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ASBESTOS EXPOSURE AND PULMONARY X-RAY CHANGES TO PIPE COVERERS AND INSULATORS AT PUGET SOUND NAVAL SHIPYARD *

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* The opinions expressed in this paper are those of the authors and do not necessarily reflect those of the Department of the Navy, the Department of Defense, or any other official agency. Reference to a company or a product name does not imply approval or recommendation of the product to the exclusion of others that may be suitable.

This paper for presentation at the annual meeting of the Pacific Northwest Section of the American Industrial Hygiene Association at Richland, Washington on October 3 - 4, 1968.

ABSTRACT.

A 2-1/2 year comparison of chest X-rays of 6571 production workers and 420 clerical workers at Puget Sound Naval Shipyard show that Pipe Coverers and Insulators handling asbestos have 21.2% diagnosed pulmonary abnormalities as a group compared to the next highest incidence of 3.5% for boilermakers, with some asbestos and silica exposure, and less than 1% for clerical workers, with no known asbestos exposure. Exposure patterns suggest that the current TLV of 5 mg/M^3 for asbestos may be too high.

1. INTRODUCTION. Numerous authors describe many case of asbestosis and the increased risk of lung cancer for insulation workers with regular exposure to asbestos or asbestos-containing materials (1,2,3,4,5,12,13,14). Fleischer (1) found few cases in 1946 of asbestosis among insulation workers in Eastern Navy Shipyards and minimized the potential hazard because dust levels were below the threshold limit value. Asbestos exposures to insulation workers during ship overhaul at Long Beach Naval Shipyard were evaluated in 1965 by Marr (4) showing asbestosis remains a serious occupational disease at Naval shipyards. Nearly all of Marr's work was done with the Bausch and Lomb Dust Counter.

Balzer (7,8) reported in 1968 that insulation workers in the San Francisco Bay area were exposed to overall environmental dust counts near the threshold limit value; however, definite radiologic lung changes were observed in the asbestos union population. Approximately 25% of the men with over 20 years work experience and a 45% time weighted exposure displayed radiologic lung changes. His studies however, did not include Naval shipyards.

Cooper (6) and Balzer (7,8) point out that the midget impinger sampling used in determining dust concentrations includes all dust, and the asbestos dust that is counted is mainly grains although some fibers are included. Lynch and Ayers (9) have suggested that the 5 mppcf threshold limit value is no longer a good measure of asbestos exposure, because short asbestos fibers are largely below the resolving power of light field microscopy.

Cralley et al (14) has suggested that asbestos fibers act as carriers for carcinogenic metals or other substances.

2. STUDY BASIS. At Puget Sound Naval Shipyard, a 2-1/2 year study was begun in 1965 to compare the chest X-ray findings and the environment of 104 insulation workers with 6571 production and 420 clerical workers. Asbestos exposures have been periodically evaluated by midget impinger sampling and counted by light field microscopy since 1956 (10,11). The same methods were used in this study to compare data collected over the past 12 years.

3. COMPARATIVE REVIEW OF CHEST X-RAYS. Most X-ray evaluations were based on several reviews over the 2-1/2 year study period. Those workers who were exposed to lung hazardous conditions received semi-annual 14 x 17 inch chest X-rays, all others received annual 70 mm chest X-rays. Physicians listed the X-ray findings as "fibrotic changes," or "pulmonary abnormalities."

Table I shows the distribution of diagnosed pulmonary fibrosis, or other abnormality for each trade category. Because the insulators had the highest incidence (21.2%) compared to all other trades, the work history was investigated. Table II compares the age, employment, and first pulmonary abnormalities for 22 insulation workers.

Table III summarizes the only case diagnosed as asbestosis reported during the study.

Table IV shows the distribution of pulmonary findings compared to the years exposed.

No cases of lung cancer were reported in the study group.

4. ENVIRONMENTAL REVIEW. Airborne samples were collected and counted according to the method adopted by the National Conference of Governmental Industrial Hygienists in 1942 (10,11). Particle size distributions were calculated for 20% of the samples. The geometric mean diameter did not exceed 2.3 microns by the light field method for any sample. Many asbestos fibers were probably below the limit of resolution of the microscope (1 micron) (9). The number of fibers observed were related to the type of material.

The materials described in Table V are used by nearly all insulators at Puget Sound Naval Shipyard. The asbestos products are similar to those used by union employees in the San Francisco Bay area (7,8) and Long Beach Naval Shipyard (4).

