



A REVIEW OF THE PUBLIC HEALTH AND ENVIRONMENTAL RISKS ASSOCIATED WITH THE USE OF TIMBER TREATED WITH CCA PRESERVATIVES

The Australasian Treated Timber Co-ordination Group (ATTCG), the Australian and New Zealand timber treatment industries, and their suppliers of CCA preservatives are committed to determining the risks, if any, associated with the use of CCA-treated timber

These Groups want to be reassured that any identifiable risks are within acceptable limits. The industry has also asked for some guidance as to what an acceptable level of risk might be and has suggested that this might best be defined with respect to benchmarks established by naturally occurring exposures.

The ATTCG, the NZ TPC, and others in the preservation industry are concerned to ensure that the review of CCA timber being undertaken by the Australian Pesticides & Veterinary Medicines Authority relies on the best science and research available. This report was therefore commissioned to summarise the existing body of research on the subject, particularly in light of a specialist Conference on CCA recently held in the USA and the impending US EPA risk assessment. To this end Dr Wayne Temple attended the 19th Annual Conference on Contaminated Soils, Sediments and Water held 20-23 October at the University of Massachusetts at Amherst. He also obtained comprehensive briefings from the US Environmental Protection Authority during a visit to that organisation.

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SUMMARY

Several human health risk assessments have been conducted for exposure to CCA treated wood particularly for the scenario for children where exposure is most likely to occur through the ingestion of dislodgeable residues from CCA treated wood or from CCA contaminated soil.

The exposure assumptions used in these published risk assessments are health protective and tend to overestimate risks. Despite animal studies indicating the true extent of bioavailability of arsenic from dislodgeable wood and contaminated soil, most risk assessments continue to assume 100% bioavailability. The use of a linear model for cancer effects at low level exposures is also controversial. Cancer risk estimates for CCA exposure are worst case, hypothetical estimates, and the true risks at low dose may be close to zero.

Background exposures to arsenic from food and water sources are significantly higher than from estimated exposure to CCA timber. The combined intake of arsenic from all these sources falls well below the WHO tolerable daily intake for arsenic.

Exposures to arsenic associated with CCA-treated wood in domestic and playground settings do not pose a significant health risk to children.

INTRODUCTION

The wood preservative copper, chromium and arsenic (CCA) has had a history of use in New Zealand since the 1950s. About 5,000 tonnes of CCA salt equivalent are used annually to treat an estimated 650,000 m³ of timber to various treatment grades. CCA treated timber has been used commercially in Australia for more than 40 years with current annual production of approximately 900,000 m³. There are no known reports of health effects associated with CCA-treated wood in New Zealand.

In February 2002, wood preservative manufacturers in the USA approached the EPA to voluntarily phase-out all CCA residential uses including CCA intended for use in treating wood destined for decks, picnic tables, landscaping timbers, gazebos, residential fencing, patios, walkways and play structures. EPA accepted the registrants' actions such that in the US CCA will not be used on residential type wood after December 30, 2003. It is important to note that the EPA has not concluded that CCA-treated wood poses unreasonable risks to the public for existing structures made with CCA-treated wood.

The US EPA is conducting a thorough review of the latest public health and environmental studies available on CCA. One risk assessment is specifically considering children's exposure at residential sites, playground settings and public parks. The other risk assessment focuses on the uses that are not subject to the phase-out agreement. These assessments are due to be released later this year.

In May 2003 the New Zealand Environmental Risk Management Authority released a report of its investigation into public health issues surrounding the use of CCA-treated timber in playgrounds and residential buildings in New Zealand. The report was conducted by a recognised public health specialist, Dr Deborah Read, and peer reviewed by three independent medical and scientific authorities. This review

established that there was insufficient evidence to conclude that these products pose an unacceptable risk.

The Board of the Australian Pesticides and Veterinary Medicines Authority (APVMA) on July 28 2003 announced that it intends to stop the use of CCA as a timber treatment in certain domestic situations such as decking and children's playground equipment by the end of 2003 unless there is conclusive proof that continued use is safe. The APVMA Board is anticipating that it will be provided with a comprehensive analysis on CCA later this year.

