PLYWOOD MILL WORKERS' MORTALITY PATTERNS 1945 - 1977

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A cohort of 2,283 plywood mill workers employed for at least one year between 1945 and 1955 was traced through March 31, 1977. Vital status was determined for 98% of the cohort. The 570 deaths observed were only 74% of the number expected based on comparable U.S. mortality rates. A statistically nonsignificant excess of deaths was observed for the category of lymphatic and hematopoietic cancer excluding leukemia (SMR=156, CI=90-252). Within this category, the excess was greatest for multiple myeloma (SMR=333, 3 deaths observed 0.9 deaths expected); however, the risk was slightly elevated for all four subgroups of lymphatic and hematopoietic cancer. These data appear to be consistent with those of other investigations of wood processing populations, including proportionate mortality reports of plywood mill workers. When analyses for duration of employment and time since first employment (latency) were conducted, the excess mortality due to lymphatic and hematopoietic cancer excluding leukemia was highest after 20 years duration of employment and latency. Based on walk-through environmental surveys in the plywood mills, no known etiologic agents were distinguishable. Plywood workers were potentially exposed to formaldehyde around the gluing operations in the mill. However, no deaths due to nasal cancer, which has been associated

with exposure to formaldehyde in laboratory animals, were observed in this cohort, although only 0.4 deaths were expected. Further surveillance of the plywood mill worker cohort was recommended.

Key Terms: Exposure, formaldehyde, lymphatic cancer, multiple myeloma, wood dust

INTRODUCTION

There were 35,300 production workers employed in the U.S. softwood veneer and plywood industry and 19,600 production workers employed in the hardwood veneer and plywood industry in 1981 (2). Softwood plywood has been produced in the United States since 1905. Through the years, the types of assembly systems have changed in many plants and hot presses are now used exclusively. The process of making plywood is similar to the veneering phase of the furniture industry (9). The process consists of gluing thin layers of wood, called plies, together with the wood grain of adjacent layers generally at right angles.

In one of two reports (10,11) of plywood mill workers in Washington State, Milham reported white male plywood workers during 1970-1979 to have statistically significant increased proportionate mortality ratios (PMR's) for cancer of the stomach, coronary heart disease, and suicide (10). For this same decade, PMR's for cancers of the liver and kidney and for lymphoma and multiple myeloma were elevated, but not statistically significant. In an earlier report of the two preceding decades, Milham (11) observed statistically significant increased PMR's for myeloid and acute leukemia and cancers of the stomach and lung. All of these reports were based on cause of death and occupational information given on death certificates.

Lymphoproliferative malignancies, including multiple myeloma, Hodgkin's disease, and other lymphomas, have been linked with occupations involving woodworking in several epidemiologic studies (Table 1). No known etiologic agent has been identified, although a Swedish study observed a significant increase of risk for Hodgkin's disease and/or non Hodgkin's lymphoma with exposure to chlorophenols contained in wood protection agents (7).

Excess Hodgkin's disease mortality was significantly associated with New York State woodworking occupations, including carpenters and lumber and sawmill workers (13). The risk of Hodgkin's disease of histological cell type, "mixed cellularity", was significantly increased for Israeli lumber and afforestation workers (1). Hodgkin's disease risk was non-significantly elevated among carpenters and joiners in their national union study (12). In Boston, woodworkers who were predominately carpenters, were observed to have a slightly elevated Hodgkin's disease incidence rate (6). In contrast, a British study of woodworkers exposed to mostly hardwoods observed a slightly decreased mortality rate for Hodgkin's disease (3).

An increase in multiple myeloma, a rare neoplasm which affects the elderly, was significantly associated with the North Carolina furniture-making industry (4,16). The Carpenters and Joiners National Union was observed to have a slightly elevated death rate

for multiple myeloma (12), and non-significant increased proportionate mortality was observed among Washington State plywood workers (10,11).

Increased mortality due to lymphomas of non-Hodgkin's origin was significantly linked to the paper and pulp industry in two studies (5,10). In addition, lymphomas were observed to be slightly increased during 1970-79 among Washington State plywood workers (10) and were found to be slightly increased during 1950-71 among Washington State carpenters and cabinet makers (11).

