UNITED STATES GOVERNMENT

MEMORANDUM

U.S. CONSUMER PRODUCT SAFETY COMMISSION
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SUBJECT: Estimating the risk of skin cancer from ingested inorganic arsenic.

I. Introduction

A procedure for estimating the average maximum ingestion exposure of children to dislodgeable arsenic from wood playground equipment was developed in a previous memo (CPSC, 1989). The present memo develops an assessment in order to estimate the risk involved with certain levels of arsenic ingestion. Dose-response relationships from human exposure data will be modeled to estimate the risk of skin cancer.

Chronic arsenic ingestion in humans has been associated with skin lesions (lightened or darkened patches of skin, hyperkeratosis, cancer), cancer of major internal organs (bladder, liver, and lung cancers), gastrointestinal distress (chronic diarrhea, vomiting), vascular degenerative alterations (generalized intimal thickening, Blackfoot disease), cardiac manifestations (lengthened QT intervals, non-specific T-wave changes, infarction), and neurological problems (weakness, paresis, aphasia, convulsions, coma) (CPSC, 1988; ATSDR, 1988; EPA, 1982, 1987, 1988; Zaldivar, 1974; Chen 1986, 1988). Of these adverse reactions, appropriate data for use in risk assessment models applicable to low dose exposures exist only for skin lesions.

Dermatological terms used to describe adverse skin effects of arsenic are as follows. "Skin cancer", as it pertains in this document, refers to malignant skin tumors, such as basal cell carcinoma, "intraepidermal carcinoma" (Fierz, 1965), or squamous cell carcinoma, but not melanoma. Arsenic induced skin cancer can arise from apparently normal cells (de novo) or may transform from keratotic lesions (Shannon, 1989). "Keratosis" or "hyperkeratosis" is an excessive thickening of the skin, similar to a corn, but not a callus. Arsenical keratosis is considered a precancerous condition by some (Sutherland, 1958; Fierz, 1965; Zaldivar, 1974) although most keratoses do not progress to carcinoma. Intraepidermal carcinoma is sometimes considered a stage of keratosis, according to EPA (1988). "Hyperpigmentation" is the abnormal over-darkening of the skin, and

"hypopigmentation" is its abnormal lightening. The two conditions may also be referred to as "melanoderma" and "leukoderma", respectively.

Skin cancers induced by arsenic histologically resemble those caused by the sun, but the arsenic-induced cancers are more invasive or more likely to metastasize (disseminate) with fatal consequences (Shannon, 1989). Arsenic-induced skin cancer is the toxicological endpoint on which the following risk assessment is based. Assessment of risk based on keratosis is also performed, although this is not a cancerous endpoint.

Arsenic is a Class A carcinogen, a classification for agents having sufficient evidence of carcinogenicity in humans. Airborne arsenic has been strongly associated with lung cancer in humans, and intratracheal exposures to experimental animals have produced tumors (EPA, 1984, ATSDR, 1987). Experimental animal studies have not demonstrated the carcinogenicity of ingested arsenic, although the lifespan of exposed animals was considerably shortened in some studies due to noncancerous causes. The details of those studies will not be presented in this document. Human studies which have relevance to serious health effects due to chronic arsenic ingestion will be discussed. The Taiwan study (section II.A) is used for risk assessment modeling. Other human studies (sections II.B to III.C) contained supplemental information, but were inadequate for use in risk assessment modeling.

II. Epidemiological studies that examined skin lesions

II.A. Taiwan (Tseng, 1968, 1977)

Artesian wells with arsenic levels ranging from 0.01-1.82 ppm were used by people living in villages on the southwest coast of Taiwan. The inhabitants had used these wells for drinking water and irrigation since 1900-1910, ending in 1956 when a new water supply was installed. A 1965 survey of the 40,421 inhabitants found the incidences of skin cancer, hyperpigmentation, and keratosis were respectively 10.6, 183.5, and 71.0 /1000. Gangrene of the extremities known as "Blackfoot disease" had an occurrence of 8.9/1000. This disease is a combination of arteriosclerosis and thromboangiitis obliterans, resulting in the impairment of blood circulation to the limbs. The accuracy of the clinical non-invasive diagnosis of skin cancer was histopathologically confirmed when the patients permitted biopsies to be taken (Yeh, 1973). Over 99% of the patients with skin cancer had multiple skin cancers. The ratios of the incidence of these disorders relative to skin cancer (=1) is 17.3 for hyperpigmentation, 6.7 for keratosis, and 0.84 for Blackfoot disease. Males were 2.9 times more likely to have skin cancer than females.

Nearby villages had wells with 0-0.017 ppm arsenic and were used as a control population. Of the 2552 persons in the control population that were examined, there were no cases of pigmentation changes, keratosis, or skin cancer. The age and sex distribution of the control population was the same as for the exposed populations.

Three categories of arsenic levels in water-- low, middle, high --were arbitrarily set at 0-0.29, 0.30-0.59, and >0.60 ppm. As the category of arsenic in water increased, the incidence of the skin disorders increased. For example, the incidences in the low, middle, and high categories were respectively 1.5, 4.3, and 22.4 /1000 males in the 20-39 yr age range. The intake of arsenic from other sources, such as from crops grown with the contaminated well water, was not recorded or estimated.

Length of exposure, as suggested by age, was a factor in increasing the incidence of hyperpigmentation, skin cancer, keratosis, and Blackfoot disease. For example, in the low arsenic in water category, the skin cancer rates were 1.5, 6.5, and 48.1 per 1000 males in the 20-39, 40-59, and 60+ yr age ranges. This suggests that long term exposure to arsenic was necessary to produce the skin and vascular disorders and/or that the disorders have a long latency period.

Analysis of the well water revealed the presence of fluorescent chemicals which were tentatively identified as ergotamine, ergocalciferol, and lysergic acid (Igrolic, 1982). Other unidentified alkaloids were also present. Ergot alkaloids are known to cause vascular disorders that can lead to gangrene (Reddy, 1989). This casts suspicion on attributing the Blackfoot disease solely to arsenic. Although arsenic ingestion has been associated with the same disorder (Zaldivar, 1974), it is not possible to determine whether the alkaloids and/or arsenic were responsible for Blackfoot disease in the Taiwanese villagers.

Speciation of metal ions in the water was analyzed (Igrolic, 1982). Pentavalent (arsenate, +5) arsenic comprised 88-91% of the total arsenic according to the hydride technique. The rest of the arsenic was trivalent (arsenite, +3). The possible differences in the carcinogenicity of the two arsenic species is discussed in section V. The Taiwan data is used for modeling in the present risk assessment.

II.B. Zurich (Fierz, 1965)

Fowler's solution was an arsenical drug prescribed for various skin disorders, such as acne, pemphigus, psoriasis, neurodermatitis, eczema, and seborrhea. It contained 1/2% arsenic trioxide (As_2O_3) and was administered as a 1:1 v/v

solution. This is equivalent to 3.75 g of trivalent arsenic per liter. A review of 262 patients at the City Polyclinic for Skin and Venereal Diseases of Zurich, who had taken Fowler's solution as treatment for various skin disorders, found that patients had developed hyperpigmentation, keratosis, and skin cancer. Hyperpigmentation was found in only 5 patients, 1 of whom had chloasma [hormonally related hyperpigmentation not associated with arsenic] (4/262 = 1.5%). No data were presented indicating that there might have been a dose related relationship for hyperpigmentation.