Against this background one of the authors of this report, Dr Wayne Temple, visited the US EPA in Washington DC to gain a first hand perspective of their review of CCA and an exposure to the latest scientific findings on CCA by attending the 19th Annual International Conference on Soils sediments and Water, October 20-23, 2003 University of Massachusetts, Amherst, which included a one day symposium on CCA Treated Wood –Regulations, Science and Risk Assessment. These findings have been incorporated into this report. Details of the symposium presentations and people that Dr Temple met with at the US EPA are given in Appendices A and B.

This report examines the key issues surrounding the risk assessment of children's exposure to CCA treated wood. Since arsenic is the most toxic of the components of CCA this report focuses on risk assessments of CCA-treated wood which are derived from parameters pertaining to arsenic exposure.

CHILDREN'S EXPOSURE TO ARSENIC FROM CCA-TREATED WOOD

There are two primary sources of possible exposure to arsenic for children at playgrounds with CCA-treated wood equipment: 1) arsenical compounds from the wood that have leached into the soils; and 2) dislodgeable arsenical compounds from the equipment. For each of these sources there are two potential pathways for exposure – dermal and ingestion. Inhalation exposure is not considered to be a significant exposure route for playground and residential exposure scenarios.

Bioavailability

A critical factor determining the magnitude of potential exposures and risks associated with a chemical is its bioavailability, i.e., the amount of the chemical that is actually absorbed into the body relative to the amount ingested or that is contact with the skin. A chemical's bioavailability is influenced by such factors as the species of the chemical, the matrix in which it is present, the amount of time that a chemical is in a matrix, and the route by which exposure occurs. When chemicals are ingested, bioavailability is determined by the amount of a chemical that is dissolved in gastrointestinal fluids and absorbed across the gastrointestinal tract into the bloodstream. An ingested chemical that is adsorbed to soil or some other solid medium like wood dust may be absorbed less completely than the same ingested dose of the chemical when dissolved in water.

There are no human studies measuring arsenic bioavailability from ingestion of CCA-treated wood, however two studies in dogs fed sawdust from CCA-treated wood gave a bioavailability of 48% (Gradient Corporation 2001). Despite these animal studies and the findings that arsenic in CCA residues appears to be complexed with the wood (Nico

2003) the US CPSC risk determination for arsenic in CCA-treated wood assumed a 100% bioavailability (Exponent 2002).

Similarly, the oral bioavailability of arsenic in soil from a wood treatment site was 16% in Cebus monkeys (Roberts et al., 2002). A juvenile swine model has also demonstrated that the absorption of arsenic from both soil and CCA-treated wood is significantly less than for soluble forms of arsenic (Casteel, 2003). Ng and Moore (1996) reported that the bioavailability of arsenic from soil from a timber treatment site at which CCA had been used ranged between 4.5% and 38% depending on the valency state of arsenic.

Despite these results, most risk assessments have taken the conservative approach and assumed 100 % bioavailability for arsenic from CCA impacted soil.

The adoption of 100% bioavailability as a default value for the ingestion of arsenic from CCA-treated wood or from soil containing arsenic will clearly lead to an overestimate of the actual amount arsenic actually absorbed and when incorporated into the risk assessment model will in turn lead to an overestimation of risk.

Dermal absorption from soil was examined in a study involving Rhesus monkeys. Two forms of arsenic were administered in this work. Soluble arsenic and arsenic from the surface of CCA-treated wood. An average of 3.6% of the soluble arsenic was absorbed as compared with only 0.01% of the arsenic from CCA –treated wood (Lowney 2003). A 2-3% value for dermal absorption is commonly employed in risk assessment models which again will lead to an overestimation of the risk.

A urinary biomonitoring study of children would overcome the uncertainty of the bioavailability of arsenic from surface residues or soil from CCA-treated wood and both Arch Chemicals (Sharma 2003) and the US EPA (Housenger 2003) are planning to conduct this exercise in the near future.

Cancer Risks

Arsenic carcinogenicity is considered the most sensitive endpoint for use in risk assessments of CCA-treated wood.