In some of the epidemiologic studies of men who were employed in wood-related industries, an increased risk of dying was found for malignancies of the digestive (5,10,11), and the respiratory system (10,11,12,14), (Table 1).

The purpose of our cohort mortality study was to follow-up the leads supplied by state mortality statistics (4,10,11,13,16) and other reports (1,5,6,7,12,14) which indicated excess deaths among workers in the wood processing industries. In particular, we wished to further investigate the reports (10,11) of increased proportionate mortality for the site-specific malignancies observed among plywood mill workers. The study was designed to examine the mortality of men who worked in four plywood mills located in the Pacific Northwest.

PLYWOOD PROCESS DESCRIPTION*

Potential exposures to the plywood mill workers included wood dust, volatiles from the wood, formaldehyde, pentachlorophenol, and carbon disulfide (Table 2). We obtained information on the historical and current processes used in our four softwood plywood mills from company officials, mill records, and observations in the mills.

In the Pacific Northwest, softwood plywood is manufactured principally from Douglas Fir, although small amounts of cedar, pine, spruce, hemlock, larch, true firs, and redwood are also used. The wet logs are sawed into blocks, debarked, and peeled into veneers. The most common method of producing softwood veneers is rotary peeling done by a knife held parallel to the log axis. A continuous sheet of veneer is peeled and then is dried at about 325°-400°F. Some mills edge-glue dry veneers into 4 by 8 foot sheets. During this study period, an odd number of plies typically were glued together to make a plywood panel. Today, much 4 and 6-ply plywood is also produced.

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*The information in this section was taken from Stanford Research Institute (SRI) Project 5847 Report; see the appendix to this report for more detail.

Glue was usually mixed in the mill (Table 3), and the type of glue used was dependent on the final product desired. Glues were soy or soy-blood blends with some straight blood also used in the production of interior plywood panels in the 1940's and 1950's. Pentachlorophenol was often added to the protein glues as a mold preventative. Carbon disulfide increased water resistance of cold pressed glues, and carbon tetrachloride was premixed with carbon disulfide to reduce fire hazards.

Phenol formaldehyde resin was the most commonly used glue in the production of exterior panels in the 1940's-1950's. Both urea and resorcinol formaldehyde resins were commonly used as patching glues. Ammonium chloride was added to urea glues as a catalyst; it was not used with resorcinols. There was potential worker exposure to the free formaldehyde released by these resins around the patching operations.

A hot or a cold press process was used to bond the panel, depending upon glue type and wood species. During the 1940-1950 period, when soy-blood glues were predominant, the cold pressing process was most common with hot pressing used to some extent. Now, hot pressing is almost universal in Pacific Northwest mills. This is due to virtually a complete switch from soybean and blood-albumin glues to phenol formaldehyde resins which require hot pressing. Hot presses

are usually hydraulically driven and steam-heated to a temperature between 240°-330°F. Panels are pressed for 1-1/2 to 14 minutes for softwoods. Time and temperature varies with the panel thickness and glue type, while the pressure varies with wood species. In softwood plywood mills, panels are usually stacked after removal from the press. This permits retained heat to be used for completing the cure of the adhesive.

After pressing, the panels are trimmed to exact dimensions. Depending upon the type of product, the panels may also be sanded, patched and resanded. Prefinishing and treating are usually not done at the plywood plant but in an operation located elsewhere. The exception is concrete form oiling. Pentachlorophenol was occasionally added to the oil.

METHODS

All softwood and hardwood plywood mills located in California, Washington, and Oregon, which had start-up dates prior to 1950, were contacted. Four softwood plywood mills three of which were located in Washington and one of which was located in Oregon, were selected for study on the basis of the age of the mill, the completeness of employment records, and the existence of a large work force. All four mills began operations before 1941.

The study cohort was defined to include white males who worked at least one year between 1945-1955 inclusive. These criteria allowed the investigation to focus on persons with long latency and whose follow-up would be facilitated due to their occupational stability and their having worked after World War II. Salaried office workers were excluded due to their minimal exposures in the mills. Females and non-white males were excluded from the study due to their small numbers. Employment records consisting of demographic data and the daily detailed work histories of each worker, including departments, job titles, dates, and layoffs, were coded from these records.

