA-treated wood for general use. The reasons given for a ban were research showing arsenic to be more carcinogenic than previously recognised, past risk assessments were incomplete, health risks are greater than previously recognised, and arsenic is present in significant concentrations on CCA-treated wood and in underlying soil.

In response the CPSC has reassessed the possible health risk to children associated with CCA-treated wood use in playground equipment (US CPSC, 2003).

Data limitations led the CPSC to undertake a study to determine levels of dislodgeable arsenic that occur on CCA-treated wood surfaces in existing and new playground equipment to provide data for a risk assessment. Sampling techniques were similar to those used in the CPSC (1990) study.

Sampling protocols for determining dislodgeable CCA residues (US CPSC, 2001) and arsenic, copper and chromium in soil and barrier materials eg wood chips adjacent to playground equipment to prevent injuries from falls (EPA, 2001b) were developed and consulted on.

Although the study was small, involving only 20 CCA-treated wood structures, and limited to one metropolitan area it is the most comprehensive study carried out to date, particularly in terms of the experiments that were done to develop and validate the study protocol (See Risk Assessments of Children's Exposure to Arsenic from CCA-treated Wood Structures section).

The CPSC held a public briefing to consider the petition to ban the use of CCA-treated wood in playground equipment on 17-18 March 2003. The Commissioners have yet to vote on the petition. The CPSC recommended action on the petition be deferred pending finalisation of the agreement between the CCA industry and the EPA to phase out CCA-treated wood in most residential settings by the end of 2003, and an assessment of its impact.

In conjunction with the EPA, the CPSC are also investigating the effectiveness of coatings in reducing exposure from CCA-treated wood (US CPSC, 2002).

11.2.3 Canada

In Canada four federal government departments, Health Canada, Environment Canada, Agriculture Canada and Natural Resources Canada, are involved in making regulatory decisions on wood preservatives.

Health Canada reviewed and agreed with the earlier assessments of CCA undertaken by the US EPA in the 1980s and US CPSC in 1990.

There is a co-operative approach between the PMRA of Health Canada and the EPA to the re-evaluation/re-registration of heavy duty wood preservatives. The EPA is leading the co-

ordination of work sharing and harmonisation issues for the health risk assessment whereas PMRA is leading co-ordination of the environmental risk assessment (PMRA, 2002c).

Following the US EPA's announcement of the CCA industry's voluntary decision to cancel residential use of CCA-treated wood the PMRA facilitated a similar voluntary decision with industry in Canada (PMRA, 2002a and 2002b). Whether the basis for this decision by the PMRA was scientific, political, or market-driven was not determined as part of this review.

Retail outlets have been providing information on proper use and handling of CCA-treated wood voluntarily. An enhanced consumer awareness programme eg end-tag labelling, similar to that in the United States, was implemented in 2002.

11.2.4 Australia

In Australia the APVMA[22] is responsible for the regulation of pesticides. In 1995 a scoping exercise to determine whether the use and registration of CCA should be reviewed resulted in a decision not to proceed. This decision was confirmed again in 2002.

Public concern about human exposure to CCA-treated wood has been expressed, particularly in Victoria, following the United States and Canadian decisions to phase out most residential uses of CCA-treated wood. State and Commonwealth health officials discussed the issue nationally at EnHealth Council, the premier advisory body on environmental health in Australia, in late 2002.

Following the release of the CPSC findings (2003) the APVMA announced on 13 March 2003 that it was undertaking a review of the health and environmental effects and labelling of products containing arsenic, including CCA. The Therapeutic Goods Administration and Environment Australia will carry out the technical assessment and advise the APVMA. Submissions close on 31 May 2003 and it is anticipated that a draft report will be available for public comment in mid-2004.

11.2.5 Other

There are restrictions on use of CCA in other countries including Japan and Indonesia. Details of the decision-making behind these decisions have not been obtained as part of this review.

12 Health Risk Assessment

Health risk assessment is the process by which toxicological and epidemiological data are evaluated, the dose-response relationship is determined and a model is applied to predict response at low doses, and information about exposure is used to estimate the probability an adverse effect will occur in a population (Paustenbach, 2000). Regulatory policy regarding chemical carcinogens generally gives more weight to epidemiological data, if available and of adequate quality (Goldman, 2001; Rodricks et al. 2001).

[22] The APVMA is also known as the National Registration Authority for Agricultural and Veterinary Chemicals until legislative amendment to reflect the change of name occurs later in 2003.