Cancer risks are characterised as the incremental probability that an individual will develop cancer during his or her lifetime due to chemical exposure under the specific exposure scenarios evaluated in the human health risk assessment. The term “incremental” implies the risk of above the background cancer risk experienced by all individuals in the course of daily life. Approximately one in four New Zealanders die of cancer, so the background cancer risk is 0.25, or 250,000 in one million. The incremental risk is a measure of the additional estimated cancer risk due to a specific exposure. Cancer risks are expressed as a unitless probability (e.g. one in a million, or 1×10^{-6}) of an individual developing cancer over a lifetime, above background risk, as a result of exposure to arsenic associated with CCA-treated wood.

In the US excess cancer risks for the exposure pathways (oral ingestion, dermal absorption and inhalation) evaluated in the human health risk assessment are calculated using intake estimates (lifetime average daily doses), and cancer slope factors (CSF). Estimated intakes and cancer slope factors are combined to calculate excess cancer risk according to the following equation:

$$\text{Cancer risk} = \text{Intake (mg/kg.day)} \times \text{CSF(mg/kg.day)}^{-1}$$

The CSF is an upper bound estimate of carcinogenic potency used to calculate risk from exposure to carcinogens by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over their lifetime. The CSFs recommended by the US EPA are conservative upper bound estimates, which means that the EPA is reasonably confident that the “true” cancer risk does not exceed the estimated risk based on the CSF, and may be as low as zero. The US EPA-recommended CSF value for arsenic is $1.5 \text{ (mg/kg.day)}^{-1}$ but this value is subject to considerable controversy particularly in the context of low level exposure to arsenic (see below).

The estimated cancer risks are compared to the US EPA’s “acceptable cancer risk range” of 1×10^{-6} to 1×10^{-4} (A cancer risk of 1×10^{-6} represents 1 case of cancer in every 1,000,000 population and a cancer risk of 1×10^{-4} represents 1 case of cancer in every 10,000 population).

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 1 in 100,000.

The definition of an acceptable risk in the US federal government is not a single precise value, but rather this range of values (1×10^{-6} to 1×10^{-4}) that allows the selection of an acceptable risk within this range based on a number of considerations. However, based on a review of the published literature and several federal regulatory decisions, it is clear that cancer risks associated with US EPA-approved site remediation, and air and drinking water standards often exceed this range (Gradient Corporation 2001).

The primary source of evidence that inorganic arsenic in drinking water is associated with increased mortality from cancer at internal sites (bladder, liver, lung, and other organs) is a large ecologic study conducted in regions of Southwest Taiwan endemic to Blackfoot disease. This study has served as the basis of the quantitative risk analysis by both the US EPA and the US National Research Council for arsenic in drinking water.

A number of concerns have been raised including the adequacy of the model used by the US EPA and the accuracy and reliability of the exposure data (Brown et al. 1997); a number of host and environmental factors among the Taiwanese not applicable elsewhere (Carlson-Lynch et al. 1994); a possible threshold for arsenic carcinogenicity and nonlinearities in the dose-response curve (Abernathy et al. 1996; Slayton et al. 1996); differences in health and nutrition between Taiwan and the United States that might increase cancer risk in Taiwan (Beck et al. 1995); the possibility that arsenic is an essential nutrient at lower doses (EPA 1988; NRC 1999); and the possibility of significant exposure to arsenic from sources other than the well water (Chappell et al. 1997). These factors, many of which were recognized by the US EPA at the time of the assessment, all contribute to significant uncertainty in the risk assessment.

In view of these concerns, estimates of risk based on a CSF derived by using the Taiwanese data, such as those of the US EPA and WHO (1996), need to be interpreted with caution. Whilst adequate for risk assessment of the oral intake of “high” levels of inorganic arsenic in water, their relevance to risk at the comparatively low arsenic concentrations found in CCA-treated timber is less clear. Valberg et al.

(1997), for example has questioned also the relevance of using toxicity factors derived from studies of ingestion of high arsenic-containing drinking water to assess toxicity of arsenic in soil.