The 2,283 plywood workers who met the study cohort criteria were traced from their last date of employment through March 31, 1977 (Table 4). Their vital status was determined through records maintained by various government agencies, directories, and other sources for 98% of the study cohort. Death certificates were obtained for 97% of those deceased. Underlying causes of death were interpreted and coded by a nosologist, according to the revision of the International Classification of Diseases, Adapted (ICDA), in effect at the time of death. Individuals with unknown vital status were assumed to be alive as of March 31, 1977. The 15 deceased individuals for whom no death certificates were available were assumed to be deceased, cause unknown.

Because of the potential exposures to formaldehyde and to pentachlorophenol which occurred together during drying, glue mixing and application, a subcohort of these workers was identified for a separate analysis. Workers were included in this subcohort if they had worked for one year or more in the relevant exposure categories which were veneer pressing and drying, glue mixing, veneer and panel gluing and panel patching (Table 2).

A modified life table technique (18) was used to obtain person-years at risk of dying for white males in 5-year calendar time periods, age groups, duration of employment periods, and latency (time since first employment at the plant) periods. Person-years were multiplied by the appropriate U.S. mortality rates to obtain the number of expected deaths. Since the cohort was limited to white male production employees who had at least one year of employment between 1945 and 1955, person-years at risk began either at one year after first date of employment or on January 1, 1946, whichever came later.

Standardized mortality ratios (SMR's) were calculated by dividing the number of observed deaths by the number of expected deaths and multiplying by 100. SMR's were not calculated when the observed and expected numbers of deaths were both less than two. Based on our literature review, our a priori hypotheses were for excess risks of

death due to cancer of the stomach, liver, lung, and kidney, and multiple myeloma, leukemia, coronary heart disease and suicide. One-sided hypothesis testing was performed for these and other causes of death at the .05 level, and two-sided 90% confidence intervals were then calculated for the SMR's. These calculations were performed only for SMR's greater than 100. The Fisher exact method was used to calculate the intervals if the observed deaths were less than eight; and an approximate method was used if the observed number of deaths was eight or more (15).

RESULTS

For the entire cohort, 57,588 person-years of observation were accrued (Table 5). A deficit of deaths from all causes combined was observed (SMR=74, 570 observed, 771.9 expected) (Table 6). Mortality due to circulatory system disease (SMR=74, 260 observed, 360.1 expected) and all malignant neoplasms (SMR=70, 100 observed, 141.8 expected) was also below expected. However, mortality for lymphatic and hematopoietic cancer (excluding leukemia) was slightly elevated. (SMR=156, 12 observed, 7.7 expected). Two other site specific malignancies had statistically nonsignificant elevated SMR's: pancreas (SMR=138, 11 observed, 8.0 expected) and kidney (SMR=114, 4 observed, 3.5 expected). No deaths due to nasal cancer were observed, but only 0.4 deaths were expected. Table 7 provides

SMR's for the four major subgroups of lymphatic and hematopoietic cancer (excluding leukemia): multiple myeloma* (SMR=333, 3 observed, 0.9 expected) lymphosarcoma and reticulosarcoma (SMR=103, 4 observed, 3 expected); Hodgkin's disease (SMR=111, 2 observed, 1.8 expected), and other lymphatic cancers (SMR=272, 3 observed, 1.1 expected).

Table 8 shows work history, date of death, and exact cause of death as stated on the certificate for all 12 cases of lymphatic and hematopoietic cancer excluding leukemia.

Mortality for lymphatic and hematopoietic cancer excluding leukemia was further analyzed by duration of employment and latency (Table 9). The excess risk was most elevated after 20 years duration of employment (SMR=250 and latency (SMR=195).

After a careful review of all 555 death certificates, no diagnoses of nasal cancer was observed among the plywood workers cohort as either an underlying or contributory cause of death.

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*Since the life table analysis program does not have a separate category for multiple myeloma, the proportion of multiple myeloma deaths in U.S. vital statistics (17) was used to estimate the expected number of deaths.

Of the 818 members of the formaldehyde-pentachlorophenol subcohort, 151 were deceased and only 2 had unknown vital status at the end of the study. Statistically nonsignificant increased risks of death based on small numbers were found for lymphosarcoma (SMR=250, 3 observed, 1.2 expected) and Hodgkin's disease (SMR=333, observed, 0.6 expected) (Table 10).