Keratosis was seen in 106 (40.4%), particularly on the palms of the hands and soles of the feet. A relationship between the amount of Fowler's solution consumed over the lifetime of treatment and the incidence of keratosis was observed. The shortest latency (time to appearance) was about 2.5 yr. An average latency or range was not stated.

Skin cancer occurred in 21 (8%), with the most common form being basal cell carcinoma. The average latency was 14 yrs (range 6-20). The patients were widely distributed among the dose range from 10-2600 ml of Fowler's solution (37.5-9750 mg arsenic). The length of time that the patients ingested Fowler's solution ranged from 6-26 years. Other possible sources of arsenic exposure, such as in food or water, were not mentioned by Fierz. The Fierz data was not used for risk assessment modeling because of the lack of control groups and multiple dose-response points.

II.C. Lane County, OR (Morton, 1976)

The Eugene-Springfield area in the Willamette Valley of Oregon has drinking water wells which have concentrations of arsenic averaging 0.0038-0.033 ppm due to the underlying Fisher formation. The water concentrations were substantially less than the 0.01-1.82 ppm found in the Taiwan study (section II.A). Five percent of the water samples were greater than 0.1 ppm in Lane County, even though the EPA maximum concentration level is currently 0.050 ppm. Basal cell and squamous cell carcinoma skin cancer cases were collected for the years 1958-1971 from the records of a local hospital, 2 dermatologists, a pathologist, and a Portland, OR pathology lab.

Among a population of 190,871 in Lane County, 3,691 cases of non-melanoma skin cancer were found. The overall skin cancer rates for males were 0.675/1000 for squamous cell carcinoma and 0.966/1000 for basal cell carcinoma. The rates for females were lower (0.323/1000 for squamous and 0.795 for basal cell carcinoma), but this was not associated with a difference in arsenic ingestion. These rates are much less than those found in

the Taiwan studies (section II.A). However, the greater frequency of males with skin cancer, compared to females, is apparent in both the Taiwan and Lane County studies.

Districts in the county were divided according to drinking water arsenic levels. No correlation was found between the skin cancer rates and the levels of drinking water arsenic. The authors felt that the lack of correlation was due to the lower concentrations of arsenic than in Taiwan (section II.A). The Lane County data will not be used for risk assessment modeling due to the lack of a dose response correlation.

II.D. Northern Mexico (Cebrian, 1983)

Chronic arsenic toxicosis in northern Mexico was investigated by comparing two rural populations. The water supply in the "exposed" town of El Salvador de Arriba (population 998) had an arsenic concentration of 0.41 ppm (sd=0.11), whereas the "control" town of San Jose del Vinedo (population 1488) had 0.005 ppm (sd=0.007). The arsenic in the water was 70% pentavalent and 30% trivalent. About 1/3 of the households were surveyed (296 in El Salvador, 318 in San Jose) and skin lesions were classified according to the descriptions of Yeh (1973). Skin biopsies were not taken when lesions were found. However, lesions tentatively identified as ulcerative lesions and papular keratoses were found to be carcinoma in later postmortem studies.

The exposed town had a significantly greater overall incidence of cutaneous signs of arsenicism. Males had no greater tendency to be afflicted than females. The rate per 1000 persons for hypopigmentation was 176 (vs. control =22), hyperpigmentation 122 (vs. 19), palm or sole keratosis 112 (vs. 3), papular keratosis 51 (vs. 0), and ulcerative zones 14 (vs. 0). If Cebrian's conclusion that papular keratosis and ulcerative lesions should be considered skin cancers is followed, then the incidence of skin cancer in the exposed town was 64/1000 (vs. 0 in the control town). However, the number of postmortem cases that were examined was not stated, so the validity of considering papular keratosis and ulcerative zones as skin cancer can not be assessed. Therefore, the data will not be used for risk assessment modeling.

The minimum latency was 8 yr for hypopigmentation, 12 yr hyperpigmentation, 12 yr palm or sole keratosis, 25 yr for papular keratosis, and 38 yr for ulcerative lesions. Based on a lifetime exposure of consuming 2.5 L of water for females and 3.5 for males, Cebrian estimated the minimum cumulative lifetime dose of arsenic for hypopigmentation to be 2 g, hyperpigmentation 3 g, palm or sole keratosis 3 g, papular keratosis 8 g, and ulcerative lesions 12 g.

II.E. Antofagasta, Chile (Borgono, 1972, 1977; Zaldivar, 1977a)

A new water system in Antofagasta, Chile (population 137,917) supplied water containing 0.6-0.8 ppm arsenic beginning in 1958. Locally processed beverages became contaminated, such as milk (0.08 ppm), cola (0.20 ppm), and beer (0.55 ppm) (Zaldivar, 1974). Pentavalent arsenic was 98-99% of the arsenic in the water (Irgolic, 1982). Changes in water treatment beginning in May 1970 lowered the arsenic to 0.082 ppm, the weighted average value from June 1970-Mar 1972 (Zaldivar, 1977b).

Investigation into skin lesions which were observed in children in the 1960s led to the comparison of 180 Antofagasta inhabitants with those of a community without exposure to the drinking water system (Borgono, 1972). Hypo- or hyper-pigmentation was observed in 80% and keratosis in 36% of the of the Antofagasta people. The controls had no pigmentation or keratotic lesions. Exposures were not estimated, so it was not possible to determine whether a dose-response relationship existed.

Skin cancer was not reported in the follow-up report (Borgono, 1977). For some cohort members, this may be due to the lengthy period of time required for skin cancer to appear (Cebrian, 1983; Fierz, 1965). Also, in 1970, a water treatment plant was placed into service, which reduced further exposure to arsenic in the water. The arsenic in carbonated drinks dropped from 0.24 ppm in 1968 to 0.06 in 1976. Although the arsenic in hair (0.92 decreased to 0.27 mg/100 g) and nail clippings (2.86 decreased to 1.41 mg/100 g) suggest that exposure to arsenic had decreased in the same time period, the level of arsenic in the water in 1976 was not stated. Of 306 children who were born after the 1970 water treatment had begun and were examined, none had arsenical skin lesions.

A likely reason for the lack of skin cancer cases in Borgono (1977) could be that his diagnoses may not have had histopathological confirmation. Cebrian (1983) found that papular keratoses in an unstated number of postmortem examinations were actually skin cancers. Zaldivar (1977a) surveyed 457 patients from Antofagasta in 1968-1971 and noted 55% had skin lesions indicative of arsenic poisoning. Squamous cell carcinoma did occur, but was not reported as a separate category. Instead, he observed a linear dose response between the daily arsenic dose per kg body weight and "chronic arsenical dermatoses". This category combined all the different types of arsenical skin lesions, so that it could not be used for assessing the risk of cancer-related lesions. The mean daily doses in each 10 yr age cohort (starting with 0-10 yr) were 63.3,

20.2, 16.6, 11.7, 9.5, 7.5, 5.5, 4.3, and 2.2 ug. Respective prevalence rates per 100,000 were 727, 413, 203, 183, 162, 162, 129, 46, and 0.