It is therefore apparent that the choice of exposure assumptions and parameters can have a dramatic effect on the estimated risks. The estimated cancer risks for exposure to CCA-treated wood conducted by several organisations have been summarised in the Read (2003) report:

Cancer risk estimates for children exposed to dislodgeable arsenic residues

| Risk assessor(s) | Risk estimates |
|-----------------------------|-----------------------------------------------------------------------------------------------------------------------|
| CDHS (1987) | $1 \times 10^{-4} - 6 \times 10^{-3}$ (skin) |
| CPSC (1990) | $3-4 \times 10^{-6} - 8-9 \times 10^{-6}$ (skin) $2 \times 10^{-6} - 1 \times 10^{-4}$ (lung/bladder) ¹ |
| Roberts and Ochoa (2001) | $4 \times 10^{-6} - 1 \times 10^{-3}$ (skin) |
| Gradient Corporation (2001) | $9.6 \times 10^{-7} - 1.5 \times 10^{-6}$ (skin) |
| EWG (2001) | 2×10^{-3} (lung/bladder) |
| Maas et al (2002) | 1.2×10^{-3} (lung/bladder) ² |
| CPSC (2003) | $2 \times 10^{-6} - 1 \times 10^{-4}$ (lung/bladder) $2 \times 10^{-7} - 5 \times 10^{-3}$ ³ |

¹In 2003 CPSC revised the estimates using the EPA and NRC unit cancer risks.

²Based on 1 in 850 not the incorrect 1 in 180 stated in the report.

³Risk estimates from a sensitivity analysis.

These estimates vary widely reflecting the exposure parameters chosen. 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 (Read 2003).

Uncertainties associated with evaluating exposure to dislodgeable arsenic include residue concentrations, relative oral and dermal bioavailability from residues, residue ingestion rate, skin exposure area, surface area for hand-to-mouth contact, and exposure frequency and duration. Different combinations of alternate assumptions (which act multiplicatively) can affect the total risk by an order of magnitude or more. For example, selection of alternate assumptions for unit risk and bioavailability in the CPSC (2003) risk assessment would reduce the high end risk of 10^{-4} to risks ranging from 3×10^{-7} to 8×10^{-6} , i.e. well within the “acceptable cancer risk range” for the US EPA (Beck, 2003) and tolerable risk for carcinogens of less than 10^{-5} by New Zealand regulatory agencies.

An examination of the USA national cancer statistics regarding lung/bronchus and bladder cancer (the two cancers of interest in the risk assessment of CCA) incidence for males and females, ages 39 years or less, from 1973 to 1998 show either stable or, in one case (lung/bronchus in males ages 35 - 39 years), decreasing cancer rates over

time. Thus, there is no evidence of increasing lung/bronchus or bladder cancer in populations exposed to CCA-treated wood as young children (Beck 2003).

Background inorganic arsenic exposures in children.

Understanding where arsenic exposures fall with respect to natural background is important for communicating the relative magnitude of arsenic exposure and risk in relation to everyday exposures. Arsenic is naturally ubiquitous in the environment.

The primary background sources of inorganic arsenic to the general population are food, water, and to a lesser extent, soil. In the US using reported inorganic arsenic data for food types and the USDA food intake survey for children ages 1-6 years, the probabilistically modelled average inorganic arsenic intake was 3.2 $\mu\text{g}/\text{day}$ (95th percentile of 6.2 $\mu\text{g}/\text{day}$; 99th percentile of 9.5 $\mu\text{g}/\text{day}$). Foods contributing the most inorganic arsenic were rice, other grains, and fruit. One bowl of rice (1 cup cooked) contributes nearly 4 μg of arsenic. Inorganic arsenic intake by Asian or other populations with much greater rice consumption compared with the typical US population would be considerably higher (e.g. 14 $\mu\text{g}/\text{day}$ for Japanese adults versus 3.2 $\mu\text{g}/\text{day}$ in US adults). At an average arsenic level in drinking water in the US around 1-2 $\mu\text{g}/\text{L}$, drinking water adds an additional 1-2 $\mu\text{g}/\text{day}$ assuming 1 L/day consumption. At the EPA's drinking water standard for arsenic (10 $\mu\text{g}/\text{L}$) this would contribute 10 $\mu\text{g}/\text{day}$. By comparison ingestion of soil containing arsenic at a background level of 20 mg/kg would result in a reasonable maximum estimate of 2 $\mu\text{g}/\text{day}$ (assuming 50 percent relative bioavailability and an average that is below 1 $\mu\text{g}/\text{day}$ (Tsuji 2003).