DISCUSSION:

Mortality due to the category of lymphatic and hematopoietic cancer excluding leukemia was elevated among the plywood workers cohort, but this finding was not statistically significant (SMR=156, CI 90-252). The SMR's for all four subgroups within the category of lymphatic and hematopoietic cancer excluding leukemia were slightly elevated, suggesting that excess risk may exist for all four subgroups (Table 7). Even though the excess was greatest for multiple myeloma (SMR=333), the data are at present too sparse to clearly distinguish between subgroups.

Overall, our data appear independent of, but consistent with those of the two previous reports of plywood workers (10,11) which found excess proportionate mortality from lymphatic cancer, including multiple myeloma and non Hodgkin's lymphoma. The excess mortality due to lymphatic and hematopoietic cancer (excluding leukemia)

observed among our cohort of plywood workers was found to be independent of the excess proportionate mortality reported among Washington State plywood workers (10). Potential overlap was limited to only four deaths between the two studies. Of 12 deaths due to these malignancies among our cohort, four occurred in Oregon, one occurred in Arkansas, and seven occurred in Washington. Three of the seven deaths from Washington did not have "plywood" stated as the occupation on the death certificate. Thus, only four deaths from this cohort would have been eligible for inclusion in the previous study of Washington State plywood workers (10). Additionally, our data for plywood workers are somewhat consistent with those of other wood processing populations which have observed excess mortality for multiple myeloma, Hodgkin's disease and lymphoma (1,4,5,6,7,12,13,16).

Our study observed that excess mortality due to these causes was highest after 20 years of latency or exposure in the plywood mill. Thus, the excess mortality was not inconsistent with an occupational etiology. None of the previous studies was able to utilize actual work history dates or job titles to calculate the risk by duration of employment or latency. In addition, studies based on occupations abstracted from death certificates or based on recalled occupation are subject to potential selection or information biases, which may have the effect of increasing or decreasing estimates of risk. Our

study avoided these potential problems by using company work history records to calculate duration of employment and latency.

Although Milham reported statistically significant excess proportionate mortality for stomach cancer among plywood workers for each of the last three decades (10,11) we observed a deficit of stomach cancer (SMR=66, 4 observed, 9.1 expected) among our plywood worker cohort. However, the SMR for another site of digestive tract cancer, the pancreas, was observed to be slightly elevated (SMR=138, 11 observed, 8.0 expected).

Most of the numbers in the formaldehyde-pentachlorophenol subcohort were too small to interpret. It is noteworthy that the SMR for Hodgkin's disease was 333, based on 2 deaths observed for the subcohort. The subcohort SMR for lymphosarcoma and reticulosarcoma was 250, based on 3 deaths observed, although it was not elevated among the full cohort; while the SMR for pancreatic cancer remained slightly elevated among the subcohort. Elevated risk for Hodgkin's disease and non Hodgkin's lymphoma were observed in a Swedish study (7) of men exposed to pentachlorophenol in wood protection agents. Although excess risk was reported for lymphatic cancer (8), Hodgkin's disease has not been associated to date with exposure to formaldehyde.

Based on the preceding discussion, it would seem appropriate to continue surveillance on the plywood mill worker's cohort. In particular, the apparent mortality excess due to lymphoproliferative neoplasms should be further evaluated again when the follow-up period may be extended to include additional deaths among plywood workers. The investigation of the potential health effects to plywood mill workers of chronic low level exposures to wood dust, wood resin volatiles, formaldehyde, pentachlorophenol, herbicides, and other agents should be continued.

CONCLUSION:

Statistically nonsignificant excess mortality for lymphatic and hematopoietic cancer (excluding leukemia) was observed among the cohort of plywood mill workers. This finding appears to be consistent with those of previous studies of wood processing populations, including proportional mortality reports on plywood workers. The excess mortality risk was highest after 20 years' duration of employment and latency. Based on small numbers, an elevated risk for Hodgkin's disease was observed for the formaldehyde-pentachlorophenol subcohort.

