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Project conducted on behalf of
South African Wood Preservers Association
on request of the Registrar of Act 36 of 1947 (DAFF)

Health Risks of Children Associated with Exposure to CCA at Playgrounds

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31 January 2014

Expertise and Declaration of Independence

This report was prepared by INFOTOX (Pty) Ltd ("INFOTOX"). Established in 1991, INFOTOX is a professional scientific company, highly focused in the discipline of ecotoxicological risk assessment. Both occupational and environmental human health risks, as well as risks to ecological receptors, are addressed.

Dr Willie van Niekerk, Managing Director of INFOTOX, has BSc, Hons BSc and MSc degrees from the University of Potchefstroom and a PhD from the University of South Africa. He is a Qualified Environmental Professional (QEP), certified by the Institute of Professional Environmental Practice (IPEP) in the USA (No 07960160), and a registered Professional Natural Scientist (Pr Sci Nat, Environmental Science, No 400284/04). Dr Van Niekerk has specialised in chemical toxicology and human health risk assessments, but he has experience in many other areas in the disciplines of analytical and environmental sciences.

Dr Marlene Fourie has BSc and Hons BSc degrees from the University of Stellenbosch and MSc and PhD degrees from the University of Pretoria. Her field of specialisation is reproductive biology/toxicology. Dr Fourie also has an MSc-degree in epidemiology from the University of Pretoria. Following positions as Medical Natural Scientist at the Andrology Unit, Department of Urology, University of Pretoria and the Pretoria Academic Hospital from 1987 to 2001, she joined INFOTOX as a Medical Biological Scientist. Dr Fourie has conducted many health risk assessments and projects relating to the health status of communities. She is registered as a Medical Biological Scientist with the South African Health Professions Council (No MW 0003700).

This specialist report was compiled for the South African Wood Preservers Association. We do hereby declare that we are financially and otherwise independent of the South African Wood Preservers Association.

Signed on behalf of INFOTOX (Pty) Ltd, duly authorised in the capacity of Managing Director:

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31 January 2014

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1 Introduction and terms of reference

Chromated copper arsenate (CCA) is a water-based preservative containing arsenic, chromium and copper. It is used for the long-term protection of wood against attack by fungi and insects.

Physical contact with the treated wood surface and dislodging of arsenic may thus lead to human exposure. This is of particular concern at children's playgrounds where wood structures have been treated with CCA.

The Registrar of Act 36 of 1947 (DAFF) notified the South African Wood Preservers Association (SAWPA) to submit an independent scientific opinion on potential health risks of children at playgrounds with CCA-treated wood structures. In response, SAWPA appointed INFOTOX (Pty) Ltd (INFOTOX) to conduct a study of the scientific literature on the subject and to advise SAWPA on the matter.

This INFOTOX report presents a review of research reports on arsenic exposures and health risks to children at playgrounds with CCA-treated wood structures. It has not been the purpose to review attributes of CCA and its history and standing in wood preservation technology. The review and interpretations are limited to documented studies dealing with childhood exposure to arsenic at playgrounds. It has not been the purpose of this INFOTOX study to defend or promote the use of CCA in the treatment of wood for use at playgrounds. The interpretations express the professional opinion of INFOTOX on potential risks of children at playgrounds, based on the scientific literature that was consulted in the subject.

2 Chemical description

The three common aqueous formulations of CCA are listed in Table 2.1. These are designated as types A, B and C, which differ in the proportions of the active ingredients (Chou et al. 2007).

Table 2.1: Composition of CCA formulations expressed on an oxide basis.

CCA formulations	CrO_3	CuO	As_2O_5
	Weight %		
CCA-A	65.5	18.1	16.4
CCA-B	35.3	19.6	45.1
CCA-C	47.5	18.5	34.0

According to Hemond and Solo-Gabriele (2004), the type C formulation is used in the majority of applications for wood treatment in the USA. It is understood that this is also the case in South Africa (personal communication: B Breedt, SAWPA).

CCA is forced into the wood in the form of an aqueous solution. The chemical compounds that are used in the formulations represent chromium as Cr (VI), copper as Cu (II) and arsenic in the oxidation state As (V).

As described by Hemond and Solo-Gabriele (2004), within a period of hours to weeks the chromium is reduced from Cr (VI) to Cr (III) in the wood matrix. Copper is immobilised by

complexation with solid wood constituents. The resulting Cr (III) ions combine with the arsenic to form chromium (III) arsenate ($CrAsO_4$).

From a review of the scientific literature conducted by Katz and Salem (2005) it was concluded that the chemical mechanism of fixation of CCA in wood during and after treatment is not fully

understood. It was suggested that the fixed CCA may be in the form of a cross-linked polymer of $–(O\text{-}CrO)_x\text{-}(O\text{-}Cu)_y\text{-}(O\text{-}AsO_2\text{-}O)_z$-complexed with cellulose.

Whatever the exact mechanism of fixation may be, the outcome is that the solubility of arsenic is much reduced through this chemical reaction compared to the solubility of the arsenic salt, but some arsenic is available in the wood matrix to exert its pesticidal action. This surface exposure may continue for years because the surface arsenic is renewed over time through diffusion mechanisms. This has been confirmed in several studies (Barraj et al. 2009; Shalat et al. 2006; Stillwell et al. 2004; Hemond and Solo-Gabriele 2004; Kwon et al. 2004).

Generally, studies of exposure focused on arsenic. Where assessments included chromium (Cr(VI)), the results showed insignificant noncancer and cancer risks. Copper is not as hazardous as arsenic and chromium and is not considered in risk assessments pertaining to CCA exposures. Although reference is made to Cr (VI) in Section 3.6, this study focused on the assessment of exposure to arsenic.

3 Exposure assessment

3.1 Methodology

Two approaches have been reported in the literature to estimate dislodging of arsenic from CCA-treated wooden structures and exposure to arsenic.

- The first approach is to quantify dislodging of arsenic by rubbing or wiping wood samples with a piece of cloth or paper and to determine the amount of arsenic that was collected. The result is expressed as the mass of arsenic per surface area that was wiped ($\mu g/cm^2$) (Barraj et al. 2009). Some relationship is then established between the collected arsenic and the loadings to be expected on the human skin when in direct contact with the treated wood.

- The second approach is to measure the arsenic that has accumulated on the skin on hands directly following a period of contact with the CCA-treated wood. The measured arsenic loadings are then related to intake of arsenic through incidental ingestion as a consequence of hand-to-mouth contact.

- Where arsenic exposures through skin contact with dislodged residues and soil were estimated, these were lower than through direct ingestion of dislodged residues. Exposure through incidental ingestion of soil at CCA-treated playgrounds was also shown to be lower. These aspects are dealt with in Section 3.6.

- The most direct way of measuring exposure is through biomonitoring of arsenic in urine. As summarised by CDC (online), urinary arsenic levels reflect recent exposures and the concentrations correlate moderately to highly with arsenic intakes from drinking water and dietary sources. Calderon et al. (1999) reported that daily variation in creatinine-corrected

urinary arsenic is relatively small when intake is constant. Ahsan et al. (2000) observed that urinary arsenic levels would be a better predictor for risk of arsenical skin lesions than levels in drinking water in Bangladesh, on which the USEPA arsenic cancer slope factor is based (see Section 4.2.2). Generally, urinary arsenic levels have been accepted as a good biomarker of dose (WHO, 2001). Lew et al. (2010) also used arsenic levels in saliva to estimate arsenic exposures in children at playgrounds, considering the prominent hand-to-mouth exposure of young children.