Zaldivar (1977a) also found that the effects of age were the inverse of that reported by Fierz (1965) and Tseng (1968). In Antofagasta, age was inversely proportional to the incidence of skin lesions. Zaldivar attributed this to the higher dose relative to body weight in the young. The method of determining the dose was stated, but the distribution of arsenic in the diet at each age cohort was not presented. The data also suggest three other possible explanations.

First, the dietary habits of the inhabitants of Antofagasta may be such that as one becomes older, fewer arsenic containing substances are consumed. In the age group 1-10 yr, the incidence rate of arsenic dermatoses is 727/100,000 (Zaldivar, 1977a). The next age group, 11-20 yr (median age 18.6) has a rate of 413/100,000. If it is assumed that the children who experienced arsenic dermatoses had the same median age as all of the children in their respective age cohort, then ingestion of arsenic contaminated dietary substances, perhaps human milk (0.21 ppm; Zaldivar, 1974) or cow milk (0.08 ppm; Zaldivar, 1974), might be suspected due to the 1.7 year median age of the first cohort.

Second, considerable variability in arsenic consumption appears to have occurred in the 1-10 yr age cohort. The mean daily consumption was 0.0633 mg/kg/day, but the standard deviation of 0.0351 was 4-5 times the standard deviations of the other age cohorts. This suggests that outlying data points may have had an overrepresentative influence in determining the mean arsenic consumption, or that there were subgroups which were not represented by the mean.

Third, the children might have been more sensitive to arsenic dermatoses than adults. However, since the dose per body weight was greater in the children and age-specific incidence vs. body weight data was not presented, it was not possible to determine whether age and/or body weight were associated with the decreased incidence of skin lesions.

II.F. A US wood preserving plant (Tabershaw, 1979)

The medical surveillance team for an unspecified wood preserving plant conducted a survey to identify health problems that may have been the result of exposure to CCA (chromated copper arsenate). Workers who had been exposed to other wood preservatives, such as pentachlorophenol and fluorinated chrome arsenic phenol, were excluded from the study. Of the 55 workers undergoing a skin examination, 9 (16.4%) had benign growths, 0 had malignant tumors, and 6 (10.9%) had keratosis. Incidences in

the general US population for the same conditions were respectively listed as 3.8%, 0.6%, and 1.0%. These findings may initially appear to be consistent with precancerous skin conditions caused by arsenic.

However, not all of the conditions within these categories could be considered precancerous. Skin lesion categories for the Tabershaw study were defined by the New York Univ./ Natl. Ctr. for Health Statistics for use in NHANES surveys, according to Butala (1989). "Keratosis" contained actinic keratoses, such as those induced by the sun. It is presumed that the examining physicians were able to distinguish between actinic and early arsenical keratoses. Benign growths were considered skin tags, ordinary nevi, papillomas, lipomas, epitheliomas, and etc. The nature of "etc." growths was not described. Although there were no malignant tumors, a basal cell carcinoma, squamous cell carcinoma, melanoma, eroded ulcer, pearly papule with telangiectasis, or a fungating mass would have fallen under this category (Butala, 1989).

Estimates of CCA exposure were not made so that it was not possible to determine whether the incidences of skin lesions were dose or age-related. It is also not known whether former employees of the plant developed arsenical skin lesions in later life. Therefore, the Tabershaw data will not be used for risk assessment modeling. The results presented by Tabershaw do not indicate that the CCA exposed workers had an abnormal incidence of skin lesions. Because there are lacks of exposure and post-employment data, the study should not be interpreted as demonstrating the lack of CCA carcinogenicity.

II.G. Hawaii wood preserving plants (Gilbert, 1983)

Mortality and morbidity were evaluated for 88 wood preservers from 7 companies in Hawaii and 61 matched controls for the years 1960-1981. None of the wood preservers or controls were female since there were no female employees on the wood treatment lines. The wood treaters had been exposed to CCA, tributyl tin oxide, and pentachlorophenol wood preserving chemicals as well as the solvent carriers for the chemicals. In-depth personal interviews collected data on exposure, frequency of health problems, and personal lifestyle. The median length of work was 80.5 months with 37.5% having >10 years. It was estimated that 26-50% of the workweek had been spent in activities resulting in exposure to the preservatives.

The Univ. of Hawaii Pesticide Hazards Assessment Project lab analyzed urine samples for arsenic, copper, chromium, tin, and pentachlorophenol. The values were normalized to 1000 mOsm/kg to correct for the lack of 24 hr urine collections. (Multiply by osmolarity to convert back to absolute values). Urine metal

levels reflect only recent exposure and may have little significance when taken weeks to months after exposure to the wood preservatives has been terminated. Therefore, the significance of urinary metals may be limited even though urine can be conveniently sampled and analyzed.

The normal range for arsenic in urine was considered to be 67 72 (ppb sd) based on Perry (1959). Twenty percent of both the workers (17/87) and controls (12/61) had above normal urinary arsenic levels. The above normal urinary arsenic levels occurred most frequently in non-Caucasian ethnic groups. Gilbert suggested the Asian populations consume more fish and shellfish, which results in greater [organic] arsenic intake and is reflected by the arsenic excreted in the urine. Dietary data were recorded according to the questionaire administered to persons in the study. However, no data were presented to support the existence of differences in fish and shellfish consumption.

Placing workers who were exposed to different wood preservatives in the same group may possibly dilute the ability to associate possible health effects with exposure to a specific chemical. However, the number of workers who had been exposed to only arsenicals may have been too small to derive meaningful data since the data suggest that the plants have used various preservatives over 1960-1981. No arsenical skin lesions, such as keratosis or carcinoma, were reported and no other clinically significant differences were found by the interviews or standard physiological and blood/urinalysis tests. Although estimates of exposure time were made by Gilbert, the lack of air concentration and skin contact data prevent estimation of actual exposure. Therefore, while the only significant health effect found by Gilbert was hypertension, the report should not be interpreted as supporting the noncarcinogenicity of CCA.

III. Epidemiological studies that examined internal cancers and heart attacks

Inhaled arsenic from occupational exposures may be associated with lung cancer (EPA, 1984). Two recent reports (Chen, 1988; Tsuda, 1989) indicated that ingested arsenic may lead to lung and other internal organ cancers and heart attacks. A third study (Gilbert, 1983) found no excess cancer in occupationally exposed persons, but might suggest that a possible link between arsenic-associated heart attacks and hypertension. Risk assessments for these effects will not be conducted due to insufficient exposure data and the uncertain nature of these studies with respect to linking arsenic exposure with these endpoints.