Surveillance of the plywood mill workers cohort should be continued to investigate the potential health effects in plywood mill workers

of chronic low level exposures to wood dust, wood resin volatiles, formaldehyde, and pentachlorophenol. It is recommended that the mortality excess due to lymphoproliferative neoplasms be evaluated again when the period of follow-up can be extended over several more years.

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TABLE 1

PREVIOUSLY REPORTED ASSOCIATIONS* BETWEEN WORKING IN THE FURNITURE, PLYWOOD, CARPENTRY AND CABINET INDUSTRIES AND SITE SPECIFIC CANCERS.

SITE OF CANCER	PLYWOOD	FURNITURE	CARPENTRY & CABINET
Stomach	(10,11)	(5)	(10,11)
Liver	(10)		(11)
Lung	(10,11)	(12,14)	(10,11)
Kidney	(10)		
Hodgkin's Disease			(1,6,10,12-13)
Multiple Myeloma	(10,11)	(4,16)	(10,11,12)
Leukemia	(10,11)		
Other Lymphomas	(3)		(11)

*Relative risk greater than 1.2 or statistically significant. The numbers in parentheses refer to the studies cited in the references.

TABLE 2

PLYWOOD MILL EXPOSURES

EXPOSURE CATEGORY	POTENTIAL EXPOSURES DURING STUDY PERIOD
Raw wood preparation, veneer cutting	wood dust, volatile wood resins
Pressing, drying of veneer	neoabietic acid and other wood volatiles, formaldehyde
Glue mixing, veneer and panel Glueing	pentachlorophenol, carbon tetrachloride, carbon disulfide, flake caustic, lime, sodium silicate, phenol, formaldehyde from phenolic resins
Panel patching of flaws, knotholes, etc.	resorcinol formaldehyde resins, ammonium chloride
Panel finishing, trimming, sanding	wood dust, wood volatiles, neoabietic acids, pentachlorophenol
Utility, power, maintenance, unknown jobs	general plant exposures, pentachlorophenol

TABLE 3

PROTEIN GLUE MIXING OPERATIONS*

(All in open vessel)

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1. Water
2. Dry Glue Mix
3. Additional Water (pentachlorophenol when used, sometimes added here)
4. Lime and Water
5. Flake Caustic, Sodium Silicate, Water
6. CS2, CCl4
7. Pentachlorophenol, when used usually added here
8. Agitated
9. To Glue Spreaders

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* Refer to appendix for description

TABLE 4

VITAL STATUS OF WHITE MALES EMPLOYED IN FOUR NORTHWEST PLYWOOD MILLS

Number Alive	1676	(73%)
Number Deceased	570	(25%)
With Death Certificates	555	
Without Death Certificates	15	
Unknown	37	(2%)
	2283	(100%)

TABLE 5

PERSON YEARS AT RISK BY AGE FOR PLYWOOD MILL WORKERS COHORT

AGES	Total
15-24	1,498
25-34	9,066
35-44	15,538
45-54	15,908
55-64	10,063
65-74	4,203
75-84	1,119
35+	193
TOTAL	57,588

TABLE 6

CAUSE-SPECIFIC DEATHS AMONG WHITE MALES EMPLOYED IN FOUR NORTHWEST PLYWOOD MILLS.

Cause of Death	ICDA*	Observed Deaths	Expected Deaths	SMR	90% Confidence Limits
All Causes		570	771.9	74	.
Malignant Neoplasms	140-205	100	141.8	70	.
Buccal cavity & Pharynx	140-148	3	4.7	64	.
Stomach	151	4	9.1	44	.
Intestines	152,153	8	13.2	61	.
Pancreas	157	11	8.0	138	77-228
Nasal	160	0	0.4	.	.
Larynx	161	1	2.2	45	.
Lung	162-163	33	41.7	79	.
Prostate	177-179	2	10.1	20	.
Kidney	180	4	3.5	114	39-261
Bladder	181	1	4.6	22	.
Brain	193	4	4.1	98	.
Lymphatic and Hematopoietic	200-203,205	12	7.7	156	90-252
Leukemia	204	5	5.8	86	.
Residual Sites		12	27.1	44	.
Diseases of Blood Forming Organs	290-299	2	2.1	95	.
Vascular Lesions of the Central Nervous System	330-334	54	60.2	90	.
Diseases of Circulatory System	400-468	266	360.1	74	.
Nonmalignant Respiratory Disease	470-527	32	42.4	75	.
Accidents	800-962	40	47.0	85	.
Transportation	800-866	24	24.5	98	.
Poisoning	870-895	1	1.7	.	.
Falls	900-904	4	6.7	60	.
Other	910-936 960-962	10	13.1	76	.
Suicide	963,970-79	17	15.7	108	69-163
Residual Causes, Unknown		59	102.6	57	.