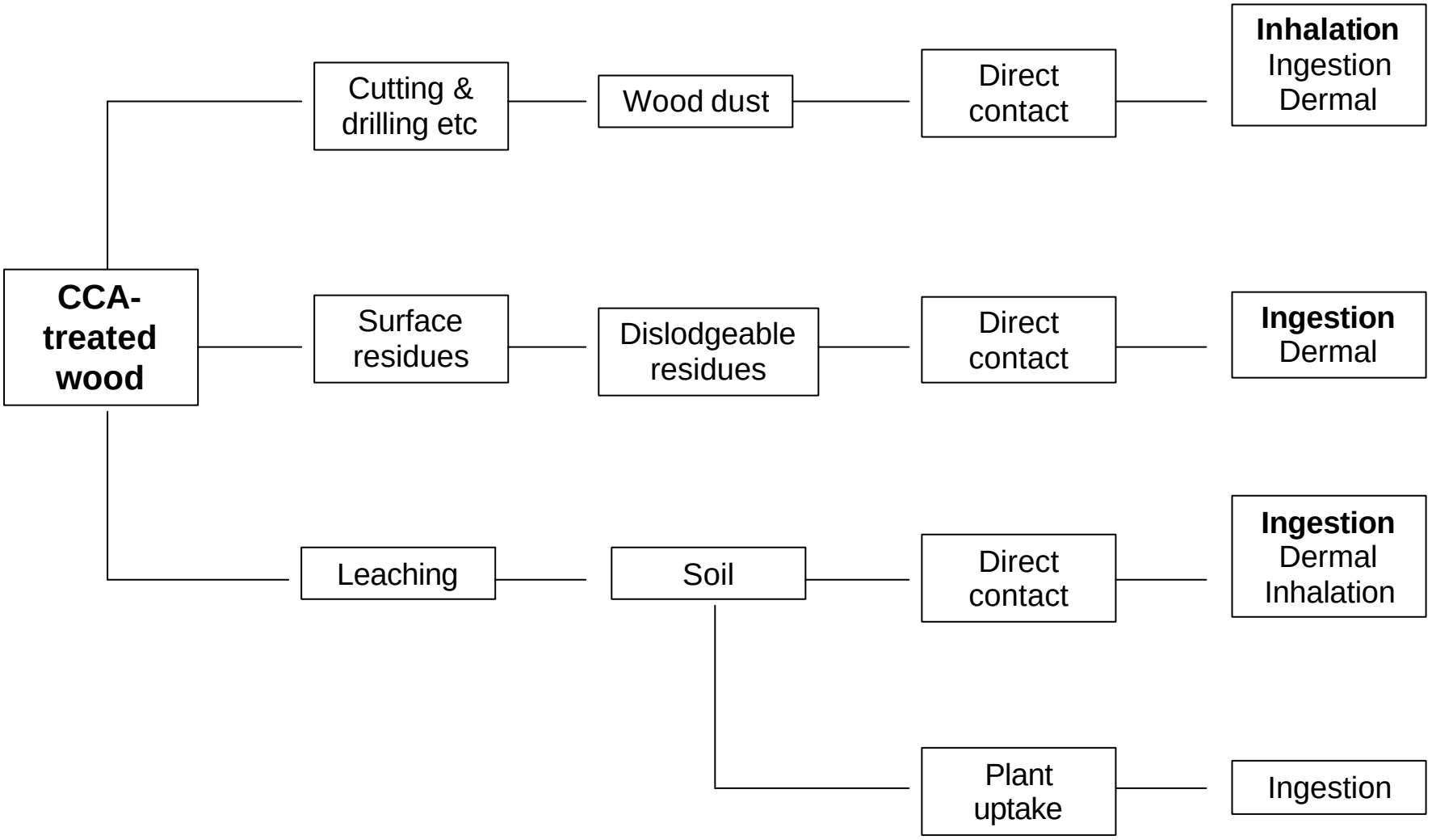
The potential health risks of CCA-treated wood depend on the toxicity including susceptibility (See Arsenic, Chromium and Copper sections) and exposure.

Exposure assessment estimates the magnitude of the absorbed dose that occurs from contact with environmental media. This depends on exposure duration, exposure route, concentration in and bioavailability from the medium, and population characteristics (Paustenbach, 2000). Potential human exposure pathways for CCA-treated wood are illustrated in Figure 1.

13 Exposure Assessment of Children

Children are typically assumed to be at greater risk for adverse health effects than adults because of greater exposures resulting from physiological factors and their behaviour. There are few empirical data to support the claim of greater exposure. In general, exposure assessments are based on activity pattern data, algorithms and default assumptions (Juberg, 2003).

Figure 1: Exposure pathways



Bold: principal exposure routes

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

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

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

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

Table 4: Potential exposure scenarios for children

Exposure route-medium	
oral/wood	dermal/wood
oral/soil	dermal/soil
inhalation/wood	inhalation/soil
oral/barrier material	dermal/barrier material

Other exposure scenarios are indirect hand-to-mouth transfer of residues on clothing, toys and food; direct mouthing of toys and CCA-treated wood structures; and splinters. Behaviours such as direct mouthing of wood and contaminated toys which are usually confined to very young children are typically omitted from risk assessments of CCA-treated wood due to lack of data.

Inhalation from contact with CCA-treated wood or CCA-contaminated soil is assumed to be negligible for child playground exposure assessment (FIFRA SAP, 2001; Exponent 2002b). Gradient Corporation (2001) and Hazardous Substance and Waste Management Research (HSWMR (2001) have carried out the only health risk assessments including exposure through inhalation of arsenic-contaminated dust from CCA-treated wood structures.

Playgrounds frequently have protective ground cover against injuries from falls from the equipment potentially reducing soil exposure. Currently available exposure assessment methods are not directly applicable to assessing exposure from non-soil media such as wood chips (Exponent, 2002b). To date these media have not been included in risk assessments.

Arsenic exposure from burning of CCA-treated wood is not included as this is a less likely exposure scenario. If it does occur exposure is likely to be short-term and unless the wood is burned in a barbecue it will usually not present a significant health risk.

Although most of the risk assessments that have been carried out focus on children's exposure to playground equipment in settings such as schools and parks this exposure may also occur at home and in early childhood centres. These locations may also have sand-pits made from CCA-treated wood. Since arsenic does not bind as well to sand as it does to soil it disperses through the sand raising the possibility of ingestion or inhalation of sand particles containing high arsenic concentrations.

Data are required on exposure factors including contaminant concentrations in the wood and soil where the child spends time, contact rates of the child with the wood and soil, contaminant transfer efficiency from the wood to the skin and mouth, contaminant uptake rates and children's activity patterns (Table 5).

Table 5: Input exposure parameters for each playground scenario

Playground soils	Playground equipment wood surfaces
soil arsenic concentration	arsenic concentration on wood
play area ground covering	transfer from wood to hands
soil ingestion rates	hand skin surface area
outside activity time[23]	hand surface area for dermal absorption
soil arsenic bioavailability	outside activity time
exposure frequency	bioavailability of dislodgeable residues
exposure duration	dermal arsenic absorption
skin surface area for soil contact	
soil-to-skin adherence factor	
dermal absorption from soil	

(Exponent, 2002b)

Estimates of the amount of time a child spends in the presence of the contaminants are needed for exposure assessment. To achieve any precision account must be taken of children's activity patterns. There are no comprehensive data on New Zealand children's activity patterns. Most of the available information relates to American children and focuses on macro-activity.