III.A. Taiwan (Chen, 1988)

Inhabitants of the Blackfoot disease area of Taiwan also suffered an increase in deaths due to cancers of internal organs and heart attack compared to persons in the surrounding unexposed villages. The statistically significant standard mortality ratios (normal =100) were: bladder 3880, kidney 1953, lung 1049, liver 466, colon 381, and cardiovascular disease 209. The relationship of those effects to arsenic exposure is unknown since arsenic exposure data were not reported for the affected and control populations.

III.B. Nakajo-machi, Japan (Tsuda, 1989)

Well water in the village of Nakajo-machi, Japan (population 467), was contaminated with wastes from a nearby factory producing "King's Yellow" (As_2O_3 and flecks of As_2S_3). The inhabitants drank the arsenite-contaminated water from 1954-1959. Twelve cases of squamous or basal cell skin cancer were seen, although none of the skin cancers resulted in death. The 281 residents in the reported study were divided into 3 cohorts, according to the concentration of well water. Standard mortality ratios indicated that two risk factors interacted to cause death by lung cancers and all cancers. First, well water arsenic concentration >0.5 ppm increased deaths due to all cancers (SMR=631, normal=100). Second, smoking increased deaths by lung cancer when well water arsenic was >0.5 ppm (SMR=1873). No cancer deaths occurred in the 0.05-0.5 or <0.05 ppm nonsmoker cohorts. The report suggests arsenic promoted smoking-induced cancers or that arsenic and smoking are co-carcinogens. The study was unable to link arsenic alone with a specific cancer.

III.C. Hawaii wood preserving plants (Gilbert, 1983)

High systolic blood pressure (>140 mmHg) was more frequent among 88 (23%) workers in seven Hawaii wood preserving plants compared to 61 matched controls (7%). In section III.A, Chen (1988) noted increased heart attack deaths in association with drinking arsenic-contaminated water. The risk of serious cardiovascular disease is known to increase with blood pressure (McGee, 1976; EPA, 1985). This raises the hypothesis that arsenic exposure increases blood pressure, which increases the risk of heart attack. There was insufficient data in the Hawaii study to determine whether the exposure to arsenic could be associated with increased blood pressure.

No excess mortality or cancer was found among the wood preserving plant workers compared to controls. Gilbert requested a search from state cancer registry for the names of wood treaters known to have been chronically exposed to wood preservatives for at least 3 months in the years 1960-1981. The

state health department provided death certificates for workers who had died in Hawaii. One urinary bladder cancer and 2 cases of colorectal cancer were found. Four deaths from coronary arteriosclerosis (narrowing of the heart arteries) and 1 death from cerebral thrombosis (clot in a brain vessel) occurred. One death was due to an unknown cause. The cancer morbidity and disease mortality was the same or less than the 8 deaths (4 from cardiovascular disease, 3 from cancer, and 1 from other) anticipated according to a 1969-1971 life table for Hawaiian males (Gilbert, 1983).

IV. Risk assessment modeling

IV.A. Basic assumptions

Four basic assumptions were made to enable the use of the epidemiologic data from Taiwan for risk assessment:

1. Arsenic in the water was the major source of arsenic exposure, although food crops grown with the contaminated water may have contributed substantial arsenic to the diet, as in Antofagasta (Zaldivar, 1974).

2. The incidences of skin cancer were not significantly affected by immigration and emigration in the surveyed area or premature deaths among persons who would have developed skin cancer due to arsenic exposure.

3. The arsenic concentrations measured at the time of the studies were representive of the levels in the water that were drunk by the inhabitants.

4. Ingested trivalent and pentavalent arsenic are capable of producing skin cancer and keratosis and are equipotent species of arsenic (section V).

IV.B. Cumulative model based on Tseng (1968, 1977)

Separate unit risk determinations were done for males and females because of the difference in skin cancer responses between the sexes (Tseng, 1968).

The weighted average arsenic levels in the 3 categories that the 114 wells were divided among were estimated as 0.17 ppm "low", 0.47 "middle", and 0.77 "high". Division of incidence data across more categories of arsenic in the water might have provided more data points for curve fitting and records of the ages when arsenic-induced skin lesions intially appeared could have been used in time-to-tumor equations. However, only 3 exposure levels and 3 age ranges were presented by Tseng.

Incidences of skin cancer in relation to age in the contaminated water area (Tseng, 1968) are listed in Table 1. The incidence increased with age, arsenic level in the water, and was greater in males.

Weighted averages for age cohorts are calculated from Tseng (1968) and listed in Table 2. The period of exposure to arsenic is from 1900-10 to 1956, or a maximum of 56 years. [The beginning years were originally stated as 1910-20, but corrected to 1900-10 in Tseng (1977).] The people were surveyed in 1965.

Cumulative lifetime doses of arsenic were estimated by multiplying the arsenic consumption rates by the times of exposure. The amount consumed per year is shown in Table 3 and the cumulative amount consumed in Table 4. Tseng (1968) reported that the arsenic in wells outside of the contaminated water area were nearly free of arsenic (0.001-0.017 ppm). Therefore, it was assumed for modeling purposes that people in those areas had no arsenic exposure (Tseng, 1968).

The number of skin cancer cases per persons at risk for each group (Brown, 1989) were ordered by estimated cumulative exposure and grouped and weighted (Table 5) for input into the multistage modeling program, Global83. Since Brown (1989) estimated that the skin cancer induction period was 6.9 years, groups in the contaminated water area with an exposure time of <6 years were omitted from the model since those persons would not have had sufficient time to develop skin cancer before they were surveyed by Tseng (1968).

Table 1a
Incidence of Skin Cancer per 1000 males

age	low	middle	high
0-19 yr	0	0	0
20-39	1.5	6.5	48.1
40-59	4.3	47.7	163.4
60+	22.4	98.3	255.3

Table 1b
Incidence of Skin Cancer per 1000 females

age	low	middle	high
0-19 yr	0	0	0
20-39	0.1	0.7	3.5
40-59	3.6	19.7	48.0
60+	9.1	62.0	110.1

Table 2a
Weighted average years of exposure for males
in the contaminated water area

age	exposure time	# of males	avg exposure time
0- 9 yr	0 yr	6763	0.0 yr
10-19	1-10	3564	5.5
20-29	11-20	1715	
30-39	21-30	1850	20.7
40-49	31-40	1387	
50-59	41-50	1098	39.9
60-69	51-56	655	
70+	56	250	54.2

		19269	

Table 2b
Weighted average years of exposure for males outside of the contaminated water area

age	exposure time	males at risk	avg exposure time
0- 9 yr	0 yr	1660	0.0 yr
10-19	1-10	1018	5.5
20-29	11-20	389	
30-39	21-30	456	20.9
40-49	31-40	373	
50-59	41-50	234	39.4
60-69	51-56	131	
70+	56	45	54.1

		4308	

Table 2c
Weighted average years of exposure for females in the contaminated water area

age	exposure time	females at risk	avg exposure time
0- 9 yr	0 yr	6515	0.0 yr
10-19	1-10	3850	5.5
20-29	11-20	2602	
30-39	21-30	2596	20.5
40-49	31-40	1861	
50-59	41-50	1227	39.5
60-69	51-56	635	
70+	56	296	54.3