3.2 The study by Gradient Corporation

Gradient Corporation (2001) based hand loading of arsenic on a study by Scientific Certification Systems (SCS 1998). This report was not available to INFOTOX for assessment, but the reference is listed. Five male volunteers (ages 18 to 40 years) rubbed various wood samples 10 times with each hand. Each hand was subsequently rinsed with reagent grade water, which was analysed for arsenic, chromium and copper. Daily hand transfer efficiencies were estimated using default skin adherence factors to quantify ingestion exposure to dislodgeable arsenic. The hand transfer efficiencies were based on empirical measurements of soil adherence to the palmar surface of children's hands following a variety of activities. Standard USEPA health risk assessment methodologies were followed in the assessment. The USEPA oral cancer slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$ was applied (IRIS online). The authors pointed out that a number of researchers believe that the USEPA cancer slope factor may overestimate cancer risks. This aspect is elaborated on in Section 4.2.2 below. Assessment of noncancer risk was based on the USEPA reference dose of 0.0003 mg/kg-day (IRIS online).

Gradient Corporation followed USEPA guidance on how to characterise exposures and risks when conducting risk assessments (USEPA 1998), following the concepts of CTE and RME. CTE and RME refer to central tendency exposure and reasonable maximum exposure, respectively. CTE represents the most likely exposure scenario under which conditions the majority of individuals would be subjected. The RME is the maximum exposure that is reasonably expected to occur in the scenario under investigation. Under this approach, some intake variables (e.g., exposure frequency, duration of exposure and intake rate) may not be at their individual maximum values, but when in combination with other variables, will result in estimates of the RME (USEPA 1989). USEPA guidance recommends estimating the high-end exposure by *"...identifying the most sensitive parameters and using maximum or near-maximum values for one or a few of these variables, leaving others at their mean values…*" (USEPA 1992). The recommendation is based on the fact that maximising all variables would result in an estimate that is above the range of actual values observed in the population.

Gradient Corporation concluded that the highest cancer risk for the playground scenario for the age group 2 to 6 years ranged between 1.2E-06 for CTE and 3.3E-06 for the RME scenario. These are well within the USEPA's acceptable cancer risk range of 1.0E-06 to 1.0E-04.

The highest noncancer risks, expressed as hazard quotients, were 0.00072 for CTE and 0.002 for the RME scenario. These hazard quotients are much lower than the noncancer target hazard quotient of 1.

3.3 The study by Kwon et al. (2004)

In a study conducted in Canada, Kwon et al. (2004) compared arsenic levels on the hands of 66 children playing at 8 playgrounds with CCA-treated wood structures with levels of arsenic on the

hands of 64 children at 8 playgrounds where no CCA-treated wood structures were present. At the end of each playtime the children's hands were washed in a bag with a known volume of deionised water. The children's age and duration of play were recorded. The two populations in the CCA-treated and untreated playgrounds were of the same age. The authors reported mean water-soluble arsenic loads of 0.50 µg (range 0.0078 to 3.5 µg) for the CCA-treated playgrounds and 0.095 µg (range 0.011 to 0.41 µg) for the untreated playground, respectively.

Kissel (2005) suggested that urine samples could solve uncertainties about arsenic ingestion rates, but Wang et al. (2005) correctly pointed out that this could be useful if the ingestion of arsenic from dietary sources would not be a major confounder.

3.4 Review of studies by Hemond and Solo-Gabriele (2004)

Hemond and Solo-Gabriele (2004) reviewed studies conducted to determine children's exposure to arsenic from CCA-treated wooden decks and playground structures. The authors made reference to "numerous protocols" that have been followed to obtain wipe samples from wood surfaces to determine the amount of dislodgeable arsenic. These have been reported in the range of "tens of micrograms per 100 cm^2 of the wiping device". Hemond and Solo-Gabriele (2004) discussed comparisons between arsenic collected on a wiping device with arsenic collected on persons' hands, which represent the substrate through which intake would occur. The authors concluded that due to the variety of protocols in use, quantitative relationships between wipe samples and hand loadings can be made only in cases where both methods were applied in the same study.

Hemond and Solo-Gabriele (2004) also discussed results of direct measurements of arsenic loadings on hands. Both wet and dry hands were used. The results suggest that moist hands collected higher loadings, but it must be noted that sample sizes in these studies were small and the confidence in the data should not be high.

Hemond and Solo-Gabriele (2004) referred to a study by Carlson-Lynch and Smith (1998) in which three measurements were made of arsenic accumulated on a child's hand during play on a CCA-treated structure and compared the loadings to 10 data points obtained from accumulation on the hands of adults. On the basis of these results the authors concluded that "*the total arsenic accumulation on the child's hands was not very different on an areal basis from the amounts accumulated on adult hands*". At the most, this conclusion should be presented as tentative due to the low number of data points used in the comparison.

The authors concluded that the potential inorganic arsenic dosages for children playing on CCA-treated structures could be "*in the range of tens of micrograms per day*".

Boyce et al. (2005) in a letter to the editor criticised the publication of Hemond and Solo-Gabriele (2004) by saying that the calculations were likely to overestimate exposures associated with CCA-treated wood, in particular in view of other scientific information, including more recent relevant studies. The response by Hemond and Solo-Gabriele (2004) correctly disputed some of the comments by Boyce et al. (2005), but the statement of doses "*in the range of tens of micrograms per day*" is questionable. This exposure would lead to cancer risks higher than the risks predicted by any of the studies covered in this INFOTOX review, which cannot be justified on the basis of the available information. For example, the lifetime average daily doses to arsenic in this range would be an order of magnitude higher than the doses reported by Zartarian et al. (2006), reviewed in Section 3.6 below.

3.5 Pilot study by Shalat et al. (2006)

Designed as a pilot study, Shalat et al. (2006) evaluated arsenic exposures of 11 children of which 7 played on CCA-treated playground sets and 4 on non-treated sets. The age group was 1 to 6 years, with mean and median ages 34.5 and 33 months, respectively. Hand rinse samples for the CCA-treated playgrounds showed a mean load of 0.6 µg arsenic (range < 0.2 to 1.9 µg) and the mean arsenic hand load for children not exposed to CCA-treated wood was below 0.2 µg. Shalat et al. (2006) also determined dislodgeable arsenic using synthetic swipe samples and determined arsenic in urine samples of the children. Although the study showed greater amounts of arsenic on the hands of children who played on the CCA-treated structures, no correlation for such increase was found in the urine. The authors provided several explanations for this lack of correlation, but the most likely reason is that the exposures to arsenic were simply too low to distinguish from background intakes of arsenic from food and drinking water.

Shalat et al. (2006) applied a mathematical relationship established from observed data to convert between arsenic determined from swipe samples and arsenic loads on the hands of children, expressed in the unit µg As/100 cm^2. The authors clearly stated that this was a pilot study and the results of the small number of test subjects should be evaluated on this basis. Overall, the hand rinse results indicated arsenic exposures on the low side of the reported range in the studies reviewed in this INFOTOX document.