Silvers et al. (1994) carried out a six state survey of children aged 5-12 years in the United States. The state with the most similar climate to New Zealand was California. The average time Californian children spent outdoors during a year was 2.03 hours/day compared to 2.65 hours/day for the other five states. Results were similar to those obtained in a large study in California of children aged 0-11 years. However they spent about the same amount of time

[23] The proportion of soil ingested contributed from playgrounds is assumed to be proportional to activity time.

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

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

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

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

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

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

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

However there is no standardised validated method of determining dislodgeable arsenic on hands. The best available is that recently developed by CPSC (2003). In this study deck boards from eight decks up to 18 years old were rubbed with adults' hands and dry polyester wipes to establish a correlation between the results of the two methods (and therefore a conversion factor[25]). The maximum amount of arsenic that can be loaded onto a hand was reached after rubbing hands just a few times over wood. This finding suggests that the amount of arsenic picked up by the hand should be reported as total arsenic on hands rather than standardised to an area, typically 100 cm^2. Significant deck variation in hand levels was found

[24] The hand load value represents the equivalent hand-to-mouth activity that results in the estimated amount of soil ingested per day.
[25] Conversion factors for converting measurements to hands are 0.20 for dry polyester and 0.08 for wet polyester.

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

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

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

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

14 Epidemiological Studies

14.1 At risk population groups

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

1) Workers in timber treatment plants

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

[26] Soil pica is the eating of soil.

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

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

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

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

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

14.2 Occupational studies

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

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

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

Industry frequently cites the results from a case control study of timber treatment workers in Hawaii who had been chronically exposed for at least three consecutive months from 1960 to 1981 to up to three types of wood preservatives. Median exposure was 6½ years. Medical histories, physical examinations, physiological and laboratory tests including urinary arsenic, copper and chromium showed no significant differences between those exposed to wood preservatives and those not exposed (Gilbert et al. 1990). However the study had a number of significant limitations such as a 48% response rate, 25% of participants were no longer exposed, exposure was poorly characterised, and there was a low number of controls. In addition urine concentrations reflect recent exposure and are of little significance when taken weeks to months after exposure to CCA has ceased, the urinalysis appears to have been for total arsenic, it is unclear to what extent dietary differences particularly seafood intake between the groups were examined, and results were pooled and included some workers who were not exposed to CCA. Selection of the control group (n = 58) also included 14 carpenters (who may or may not have been exposed to CCA-treated wood), although their urinary arsenic levels did not differ significantly from those of the other controls.

A study of cancer incidence among 1,042 CCA-exposed timber treatment workers in Sweden and Norway in 1990-1991 suggests there is no increased risk overall or for any cancer site. Cancer incidence was lower than expected compared to the national rates even when allowing

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

A small study in Australia showed statistically significant increases in urinary chromium and arsenic levels between timber treatment workers exposed to CCA and non-exposed workers, although the difference for arsenic appeared to be due to diet. Limited medical assessment of exposed workers indicated no acute or chronic health effects. The author concluded that both urinary chromium and arsenic would constitute the best monitoring programme for CCA exposure and also recommended research to determine the extent of exposure among workers processing CCA-treated wood (Szafraniec, 1991).

A review of industry biomonitoring and HSE data found urinary arsenic and chromium levels in timber treatment workers in the United Kingdom were generally within the normal range (HSE, 2001). An exposure survey found contamination of work clothing, including inside impermeable gloves, was common with about 10% penetration of CCA as estimated by a sampling patch inside the worker's overalls. Periodic removal of gloves and replacement on potentially contaminated hands was thought to be the mechanism of entry into gloves. However urinary arsenic and chromium results from 10 workers suggest dermal uptake is low (Garrod et al. 1999).

Mean urinary arsenic level (222 µg/L) of 30 New Zealand timber treatment workers was an order of magnitude higher than that for controls (Gollop and Glass, 1979). However the study was carried out more than 20 years ago and there have been significant improvements in occupational health and safety since this time. Of note is the authors' comment that there was a need to determine whether the main route of exposure was inhalation or oral and dermal contact from wet preserved wood. No other published studies of urinary arsenic in New Zealand timber treatment workers have been found. Jensen et al. (1991) found no difference in urinary arsenic concentrations between Danish timber treatment workers and controls though the number of workers was small (n = 3). The mean urinary arsenic concentration of a small group (n = 5) of timber treatment workers was elevated compared to that for the general adult population. For two of these workers the concentration was above the level considered to give rise to presumptive toxicity but well below levels associated with inorganic arsenic-related diseases (Farmer and Johnson, 1990). In contrast, Nygren et al. (1992) found no increase in urinary arsenic concentrations among five joinery workers using sawing, sanding or cutting machines. In the wood dust arsenic was present only in its pentavalent form and less than 0.1% of chromium was hexavalent.

A statistically significant correlation was found between air and urinary arsenic concentrations among people working indoors with CCA-treated wood. For outdoor workers, air concentrations were undetectable (limit of detection was 0.2 µg/m^3) for six workers building new houses with periodic daily contact with CCA-treated wood and 2.8 µg/m^3 for one worker sawing CCA-treated wood all day. The median urinary arsenic level for these workers was slightly increased but not significantly different from the control group. In eight wood workers producing garden fences indoors the median urinary arsenic level was 2.9 times higher than that of the control group (Jensen and Olsen, 1995).

[27] Latency is the time from initiation of a cancer cell to disease manifestation.

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

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

Mean total air arsenic, but not chromium or copper, concentration exceeded current recommended occupational exposure limits at an indoor site making wood components for playground equipment, and occasionally at three outdoor residential deck sites. Dust concentration for deck builders was low (mean 0.57 mg/m^3).

Indoor dust concentrations were significantly higher than those measured outdoors despite local exhaust systems, and often exceeded the exposure limit. Concentrations were highest for workers undertaking sanding. Although the respirable fraction[28] of the dust was considerably less than the inhalable fraction, the health significance of the respirable fraction is unknown due to lack of research (Decker at al, 2002). The small sample size limits the conclusions that can be drawn from this study but the findings suggest significant occupational exposure to airborne metals can occur in indoor and outdoor settings involving CCA-treated wood. The potential effects of combined inhalable wood dust, chromium and arsenic are unknown.