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Table 2d
Weighted average years of exposure for females
outside of the contaminated water area

age	exposure time	females at risk	avg exposure time
0- 9 yr	0 yr	1284	0.0 yr
10-19	1-10	752	5.5
20-29	11-20	350	
30-39	21-30	358	20.6
40-49	31-40	237	
50-59	41-50	109	38.7
60-69	51-56	71	
70+	56	30	54.2
		3192	

Table 3
Conversion of arsenic level in water
to estimated amount of arsenic consumed per year

water	Taiwan males	Taiwan females
0 ppm	0 mg/yr	0 mg/yr
0.17	217	124
0.47	601	343
0.77	984	562

Table 4a
Cumulative exposure to arsenic for Taiwan males
in the contaminated water area

avg exposure time	cum exposure low	middle	high
0 yr	0 mg	0	0
5.5	1194	3306	5412
20.7	4492	12441	20369
39.9	8658	23980	39262
54.2	11761	32574	53333

Table 4b
Cumulative exposure to arsenic for Taiwan females
in the contaminated water area

avg exposure time	cum exposure low	middle	high
0 yr	0 mg	0	0
5.5	682	1887	3091
20.5	2542	7032	11521
39.5	4898	13549	22199
54.3	6733	18625	30517

Table 5a
Exposure and skin cancer in males-
Grouped and weighted for risk modeling

cum exposure	cases	at risk
0 mg	0	4308
4492	1	935
8658	4	653
11761	11	236
12441	2	531
10588	17	1420
20369	18	810
23980	18	371
21503	36	1181
32574	22	134
39262	56	566
37982	78	700
53333	52	204

Table 5b
Exposure and skin cancer in females-
Grouped and weighted for risk modeling

cum exposure	cases	at risk
0 mg	0	3192
2542	0	1306
4898	3	792
6733	2	239
7032	1	742
6038	6	1773
11521	4	1131
13549	9	450
12098	13	1581
18625	8	136
22199	33	686
21608	41	822
30517	22	207

According to the modeling results, the estimated risk of consuming 3.73 mg over a lifetime is 10^{-6} for males (maximum likelihood estimate). A linear at low dose maximum likelihood estimate could not be derived from the female data; however, the lower confidence limit on the dose for females would be higher (even closer to the male value) if it was a maximum likelihood estimate. The 95% lower confidence limit on dose for females was 1.84 mg. The lower confidence limit on dose for females is only 2-fold lower than the maximum likelihood estimate for males. Thus, the value for males will be used to derive the final unit risk estimate for arsenic ingestion.

Applying the unit risk to Taiwan males having 56 years of exposure and an average body weight of 55 kg, the estimated unit risk from consuming 1 ug/kg/day is 3.02×10^{-4}. When a 70 kg body weight is assumed for the consumption of 1 ug/kg/day for a 70 year lifetime, the unit risk estimate is 4.8×10^{-4} or about 5 in 10,000. The risk of keratosis, roughly estimated by multiplying the risk of skin cancer by 6.7 (section II.A), produces a unit risk of 3.2×10^{-3}, or about 3 in 1000.

An additional correction to the unit risk estimate for differences in background skin cancer rates was not made. A correction for the background skin cancer rate based on the non-melanoma skin cancer rate for Singapore Chinese was considered to be of little significance by EPA (1988), but was used by Brown (1989). It is not clear if that non-melanoma skin cancer category was similar to the NCI (1987) category, which omits basal and squamous cell carcinomas (the types related to arsenic ingestion). The skin cancer rate in the population outside the contaminated water area (males = 0/4308, females = 0/3192) had been included in the present modeling by Health Sciences.

IV.C. Comparison to the EPA (1988) and Brown (1989) risk assessments

Brown (1989) explained the derivation of the EPA (1988) risk assessment modeling which had been conducted under contract to EPA. The multistage Weibull time/ dose-response model was used to estimate unit risks based on data from Tseng (1968). The distribution of skin cancer cases by age group according to well water arsenic concentration derived from Tseng (1968) was presented by Brown (1989). Three curves representing the effect of "low, medium, and high" concentrations of arsenic in the water were plotted onto age vs. incidence axes. The curves described a dose and age dependent relation to skin cancer. The unit lifetime risk estimated by Health Sciences (4.8×10^{-4}) is about a factor of two lower than the final unit lifetime risks of $1\text{-}2 \times 10^{-3}$ estimated by EPA (1988) and 1.3×10^{-3} estimated by Brown (1989). Differences in the unit risks might be associated with the use of age (EPA, 1988; Brown, 1989) rather

than actual years of exposure, 76 yr lifespan (Brown, 1989) instead of 70 yr, correction for a background rate of skin cancer (Brown, 1989), and the grouping and weighting of data.

IV.D. Skin cancer estimated risk due to inorganic arsenic ingestion in the normal diet

The typical ingestion of inorganic arsenic for US adults is estimated as 18 ug/day (EPA, 1988). An estimate of the lifetime risk of ingesting arsenic at this rate for a 70 kg male, according to the present risk model, is 18 ug/day x 0.00048 /70 kg = 1.2×10^{-4} or about 1 in 10,000.

V. Carcinogenicity of arsenic species

The carcinogenic potency of pentavalent (+5) arsenic has not been distinguished from that of trivalent (+3) arsenic. EPA (1984, 1988) documents contain substantial evidence that trivalent arsenic is carcinogenic. Much less is known about pentavalent arsenic. A few animals studies, as reviewed by EPA (1984, 1988) indicate parenteral pentavalent arsenic is carcinogenic, but none have shown carcinogenicity for oral exposure. In chronic feeding studies, the animals experienced a shortened lifespan due to noncancerous causes which may not have allowed sufficient time for tumors to develop. An additional problem is that standard animal models may not be appropriate for testing metal carcinogens, such as arsenic, which appear to act via epigenetic mechanisms. The epidemiological data from smelter and wood preserving plant studies have also been difficult to interpret since those exposures were to a mixture of arsenic valences, as well as other metals, such as lead and chromium.

The American Wood Preservers' Institute (AWPI, 1981) suggested that the arsenic-induced skin lesions, including skin cancer, in the Taiwan study were due to trivalent arsenic, which was 10% of the total arsenic in the water (Irgolic, 1982). However, drinking water arsenic in Antofagasta was 98-99% pentavalent (Irgolic, 1982) and skin "cancer" occurred at rates (Borgono, 1972, 1977; Zaldivar, 1977) higher than would be predicted from the 1-2% trivalent arsenic alone.

For example, the 10-20 yr old cohort in Antofagasta consumed about 20 ug/day and had an average age of 18.6 yrs. The estimated risk for a daily ingestion of 20 ug arsenic /70 kg body weight = 0.29 ug/kg/day, using the unit risk based on the Taiwan data (section IV.B), would be 0.29 ug/kg/day x 4.8×10^{-4} x 18.6/70 = 3.7×10^{-5} or about 4 per 100,000. If this is divided by 10, since there was about 10 times less trivalent arsenic in the Antofagasta water than the Taiwan water, the estimated risk would be <1 per 100,000. However, the incidence rate for that

particular cohort was reported as 413 per 100,000 (section II.E; Zaldivar, 1977a), which is well above the expected rate estimated on the basis of only trivalent arsenic, despite the possibility that some of the 413 may be keratoses rather than cancers. This indicates that some other factor (pentavalent arsenic) was responsible for the skin cancers in Antofagasta. Therefore, ingested pentavalent arsenic was considered to be carcinogenic for risk assessment purposes.