3.6 The probabilistic Stochastic Human Exposure and Dose Simulation Model for wood preservatives

USEPA's Office of Research and Development developed and applied the probabilistic Stochastic Human Exposure and Dose Simulation Model for wood preservatives (SHEDS-Wood) to estimate children's absorbed dose of arsenic from CCA-treated playsets and decks (USEPA 2005a). The primary population of interest in the assessment was children in the US in the age group 1 to 6 years who might frequently come into contact with CCA-treated residues at playfields (Zartarian et al. 2006).

The arsenic values used in the exposure analysis in SHEDS-Wood model were based on a structured experimental protocol for wiping wood surfaces and the application of residue-to-skin transfer efficiencies of adults (Zartarian et al. 2006). Barraj et al. 2007 suggested that these experimental methods may overestimate the amount accumulated during actual play of children. The study by (Zartarian et al. 2006) also did not consider removal of arsenic from the skin that may occur due to contact with other materials such as clothes, other parts of play structures, or grass. Not including this loss factor may overestimate exposure.

Zartarian et al. (2006) mentioned "unresolved issues" with the Kwon et al. (2004) study and referred to Kissel (2005) and Wang et al. (2005) in this regard. Kissel (2005) expressed concern that water-insoluble arsenic was not accounted for in the Kwon et al. (2004) study, but Wang et al. (2005), one of the authors of the Kwon study, responded by explaining that they

actually measured insoluble arsenic as well. This changed the total amount of collected arsenic from 3.9 µg in the original report to 4.7 µg, which does not have much effect on the conclusions.

3.7 Barraj et al. (2007) and the SHEDS-Wood approach

Barraj et al. (2007) discussed the USEPA SHEDS-Wood approach for assessing residential and playground exposures to wood preservatives (Zartarian et al. 2006). This model was applied to assess children's exposure to arsenic from CCA-treated structures, but hand loads were interpreted from an experimental study with adult volunteers in a design that aimed to obtain maximum hand and wipe loadings of arsenic from contact with the wood structures. The purpose was to estimate the residue-skin transfer efficiency.

Barraj et al. (2007) assessed the feasibility of using child hand-loading data in SHEDS-Wood and the effects on exposure estimates. The results showed that the data from observational studies of children produced mean values of 27 per cent of the USEPA estimates of the lifetime average daily dose and the 95th percentile of the lifetime average daily dose was 10 per cent of the USEPA estimates. Barraj et al. (2007) recommended that the SHED-Wood model apply data from children actively playing on playsets to more accurately estimate children's actual exposure to CCA.

3.8 The ACC and Edmonton-WPSC studies

Barraj et al. (2009) referred to studies by the American Chemical Council (ACC 2003), which obtained distributions of arsenic residues on deck board surfaces and drawing comparisons between the dislodgeable arsenic determined from wipe sampling versus hand sampling.

Barraj et al. (2009) also referred to an unpublished study by the Wood Preservative Science Council (WPSC 2007). The study was designed to estimate dislodgeable arsenic, chromium and copper from the structures included in the observational exposure study of children by Kwon et al. (2004), discussed in Section 3.3 above. The study was conducted in Edmonton, Alberta, Canada, and is generally referred to as the Edmonton-WPSC study.

The data analysis confirmed that the dislodgeable arsenic from CCA-treated wood structures in the playgrounds included in the Kwon et al. (2004) study were similar to those measured in the study by Shalat et al. (2006) (Section 3.5 above). Barraj et al. (2009) suggested that the difference between the USEPA study (Zartarian et al. 2006) and the Kwon et al. 2004 study was due to the higher loads in experimental studies by Zartarian et al. (2006) using adults and not due to lower dislodgeable arsenic in the Kwon et al. (2004) study.

3.9 USEPA probabilistic risk assessment

3.9.1 Scope of the study

Chen et al. (2008) published a report on probabilistic risk assessment for children exposed to arsenic on contact with playsets and decks in 2008. This is based on the exposure assessment conducted by Zartarian et al. (2006) discussed in Section 3.6 above. The study considered risks associated with exposure to both arsenic and chromium, and also presented arsenic cancer risks based on the observational data published by Kwon et al. (2004), as recommended by Barraj et al. 2007. The results were presented for warm and cold climates.

3.9.2 Noncancer risks

Noncancer effects were evaluated by calculating the ratio of the no-observed-adverse-effect level (NOAEL) or the lowest-observed-adverse-effect level (LOAEL) to the estimated dose. The resulting value is termed the margin of exposure (MOE). The calculation is shown in Equation 3.9.2.1. The larger the MOE, the more unlikely it is that a noncancer adverse effect would occur. USEPA has established an acceptable MOE value of 30 for arsenic and 100 for hexavalent chromium. These values take into account uncertainties associated with the toxicity data and other factors.

$$MOE = (NOAEL\ or\ LOAEL)/ADD \qquad (3.9.2.1)$$

Where:

MOE	Margin of exposure (unitless)
NOAEL	No-observed-adverse-effect level (mg/kg-day)
LOAEL	Lowest-observed-adverse-effect level (mg/kg-day)
ADD	Averaging daily dose (mg/kg-day)

For arsenic, the LOAEL used was 0.05 mg/kg-day.
For Cr (VI), the NOAEL used was 0.5 mg/kg-day.

The MOEs for arsenic for all conditions were found to be substantially greater (minimum factor of 2) than the guidance MOE of 30. All the chromium MOEs were found to be at least two orders of magnitude above the target MOE of 100.

Noncancer risks associated with arsenic and chromium at CCA-treated playfields were thus shown to be insignificant.

3.9.3 Cancer risks

Generally, cancer risks were shown to be higher under warm conditions than under cold conditions. Considering the relatively warm climate of South Africa, the results for warm conditions were selected for discussion. The results for arsenic exposure published by Chen et al. (2008) are presented in Table 3.9.3.1. Percentiles in this table represent the percentages of the exposed population that have cancer risks less than or equal to the stated risk level. For example, in the case of exposure at playsets and decks, the 23rd percentile means that 23 per cent of the exposed population would have a cancer risk equal to or lower than one in a hundred thousand (10^{-5}). The other 77 per cent have risks above one in a hundred thousand.

Table 3.9.3.1. Cumulative percentiles at specified risk levels.

Scenario	Cancer risk level		
	10^{-6}	**10^{-5}**	**10^{-4}**
Playset and deck	<1st	23rd	91st
Playset only	2nd	46th	97th

Mean, median and the 95th percentile cancer risks associated with exposure to arsenic are presented in Table 3.9.3.2.

Table 3.9.3.2. Cancer risks associated with exposure of children to arsenic at CCA-treated playgrounds.

Scenario	Mean	Median	95th Percentile
Playset and deck	4.2E-05	2.3E-05	1.4E-04
Playset only	2.2E-05	1.1E-05	7.7E-05

Cancer risks associated with exposure to hexavalent chromium were shown to be much lower, in that 99.9 per cent of the exposed population had a cancer risk equal to or lower than one in a million (10^{-6}).

3.10 Biomonitoring study by Lew et al. (2010)

The biomonitoring study was based on 125 saliva samples from 61 children and 101 urine samples from 45 children following play at 8 CCA and 8 non-CCA playgrounds. The same playgrounds as previously described by Kwon et al. (2004) were selected for the study. The first urine samples were collected during the evening of the day of play. The second samples were collected during the first urination the next morning and a final were samples collected later that same morning or early in the afternoon following the day of play.