Thoroughly wetting amosite or other asbestos materials was found to reduce airborne particulate levels by one-half to two-thirds compared to dry handling techniques. Wetting amosite or magnesia block before ripout of old insulation aboard ship is not practical because the adhesive and paint coating is water resistant. Installation or ripout of asbestos insulation aboard ship produces high concentrations of airborne asbestos dust. Adequate ventilation and wet handling techniques in the fabrication shop keep the dust levels well below the TLV.

Table VI shows the estimated time weighted exposure to asbestos determined from work schedules and field sampling. The exposure patterns for each insulation job on new ship construction, overhaul, and pre-fabrication are estimated in Tables VII, VIII, and IX.

Graph A shows a typical sawing operation where excessive exposures occur for brief periods.

Although dust respirators have been required for installation or ripout of asbestos aboard ship since 1956, this survey found that 76% of the insulation workers did not use dust respirators. Over 50% did not possess respirators which are readily available at tool rooms.

5. DISCUSSION. The exposure patterns of insulators at Puget Sound Naval Shipyard are intermittent, largely chronic low level, but include brief acute high level episodes during sawing or overhaul of ships when old insulation is ripped out without pre-wetting the material. Individual exposure patterns vary widely within work groups because of high mobility, individual work patterns, variety of job assignments, and the use of many different materials.

The insulators have a comparatively light exposure when compared to asbestos miners or fabric manufacturers, yet after about 10 years of exposure chest X-rays begin to reveal increased diffuse markings. Change of job status, termination, or removal from the hazardous duty list may obscure the past occupational history of insulation workers. There is a need to develop a uniform classification for reading and identifying such X-rays taken of insulation workers (14) and to maintain their occupational history. The individual habits of workmen appear to play a large part in their exposure patterns. Some workmen wear their respirators, while others will not. Most insulation workers were aware that exposure to asbestos, even at low concentrations, is hazardous. The attitude prevails that the hazards are unavoidable and must be accepted as part of the trade.

Total dust counts are believed to be important because some of the grains are actually particles from the asbestos fibers or the parent rock in the asbestos vein. They are in fact crystallographically indistinguishable from fibrous asbestos (9). Binders, metals from processing (14) and surface absorption may play yet another role. Until these uncertainities are resolved, the fines associated with asbestos products should not be treated as inert material.

The 21.2% diagnosed lung abnormalities found in insulation workers compared with 25% (7,8) found in union insulation workers in the San Francisco Bay area. The 3.5% occurrence of pulmonary abnormalities among boilermakers with marginal exposures to asbestos and silica may be partially due to asbestos.

6. CONCLUSIONS. A 2-1/2 year comparison of chest X-rays of 6571 production workers and 420 clerical workers at Puget Sound Naval Shipyard showed that 104 insulators handling asbestos have 21.2% diagnosed pulmonary abnormalities compared to the next highest incidence of 3.5% for boilermakers with some asbestos and silica exposure, and less than 1% for clerical workers with no known asbestos exposure.

Only one case of asbestosis was diagnosed since July 1965 when the study was begun.

Exposure patterns and the high incidence of lung abnormalities suggest that the current threshold limit value of 5 mg/M^3 for asbestos may be too high.

Lung abnormalities of insulators are first evident after about 10-15 years exposure and progress until retirement or termination.

Insulation workers handle asbestos materials between 70 - 90% of the work shift. The exposure to asbestos dust is intermittent, although brief episodes at concentrations considerably above the TLV do occur during sawing or ripout. The total airborne particle count ranges about 5 mppcf for about 50% of the work day. The asbestos content of the materials ranges from 15 to 95%. The work day has periods of inactivity, time between job changes, and time for material acquisitions, which reduce the actual exposure to about 6 hours.

A high incidence of lung abnormalities would not be expected according to time weighted exposures compared to the present TLV. This is contrary to the 21.2% pulmonary abnormalities among insulators shown in Table I.

Possible explanations are:

a. The pulmonary abnormalities are the result of intermittent peak exposures throughout each work day for more than 10 years. The time weighted exposure estimates are low and do not reflect the true exposure.

b. The threshold limit value has been set too high.

c. Methods of evaluation do not properly access the exposure pattern.

The 21.2% pulmonary abnormalities found among insulation workers at Puget Sound Naval Shipyard compare to 25% found in union insulation workers in the San Francisco Bay area (8).