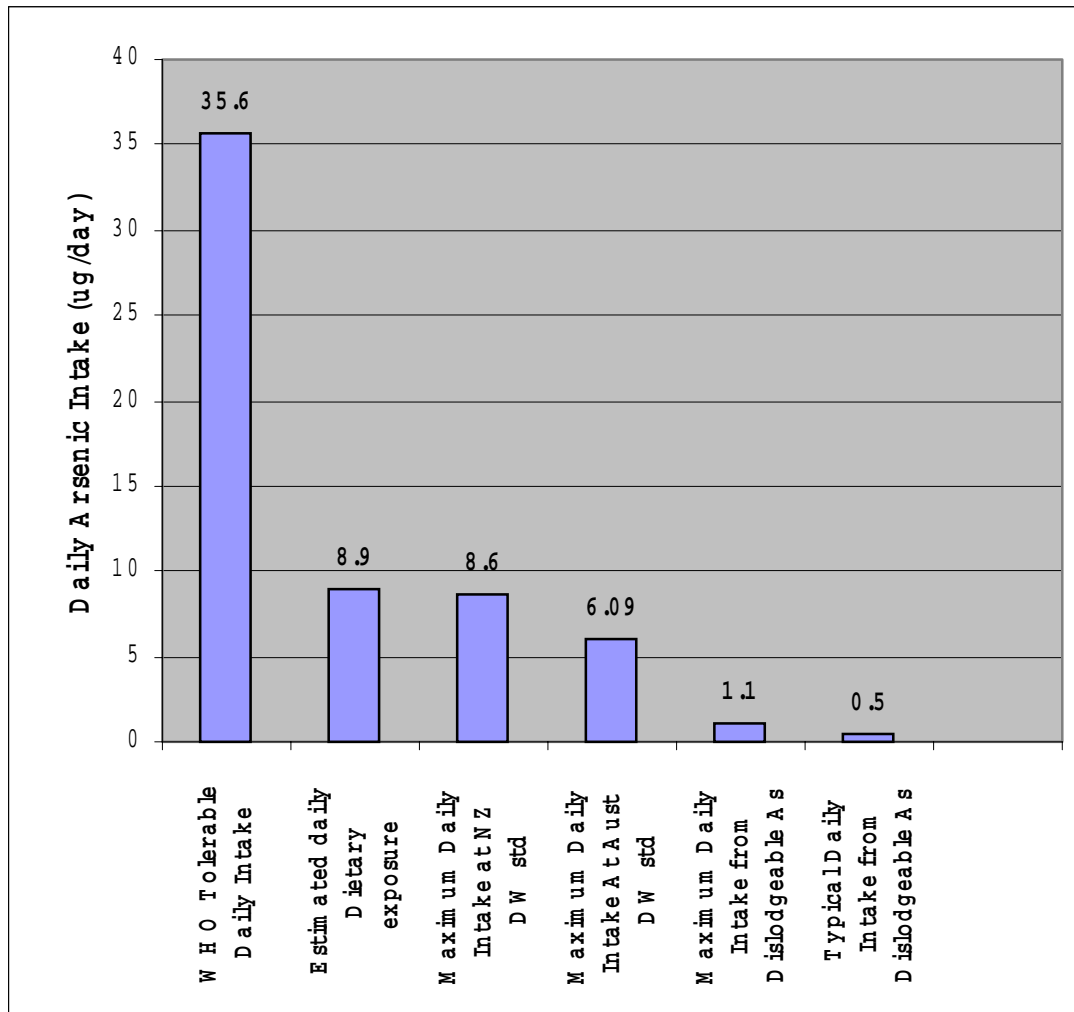
Epidemiological studies show no evidence of adverse health effects in United States populations with elevated arsenic drinking water or soil levels. (Exponent 2002)

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 $\mu\text{g}/\text{kg}$ body weight per 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 WHO Joint Expert Committee on Food Additives (JECFA) provisional tolerable weekly intake (PTWI) of 15 $\mu\text{g}/\text{kg}$ bodyweight/week for oral exposure to inorganic arsenic (Vannoort 2000). Similar estimates can be derived for background intake from food in Australia using data reviewed by WHO (2001), with estimated intakes for children of 17 $\mu\text{g}/\text{day}$ total arsenic equivalent to about 7 $\mu\text{g}/\text{kg}$ body weight per week.