Pre-play saliva samples were collected from children not already playing on the playgrounds. Saliva samples were collected later in the afternoon following play and again the next morning.

The authors provided clear descriptions of the sampling and analytical methodologies that were followed in the study. The age of children, duration of play, frequency of play at the playground of sampling, and the number and detail of urine and saliva samples were recorded. Well established statistical methods were applied for data interpretation.

It was concluded that that there was no significant difference in the arsenic concentrations and speciation distributions in urine and saliva samples of children playing on CCA-treated and non-CCA-treated playgrounds.

4 Toxicological review

4.1 Noncancer risks

Noncancer risks associated to exposure to arsenic were shown to be insignificant in the available literature and review of risk associated with short and intermediate exposures would not add value to this report. The study thus focused on carcinogenicity and the interpretation of cancer risks.

4.2 Carcinogenicity

4.2.1 Cancer classification

The International Agency for Research on Cancer (IARC 2012) evaluated cancer risks associated with exposure to arsenic and concluded as follows:

- For inorganic arsenic and its metabolites, available evidence suggests weak or non-existent direct mutagenesis (genotoxicity), which has been observed only at highly cytotoxic concentrations.

- Long-term, low-dose exposure to inorganic arsenic is considered likely to cause increased mutagenesis as a secondary effect of genomic instability. The mechanism of action is not fully understood, but IARC (2012) suggested that it may be mediated by increased levels of reactive oxygen species, as well as co-mutagenesis with other agents. The major underlying mechanisms observed at low concentrations have been identified as the rapid induction of oxidative DNA damage and DNA-repair inhibition, and slower changes in DNA-methylation patterns, aneuploidy, and gene amplification.

- IARC (2012) suggested that inhibition of DNA repair leads to co-carcinogenicity.

4.2.2 Cancer slope factor

In its derivation of an oral cancer slope factor for arsenic, USEPA (IRIS online) interpreted data provided in Tseng et al. (1968) and Tseng (1977) on approximately 40 000 persons exposed to arsenic in drinking water and 7 500 relatively unexposed controls. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalence rates associated with ingestion of inorganic arsenic. The references are listed to provide context, but INFOTOX did not consult the original publications for the purpose of this review. The USEPA oral cancer slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$ was published in the Integrated Risk Information System (IRIS) in 1998. This is still the official slope factor listed in IRIS (online), although USEPA has published other documents stating and applying different, more conservative, slope factors. The National Research Council (NRC 2001) concluded that "… *internal cancers are more appropriate as end points for risk assessment than non-melanoma skin cancers*". Slope factors relevant to these end points range from 0.4 to 23 $(mg/kg\text{-}day)^{-1}$.

The USEPA cancer slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$ has been the subject of much debate (Carlson-Lynch et al. 1994; Lamm et al. 2006; USEPA 2005b; Chappell et al. 1997). For example, the normal background intake of inorganic arsenic of 0.1 to 0.70 µg/kg-day by Canadians (Health Canada 1993), would lead to a calculated cancer risk of 1.5 in ten thousand to 1 in a thousand, which seems unrealistic. Risks of one in a million to one in ten thousand are normally deemed acceptable. This criterion cannot be met in the case of arsenic for the average Canadian resident. This was highlighted in a review document prepared by Nieboer and Fletcher (2001) for physicians on behalf of the Regional Niagara Public Health Department.

As an example of the impact of background arsenic intake on estimates of cancer risk, Yost et al. (2004; 1994) have shown that USEPA's current cancer slope factor for arsenic, calculated using a linearised multistage model, would be lowered to as little as 0.13 per (mg/kg-day) by correcting the study data on which the slope factor is based for background dietary sources of inorganic arsenic. This implies a cancer risk of more than a factor 10 lower for a particular exposure scenario than the risk estimated using the USEPA slope factor of 1.5.

Schoof et al (1998) reported on dietary intakes of the Taiwanese population where arsenic was elevated in drinking water, on which the USEPA based their slope factor. A dietary intake of 50 µg/day was determined in field studies instead of the 10 µg/day applied by USEPA. Schoof et al. concluded that consideration of dietary intake may result in a downward revision of the

assumed potency of ingested arsenic as reflected in USEPA's toxicity values, which is in agreement with Yost et al. (1994).

The New Zealand Ministry for the Environment (NZME 1997) proposed a cancer slope factor of 0.15 per (mg/kg-day) for inorganic arsenic, reasoning that the USEPA's slope factor overestimates risk by at least a factor of 10. This may seem arbitrary, but based on the available data and the well-documented scientific arguments against the validity of the USEPA cancer slope factor for arsenic as presented above, the slope factor developed by the NZME is likely to be more realistic than the current USEPA slope factor. It can even be argued that an arbitrary ten-fold adjusted slope factor may still represent an overly conservative interpretation of the available dose-response information.

The USEPA subsequently requested the USEPA Science Advisory Board (SAB) to review and comment on key scientific issues in the USEPA's approach to the development of the cancer slope factor for inorganic arsenic. The SAB convened an expert panel for this purpose. The panel supported the use of a linear cancer risk model and supported the use of the epidemiologic data on the Taiwanese population (Tseng et al. 1968 and Tseng 1977, cited in IRIS online) for estimating human cancer risk for inorganic arsenic. However, the panel recognised limitations in these data, and concluded that there is some evidence suggesting a bladder cancer dose-response curve of a shape other than a linear. Therefore, the panel urged the USEPA to consider other epidemiologic studies, and recommended sensitivity analyses to account for certain assumptions used in USEPA (IRIS online) assessment, amongst others, human variability in drinking water consumption rates and in dietary intake of inorganic arsenic from food (USEPA SAB 2007).

The USEPA responded with the publication of a draft toxicological review of inorganic arsenic, focused on oral waterborne arsenic exposure (IRIS 2010). The review confirmed significant uncertainty with regard to non-water arsenic intake, and concluded that the best approach to handle the uncertainty in the assessment was to conduct sensitivity analyses based on a reasonable range of non-water arsenic intake values, up to 200 µg/day for a dietary arsenic intake assumption in exposed populations. This was in agreement with the recommendations by the USEPA SAB (2007). Overall, the USEPA reported that cancer slope estimates for male and female lung cancer and male bladder cancer were relatively insensitive to assumptions related to non-water arsenic intake and varied more or less inversely with the assumed daily water consumption and with drinking water arsenic concentration estimates (IRIS 2010).

The cancer slope factors for ingestion of arsenic in drinking water, developed in the toxicological review, were based on more recent exposure response data on internal cancers. Therefore, the important difference was that the 2010 assessment was based on bladder and lung cancer, and not on skin cancer as was previously done. The carcinogenic mode of action (MOA) of inorganic arsenic was specifically reviewed. The MOA is important; since USEPA guidance for assessing risks associated with early-life exposure to carcinogens (USEPA 2005b) directs the application of age-dependent adjustment factors to the cancer slope factor when carcinogens have a mutagenic MOA. Insufficient data were available to adequately demonstrate a mutagenic mode of action for inorganic arsenic; therefore, the application of age-dependent adjustment factors to the slope factor was not recommended (IRIS 2010).