Penetration of CCA and surface concentration is higher in sapwood than heartwood resulting in variability of the CCA components in dust depending on the type of timber used (Nygren et al. 1992).

The group with the highest exposure to CCA-treated wood dust is likely to be builders. This wood is more likely to be cut or drilled and a builder is less likely to use protective equipment and methods to reduce exposure to dust.

The risk assessment carried out for the EC estimated the lung cancer risk for such workers from inhalation. Using a value calculated by the WEIWP of 2.7 µg for the arsenic concentration in 2 mg CCA-treated wood dust (with a CCA retention of 6.1 kg/m^3), exposure to the maximum permitted occupational concentration of 2 mg/m^3 for wood dust in Denmark would result in exposure to 2.7 $\mu g/m^3$ arsenic. This gave a lifetime occupational exposure of 0.37 $\mu g/m^3$, and an additional lung cancer risk of 4×10^{-4} ($1.5 \times 10^{-3} \times 0.37$)[29] (WS Atkins, 1998).

There have been several New Zealand studies that have looked at occupational cancer incidence and mortality.

Firth et al. (1996) examined cancer incidence (excluding non-melanoma skin cancer) for males aged 15-64 years in full or part-time employment in New Zealand from 1972-1984.

[28] The respirable fraction comprises small particles which reach the gas exchange area of the lung.
[29] The WHO's estimate of the lung cancer risk associated with arsenic inhalation is 1.5×10^{-3} deaths per $\mu g/m^3$ over a lifetime exposure.

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

Case-control studies based on the New Zealand cancer registry from 1980 to 1984 found increased risks for lung cancer (OR = 1.27; 95% CI: 1.05 - 1.54) and the buccal cavity specifically lip cancer (OR = 2.28; 95% CI: 1.23 - 4.14) among carpenters (Kawachi et al. 1989).

In other New Zealand studies occupations were not examined at the unit group level where occupational description is more specific but at the minor group level of bricklayers and carpenters. For bricklayers and carpenters, observed mortality was less than that expected for employed males (RR[31] = 0.98; 95% CI: 0.93 - 1.04) and males in the same social class (RR = 0.91; 95% CI: 0.86 - 0.96), and no significantly elevated relative risks (i.e. RR > 1.5)[32] were found for any major disease grouping (Pearce and Howard, 1985). No significantly elevated relative risks for cancer mortality for specific cancer sites were also found for bricklayers and carpenters (Pearce and Howard, 1986).

Using data from a longer time period that included those analysed by Pearce and Howard (1986), Firth et al. (1993) reported that bricklayers and carpenters had increased cancer mortality for lung cancer (SMR[33] = 125; 95% CI: 110 - 141) after standardising for age and socio-economic level.

Studies such as these have methodological limitations such as misclassification bias between numerator and denominator, occupation at time of registration or death may not be the occupation where exposure occurred because of the long latency period for cancer, and confounding from factors such as diet, smoking and alcohol consumption. Adjusting for socio-economic level attempts to adjust for lifestyle factors. Occupational data for those aged 65 or more years are excluded from these type of studies as it tends to include a relatively high proportion of people recorded as retired who cannot be assigned to an occupation.

There has been no recent analysis of New Zealand occupational data and cancer incidence or mortality. If use of CCA-treated wood peaked in the 1980s data would only now be coming available that take account of that increased exposure and give a sufficient latency period.

[30] A standardised incidence ratio (SIR) is 100 times the ratio of the number of registrations observed in the occupational group to the total number of expected registrations.
[31] The relative risk (RR) is the ratio of the observed and expected numbers of deaths.
[32] Relative risks less than 1.5 are likely to be due to bias or confounding.
[33] A standardised mortality ratio (SMR) is 100 times the ratio of the number of deaths observed in the occupational group to the number that would be expected if the group under study had the same rates as the comparison population.

14.3 Non-occupational studies

Among the non-occupationally exposed general population it is likely that children using CCA-treated wood structures will have the highest exposure because the main route of exposure is transfer of residues from hands to the mouth. Children also have a lower PTWI than adults. Assuming the amount of transfer from wood to skin decreases in the same way that the leaching rate decreases after initial exposure to water the amount of exposure will decline with time.

No studies of children exposed to CCA-treated wood structures were identified.

Jensen et al. (1991) found no significant difference between urinary inorganic arsenic (and its metabolites) concentrations among a small group of children aged 3 - 11 years living in an area with soil arsenic levels above 20 mg/kg and children living in an uncontaminated environment. Results for adults were similar to those of children in the two environments. The study is limited by its small sample size but suggests that contact with soil contaminated with arsenic, and the increased contact children have with soil compared to adults, do not result in a significantly increased absorbed dose of arsenic.

15 Risk Assessments of Children's Exposure to Arsenic from CCA-Treated Wood Structures

A number of risk assessments were identified as having been carried out in the last 15 years concerning children's exposure to arsenic from CCA-treated wood structures.

One of these, carried out by the Maine Bureau of Health in 1998, was unable to be obtained for this review but was only found cited in bibliographies as a draft document.

There are differences among the assessments with respect to parameters such as years of exposure, number of years defined as a lifetime, bioavailability, dislodgeable arsenic residue concentrations and body weight. Although there is high uncertainty for some parameters in assessing exposure of children to CCA-treated wood the final outcome on the risk estimates of some of these may not be significant. For example, dermal absorption of soil arsenic is likely to contribute only a small proportion of the total amount of arsenic compared to ingestion. The most significant differences in terms of impact on the risk estimates are probably the estimates for the amount of arsenic that is transferred to the hands, bioavailability, and the choice of unit risk used to relate the exposure to cancer risk. The range of cancer risk estimates that have been derived for children exposed to dislodgeable arsenic residues is summarised in Table 10.

A summary of the exposure routes that were evaluated for children's exposure to CCA-treated wood structures in each risk assessment is given in Table 6.