The New Zealand drinking water guidelines have a provisional MAV for arsenic of 10 $\mu\text{g}/\text{L}$ (similar to the US). ESR data for arsenic in drinking water give a mean concentration of 2 $\mu\text{g}/\text{L}$ and a maximum of 6.9 $\mu\text{g}/\text{L}$ (Read 2003). Intake from water in Australia would be comparable to that in New Zealand. The Australian drinking water guideline for arsenic is 7 $\mu\text{g}/\text{L}$, with typical values in the Australian reticulated water of less than 5 $\mu\text{g}/\text{L}$ (NHMRC/ARMCANZ, 1996).

Thus, total background arsenic intakes are available for placing arsenic exposures and risk in context with naturally occurring sources.

Comparison of inorganic arsenic doses for children aged 2-6



Notes:

- 1) Tolerable Daily Intake of 2 µg/kg bodyweight/day set by JECFA and using a bodyweight of 17.8 kg based on US EPA data for mean bodyweight of boys and girls age 2-6.
- 2) Estimated daily dietary exposure of arsenic at 25% of TDI.
- 3) Arsenic intake from drinking water is based on the current New Zealand (10 µg/L) and Australian (7 µg/L) drinking water guidelines for arsenic and was calculated using US EPA-recommended mean drinking water intake rate for children ages 3-5 (0.87 L/day).
- 4) Soil arsenic intake is based on ingestion and dermal exposure to arsenic in soil according to the Gradient (2001) risk assessment model.

This graph indicates that estimates of daily inorganic arsenic intake from CCA-treated wood for children are significantly less (17 times less) than the daily intake of inorganic arsenic from food and from drinking tap water at the current drinking water standards for arsenic.

The combined intake of arsenic from all these sources falls well below the WHO tolerable daily intake for arsenic.

When viewed in this perspective it is clear that exposures to arsenic associated with CCA-treated wood in domestic and playground settings do not pose a significant health risk to children

RISK ASSESSMENT AND COMMUNICATION

Society demands, to an increasing extent, control of the potential hazards to humans and the environment by industrial chemicals and is becoming increasingly well informed and anxiety prone about technology associated risks. The logical and traditional approach is to first estimate the risk, a scientific step. Then follows the issue of risk acceptance, a most difficult step – moving from the world of facts to the world of values. The risk decision ultimately represents the social climate of the country in which it is made. Public perception of risk is often markedly at variance with the estimates given by professional scientists.

Since there is no risk-free world and resources are limited, society must set priorities based on cost effectiveness in order to save the greatest number of lives. In 1991 the US EPA projected that the cost to society of environmental regulations in 1997 would be about \$140 billion per year (about 2.6% of the GNP) (US EPA 1991). Several economic analyses have concluded that current expenditures are not cost effective and resources are not being used so as to save the greatest number of lives per dollar. One estimate is that the US could prevent 60,000 deaths per year by redirecting the same dollar resources to more cost effective programmes (Tengs 1995). For example, the median toxin control programme costs 146 times more per life-year saved than the median medical intervention (Tengs 1995). This difference is likely to be even greater because cancer risk estimates for toxin control programmes are worst case, hypothetical estimates, and the true risks at low dose are often likely to be zero. Worst case assumptions in risk assessment represent a policy decision, not a scientific one.

Risk assessments are conducted on the basis of assumptions. In reporting results, assumptions should be clearly stated and their validity should be examined carefully to prevent confusion and unnecessary adverse impacts on society.