A Poisson regression model with additive linear dose terms and quadratic age terms was used for drinking water dose-response model fitting in the Taiwanese population (Tseng et al. 1968 and Tseng 1977, cited in IRIS online). Lifetime cancer incidence in exposed US populations was

estimated by using a modified version of the "BEIR IV" relative risk model that has the following inputs: (1) the arsenic dose-response coefficient from the Poisson model; (2) background cancer incidence data and (3) age specific mortality data. The result of this model is a direct estimate of the lifetime bladder and lung cancer incidence for the target (US adult) population (IRIS 2010).

The estimated cancer potency factors for combined (lung plus bladder) cancer incidence related to the ingestion of water-borne inorganic arsenic were 16.9 per mg/kg-day for males and 25.7 per mg/kg-day for females. The factor for females is more severe, and females are considered the more sensitive population. According to the toxicological review report, most of the difference between the cancer potency estimates in the 2010 assessment and those from previous analyses were explained by differences in dose-response models, changes in the assumptions related to the relative drinking water consumption by women in Taiwan and the United States, and the use of more recent data on US population mortality and cancer incidence in the BEIR IV relative risk model (IRIS 2010).

The draft toxicological review includes review comments by the USEPA SAB. The USEPA SAB was in agreement with the conclusions of the draft toxicological review that the studies of indirect genotoxicity strongly suggest the possibility of a threshold for arsenic carcinogenicity, but that it is uncertain where such a threshold might be, and that the shape of the dose-response curve at these low levels is not established. In addition, it was important to note that a threshold has not been confirmed by epidemiological studies. This issue was pointed out as an extremely important area for research attention (IRIS 2010).

The risk analysis in the IRIS toxicological review (IRIS 2010) was criticised as being "deficient in its exposure analysis, both in its consideration of exposure confounders and its choice of summary exposure metric" (Lamm et al. 2010). Schoof (2010) contended that there are substantial flaws in the USEPA's dose response analysis, and that the flawed analysis results in markedly overstated low dose risk estimates. Schoof also suggested that the USEPA SAB should recommend a revision of the dose response assessment, amongst other reasons, to incorporate results of recently published studies, as well as ongoing USEPA research programs.

The USEPA slope factor is best applicable to assessments of drinking water contaminated with inorganic arsenic, and not contaminated food or soil. The cancer slope factor proposed in the 2010 review (IRIS 2010) has not as yet been implemented by IRIS and has been the subject of scientific criticism from outside of the USEPA. The official IRIS oral cancer slope factor for inorganic arsenic is the factor developed in 1998; namely, 1.5E+00 $(mg/kg\text{-}day)^{-1}$, based on skin cancer (IRIS online). This is lower by one order of magnitude than the slope factor associated with cancer of the internal organs (lungs and bladder) proposed for males in 2010, and almost 20 times lower than the slope factor proposed for females.

Schoof (2010) pointed out earlier criticisms in support of her conclusion that the low dose risk estimates were markedly overstated by the USEPA. She attended the USEPA Science Advisory Board work group meeting on April 6, 2010, noting that USEPA's baseline assumption that non-water (dietary) inorganic arsenic intake of 10 µg/day in the Taiwanese study was incorrect. Subsequently she referred to her attendance of the meeting in a written submission to USEPA (Schoof 2010) and stated that *"(I)t was apparent during the SAB work group deliberations that many aspects of our 1998 study were not understood by either the work group members or the EPA staff in attendance at the SAB meeting*".

In the SHEDS-Wood probabilistic risk assessment, Chen et al. (2008) applied a cancer slope factor of 3.67 (mg/kg-day)$^{-1}$. This slope factor is 24.4-times the cancer slope factor of 0.15 per (mg/kg-day) for inorganic arsenic proposed by the New Zealand Ministry for the Environment (NZME 1997) and the calculated cancer risk would thus be more than 20-times higher when the slope factor of 3.67 (mg/kg-day)$^{-1}$ is applied. This slope factor is 2.4-times the much-criticised current USEPA slope factor of 1.5 (mg/kg-day)$^{-1}$ in IRIS (online).

An assessment of cancer risks associated with exposure to arsenic cannot be conducted with confidence without considering these controversies around the cancer slope factor. Furthermore, arsenic occurs naturally in nature and its presence in soil and background intake through food and water has to be acknowledged and taken into account, as described in Sections 5 and 6 below.

5 Arsenic intake from soil

Because of leaching of arsenic from CCA-treated wood structures into soil at playgrounds over time, the soil pathway of exposure has been considered in several studies of exposure at playgrounds, of which some are referred to in this report (Stilwell and Gorny 1997; Ursitti et al. 2004; Hemond and Solo-Gabriele 2004).

Stilwell and Gorny (1997) conducted a comprehensive study of arsenic concentrations in soil underneath CCA-treated decks at playgrounds. Average arsenic concentrations in 85 samples from under 7 different decks were found to be 76 mg/kg.

Ursitti et al. (2004) reported on soil samples collected from 217 play structures in Toronto from which 4 composite samples were prepared and analysed for arsenic. The mean concentration was 2.1 mg/kg, with a range of 0.5 to 10 mg/kg.

In an unpublished study cited by Hemond and Solo-Gabriere (2004), 84 samples were collected from under 10 decks. The average arsenic concentration was 22 mg/kg and the highest concentration was 85 mg/kg. Hemond and Solo-Gabriere (2004) reviewed other studies (not cited in this INFOTOX report) and quoted a mean arsenic concentration of 27 mg/kg, noting widely variable values up to 76 mg/kg.

Exposure to arsenic in soil at playgrounds was shown to be lower than exposure from playsets and decks and the addition from this pathway to noncancer and cancer risks would not affect the overall interpretation of cancer risk (Gradient Corporation 2001; Chen et al. 2008; Kwon et al. 2004; Zartarian et al. 2006). Exposure due to arsenic in soil is thus not regarded as of pertinent significance. This must also be viewed in the context that arsenic in soil in certain regions in South Africa is above 100 mg/kg (SRK 2001).

6 Background intake of arsenic from food

The European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain (CONTAM Panel) (EFSA 2009) assessed the risks to human health related to the presence of inorganic arsenic in food. More than 100 000 data points on arsenic in food were considered. The data covered food and water across 19 European countries, using lower bound and upper bound concentrations. The survey referred pertinently to inorganic arsenic, because organic arsenic compounds have much lower toxicity than inorganic arsenic.

The beta binomial-normal (BBN) model in the Monte Carlo Risk Assessment (MCRA) software was used to estimate long-term dietary exposures. In this approach all daily consumption patterns were multiplied with the average level of inorganic arsenic per food, and summed over foods per day. This resulted in a set of daily mean exposure levels, which were then analysed using the statistical BBN model to assess the long-term exposure. Exposures were calculated as a function of age. For the purpose of this review, INFOTOX selected the age group 1 to 6 years, because this is relevant to the ages of children at playgrounds. Figure 6.1 shows the average daily doses for the various age groups.

