HEALTH EFFECTS OF PRESERVED WOOD: RELATIONSHIP BETWEEN CCA-TREATED WOOD AND INCIDENCE OF CANCER IN THE UNITED STATES

Daniel C. West, MD

Associate Professor of Pediatrics

University of California, Davis

Correspondence to:

Daniel C. West, MD

Department of Pediatrics

UC Davis Medical Center

2516 Stockton Blvd.

Sacramento, CA 95817

Email: dcwest@ucdavis.edu

Phone: 916-734-2428

Fax: 916-734-0342

Introduction

There is growing concern about the health effects of exposure to preserved wood due to arsenate, a form of arsenic found in the chemical CCA (Copper Chromium Arsenate), which until recently was used to preserve the wood. It has been shown that CCA in preserved wood can rub off onto hands when wood structures are touched and leach from the wood into surrounding soil after exposure to rain and water.[1-3] Humans could potentially ingest CCA by transferring it from their hands to their mouth or by consuming contaminated soil. Children are particularly at risk because they frequently play on structures made from CCA-preserved wood. In addition, children demonstrate behaviors, such as increased hand to mouth activities that might increase the likelihood of ingestion.[4]

Although arsenic exposure has been linked to a variety of adverse health effects, the greatest concern is the risk of developing specific types of cancer later in life. For instance, there is epidemiological evidence that chronic ingestion of water contaminated with arsenic significantly increases the risk of developing lung and urinary bladder cancer about 15-20 years after exposure.[5-9] In the case of CCA-preserved wood, there are no epidemiological studies to indicate whether routine exposure to CCA in the wood is associated with similar risk. In the absence of such evidence, a number of investigators have attempted to estimate the risk by extrapolating information known about arsenic in drinking water. Risk calculations from these studies vary widely from as high as 1 case of cancer per 500 children chronically exposed for 5 years to CCA-preserved wood to as low as 1 case per 1,000,000 exposed children.[10, 11]

Given these disparate cancer risk calculations and the lack of direct epidemiological evidence, we sought to determine whether exposure to CCA-preserved wood is associated with an increased risk of developing cancer. As a first step towards answering this question, we

reasoned that millions of children have played on structures made of CCA-preserved wood since this product was first introduced to the U.S. market 30-35 years ago. We hypothesized that if exposure to CCA-preserved wood increased the risk of cancer, then the rate of arsenic-related cancers should be significantly higher in young adults today, who were potentially exposed to CCA-preserved wood structures as a child, than it was in young adults 30 years ago, prior to the widespread use of CCA-preserved wood.

Methods

We used cancer incidence data from the Surveillance Epidemiology and End Results 9 (SEER 9) registry maintained by the National Cancer Institute. At the time of this study the registry contained complete data on cancer incidence from 1975-1999 from 9 geographic regions of the United States. The 9 geographic regions included Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, and Utah. These areas were chosen to be included in the SEER registry because of their ability to operate and maintain a high quality population-based cancer registry and their epidemiologically significant population sub-groups. Quality control measures were in place to ensure the accuracy of data collected by the SEER registry. The SEER program is considered the standard for quality among cancer registries throughout the world.

Because CCA-preserved wood came into widespread use in the United States in the 1970s and there is a known 15-20 year latency period between exposure to arsenic and the development of arsenic-related cancer, we reasoned that 20-29 and 30-39 year old individuals between 1995-1999 were potentially exposed to CCA-preserved wood as a child. The same age individuals during the period 1975-1979 could not have been exposed. Therefore, to test our

hypothesis we compared the incidence of arsenic-related cancer in the 20-29 and 30-39 year old population in 1975-1979 (i.e. unexposed group) with the same age groups in 1995-1999 (i.e. exposed group).

We determined the incidence of all types of lung and bronchus cancer and urinary bladder cancer during the time period and in age groups described above. In addition, we separately identified the incidence of the two most common sub-types of lung cancer, Squamous Cell Carcinoma and Adenocarcinoma. We also determined the change in incidence of all lung and bronchus cancers, Squamous Cell Carcinoma and Adenocarcinoma subtypes, and all urinary bladder cancers each year from 1975-1999, the period of potential exposure to CCA-preserved wood.