VI. Conclusions

Sufficient evidence exists to link chronic oral ingestion of arsenic with skin keratosis and skin cancer (basal and squamous cell carcinoma) in humans. Although keratosis and these particular types of skin cancer are not as life threatening as internal cancers, they may result in disfigurement and should be considered adverse health effects.

Risk modeling based on Tseng (1968, 1977) was conducted by Health Sciences staff and a unit risk for ingested inorganic arsenic (4.8×10^{-4}) was derived. The modeling was based on epidemiological data relating the incidence of the skin cancer to the dose of ingested arsenic. The risk of keratosis was about 7 times higher and was estimated from the risk of skin cancer by comparing the incidences of keratoses and skin cancers in Tseng (1968). Uncertainties in the risk assessment process, especially in the extrapolation from high to low doses and in the use of epidemiological data, should be kept in mind when using these estimates.

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UNITED STATES GOVERNMENT

MEMORANDUM

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Elaine A. Tyrrell, Vulnerable Populations Project Manager (EX-PM)

JAN 26 1990

Through: Andrew G. Ulsamer, PhD, AED (HS) AGU
Through: Murray S. Cohn, PhD, Director (HSHE) MSC

FROM : Brian C. Lee, PhD (HSHE) BL

SUBJECT: Dislodgeable arsenic on playground equipment wood and the estimated risk of skin cancer.

Introduction

Samples of new playground equipment wood have been analyzed by the Health Sciences Laboratory (HSHL) for dislodgeable arsenic. Exposure due to hand contact with the wood and subsequent ingestion of the arsenic was estimated by a "handload" procedure (CPSC, 1989b). The estimated risks of skin cancer and keratosis were then assessed according to the levels of exposure (CPSC, 1989c). The following discusses the results and significance of this investigation.

I. Playground equipment wood

Major US manufacturers of residential and public playground equipment were identified by Economics (CPSC, 1989d). Field Operations collected new playground wood from six of the manufacturers, which represents a sampling of at least the majority of the US wood playground equipment manufacturing market. A minimum of 10 subsamples were taken per type of finishing per manufacturer. The type of wood, preservative, and finish was noted by the Field staff. Official samples were carefully packaged to prevent sample contamination and shipped under official CPSC seals to HSHL for dislodgeable arsenic analysis.

All playground wood samples were pine (Southern yellow, lodgepole, or nonspecific) and had been treated to a minimum retention of 0.40 lbs/ft^3 (pcf, as the oxide) with chromated copper arsenate (CCA).

One sample of unfinished 0.40 lb/ft^3 CCA-treated Southern yellow pine (L400-6961) was obtained from a hardware store for comparison. No dislodgeable arsenic was detected in samples of non-treated wood during methodology development, so non-treated wood samples were not collected.

II. Dislodgeable arsenic testing

Five subsamples were selected from each sample and 5 replicates were performed on each subsample. Each replicate represents 10 repetitive wiping cycles (1 cycle = back and forth) across a measured 400 cm^2 area. Thus, each sample generated 25 individual data units. The 5 replicates for each subsample were averaged and then reported (Table 1).

Lateral (with the grain) sides of the subsamples were wiped but not the ends (across the grain) due to the small dimensions and catching of the sampling material on the rough surface of the ends. Nylon material (Miracle Wipes 4000, International Clean Products) was securely fastened in a block having a 64 cm^2 (8 cm x 8 cm) face, so that wrinkling or "rolling" would not occur during the wiping. Backing paper was used to separate the sampling material from the block to prevent possible cross contamination. Weight on the block was adjusted to provide 1 kg /64 cm^2 (15.6 g/cm^2) pressure at the face. The block was pulled by hand, using a force parallel to the plane of the wood being tested. The wipes were not moistened since a pilot experiment at HSHL found no significant difference in dislodgeable arsenic levels between wet and dry nylon wipes.

The synthetic wipes did not leave visible nylon fibers caught in the wood and gross wood splinters did not become embedded in the material. Embedding of wood splinters and deterioration of the sampling material had been suspected as a source of error in studies using other materials, such as cotton cloth, adhesive paper, filter paper, or gauze as reviewed in CPSC (1989a). The nylon wipes did pick up small particles of wood which are applicable to the hand-to-mouth exposure route.

After wiping, the edges of the nylon material which were not in contact with the tested wood were trimmed away. Each nylon wipe was placed in 25 ml of 0.01N HCl (pH 2) for 18-24 hr with occasional agitation to dissolve the acid soluble arsenic. The solution was then analyzed by inductively-coupled plasma spectrometry using the third arsenic line (235 nm). Insoluble arsenic was assumed to be unabsorbable and was not measured due to difficulty in chemically digesting the nylon for spectrophotometry. No acid soluble arsenic was detected in unused nylon wipes. The minimum detection level for dislodgeable arsenic was 6.25 ug/100 cm^2.

The levels of acid soluble dislodgeable arsenic from the playground wood and unfinished wood samples are shown in Table 1. Five of the seven playground equipment wood samples were mostly below the minimum detection level. Of the 2 with detectable levels, one sample had received only sanding as a surface treatment and the other had been stained. The unfinished

pressure treated wood sample had the highest dislodgeable arsenic level (69 ug/100 cm^2) and also contained the subsample with the highest level (153 ug/100 cm^2).

The nylon wipes were compared against an animal skin in the ability to remove dislodgeable arsenic from the wood surface. The smooth side of oil-tanned sheep skin chamois was used with the same protocol as described for the nylon wipes. Small pieces were abraded from the chamois during the wiping. This may possibly have reduced the dislodgeable arsenic picked up by the chamois. Lab animal skins more delicate than chamois, such as guinea pig, mouse, rabbit or rat, might be expected to similarly deteriorate under the testing conditions used, and therefore were not tested.

The chamois had a "background" arsenic level of 0.175±0.050 ug/cm^2. It was not determined whether the background was due to acid soluble arsenic, other interfering elements in the chamois that mimic arsenic, or a matrix effect. Nevertheless, when chamois was used to wipe the wood, a correction for the background was made. In paired wiping comparisons between nylon and chamois wipes (Table 2), the nylon picked up the same or more dislodgeable arsenic than the chamois, assuming that there were no differences in the ability to extract the arsenic from the wipes. The difference was less than twofold, which indicates that the error due to using nylon wipes instead of human skin would probably be small. This assumes that human skin would not be much different from chamois in the abilities to pick up dislodgeable arsenic and release it into the acidic extracting solution.