If working conditions remain the same, about 20% of the insulation workers at Puget Sound Naval Shipyard can be expected to have some pulmonary abnormalities distinguishable by chest X-rays after 15 years in the trade (Table IV).

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INCIDENCE OF DIAGNOSED PULMONARY FIBROSIS

TRADE	TOTAL WORKING FORCE	WORKERS WITH PULMONARY FIBROSIS OR OTHER ABNORMAL X-RAY FINDINGS	
		NUMBER	PERCENT
Shipfitters	890	6	.7%
Sheetmetal Workers	489	6	1.2%
Forge Workers	32	-	-
Welders	998	11	1.1%
Inside Machinists	536	1	.2%
Outside Machinists	490	-	-
Boilermakers	115	4	3.5%
Electricians	574	-	-
Pipe Coverers & Insulators	104	22	21.2%
Pipefitters	765	6	.8%
Shipwrights & Joiners	228	-	-
Electronics Workers	280	-	-
Painters	263	4	1.5%
Riggers	664	1	.1%
Temporary Services	143	1	.7%
Control Group (clerical workers)	420	1	.2%

TABLE I

August 1968

INTERVAL BETWEEN EMPLOYMENT AND
FIRST X-RAY INDICATIONS OF LUNG ABNORMALITY

EMPLOYEE	AGE WHEN HIRED	YEAR HIRED	CHEST CONDITION 1st NOTED	INTERVAL BETWEEN EMPLOYMENT AND 1st X-RAY ABNORMALITIES	YEARS IN PC&I TRADE AT PSNS (WHERE KNOWN)
1	22	1933	1959	26 years	33
2	22	1933	1954	21	33
3	18	1936	1958	22	30
4	27	1939	1963	24	27
5	26	1941	1947	6	25
6	39	1941	1953	12	25
7	31	1942	1961	19	17
8	30	1944	1951	7	22
9	25	1945	1963	18	21
10	28	1947	1965	18	20
11	41	1947	1964	17	20
12	36	1951	1963	12	15
13	-	1952	1962	10	-
14	45	1952	1958	6	-
15	35	1953	1962	9	13
16	49	1960	1960	-	6
17	38	1958	1960	2	8
18	54	1961	1966	5	5
19	31	1961	1961	-	5
20	57	1961	1962	1	5
21	47	1963	1965	2	3
22	22	1965	1966	1	1

TABLE II

August 1968

EMPLOYEE NUMBER 7: CASE HISTORY

DATE	OCCUPATIONAL HISTORY	PROBABLE ASBESTOS EXPOSURE	CLINICAL HISTORY
1942-52	PC&I at PSNS	unknown	X-ray: negativ
1952-60	PC&I maintenance work, apartment houses	intermittent, less than 5 mppcf *	
1960	PC&I at PSNS	less than 5 mppcf	X-ray: negativ
1961	PC&I at PSNS	less than 5 mppcf	Pulmonary abnor ities 1st noted X-ray
1967	disability retirement		Asbestos found lung during sur
1967	compensation awarded for disability due to asbestosis		

* 5 mppcf is the current recommended threshold limit value for asbestos

TABLE III

August 1968

DISTRIBUTION OF LUNG ABNORMALITIES
AMONG PIPE COVERERS AND INSULATORS

YEARS AT PSNS	NUMBER OF WORKERS	MEDIAN AGE	INCIDENCE OF DIAGNOSED PULMONARY FIBROSIS OR OTHER ABNORMAL X-RAY FINDINGS		
			NUMBER WITH PULMONARY CHANGES	PERCENT OF INCIDENCE BY GROUP	PERCENT OF INCIDENCE BY TOTAL PC&I WORK FORCE
0-5	25	37	2	8%	2%
6-10	28	47	5	18%	5%
11-15	11	43	1	9%	1%
16-20	20	48	4	20%	4%
21-25	7	51	4	57%	4%
25+	7	56	6	90%	6%

AGGREGATE = 21.6%

TABLE IV

August 1968

INSULATION MATERIAL AND USE

AMOSITE

Use: Piping, fittings, machinery units and large or irregular surfaces

Composition:	Long fiber asbestos	95%

MAGNESIA BLOCK INSULATION

Use: Tanks, boilers, valves and pipes

Composition:	Magnesium Carbonate	85%
	Long fiber asbestos	15%

ASBESTOS CEMENT

Use: Finishing and insulating, used over bare metals or insulation and as a filler

Composition:

Johns-Manville #301

Asbestos (Crysotile)	15%
Portland Cement	17%
Clay	33%
Mineral wool	20%
Diatomaceous earth	15%

One-Cote

Nodulated fiber and Asbestos (Crysotile) -	less than 50%
Portland Cement	less than 30%
Diatomaceous earth	less than 20%
Clay	less than 30%
Calcium sulfate or nitrate	less than 5%
Organics	trace

TABLE V

August 1968

UNIBESTOS

Use: Piping up to 30 inches in diameter, very little is used at this time

Composition: Finely ground asbestos fiber

Waterproof adhesive

ASBESTOS CLOTH

Use: Final coat or lagging on piping, valves, tanks, fittings, and machinery installations

Composition: There are three weights ranging from 1.25 to 2.4 lbs/sq. y

80% to 95% Crysotile or Amosite asbestos fiber with cotton fiber

FIBERGLASS

Use: Many types such as batts, sheets, molded pieces and felt are available for different applications such as vent ducts, piping and valves

Composition: Fibrous glass with binder

CORK

Use: Refrigeration and air conditioning piping

Composition: Vegetable product

RUBBER

Use: Refrigeration and air conditioning piping, replaces cork in most applications

Composition: Foam rubber

AVERAGE TIME-WEIGHTED EXPOSURE TO INSULATION MATERIALS *

OPERATION AND MATERIAL	PERCENT OF TIME EXPOSED NEW SHIP CONSTRUCTION	SHIP OVERHAUL
Installation		
Amosite installation	10%	11%
Magnesia block insulation cutting and installing	39%	35%
Asbestos Cloth cutting, glueing and fitting	20%	15%
Asbestos Cement mixing	1%	1%
Fiberglass, Rubber, and Cork	30%	13%
Rip-Out		
Amosite		4%
Magnesia block insulation, Amosite, and Unibestos		17%
Fiberglass, Rubber, and Cork		5%
Prefabrication in Shop	**Percent of Time Exposed**	
Magnesia block insulation, Amosite and Asbestos Cloth	50%	
Fiberglass, Rubber, and Cork	25%	

* Based on 6 hour work day.

TABLE VI

August 1968

NEW SHIP CONSTRUCTION

Material and Operation	Airborne Dust Concentration in Breathing Zone (Million Particles per Cubic Foot)		Percent of Time Exposed (Average) Based on a 6 hour day
	Range	Average	
Amosite: Installation	2.6 to 8.9	5.6	10%
Magnesia block insulation: cutting and installing	2.4 to 13.3	6.1	39%
Asbestos cloth: cutting, glueing and fitting	3.1 to 4.6	3.6	20%
Mixing asbestos cement	11.5 to 82.8	47.5	1% or less
Fiberglass, rubber and cork	---	---	30%