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

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

15.1 California Department of Health Services (1987)

The California Department of Health Services (CDHS) investigated the hazard posed to children from ingestion of dislodgeable residues from playground equipment and estimated that a child could absorb 24 - 630 µg arsenic per visit (CDHS, 1987). From these data they estimated an additional lifetime cancer risk between 1×10^{-4} and 6×10^{-3}. This analysis compared estimated doses with arsenic intake associated with the drinking water standard of 50 µg/L which was not a risk based number.

It was estimated from survey data from a random sample of parks and schools that 20% of all park and school playground structures were wood and 20% of these were CCA-treated wood though in most cases the respondents did not know or did not indicate whether the wood had been treated. The report also notes that an estimated 1.4% of injuries on school playground equipment relate to structural failure of wooden components and half of these failures result in injury. In contrast there was only one report of illness related to chemical exposure from playground equipment. The proportion of failures involving untreated wood was not known. The report also states that studies undertaken by CDHS show residue levels decrease after application of sealants but no details are given (CDHS, 1987). Subsequently publicly funded playground equipment made from CCA-treated wood was required to meet AWPA's treatment standard and to be sealed every two years to minimise surface residue availability.

15.2 US Consumer Product Safety Commission (1990)

Seven playground equipment wood samples were collected from manufacturers and one comparison sample of new CCA-treated wood not specifically finished and sold for playground use was bought from a retail store. Two samples had average dislodgeable arsenic levels in the range of 21.9 - 32.1 µg/100 cm^2 compared to 68.9 µg/100 cm^2 for the unfinished wood. The estimated cancer risk for five of seven samples from manufacturers that were below the detection limit for arsenic was less than one in a million. For two samples with detectable levels the estimated risk was 3-4 $\times 10^{-6}$ and for unfinished wood 8 - 9 $\times 10^{-6}$. The latter result suggested a possible hazard if playground equipment is made from CCA-treated wood that was not specifically processed (and therefore does not meet the standard) for playground equipment (US CPSC, 1990).

This study evaluated skin cancer risk only. CPSC developed a unit cancer risk for this risk assessment that was one-third that developed by the EPA using the same epidemiological data but a different methodology. This is not known to have been peer reviewed or used elsewhere. In addition they used the daily hand loading rate of boys rather than the more conservative approach of using the higher rate of girls or an average for both sexes (Roberts and Ochoa, 2001; Sharp and Walker, 2001). The study has also been criticised for not taking into account routes of exposure other than ingestion and exposure from CCA-treated wood structures other than playground equipment (Sharp and Walker, 2001). The CPSC have subsequently updated this risk assessment using the recently derived unit cancer risks of the EPA and the NRC and report a range from 2×10^{-6} to 1×10^{-4} for lung or bladder cancer which is similar to their recent risk assessment (US CPSC, 2003).

15.3 WS Atkins International (1998)

The risk assessment carried out for the EC considered only children's dermal exposure to CCA-treated wood and concluded that in a worst case scenario children's ingestion of dislodgeable residues from playground equipment can result in arsenic intake that exceeds the tolerable daily intake for children.

Other human health risks included in this assessment were the lung cancer risk from occupational arsenic exposure including CCA-treated wood dust which was estimated to range from 4 to 6×10^{-4}, and lung cancer risk from domestic exposure as a result of burning CCA-treated wood which was estimated to be 4.5×10^{-4}.

15.4 Hazardous Substance and Waste Management Research (2000a, 2000b, 2001)

HSWMR carried out health risk assessments on behalf of the CCA industry for dislodgeable residues on and soil under decks and playground equipment. They addressed both cancer and non-cancer (eg hyperpigmentation, hyperkeratosis) effects and assumed oral, dermal and inhalation (for soil) exposure routes, 25% bioavailability of arsenic from soil and 5% transfer efficiency of residues from wood to hands. They calculated a soil arsenic level under playground equipment protective of non-cancer effects[34] of 260 mg/kg and of cancer of 90 mg/kg (HSWMR, 2000a), a soil arsenic level under decks protective of non-cancer effects of 390 mg/kg and of cancer of 170 mg/kg (HSWMR, 2000b), and a wood surface level of 40 µg/100 cm^2 protective of cancer and 420 µg/100 cm^2 protective of non-cancer effects (HSWMR, 2001). The 'acceptable' concentrations are based upon an underlying assumption of a one in a million risk.

Criticisms include the dermal absorption rate of 0.1% was too low (Roberts and Ochoa, 2001; Exponent, 2002a), 150 days/year exposure was too low (Roberts and Ochoa (2001) and some assumptions were very conservative eg 200 mg/day soil ingestion rate (Exponent, 2002a).

The assessment has also been criticised for using incorrect methodology in calculating carcinogenic risk. HSWMR calculated a time-weighted average for each exposure parameter that differed between children and adults rather than calculating the risks for children and adults separately and summing them as recommended by the EPA. This may have

[34] In other words the accepted hazard quotient of 1 for non-carcinogenic effects is not exceeded at a soil arsenic concentration of 260 mg/kg.

overestimated soil and wood surface arsenic levels considered unlikely to present an unacceptable cancer risk (Exponent, 2002a).

15.5 Roberts and Ochoa (2001)

Roberts and Ochoa (2001) calculated arsenic doses (and risk) that could potentially result from five years of daily exposure for children aged 2 - 6 years to CCA-treated wood for a range of hypothetical residue concentrations (Table 7). They concluded that potential arsenic exposure was too high in terms of excess cancer risk to protect health.

Table 7: Cancer risk estimates for various dislodgeable arsenic concentrations

Arsenic concentration	Cancer risk estimate
1 µg/100 cm^2	4.22×10^{-6}
10 µg/100 cm^2	4.22×10^{-5}
100 µg/100 cm^2	4.22×10^{-4}
250 µg/100 cm^2	1.06×10^{-3}

Hazard quotients[35] at the same concentrations were 0.14, 1.42, 14.2 and 35.4. As the authors note the risk estimates are very conservative as exposure is unlikely to be daily. On the other hand exposure may occur for longer than five years.

Exposure frequency would need to be no more than 85 days per year to reduce the risk to one in a million or less at the lowest concentration considered. These are similar risk estimates to those estimated by CDHS (1987).