We analyzed these data using SEER Stat 4.0 provided by the National Cancer Institute. We used linear regression to analyze the change in annual incidence over time. A P value < 0.05 was considered significant.

Results

Lung Cancer

We found that the incidence of all types of lung and bronchus cancer among 20-29 year old population between 1995-99 was unchanged compared with that during 1975-1979 (0.4 cases/10^5 persons)(Table 1). In the 30-39 year old population the incidence of lung cancer decreased (3.3 cases/10^5 persons [1995-1999] vs. 4.7 [1975-1979])(Table 1). We analyzed the incidence of specific sub-types of lung and bronchus cancer and demonstrated similar findings (Table 2). The incidence of both Squamous Cell Carcinoma and Adenocarcinoma of the Lung and Bronchus were not different in the 20-29 year old age group in 1995-1999 compared with

1975-1979. In the 30-39 year old population the incidence of Squamous Cell Carcinoma in 1995-1999 was less than that in 1975-1979 (0.2 cases/10^5 persons [1995-1999] vs. 0.9 [1975-1979]). Likewise, the incidence of Adenocarcinoma in the 30-39 year old group in 1995-1999 was less than that reported in 1975-1979 (1.4 cases/10^5 persons [1995-1999] vs. 1.9 [1975-1979]).

Urinary Bladder Cancer

We also compared the incidence of all types of urinary bladder cancer (Table 1). We found that the incidence of urinary bladder cancer was less in 1995-1999 in both age groups (20-29 year olds, 0.5 cases/10^5 persons [1995-1999] vs. 0.6 [1975-1979]; 30-39 year olds, 1.4 cases/10^5 persons [1995-1999] vs. 1.7 [1975-1979]). No data was available to compare the relative incidence of sub-types of urinary bladder cancer.

Annual Percentage Change Over Time

We also compared the annual percentage change in the incidence of all types of lung and bronchus cancer and urinary bladder cancer and sub-types of lung and bronchus cancer in both at risk age groups from 1973 to 1999 (Table 3). In the 20-29 year old age group there was no significant change in incidence for lung and bronchus cancer and reported sub-types. Urinary bladder cancer demonstrated a statistically significant decrease of 1.2 percent each year. In the 30-39 year old age group all lung and bronchus cancer and subtypes showed statistically significantly decreases each year (range 1.4 to 6.2 annual percent). We found a similar decreased incidence of urinary bladder cancer each year in the 30-39 year old group (1.4 annual percent).

Discussion

About 30 years ago CCA-preserved wood was introduced to the U.S. market and since that time potentially millions of children have been exposed to CCA by playing on wood

structures made from this product. With this fact in mind, we hypothesized that if exposure to CCA-preserved wood as a child increased the risk of cancer later in life, then the incidence of arsenic-related cancers in the U.S. should be greater now than it was prior to the use of CCA-preserved wood. On the contrary, we found that the incidence of arsenic-related cancers in adults who could have been exposed to CCA-preserved wood as children was the same or less than it was prior to the widespread use of CCA-preserved wood. Furthermore, we found that the incidence of arsenic-related cancers in potentially exposed individuals has not changed or has decreased each year since the introduction of CCA-preserved wood to the US market. These findings do not support our hypothesis and provide preliminary evidence suggesting that routine exposure to CCA-preserved wood does not increase the risk of developing arsenic-related cancer.

Although to our knowledge there are no other epidemiological studies that attempt to directly measure the association between CCA-exposure and the development of cancer, several groups of investigators have investigated the risk of developing cancer after long-term ingestion of arsenic in drinking water.[5-9] In contrast to our findings, high levels of arsenic in drinking water have been associated with the development of lung and urinary bladder cancers. Perhaps more concerning is that long-term exposure to even relatively low levels of arsenic in drinking water appears to substantially increase the risk of developing urinary bladder cancer. The contrast between these findings and those in our study can be explained, at least in part, by differences in the bioavailability of CCA in preserved wood compared with arsenic in drinking water. For instance, CCA from preserved wood is much less soluble than arsenic in drinking water, and therefore one would expect CCA to be absorbed less efficiently from the gastrointestinal track.