III. Coatings

Coatings, such as stains, sealants, polyurethane, varnish, and paint, may be applied to wood to improve appearance, reduce weathering, and increase the longevity of wood products. CDHS (1984) found that applying an oil-based stain to pressure treated wood reduced dislodgeable arsenic from 31-314 ug/100 cm^2 to 6-11 ug/100 cm^2. It has been hypothesized that the stain seals and "hardens" the surface thus inhibiting migration of arsenic from subsurface layers to the surface and making the wood more resistant to abrasion. Some coatings might provide a barrier between the wood and any skin that might contact the surface. However, barrier coatings are not permanent since abrasion due to normal play activities and weathering (Feist, 1984, 1986) degrade the coatings.

CDHS (1987) was unable to satisfactorily assess the risks of skin cancer due to playground equipment wood. As a result, the legislature of California State (1987) required all new state-funded playground equipment using pressure treated wood to be "sealed" and then recoated every 2 years. The 2 year period

coincides with the low estimate for the expected lifetime of clear or semi-transparent wood finishes (Feist, 1986). The type of sealant to be used was not specified by the California law.

The scope of the coating experiment at HSHL was not to determine the effectiveness of all coating types, but rather to determine whether reduction of dislodgeable arsenic levels, and thus estimated risks, could be accomplished by the application of coatings. The 2 playground equipment wood samples with detectable levels of dislodgeable arsenic were sprayed with 2 coats of either a widely available water repellant/sealer or an oil-based wood stain. The wood was allowed to dry for at least 1 week in a hood at room temperature before dislodgeable arsenic testing.

The coatings did not significantly reduce the dislodgeable arsenic levels. Levels after coating (Table 3) were not statistically different from those before the coatings were applied (Table 1). Furthermore, the levels after the oil stain were not statistically different from those obtained after the water repellant/sealant was applied.

IV. Exposure and Risk Assessments

IV.A. Specific assumptions

The lifetime risks of skin cancer due to ingestion of dislodgeable arsenic were estimated for samples that had 3 or more subsamples with levels above detection limits. The procedure developed for the exposure assessment (CPSC, 1989b) and the unit risk for ingested arsenic (4.8×10^{-4}; CPSC, 1989c) were followed using the following specific assumptions:

1) Exposure to playground equipment wood arsenic occurs in the 5 years from ages 2-7, or 1/14th of a 70 yr lifetime.

2) Absorption of acid soluble dislodgeable arsenic is 100% (ATSDR, 1987).

3) Average hand surface area on one side of one hand, between ages 2-7 years = 66 cm^2 (CPSC, 1975).

4) Average body weight between ages 2-7 years = 17.9 kg (CPSC, 1975).

5) Maximum median hand-to-mouth activity represented by soil consumption between ages 2-7 = 0.374 handloads/day (3 yrs at 0.43 + 2 yrs at 0.29; CPSC, 1989b).

6) Children play on wood playground equipment 4 of 7 days/wk during 6 months of the year.

7) Estimated risk of skin keratosis = 6.7 x estimated risk of skin cancer (CPSC, 1989c).

IV.B. Exposure and risks estimated for playground equipment wood

The estimated risks are proportional to the exposure. For example, if children played on the equipment year-round rather than only for 6 months of the year, then the estimated risks would be doubled.

Exposures and risks were estimated only for the 3 samples having 3 or more subsamples with detectable dislodgeable arsenic. Table 4 shows the estimated risks of skin cancer and keratosis associated with the estimated exposures. As an example, the exposure from a dislodgeable arsenic level of 6.3 ug/100 cm^2 would be 6.3 ug/100 cm^2 x 66.0 cm^2 x 0.374 /17.9 kg = 0.087 ug/kg/day played. The estimated risk of skin cancer associated with this exposure would be 0.087 ug/kg/day played x 4 days/7 days x 6 months/12 months x 5 yrs/70 yrs x 4.8 $x10^{-4}$ = 8.5 $x10^{-7}$. Keratosis would be estimated as 8.5 $x10^{-7}$ x 6.7 = 5.7 $x10^{-6}$.

Skin cancer estimated risks from the average levels of dislodgeable arsenic found on playground equipment wood were estimated as high as 4 per million (Table 4). Since 5 of 7 playground equipment wood samples fell below the detection limit of 6.3 ug/100 cm^2, the highest skin cancer risk estimate that could be applied to these samples would be <1 per million. The risk was greater from unfinished pressure treated wood, which was estimated as 9 per million. As a maximal case, if the unfinished pressure treated wood tested by HSHL occurred in playground equipment at its highest tested level (153 ug/100 cm^2), then the estimated risk would be 21 per million.

Table 5 estimates the dislodgeable arsenic level associated with a specific risk. For example, to achieve a risk of 50 per million, the estimated dislodgeable arsenic level would have to be below about 370 ug/100 cm^2.

CDHS (1983, 1984) found considerably higher levels of dislodgeable arsenic (some >300 ug/100 cm^2 on playground equipment and >2000 ug/100 cm^2 on non-playground equipment wood) than recorded by HSHL. Several explanations are possible. First, samples tested by HSHL were only pine which was treated with CCA system C (CCA-C). Systems A and B, which are no longer used, may have been applied to some of the wood sampled by CDHS, which might possibly affect the dislodgeable arsenic level. CDHS

also tested woods treated with ammoniacal copper arsenate (ACA), which may not bind the arsenic as tightly as with CCA (DeGroot, 1979; EPA, 1984b).

Second, the wood preservers and playground equipment manufacturers may now be more aware of the occupational hazard posed by arsenic residue on the wood and have altered preserving procedures to minimize residue formation (Fong, 1980; EPA, 1981, 1984). Although most manufacturers do not specifically order wood according to the C17 standard (AWPA, 1988), some manufacturers have indicated that they specifically order pressure treated wood that is visibly free of residues. Some manufacturers have constructed their own preserving and drying facilities to more closely control the treatment process.

Third, the sampling and testing protocols used by HSHL were different. A synthetic wipe was drawn across 400 cm^2 of wood for 10 cycles as a standard pressure was placed across the surface. CDHS wiped 100 cm^2 of wood by hand using a moistened cloth or filter paper. Although HSHL found no difference between wet and dry wiping with nylon wipes, the water may have increased the arsenic picked up with the cloth or filter paper used by CDHS. The materials used by CDHS probably would have shredded or picked up large splinters under the rigorous 10 cycles of the 1 kg/64 cm^2 pressure used by HSHL. A larger number of subsamples and replicates were analyzed by HSHL in consideration of the natural variability of wood. CDHS sampled playground equipment which had already been installed, whereas CPSC collected economically representative samples from the manufacturers before installation.

Fourth, CDHS also tested the ends of the treated wood as well as the sides. Higher levels were found on the ends. Wiping the ends was not technically feasible in the CPSC study due to the small surface area and the rough surface that would tend to catch any type of wiping material. Furthermore, some of the ends may be discarded when a manufacturer cuts the treated wood to the desired lengths. It would be difficult to determine if an end was an "original" end which was exposed during pressure treatment, or if it was an end produced by cutting the wood after treatment. One manufacturer pressure treated the wood after cutting, but since the wood was center cored and bevelled at the end, wiping would have been difficult and unrepresentative by the HSHL method.