In a report to the American Chemistry Council, Exponent illustrates the impact of varying parameters on the risk estimate calculated by Roberts and Ochoa (2001). If the exposure frequency is changed from 365 days to 150 days, transfer of arsenic from wood to hands is assumed to be 38% rather than 100%, and surface area is one-third of the total surface area as recommended by the EPA rather than the one-half approach used, an arsenic residue concentration of 10 µg/100 cm^2 represents a cancer risk of 3.6×10^{-6} rather than 4×10^{-5} (Exponent, 2002a).

15.6 Gradient Corporation (2001)

Exposure scenarios for a risk assessment carried out for two CCA manufacturers included ingestion and dermal contact with dislodgeable residues and ingestion, dermal contact and inhalation to CCA-contaminated soil. The assessment followed EPA risk assessment guidelines.

Estimated cancer risks for children aged 2-6 years exposed to CCA-treated wood structures[36] in residential or playground settings ranged from 10^{-6} to 10^{-8} and non-cancer risk estimates were less than the commonly accepted non-cancer risk (hazard quotient) limit of 1.

[35] The hazard quotient is the ratio of exposure to recommended reference exposure levels (eg the tolerable daily intake, EPA oral reference dose) for any given chemical. It indicates whether an estimated exposure is considered to be without significant non-cancer health risk. An acceptable risk is defined as a hazard quotient of 1 or less.

[36] Exposure scenarios included average and reasonable maximum exposures.

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

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

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

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

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

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

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

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

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

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

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

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

15.7 Environmental Working Group (2001)

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

described and there is no discussion in the report about hand-to-mouth transfer efficiency and relative bioavailability.

15.8 Maas et al. (2002)

Maas et al. (2002) tested CCA-treated wood for dislodgeable arsenic using wipe sampling based on the method developed by the CPSC.

The authors estimated 8.5 x 10^5 µg/year arsenic ingestion for a baby crawling on a deck (assuming 200 hand touches/week, hands are 40 cm^2, and 50% hand-to-mouth transfer efficiency). They then calculated the cancer risk for a baby crawling on a deck as 1 in 180 based on a lifetime exposure of 1.53 x 10^5 µg (6 µg/day x 365 days/year/ x 70 years). Lifetime cancer risk estimates were also calculated for an amateur builder of 1 in 612 (builds four decks over a lifetime and based on exposure by ingestion only), and an adult using a CCA-treated wood handrail (twice a day for two years with twice weekly deck surface contact) of 1 in 1275 (Maas et al. 2002). Hand-to-mouth transfer values are similar to those that have been used in other exposure assessments eg HSWMR (2001) but transfer efficiency from wood to skin (represented by wipes) is assumed to be 100%.

The authors mention 6 µg/day arsenic intake as translating to a cancer risk of approximately 1 in 1,000 and cite the National Academy of Sciences (2001). The NRC of the National Academy of Sciences (2001) states 3 µg/L of arsenic in drinking water per day equates to a 1 x 10^{-3} lifetime bladder or lung cancer risk. Although unclear in their report it appears that the authors may have misused this value in their cancer risk estimates by deriving 6 µg/day from the default water intake for adults of 2 L/day. It is unclear why the authors used this value when they had estimated 8.5 x 10^5 µg/year arsenic ingestion for a baby crawling on a deck which equates to 33 µg/day over a 70 year lifetime.

This assessment has been criticised for lack of sensitivity analysis or discussion of the assumptions made and their impact on the risk estimates, lack of standardised or validated exposure scenarios, and assuming that cancer risk for short term (from four separate seven working day periods to two years) exposure scenarios can be extrapolated linearly to a lifetime low level exposure scenario (Fowles et al. 2003). This is an area of uncertainty in cancer risk estimation as short-term exposures cannot be presumed to present no risk.[37] Whilst temporal averaging is a feature of other assessments the period of exposure is typically five years.

Following its publication the author of the Gradient Corporation (2001) risk assessment has also criticised Maas et al. (2002) citing an error in the calculation of the risk estimate for the baby crawling scenario which should be 1 in 850 and not 1 in 180, misuse of the NRC report (2001) in the risk calculations, and ignoring the EPA's calculations used to set its new drinking water standard (Beck, 2002; Mirams, 2003).

[37] There is also some evidence that suggests short-term exposures do not produce proportional decreases in cancer risk depending on the nature of the carcinogen and timing of exposure. Under-estimation of risk may be more likely than overestimation from the use of unit cancer risks derived from lifetime exposure (Halmes et al. 2000; US EPA, 2003a).

15.9 US Consumer Product Safety Commission (2003)

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

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

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

Prior to the field study laboratory experiments were carried out to develop the study protocol. The factors that had the most impact on dislodgeable arsenic levels were type of hand contact, pressure applied, hand rinsing procedure, type of wipe material, and use of a sampling template. The sampling method developed uses weighted polyester cloth wipes in a template to standardise wiping motion and the amount of pressure applied.

The CPSC also reviewed data on dislodgeable arsenic from other studies and concluded that when similar methods are used average results are relatively consistent.

The estimates derived by the CPSC change if alternate assumptions are used. For example, if the upper bound of the unit cancer risk used is that of the EPA (0.0037 µg/kg/day) rather than the NRC's risk estimate of 0.023 µg/kg/day that was calculated by the CPSC then the upper end risk of 10^{-4} reduces.

Of the assessments discussed in this report it has only been possible to confirm two (WS Atkins International, 1998; CPSC, 2003) as having undergone independent scientific peer review. The assessments are characterised by the use of different approaches and data and conclusions range from that the plausible range of children's exposure to arsenic from CCA-treated wood playground equipment does not present a significant health risk eg Gradient Corporation (2001) to an unacceptable risk eg Roberts and Ochoa (2001).

[38] This can be compared to an estimated average daily inorganic arsenic intake of a 2-6 year old child in the United States ranging from 2-46 µg depending on amounts in diet, air and soil (ATSDR, 2000b).