Several groups of investigators have performed risk calculations in an effort to estimate cancer risk of children who play on structures made from CCA-preserved wood.[10-12] Results vary widely from as high as 1 cancer case per 500 exposed children to as low as 1 case in 1,000,000 exposed children, well within the range considered safe by the US Environmental Protection Agency. The reasons for this variability are primarily due to differences in critical assumptions that are required to perform the calculations. For example, calculated risk varies depending on assumptions made by the investigator regarding the extent and duration of exposure to the wood, the efficiency with which CCA rubs off onto children's hands and is transferred into the mouth and then ingested, and the extent to which ingested CCA is absorbed from the gastrointestinal track. Our findings provide preliminary epidemiological evidence supporting risk assessments suggesting that routine play on wood structures made from CCA-preserved wood does not increase cancer risk.

Our study was designed to be preliminary, and therefore, has several limitations that must be considered. We used only data collected from the 9 geographic areas included in the 1975-1999 SEER database. Therefore, it is possible that the population represented in the SEER registry is not representative of other regions of the United States. However, this seems unlikely since the population covered by the SEER registry is comparable to the population of the entire United States with regard to education, socio-economic status, and ethnicity. In addition our study did not directly measure the population's exposure to CCA-preserved wood or other potential confounding variables, such as smoking rates. Therefore, we cannot exclude the possibility that some unmeasured confounding variable unique to the SEER registry population is responsible for our observations. More research that directly measures exposure and controls for potential confounding factors is needed to confirm our findings.

We conclude that the incidence of cancer known to be associated with arsenic exposure is either unchanged or decreased in age groups that would have been exposed to CCA-preserved wood structures during childhood. The rate of arsenic-related cancers has been the same or decreasing over the time in which CCA preserved wood has been sold in the United States. Thus, these data provide preliminary evidence suggesting that there has not been an increase in arsenic-associated cancers during the period of extensive use of CCA-preserved wood in the United States.

Table 1. Incidence of Cancer in Individuals Who Could Have Been Exposed To CCA-Treated Wood At Least 15-20 Years Ago.

CANCER TYPE	AGE RANGE	1975-1979*	1995-1999*
Lung and Bronchus	20-29 year old	0.4	0.4
	30-39 year old	4.7	3.3
Urinary Bladder	20-29 year old	0.6	0.5
	30-39 year old	1.7	1.4

From the SEER Cancer Incidence Public-Use Database 1973-1999
*Expressed as incidence per 100,000 persons.

Table 2. Incidence of Specific Types of Lung Cancer In At Risk Population

CANCER TYPE	AGE RANGE	1975-1979*	1995-1999*
Squamous Cell Carcinoma of Lung and Bronchus	20-29 year old	0.0	0.0
	30-39 year old	0.9	0.2
Adenocarcinoma of Lung and Bronchus	20-29 year old	0.1	0.1
	30-39 year old	1.9	1.4

From the SEER Cancer Incidence Public-Use Database 1973-1999
*Expressed as incidence per 100,000 persons.

Table 3. Estimated Annual Percentage Change In Cancer Incidence In The At Risk Population Over Time

CANCER TYPE	20-29 YEAR OLD*	30-39 YEAR OLD*
All Lung and Bronchus	0.6	-1.7**
Squamous Cell Carcinoma of Lung and Bronchus	0.0	-6.2**
Adenocarcinoma of Lung and Bronchus	0.0	-1.4**
Urinary Bladder	-1.2**	-1.4**

From the SEER Cancer Incidence Public-Use Database 1973-1999
*Estimated annual percentage change over time.
**Change is significantly different from zero ($P<0.05$)

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