V. Discussion

The results indicate that dislodgeable arsenic on the majority of playground equipment wood manufactured by major US firms may pose a skin cancer risk of <1 per million. The few samples with detectable dislodgeable arsenic may pose a risk on the order of 10^{-6} to children who play on the equipment and

subsequently ingest arsenic by hand-to-mouth operations. These risk estimates are based on the earlier stated assumptions. However, if the assumptions are modified, then the estimated risks will also change proportionately. For example, if it is assumed that children play on the equipment every month instead of only for 6 of 12 months, then the risk estimates would be doubled.

The large standard deviations in the dislodgeable arsenic levels (Table 1) were due to the occasional piece or side of wood with a high level. Since no association of dislodgeable arsenic levels with the manufacturers' surface preparation methods of sanding or coating was found, the variability in the playground equipment samples was probably caused by the wood preserving procedures and/or the innate variability of wood.

Although the single sample of unfinished wood which was tested can not adequately represent the national market, those results raise the possibilities that a greater risk of skin cancer might result from wood that has not undergone some type of surface preparation, such as by a commercial manufacturer, or from wood that was not preserved according to procedures for wood to be used in playground equipment. Playground equipment is not the only pressure treated wood product which children regularly contact. Rails and decking are consumer products which are not necessarily sanded or coated but might possibly contain dislodgeable arsenic.

The HSHL coating experiment results do not confirm the observations that coatings reduce dislodgeable arsenic levels (CDHS, 1984), since no effect on levels was observed with oil stain or water repellant/sealant. Possible reasons for the different outcomes may be related to the specific type of coating used by CDHS, and the higher pre-coating dislodgeable arsenic level on the wood tested by CDHS. Film forming stains, such as linseed oil or tung oil, might possibly provide a barrier between the arsenic on the wood and the skin as might coatings such as epoxy paints, spar varnish or polyurethane (J. Plattner, Chemical Hazards Info. Ctr., Cornell Univ., personal communication).

Arsenicals, such as CCA or ACZA (ammoniacal copper zinc arsenate), are the major wood preservatives used for playground equipment wood in the US (CPSC, 1989d). However, this is not the only wood preservative available to consumers. Less toxic alternatives to arsenical wood preservatives include borates, quinolinolates, naphthenates, and naturally resistant woods, such as cedar (CPSC, 1988). However, these alternatives may not necessarily have the same long term effectiveness in preventing rot and insect damage. Creosote, pentachlorophenol, or tributyl tin oxide are excellent preservatives, but are considered too toxic or irritating for use on playground equipment.

Table 1

0.1N HCl Soluble Dislodgeable Arsenic Level on Playground Equipment Wood Samples

sample #	wood treatment	dislodgeable arsenic ($ug/100\ cm^2$)
K800-9941	0.40 pcf CCA S.yellow pine stain/sealant	not detectable
		not detectable
		not detectable
		not detectable
		11.2
K830-0584	0.40 pcf CCA S. yellow pine stained	not detectable
		not detectable
		not detectable
		not detectable
		not detectable
K800-9944	0.40 pcf CCA S. yellow pine molded, sanded	not detectable
		not detectable
		not detectable
		not detectable
		10.0
K800-9942	0.40 pcf CCA S. yellow pine molded, sanded	not detectable
		not detectable
		not detectable
		not detectable
		not detectable

Table 1 (continued)

sample #	wood treatment	dislodgeable arsenic ($ug/100\ cm^2$)
K800-9943	0.40 pcf CCA S. yellow pine molded, sanded stained	
		not detectable
		not detectable
		not detectable
		not detectable
		not detectable
L830-8638	0.40 pcf CCA pine sanded	
		7.0
		12.1
		13.3
		15.3
		61.7
	avg ± sd	21.9 ± 22.5
K860-6165	0.40 pcf CCA lodgepole pine core cut, cored stained	
		16.7
		19.3
		21.9
		32.1
		70.3
	avg ± sd	32.1 ± 22.2

Table 1 (continued)

sample #	wood treatment	dislodgeable arsenic (ug/100 cm^2)
L400-6961		
	0.40 pcf CCA S. yellow pine unfinished	
		20.2
		42.7
		58.2
		70.0
		153.1
	avg sd	68.8 ± 50.7

Minimum detection level = 6.25 ug/100 cm^2.
Average of replicates shown; 5 replicates/subsample; 5 subsamples/sample.
10 repetitions/replicate across 400 cm^2 at 1 kg/64 cm^2 pressure.

Table 2
Effect of Wiping Material on Dislodgeable Arsenic Levels
from Playground Equipment Wood
(paired comparisons)

sample #	dislodgeable arsenic (ug/100 cm^2)	
	chamois[a]	nylon[b]
L830-8628	3.8	7.4
	15.9	9.5
	56.9	84.4
avg±sd	25.5±27.8	33.8±43.8
K860-6165	25.9	41.2
	22.4	37.7
	19.1	37.5
avg±sd	22.5±3.4	38.8±2.1

[a]corrected for background in chamois
[b]no background detected in nylon wipes

Table 3
Effect of Coatings on Acid Soluble Dislodgeable Arsenic
on Playground Equipment Wood

sample #	coating	dislodgeable arsenic ($\mu g/100\ cm^2$)
K860-6165	oil stain	91.9
		24.1
		34.1
	avg±sd	53.0±35.0
K860-6165	repel/sealant	23.5
		59.1
		75.0
	avg±sd	52.5±26.4
L830-8638	oil stain	8.4
		13.1
		7.6
	avg±sd	9.7±3.0
L830-8638	repel/sealant	21.3
		8.0
		12.8
	avg±sd	14.0±6.8

No statistical differences in dislodgeable arsenic levels within either wood sample were found by 1-way analysis of variance of oil stain vs. repellant/sealant or in before vs. after coating.

Table 4
Estimated Exposure and Risks of Skin Cancer and Keratosis due to Acid Soluble Dislodgeable Arsenic Ingestion

arsenic ug/100cm^2	exposure ug/kg/day[a]	skin cancer	keratosis
6.3[b]	0.087	8.5×10^{-7}	5.7×10^{-6}
21.9	0.302	3.0×10^{-6}	2.0×10^{-5}
32.1	0.422	4.3×10^{-6}	2.9×10^{-5}
68.8	0.948	9.3×10^{-6}	6.2×10^{-5}
84.4	1.163	1.1×10^{-5}	7.7×10^{-5}
153.1	2.109	2.1×10^{-5}	1.4×10^{-4}
0.0	18.0[c]	8.6×10^{-3}	5.8×10^{-2}

[a]exposure from wood on days played
[b]minimum detectable level
[c]lifetime exposure from arsenic in typical diet (EPA, 1988)

Table 5
Dislodgeable Arsenic Levels Estimated from Skin Cancer Risks

skin cancer	exposure ug/kg/day[a]	arsenic ug/100cm^2
1×10^{-6}	0.102	7.4
5×10^{-6}	0.510	37.1
1×10^{-5}	1.021	74.1
5×10^{-5}	5.104	370.6

[a]exposure from wood on days played

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