Total Person Years: 57,588

*7th REVISION

TABLE 7

LYMPHATIC AND HEMATOPOIETIC CANCER CLASSIFIED BY SUBGROUP AMONG PLYWOOD MILL WORKERS.

Cause of Death	7TH Revis., ICDA	Observed Deaths	Expected Deaths	SMR	90% Confidence Limits
Lymphosarcoma and Reticulosarcoma	200	4	3.9	103	35-235
Hodgkin's disease	201	2	1.8	111	20-350
Multiple Myeloma	203	3	0.9*	333	91-862
Other Lymphatic	202,205	3	1.1	272	74-705
TOTAL		12	7.7	156	90-252

* Based on U.S. Vital Statistics, 1975 (See footnote, page 8)

TABLE 8

WORK HISTORY INFORMATION* FOR PLYWOOD WORKERS WHO DIED FROM LYMPHATIC AND HEMATOPOIETIC CANCER.

MILL	CAUSE OF DEATH	DATE OF DEATH	YEARS OF EMPLOYMENT	YEARS OF LATENCY
1	Multiple Myeloma	1973	27	31
3	Myeloma Kidney	1973	26	26
1	Multiple Myeloma	1959	10	17
3	Lymphocytic Lymphoma	1959	4	13
1	Mycosis Fungoides	1977	6	30
1	Non-Hodgkin's Lymphoma	1976	30	30
1	Hodgkin's disease	1961	15	15
3	Hodgkin's disease	1973	30	30
1	Lymphosarcomas lung	1969	8	26
3	Reticulum Cell Sarcoma	1974	10	30
4	Lymphosarcoma of Small Intestine	1958	1	5
4	Lymphoma-mediastinal	1969	18	20

* Rounded down to nearest year

TABLE 9

OBSERVED AND EXPECTED DEATHS DUE TO LYMPHATIC AND HEMATOPOIETIC CANCER (EXCLUDING LEUKEMIA) BY LATENCY AND DURATION OF EMPLOYMENT AMONG PLYWOOD MILL WORKERS.

	LATENCY			DURATION OF EMPLOYMENT		
TIME PERIODS IN YEARS	OBS	EXP	SMR	OBS	EXP	SMR
Less than 20	4	3.6	111	8	6.1	131
Greater than 20	8	4.1	195	4	1.6	250
Total	12	7.7	156	12	7.7	156

TABLE 10

SELECTED CAUSE-SPECIFIC DEATHS AMONG WHITE MALES EMPLOYED IN AREAS OF POTENTIAL FORMALDEHYDE AND PENTACHLOROPHENOL EXPOSURE IN FOUR NORTHWEST PLYWOOD MILLS.

Cause of Death	ICDA*	Observed Deaths	Expected Deaths	SMR	90% Confidence Limits
Malignant Neoplasms	140-205	30	42.9	70	.
Buccal cavity & Pharynx	140-148	1	1.5	.	.
Stomach	151	1	2.4	.	.
Intestines	152,153	3	3.8	79	.
Pancreas	157	3	2.4	125	34-323
Larynx	161	0	0.7	.	.
Lung	162-163	12.0	13.6	88	51-143
Prostate	177-179	0	2.5	.	.
Kidney	180	0	1.1	.	.
Bladder	181	0	1.3	.	.
Lymphosarcoma and Reticulosarcoma	200	3	1.2	250	68-646
Hodgkin's disease	201	2	0.6	333	59-1049
Leukemia	204	1	1.7	.	.
Other Lymphatic	202,203-205	1	0.6	.	.
Diseases of Blood Forming Organs	290-299	0	0.5	.	.
Vascular Lesions of the Central Nervous System	330-334	15	15.1	99	.
Diseases of Circulatory System	400-468	62	102.3	61	.
Nonmalignant Respiratory Disease	470-527	12	12.1	99	.
Accidents	800-962	12	14.3	81	.
Residual Causes, Unknown		20	36.0	56	.
All Causes		151	223.3	67	.