Table 10: Cancer risk estimates for children exposed to dislodgeable arsenic residues

Risk assessor(s)	Risk estimates
CDHS (1987)	1×10^{-4} - 6×10^{-3} (skin)
CPSC (1990)	$3\text{-}4 \times 10^{-6}$ - $8\text{-}9 \times 10^{-6}$ (skin)
	2×10^{-6} - 1×10^{-4} (lung/bladder)[1]
Roberts and Ochoa (2001)	4×10^{-6} - 1×10^{-3} (skin)
Gradient Corporation (2001)	9.6×10^{-7} - 1.5×10^{-6} (skin)
EWG (2001)	2×10^{-3} (lung/bladder)
Maas et al. (2002)	1×10^{-3} (lung/bladder)[2]
CPSC (2003)	2×10^{-6} - 1×10^{-4}(lung/bladder)
	2×10^{-7} - 5×10^{-3} [3]

[1]In 2003 CPSC revised the estimates using the EPA and NRC unit cancer risks.
[2]Based on 1 in 850 not the incorrect 1 in 180 stated in the report.
[3]Risk estimates from a sensitivity analysis.

Uncertainties associated with evaluating exposure to dislodgeable arsenic include residue concentrations, relative oral bioavailability from residues, residue ingestion rate and surface area for hand-to-mouth contact, and exposure frequency. There is also uncertainty about the relationship between arsenic residue concentration and concentration on hands after contact although the recent CPSC study (2003) established a conversion factor of 0.2 for converting wipe data to hand loading.

It is difficult to compare assessments with one another as the values used for some exposure parameters vary widely. As well there has been no standardised approach for evaluating a child's exposure to dislodgeable arsenic from CCA-treated wood. For those assessments that are well described in terms of assumptions and exposure parameters, and dislodgeable arsenic levels are ≤ 100 µg/100 cm^2, risk estimates range from 10^{-6} to 10^{-4}.

The dislodgeable arsenic values used in the assessments in conjunction with other exposure parameters give some indication of levels at which no significant health risk is present against which New Zealand data, if available, could be compared.

In some instances there have been attempts to compare these risks with other risks to American children (eg the 1 in 246 risk of injury on playground equipment requiring emergency department treatment (Paling, 2001)) and to place them in the context of background lifetime risks (eg CPSC estimates increase lifetime lung cancer risk in the United States from 0.01% to between 0.0102 to 0.02% (Milloy, 2003)). However with respect to play equipment non-wood alternative materials, such as plastic, metals and composite materials, and wood treated with other preservatives are available. It is also possible to use CCA-treated wood only for the load-bearing components and other materials for the decking and handrails.

When considering the various lifetime cancer risk estimates that have been derived it is important to take account of the level of risk that is regarded as tolerable or acceptable in New Zealand regulatory decision-making. In New Zealand the level of health protection provided for carcinogens equates to a lifetime (based on 70 years of exposure) risk of additional cancer of no greater than one in 100,000 for a given exposure scenario eg drinking water. In the United States this level is usually no greater than one in a million.

There was public consultation on the level of tolerable cancer risk in New Zealand as part of the development of the drinking water standards (Ministry of Health, 2000) and soil criteria for timber treatment sites (Ministry for the Environment and Ministry of Health, 1997).

16 Aggregate Exposure of New Zealand Children to Arsenic

The inorganic arsenic intake from exposure to CCA-treated wood needs to be considered in the context of total inorganic arsenic exposure from all media. The main contributors to total intake in New Zealand are food and drinking water. Table 11 compares the estimated intake from food and water to that from a playground visit using the approximate value for ingested dislodgeable arsenic calculated by the CPSC (2003).[39]

For example, for a 15 kg child (3 year old) daily arsenic intake is no more than 18.5 µg(<6 µg from food, =9 µg from drinking water and 3.5 µg from playground equipment).

For an 18 kg child daily arsenic intake is no more than 19.7 µg (<7.2 µg from food, =9 µg from drinking water and 3.5 µg from playground equipment).

Table 11: Inorganic arsenic intake of New Zealand children

Age group	Daily intake	Medium	
1 - 6 years	< 0.4 µg/kg	Food	1997/98 total diet survey; assumes 25% dietary arsenic is inorganic
2 - 6 years	= 9 µg	Water	0.9L/day;[1] = MAV of 10 µg/L
2 - 6 years	3.5 µg	CCA-treated wood	Ingested residues/ playground visit (US CPSC, 2003)

[1]The mean drinking water intake of children aged 3 - 5 years is 0.87 L/day (US EPA, 1997).

However both of these results are below the approximate daily proportion of the PTWI of 2 µg/kg/day (30 µg and 36 µg for a 15 kg and 18 kg child respectively) and well below the Lowest Observed Adverse Effect Level (LOAEL) of 50 µg/kg recommended for assessing non-cancer effects to children from short-term exposure (oral and dermal exposures up to 180 days) by the SAP (2001). If an uncertainty factor of 10 is applied to this LOAEL to extrapolate to a No Observed Adverse Effect Level the levels are 75 µg for a 15 kg child and 90 µg for an 18 kg child.

Most children in New Zealand will have a daily arsenic intake from drinking water much less than 9 µg. For example the mean arsenic concentration has been reported as 2µg/L (personal communication to the Ministry of Health, ESR, 6 August 2002).

These calculations also assume that a child visits a playground every day.

These calculations do not include ingestion, dermal contact or inhalation of arsenic in soil from whatever source, dermal absorption of arsenic in wood surface residues, or ingestion and

[39] This value was derived from a mean arsenic concentration of 7.6 µg and assumed hand-to-mouth transfer efficiency is 43% and bioavailability is 100%.

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

17 Information Gaps and Uncertainties

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

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

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

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

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

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

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

18 Precautionary Health Advice

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

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

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

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

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

Children's sandpits should not be constructed from CCA-treated wood as any arsenic leached into the sand will not bind as well as it does to soil and will disperse through the sand. Alternatively sand-pits may be lined with plastic to prevent leachate contaminating the sand. Sand-pits that were built years ago and in which the sand has been replaced are unlikely to present a significant risk as evidence suggests leaching decreases with time. Any risk, if present, could be reduced by lining the sand-pit with plastic and replacing the sand. Local authority advice should be sought about the appropriate means of disposal of potentially contaminated sand.