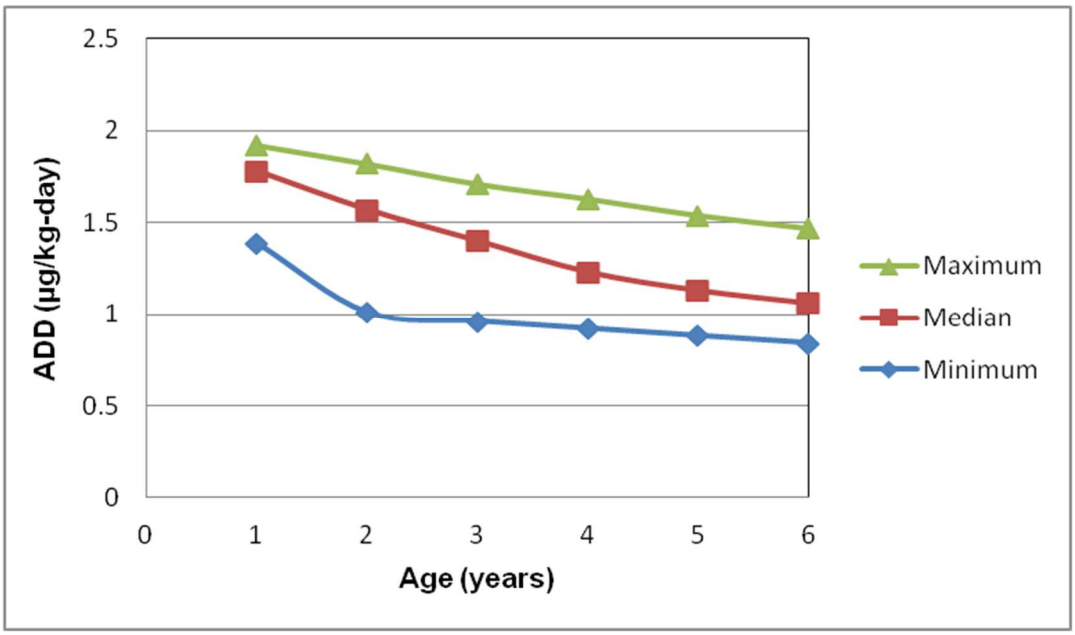

Figure 6.1: Average daily doses of arsenic from intake of food for the age groups up to six years.

The average daily dose (ADD) is not the same as the lifetime average daily dose (LADD) reported for exposures at CCA-treated play sets by Zartarian et al. (2006). For lifetime simulations it was assumed that exposure to CCA-treated wood is limited to the 6-year life span. The total dose over 6 years was averaged over a lifetime of 75 years to obtain the LADD. In risk quantification the LADD calculated in this way would represent the excess cancer risk that may be associated with the exposures to arsenic over the period of 6 years at CCA-treated wood play fields.

Table 6.1 shows cancer risks calculated from background arsenic intakes. The ADD was simplified by calculating the mean ADD over the age groups.

Table 6.1: Cancer risks associated with intake of inorganic arsenic from food and water (EFSA 2009).

	Mean ADD	LADD	LADD	Cancer risk
	µg/kg-day	µg/kg-day	mg/kg-day	Q* = 1.5 (mg/kg-day)$^{-1}$
Minimum	1.00	0.09	8.57E-05	1.29E-04
Median	1.36	0.12	1.17E-04	1.75E-04
Maximum	1.68	0.14	1.44E-04	2.16E-04

The significance of these data is dealt with in Section 10.1 below.

7 Status of information

There is not an abundance of studies on health risks to children at playfields with CCA-treated playsets and decks. As indicated in Section 3, each study is characterised by specific strengths, but also by some limitations. It is appropriate to summarise the status of information in this section of the INFOTOX review.

- Gradient Corporation (2001) conducted an exposure assessment based hand loading of arsenic on a study by Scientific Certification Systems (SCS 1998) in which male adults rubbed their hands on CCA-treated wood. It was concluded from the study that cancer risks associated with arsenic exposure would be well within the USEPA's acceptable cancer risk range of 1.0E-06 to 1.0E-04. Estimated noncancer hazard quotients were also much lower than the target hazard quotient of 1. However, the study was based on a small number of test subjects and the conclusions thus have limited confidence. The exposures also did not represent children's activities and exposures at playgrounds. Because of these limitations, INFOTOX did no rely solely on this publication, but it has to be acknowledged in the overall set of reports.

- The study by Kwon et al. (2004) is one of the few observational studies of children at CCA-treated structures at playgrounds and as such has particular relevance. The study has been criticised by Kissel (2005), Zartarian et al. (2006) and Chen et al. (2008), primarily because of potential underestimation in the quantification of exposure. However, Wang et al. (2005) clarified uncertainties. Barraj et al. (2007 and 2009) supported the Kwon results and pointed out that the Zartarian et al. (2006) and Chen et al. (2008) studies were likely to overestimate exposure and risk. The study by Kwon et al. is regarded as one of the important studies to consider in the overall assessment, but it is not singled out as the most relevant or most important study.

- Hemond and Solo-Gabriele (2004) reviewed studies conducted to determine children's exposure to arsenic from CCA-treated wooden decks and playground structures. The statement that arsenic doses at playgrounds are likely to be "*in the range of tens of micrograms per day*" is questionable in view of other studies and the report has been challenged in a letter to the editor by Boyce et al. (2005). INFOTOX considered the exchange of opinions between Boyce et al. (2005) and Hemond and Solo-Gabriele (2004) and also estimated the lifetime average daily dose to arsenic where exposures could be in the "*in the range of tens of micrograms per day*". This level of exposure to arsenic would be approximately an order of magnitude higher than the lifetime average daily dose reported by Zartarian et al. (2006). The study by Hemond and Solo-Gabriele (2004) is thus not regarded as a benchmark in the assessment of children's exposure to arsenic from CCA-treated wooden decks and playground structures. INFOTOX did not use this study in the general assessment.

- Designed as a pilot study, Shalat et al. (2006) evaluated arsenic exposures of 11 children of which 7 played on CCA-treated playground sets and 4 on non-treated sets. Chen et al. (2008) pointed out some limitations in the Shalat et al. study, but INFOTOX agrees with Barraj et al. that this study, despite its limitations, may be used together with Kwon et al. (2004) and Wang et al. (2005) to provide perspectives on the spread of exposures found in various studies. The results also fit in with the conclusions of the more recent study by Lew et al. (2010) (Section 3.10).

- USEPA's Office of Research and Development developed and applied the probabilistic Stochastic Human Exposure and Dose Simulation Model for wood preservatives (SHEDS-Wood) to estimate children's absorbed dose of arsenic from CCA-treated playsets and decks (USEPA 2005a). The primary population of interest in the assessment was children in the US in the age group 1 to 6 years who might frequently come into contact with CCA-treated residues at playfields (Zartarian et al. 2006). This is regarded as an important study, which was followed up by Chen et al. (2008).

- Chen et al. (2008) published a report on probabilistic risk assessment for children exposed to arsenic on contact with playsets and decks in 2008. This is based on the exposure assessment conducted by Zartarian et al. (2006) discussed in Section 3.6 above. The study considered risks associated with exposure to both arsenic and chromium, and also presented arsenic cancer risks based on the observational data published by Kwon et al. (2004), as recommended by Barraj et al. 2007. The results were presented for warm and cold climates. The primary issue about these studies (Zartarian et al. 2006 and Chen et al. 2008) is that exposure estimates relied on experimental data acquired using adult volunteers in a study designed to obtain maximum hand and wipe loadings (Barraj 2007). The results of the Chen et al. (2008) were used by INFOTOX as part of the overall volume of information to assess potential risks of children at playgrounds with CCA-treated playsets and decks.