*7th Revision

APPENDIX

Chemicals Used in 1940-1950's and Potential Exposures in the Plywood Study Mills

Soy glues for interior plywood were mixed in the mill. Carbon disulfide and carbon tetracholoride in a 3:1 mix were added to the soy glues at a rate of 1-3/4 lbs. per 100 pounds of dry glue. [100 lbs. of dry glue powder, after mixing, produced 500-600 lbs. of mixed glue.] The carbon disulfide was added to soy glues to provide a degree of cross-linking to soy proteins, thus increasing their water resistance. Carbon tetrachloride was premixed with the carbon disulfide to reduce fire hazard. The glue mixer was probably the only individual exposed to these solvents in liquid form, although all of those workers in the gluing process exposure categories were exposed to the vapors. The exposures were measurable around the glue spreaders. According to the industrial hygienists in the industry during the study period, the exposures were above the exposure limits for 1957 which were 20 ppm for carbon disulfide and 25 ppm for carbon tetrachloride. Because of its extreme flammability and wide range of explosive limits, carbon disulfide was kept outside, with small quantities in a flashproof can brought into the glue room for mixing. Pine oil was also used in these glues as a defoamer.

Blood and soy powder were used as the base of the interior panel veneer glues and were added dry to the glue mixing vessel, after the first water was charged. (Table 3) Most hot pressed protein glues were actually soy-blood blends. Flake caustic and lime were used in small quantities and sodium silicate was occasionally added. Water was added to the dry mix and it was then piped to the glue spreaders. Limited skin contact was reported with this glue, which was spread on the veneers.

Phenol formaldehyde resin was the most common glue used for exterior panel veneers. In one of the study mills, an adhesive based upon cresylic acid was used. The phenolic resins were occasionally purchased in drums, but were more commonly supplied in tank trucks or tank cars. Final mixing of the phenolic resins was almost always done in the mill.

Panel patching glues used included urea and resorcinol formaldehyde resins. Ammonium chloride was added to the urea resins as a catalyst (with extenders such as wheat flour or ground walnut shells). In the hand-patching operation, there was significant skin contact with the glues, and para-formaldehyde was released into the atmosphere in measurable quantities around the operation. Hand-patchers frequently complained of severe local dermatitis. This was attributed to the formaldehyde by the mill industrial hygienists.

Pentachlorophenol (or sodium pentachlorophenate) was used in two ways in the industry during this time. It was added to the glue mix toward the end of the mixing process at 5% by weight of the dry glue weight for the soy and blood based glues used in some of the interior sheathing panels produced by the study mills. In this case, sodium pentachlorophenate was commonly used, because of its greater water solubility. Pentachlorophenol was also occasionally added to the oils applied to concrete form panels, although "plain pale oil" was more commonly used. This latter operation was often a diptank operation in the warehouse areas, with probable significant skin and inhalation exposures.

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16. Abstract (Limit: 200 words)

A cohort of 2,283 plywood mill workers employed for at least one year between 1945 and 1955 was traced through March 31, 1977. Vital status was determined for 98% of the cohort. The 570 deaths observed were only 74% of the number expected based on comparable U.S. mortality rates. A statistically nonsignificant excess of deaths was observed for the category of lymphatic and hematopoietic cancer excluding leukemia (SMR=156, CI=90-252). Within this category, the excess was greatest for multiple myeloma (SMR=333, 3 deaths observed 0.9 deaths expected); however, the risk was slightly elevated for all four subgroups of lymphatic and hematopoietic cancer. When analyses of duration of employment and time since first employment (latency) were conducted, the excess mortality due to lymphatic and hematopoietic cancer excluding leukemia was highest after 20 years duration of employment and latency. Based on walk-through surveys in the plywood mills, no known etiologic agents were distinguishable. Plywood workers were potentially exposed to formaldehyde around gluing operations in the mill. However, no deaths due to nasal cancer, which has been associated with exposure to formaldehyde in laboratory animals, were observed in this cohort, although only 0.4 deaths were expected.

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