CCA-treated wood sawdust or wood chips should not be used as barrier materials around playground equipment.

Limited data suggest that playground equipment be coated with an outdoor grade penetrating sealant eg polyurethane every 1-2 years depending on wear and weathering.

Limited data suggest strong oxidising commercial deck treatments or brighteners should be avoided as they result in leaching of hexavalent chromium from CCA-treated wood.

Any possible concern about arsenic uptake in root vegetables can be eliminated by growing these vegetables more than 100 mm from treated wood garden edgings or lining the wood interior with plastic (CSIRO, 2002).

CCA-treated wood should not be burnt as arsenic is released in the smoke and ashes, or used as mulch or compost.

19 Conclusion

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

Few well-designed epidemiological studies have been carried out of timber treatment workers using CCA or workers using CCA-treated wood. Results from studies of urinary arsenic levels have been mixed with studies by Jensen et al. (1991; 1995) reporting increased levels among workers processing CCA-treated wood indoors compared to controls. Given the higher exposure of workers compared to children in terms of duration, frequency and amount of arsenic these studies suggest that it is unlikely that CCA-treated wood structures would pose an unacceptable risk to children.

A number of risk assessments of CCA-treated wood have been undertaken internationally with estimated additional lifetime lung and bladder cancer risks ranging from around one case in a million or less to as high as one case in a thousand children exposed. The upper bound of the range reduces to one in ten thousand (i.e one order of magnitude higher than the risk level regarded as tolerable by New Zealand regulatory agencies) if only the most well described assessments in terms of assumptions made and exposure parameters used are considered. The risk estimates change depending on the exposure assumptions that are made and are influenced particularly by uncertainty relating to the amount of arsenic from residues and soil that is eventually absorbed into the body.

Any health risks to children may be greater from new CCA-treated wood structures assuming the amount of transfer from wood to skin decreases in the same way that the leaching rate into soil decreases after exposure to rain. However it is not known how long it takes for readily dislodgeable arsenic residues once removed to be replaced by subsurface arsenic, and over what time period this continues. Limited data suggest exposure to dislodgeable surface residues and leaching into soil is reduced if the wood is sealed every 1-2 years.

In general, the risk assessments do not put arsenic exposure assessment from CCA-treated wood into the context of an average individual's aggregate arsenic exposure from all sources. Most children in New Zealand will have a daily arsenic intake from drinking water, food and visiting a playground (assuming the CPSC value of 3.5 µg ingested arsenic from surface residues applies in New Zealand) much less than the TDI. These calculations do not include ingestion, dermal contact or inhalation of arsenic in soil from whatever source, dermal absorption of arsenic in wood surface residues, or the potential contribution from other CCA-treated wood structures such as decks or sand-pits. Aggregate exposure may be high for some children such as a toddler in an area where there is a higher arsenic concentration in drinking water, a warm climate resulting in more outdoor play, a high natural background level of arsenic in the soil, and an early childhood centre or home with decks, sand-pit and/or play equipment made of CCA-treated wood.

There are no epidemiological studies or human case reports involving disease related to direct contact with CCA-treated wood and the low level exposures that most of the general population will experience from contact with CCA-treated wood are extremely unlikely to result in acute health effects. CCA-treated wood has also been in use for many years without discernible adverse health effects suggesting that if there is a true increased risk it is very small. This is supported by the finding that epidemiological studies do not show increased

cancer risk among populations with low levels of arsenic exposure from drinking water though to some extent this is limited by the lack of statistical power of the studies.

New Zealanders are exposed to low levels of arsenic present in food, water, air and soil, particularly in the central North Island where background levels are naturally high in the volcanic soil. Available New Zealand data on inorganic arsenic intake are insufficient for a risk assessment to be carried out with reasonable certainty. The issue for regulatory agencies is then to what extent a precautionary approach should be adopted to the use of CCA, as one source of potential inorganic arsenic exposure, in New Zealand. 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.

Recommended actions include:

- enhanced monitoring of treatment plants to ensure "best practice" is carried out;
- branding or run-on printing of each board and/or labelling of CCA-treated wood;
- having new playground equipment in schools, early childhood centres, and public parks built of alternative materials to restrict public 'involuntary' contact with CCA-treated wood since alternative materials are available;
- sealing recently constructed (i.e. = 6 months old) CCA-treated wood playground equipment in schools, early childhood centres and public parks;
- consumer information at the point of sale;
- greater dissemination of precautionary health advice to the public and builders; and
- research – analysis of cancer incidence and mortality data for builders, and a urinary biomonitoring study of children or builders.

The weight of current evidence with respect to health risk seems insufficient to support measures such as replacement of CCA-treated wood structures in current use or banning all future use.

In the United States, Canada and the European Union a ban on the use of CCA-treated wood in residential and recreational settings will take effect in 2004. The regulatory decisions in the United States and Canada resulted from a request from the CCA industry and risk assessments of CCA and children's exposure to CCA-treated play equipment are still in progress. In the European Union the decision followed an assessment of health and environmental risks and consultation. It is difficult to ascertain with certainty to what extent potential risks to health have influenced regulatory decision-making in the EC and its member states which already have restrictions in place. It is possible that in some instances potential environmental risks such as the aquatic ecotoxicity of arsenic, copper and chromium and concerns about disposal of CCA-treated wood have been more influential though it is noted that the EC took the precautionary principle into account. CCA is also currently undergoing a full evaluation by the EC so it is possible that further restrictions may result. Of note is the fact that none of these regulatory decisions apply to CCA-treated wood already in use.

Perspectives differ as is illustrated by the many risk assessments that have been carried out in the United States. A very small amount of uncertainty is unacceptable to some and leads to action in the absence of affirming evidence based on a precautionary approach. In contrast applying a precautionary approach within a risk management framework would involve consideration of the costs of more restrictive regulatory standards, and the risks (including the health consequences of failure of a wooden structure), costs and benefits of alternatives.

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