- The studies conducted by Barraj et al. (2007 and 2009) provided context between the studies suspected to underestimate exposure and the SHEDS-Wood modelling results that represented the upper limit of exposures. These are important studies and were taken into consideration by INFOTOX.

- Results of the biomonitoring study by Lew et al. (2010) supported the observations of the pilot study of Shalat et al. (2006) and made an important contribution to reducing uncertainties in the available scientific data. The study design and interpretations are regarded as robust and the conclusions of the study have a significant influence on the level of confidence in the interpretation of potential cancer risks of children at playfields that have CCA-treated wood structures.

8 Summary of exposures and risks

For ease of reference, the risk assessment results presented in Section 3.9.3 are presented again below.

Table 8.1: Cumulative percentiles at specified risk levels (Chen et al. 2008).

Scenario	10^{-6}	10^{-5}	10^{-4}
Playset and deck	<1st	23rd	91st
Playset only	2nd	46th	97th

Table 8.2: Cancer risks associated with exposure of children to arsenic at CCA-treated playgrounds (Chen et al. 2008).

Scenario	Mean	Median	95th Percentile
Playset and deck	4.2E-05	2.3E-05	1.4E-04
Playset only	2.2E-05	1.1E-05	7.7E-05

The excess cancer risk determined by Chen et al. 2008 is close to one in ten thousand (10^{-4} or in scientific notation, 1.0E-04). In terms of probabilistic risk, it was shown that 91 per cent or fewer of children exposed to CCA-treated playsets and decks would experience an excess cancer risk of equal to or less than one in ten thousand. The term "excess risk" refers to the lifetime cancer risk attributable to exposures during childhood (1 to 6 years). The risks associated with playsets only are lower, as shown in the tables.

According to Barraj et al. (2007 and 2009), the study reported by Zartarian et al. 2006 overestimated exposure by up to ten-fold. This would lower the risk accordingly. This comment is supported by several studies, as outlined in Section 3.

Furthermore, Chen et al. (2008) stated in their uncertainty review that the cancer risks represent conservative estimates of risk, due to the choice of the cancer slope factor. The cancer slope factor that was applied in the assessment is not a mean value. It is at the high end of the distribution, based on the various conservative factors that are included in the formulation of the final value (e.g., low dose extrapolation and extrapolation from adults to children). Therefore, according to Chen et al. (2008), the "*uncertainty cloud*" around the slope factor is asymmetrical. Therefore, more of the uncertainty cloud is below the estimated risk than above it. There is thus high confidence that the estimated cancer risk is not higher than the calculated value and, in fact, might be lower.

Considering the discussion about the USEPA cancer slope factor in Section 4.2.2, it is likely that the estimated cancer risks could be much lower than the values reported by Chen et al. (2008). This fact, together with the interpretations about exposure by Barraj et al. (2007 and 2009), would lead to even lower risks. It is expected that the cancer risks due to exposure at playgrounds would be lower than the risks estimated for background exposure to food and water and it is reasonable to estimate these risks in the range between 1.0E-06 and 1.0E-04 (between one in a million and one in ten thousand).

9 Regulatory status in the western world

9.1 Introduction

It has not been the intention of this report to review the regulatory status of CCA in other countries in detail. Rather, it is considered important to understand the reasons why certain countries in the western world have placed restrictions on its use for certain applications.

There is no indication of life-threatening exposures or large-scale poisoning of children at playgrounds with CCA-treated wood structures. That is why regulators have not instructed the removal of existing CCA-treated structures in countries where restrictions on the use of CCA have been imposed, either voluntary or through regulations.

9.2 The European Union

It appears that much of the concerns about CCA are based on a report by WS Atkins International published in 1998. INFOTOX did not review this report, but The Scientific Committee on Toxicity, Ecotoxicity and the Environment of the European Commission (CSTEE 1998) formulated an opinion of the report by WS Atkins International Ltd and expressed some criticism. It was agreed that the methodological approach and conclusions were valid, but it was pointed out that much of the information on the subject of wood preservation was available only in the form of technical reports, which WS Atkins International referred to, but there was also substantial information in the open literature to which no reference was made. Of the 65 or so references in the report, only 9 were papers published in the open literature. Thirty were technical reports, 5 were papers from meeting proceedings (presumably not subjected to peer review) and there were even 9 references to personal communications, some of which concerned issues of major importance. As presented in Section 3, more studies of significant relevance have been published since the report by WS Atkins International.

According to Read (2003), it is difficult to ascertain with certainty to what extent potential risks to the health of children have influenced regulatory decision-making in the EC and its member states that already had restrictions on CCA-treated structures in place. It is likely that the precautionary principle was applied, considering general public concern and the limited data that were available on children's health risks at the time. 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. 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 Health and Safety Executive (HSE 2001) reviewed available information and submitted a recommendation to government for continued use of CCA-treated wood subject to certain environmental data and occupational health requirements.

It appears that these steps in the EU were implemented primarily because of public concerns about potential exposure to arsenic rather than indisputable evidence of health risks. It is important to note that none of these regulatory decisions in the EU on the use of CCA-treated wood apply to CCA-treated wood already in use (Read 2003). In itself this suggests that the steps are precautionary and that the estimated cancer risks are low, if not insignificant.

9.3 Canada and the United States of America

As described by Read (2003), there is a co-operative approach between the Pest Management Regulatory Agency (PMRA) of Health Canada and the US Environmental Protection Agency to the re-evaluation/re-registration of heavy duty wood preservatives. Following the USEPA's announcement of the CCA industry's voluntary decision to cancel residential use of CCA-treated wood in 2002, the PMRA facilitated a similar voluntary decision with industry in Canada. It is unclear whether the basis for this decision by the PMRA was scientific, political, or market-driven.

More recently, USEPA (2011) concluded that wood preservation compounds, including CCA, contribute benefits to society and are eligible for reregistration provided the mitigation measures and associated label changes identified in the re-registration eligibility decisions (REDs) are implemented and required data are submitted. Pesticide manufacturers voluntarily agreed to

phase out certain CCA-treated wood products around the home and in children's play areas. Treated structures already in use are not affected by these agreements.

9.4 Australia

In March 2005, the Australian Pesticides and Veterinary Medicines Authority (APVMA 2005) reviewed the safety of CCA-treated timber use in Australia. It was concluded that CCA-treated timber should not be used to build children's play equipment, patios, domestic decking, handrails, new garden furniture, exterior seating or picnic tables. Several uses were supported, e.g., CCA-treated for poles, fencing, landscaping timbers, piling and other structure foundations, residential construction, industrial and commercial construction, rural and farm use, fresh and salt water structures, signage and boat construction.

It is important to note that existing structures made from CCA-treated timbers do not need to be removed and replaced until they reach the end of their functional life. It is also important to point out that these recommendations were made as a precaution since there is no evidence to suggest that CCA-treated timber is harmful when handled or used properly.

9.5 New Zealand

Read (2003) was commissioned by the Environmental Risk Management Authority, the New Zealand Environmental Protection Authority's (EPA's) predecessor organisation. The final report was peer reviewed and released to the public on 2 May 2003. New Zealand has not banned CCA-treated timbers. The mechanism for banning a substance is through a reassessment process, but on the basis of the Read (2003) report and other reports available on the EPA's website it was concluded that there was not sufficient evidence to demonstrate that the health risks associated with exposure to CCA treated timber warrant the substance being reassessed (EPA 2013). However, considering the fact that arsenic is carcinogenic, the EPA acknowledged that it would be prudent to avoid unnecessary exposure.