Recently, a report on an assessment of the cancer risk associated with consumption of oysters caused a panic among consumers in Taiwan and produced significant effects on related industries. A group of researchers measured the arsenic content in oysters in the Taiwan area and conducted a cancer risk assessment accordingly. The results, published in a research article in an international journal (Guo, 2002), included a lifetime cancer risk estimate of 5.1×10^{-4} calculated based on the assumption that a person consumes oysters with the highest arsenic level (19.3 mg/g dry weight) at the highest rate (139 g/day) for 30 years. A national newspaper in Taiwan translated part of the article and published results that focused on the finding that this estimate was more than 500 times higher than what would be considered acceptable by the US EPA. As a result, most consumers stopped purchasing oysters, and the related industries suffered substantial losses. The newspaper's omission of the key assumptions in the risk assessment and the extreme assumptions made in the risk assessment led to this tragedy. This event demonstrated the importance of careful communication of risk assessment results.

Aaron Wildavsky (1995) discusses worst-case risk assessment in his book - *But Is It True?: A Citizen's Guide to Environmental Health and Safety issues*.

“We should be guided by the probability and extent of harm, not by its mere possibility. The search for possibilities is endless and it trivialises the subject. There is bound to be a great diversion of resources without reducing substantial sources of harm. Consternation is created but health is not enhanced.....

Weak causes are likely to have weak effects. Our search should be for strong causes with palpable effects like cigarette smoking. They are easier to find and their effects are much more important to control.... The past necessity of proving harm has been replaced by a reversal of causality: now the individuals and businesses must prove that they will do no harm.”

In the context of possible adverse health effects through exposure to CCA treated wood, the scientific evaluation is clear – there simply is no significant risk. Any attempt to quantify possible cancer risks from exposure to CCA-treated timber are incongruous when viewed against background exposures to arsenic.

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Appendix A

The 19th Annual International Conference on Soils sediments and Water, October 20-23, 2003 University of Massachusetts, Amherst.

CCA Treated Wood –Regulations, Science and Risk Assessment

CCA and Regulatory History

Dr. Raj Sharma, Arch Chemicals, Inc., Norwalk, CT.

XFAS Analysis of CCA Dislodgeable Material and Residues on Wood

Dr. Peter Nico, California State University, Stanislaus, Turlock, CA.

As and Speciated Cr Levels in Soil Surfaces Near Treated Wood Playscapes.

John H. Butala, M.S., Toxicology Consultants, Inc., Gibsonia, PA.

Do Arsenic-Containing Products Influence Arsenic Concentrations in Subsurface Drinking Water Supplies?

Dr. Jennifer K. Saxe, Gradient Corporation, Cambridge, MA.

Hand-Wipe Sampling of CCA-Treated Wood with Comparison to CPSC Hand-Wipe Study.

Dr. William F. Gutknecht, Research Triangle Institute, Research Triangle Park, NC

In Vivo Relative Oral Bioavailability of Arsenic from CCA-Affected Soils and Dislodgeable CCA in the Swine.

Dr. Stan W. Casteel, University of Missouri, Columbia, MO.

In Vivo Relative Oral Bioavailability of Arsenic from CCA-Affected Soils in the Primate.

Dr. Stephen M. Roberts, University of Florida, Gainesville, Florida.

USEPA SHEDS Model: Methodology for Exposure Assessment.

Dr. Valerie G. Zartarian, USEPA, Office of Research & Development, Boston MA

CPSC: Risk Assessment of CCA-Treated Wood

Dr. Kristena Hatlelid, US Consumer Product Safety Commission, Bethesda, Maryland

Comparison of a Probabilistic/Mechanistic Method (SHEDS) to a Deterministic/Empirical Method for Evaluating Exposures to CCA Treated Wood

Dr. Barbara D. Beck, Gradient Corporation, Cambridge, Massachusetts

Background Exposures to Inorganic As

Dr. Joyce S. Tsuji, Exponent, Bellevue, Washington

Arsenic Epidemiology USA Data

Dr. Floyd J. Frost, Jr., Lovelace Respiratory Research Institute Albuquerque, New Mexico

Arsenic Epidemiology & Cancer Slope Factor

Dr. Steven H. Lamm, Consultants in Epidemiology and Occupational Health, Inc., Washington, D.C.

Appendix B

Dr Jack E Housenger, Associate Director, Antimicrobials Division, Office of Pesticide Programs, US EPA.

Dr Norman J Cook US EPA