10 Conclusions

10.1 The level of confidence in the risk of children at CCA-treated wood structures at playgrounds

As outlined in Section 4.2, there is no doubt about the carcinogenicity of arsenic. The reasoning in this INFOTOX review is about the assessment of exposure at playgrounds and risk interpretation considering the cancer slope factor that is applied.

INFOTOX is in agreement with Read (2003) that at the time of publication in 2003, research reports were inconclusive as to whether exposure to arsenic from CCA-treated structures at playfields poses a significant health risk to children. More recent studies have been published since the review conducted by Read (2003). The various studies reported in the scientific literature applied different methods for quantification of exposure to arsenic at playgrounds and reviews have expressed opinions of either overestimation or underestimation of exposure. In most cases it was not possible to compare the findings of the various studies directly, because different methodologies and exposure factors were applied to estimate arsenic exposure and health risks.

Although comments were made in the literature about uncertainties in the mode of action of arsenic, USEPA cancer slope factors were applied in most of the publications. Some of the risk assessments selected the slope factor that is currently listed in IRIS (online). As discussed in Section 4.2.2, this slope factor is under much dispute and it has been estimated that its application may overestimate risk up to a factor 10. The prominent USEPA probabilistic risk assessment discussed in Section 3.6 (Chen et al. 2008) applied an even more conservative cancer slope factor. Even under these conditions the highest cancer risk at the 95 percentile was estimated as 1.4E-04, which is only marginally outside the USEPA acceptable risk range.

Considering the discussion in Section 8 of this INFOTOX report, the most likely cancer risk range is roughly between one in a million (1.0E-06) and one in ten thousand (1.0E-04) for childhood exposures at playgrounds with CCA-treated wooden structures. This risk range is acceptable according to USEPA criteria.
The factor of background exposure must also be taken into account. As shown in Section 6, background exposures from food and water are estimated to be higher than from exposure to arsenic at playgrounds. The aggregate cancer risk from background intake and from exposure at playgrounds with CCA-treated playsets and decks would not be remarkably different from the risk associated only with background exposures. The cancer risk would still be slightly higher than one in ten thousand, but lower than one in a thousand if the USEPA cancer slope factor is applied (IRIS online). Considering the discussions about the arsenic cancer slope factor in Section 4.2.2, it is even likely that the aggregate cancer risk would be an order of magnitude lower, thus in the range between one in a hundred thousand and one in ten thousand, which falls within the acceptable risk range.

In commenting on the Kwon et al. (2004) study, Kissel (2005) remarked that biomonitoring based on urine samples from children who do and do not play on CCA-treated structures would remove uncertainties about arsenic ingestion rates at playgrounds. The pilot study by Shalat et al. (2006) and the more recent study by Lew et al. (2010) convincingly provided this clarification and showed that arsenic levels in urine of children at playgrounds are due primarily to background ingestion of food and water. The studies confirmed that there was no statistically proven difference in arsenic exposure of children between playgrounds with CCA-treated wood structures and those that do not have CCA-treated wood structures. Lew et al. (2010) also did not find any differences in the arsenic content of saliva samples between the two groups of children, which supports the conclusion that the intake of arsenic is not significantly different between the two groups.

The studies on biological monitoring support the results of the Kwon et al. (2004) study and the reviews of various studies by Barraj et al. (2007 and 2009). There is thus a coherent set of studies that indicate insignificant cancer risks associated with exposure to arsenic at CCA playgrounds.

10.2 The background and significance of voluntary and regulatory bans on CCA-treated wood structures

It is acknowledged that there is public concern over exposure of children, not factually based on what is known about cancer risks associated with arsenic exposure at playgrounds, but more significantly about what is not known about the levels of exposure. Furthermore, there is a general objective in the world to limit exposures to carcinogens to levels as low as reasonably achievable. However, note has to be taken of background exposures of children to arsenic from food and water, due to naturally occurring arsenic. No action, whether voluntary or through regulatory processes, is warranted where the contribution of any potential additional source of exposure is

so low that it is not measurable in an exposure assessment. Several studies reviewed in this INFOTOX report support this conclusion about arsenic exposures at playgrounds.

As pointed out earlier in this report, the various reports about arsenic exposure at playgrounds vary in methodologies that were applied in the quantification of exposure. Different techniques were applied to quantify the extent to which arsenic can be dislodged from wood surfaces and exposure parameters such as contact times and contact frequencies of test subjects varied. It is also uncertain to what extent the type of wood, the age of structures and weathering conditions may influence dislodging of arsenic. Nevertheless, the value of the available studies should not be played down and some balance has to be sought between uncertainties and the current understanding of potential risk. In particular, the biomonitoring studies by Shalat et al. (2006) and Lew et al. (2010) have significantly reduced the uncertainties introduced by assumptions and methodologies in some of the other studies. Furthermore, the data from Kwon et al. (2004) emphasised the importance of applying empirical data from actual exposures of children. These exposure values from Kwon et al. (2004) were applied by Barraj et al. (2009) in the SHEDS-Wood model (Section 3.7), which confirmed exposure levels that would be in agreement with the observations of Shalat et al. (2006) and Lew et al. (2010).

10.3 Assessment of the situation in South Africa

Regulators in the world tend to apply the precautionary approach where there is uncertainty about potential risk. The purpose of the precautionary principle is to provide the motivation to take a decision notwithstanding scientific uncertainty about the nature and extent of the risk. This is not an easy concept, because there are many uncertainties about risk in everyday life and an item or activity cannot be banned without considering the implications of such action. Given that every risk involves some uncertainty, it is not clear when risk is sufficiently uncertain to justify application of the precautionary principle.

Characteristics of risk that may affect the appropriate level of precaution may include irreversibility of the consequence, the magnitude of possible consequences, the probability of occurrence, the amount and types of uncertainty associated with the risk, the societal benefits of the risk-causing activity, the difficulty and costs of reducing the risk, potential alternatives to the risk-causing activity, potential risks associated with the alternatives, potential risk-risk trade-offs, and public perception of the risk (Marchant 2003). In many cases public perception of a risk carries a disproportionate weight in the decision to institute the precautionary principle, which may lead to decisions that have unsustainable and adverse financial and societal impacts.

It must be taken into account that voluntary and regulatory restrictions on the use of CCA-treated wood in several countries were introduced on the basis of uncertainties in the available arsenic exposure data at the time. This is not the case anymore. The studies of Zartarian et al. (2006) and Chen et al. (2008) provided conservative estimates of exposure and cancer risk. There are valid reasons to believe that cancer risks associated with arsenic at playgrounds would be lower than the values estimated by Chen et al. (2008), based on the exposure assessment methodology and the likely overestimation of cancer risk by the USEPA arsenic cancer slope factor. Furthermore, biological monitoring has confirmed insignificant exposure of children at playgrounds with CCA-treated wood structures. Arsenic measured in urine and saliva samples were associated with background exposure to arsenic in food and water, with no measurable difference between children at CCA playgrounds and those at playgrounds without any CCA-treated wood structures.

The level of confidence in the available exposure and health risk data at this time does not warrant application of the precautionary principle. Any restriction in the use of CCA-treated wood structures is thus not warranted.

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