TABLE VII

SHIP OVERHAUL

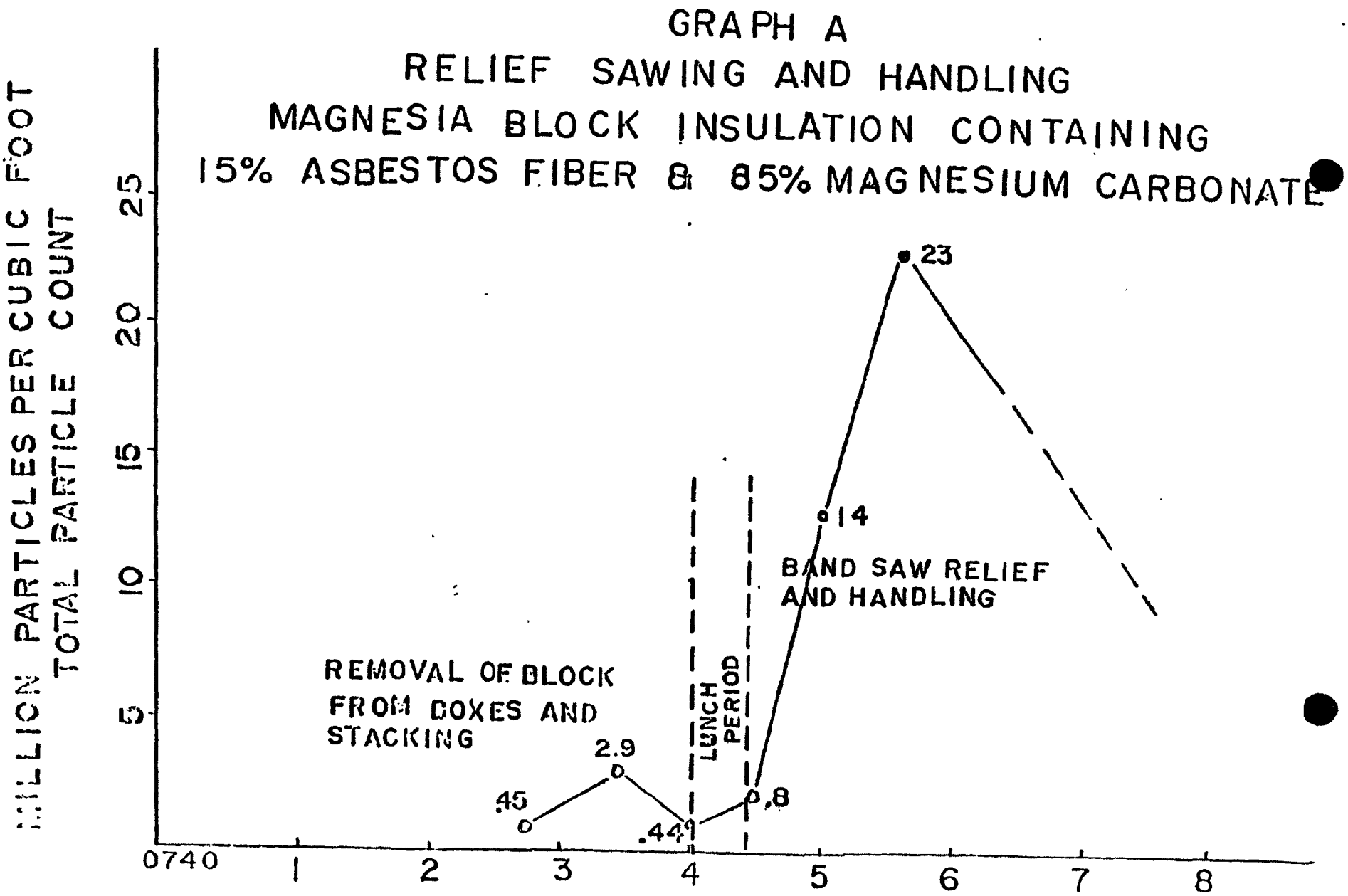
Material and Operation	Airborne Dust concentration in Breathing Zone (Million Particles per Cubic Foot) Range	Average	Percent of Time Exposed (Average) Based on a 6 hour day
INSTALLATION			
Asbestos cloth; cutting, glueing and fitting	2.8 to 5.7	4.3	15%
Magnesia block insulation: cutting and installing	3.5 to 40.5	16.7	35%
Amosite	2.6 to 8.9	4.5	11%
Fiberglass, rubber and cork	---	---	13%
RIPOUT			
Amosite	1.0 to 7.7	4.4	4%
Magnesia block insulation and asbestos cloth	2.6 to 6.8	5.0	17%
Asbestos cloth and unibestos	2.8 to 17.4	6.7	17%
Fiberglass rubber			

PREFABRICATION IN SHOP

Material and Operation	Airborne Dust Concentration in Breathing Zone (Million Particles per Cubic Foot)		Percent of Time Exposed (Average) Based on a 6 hour day
	Range	Average	
Magnesia block insulation	1.1 to 1.3	1.2 (cutting) .32 (glueing)	67%
Amosite		2.4 (cutting)	
Asbestos cloth	.39 to .41 .07 to .1 .1 to .15	.4 (sewing) .09 (glueing) .13 (pinning and marking)	
Fiberglass, rubber and cork	---	---	33%

TABLE IX

August 1968


GRAPH A
RELIEF SAWING AND HANDLING
MAGNESIA BLOCK INSULATION CONTAINING
15% ASBESTOS FIBER & 85% MAGNESIUM CARBONATE
MILLION PARTICLES PER CUBIC FOOT
TOTAL PARTICLE COUNT
5
10
15
20
25
0740
1
2
3
4
5
6
7
8
23
14
.8
2.9
.45
.44
REMOVAL OF BLOCK FROM BOXES AND STACKING
LUNCH PERIOD
BAND SAW RELIEF AND HANDLING

SELECTED REFERENCES RELATED TO ASBESTOS
EXPOSURES OF PIPE COVERERS AND INSULATORS

1. Fleischer, et al, A Health Survey of Pipe Covering Operations in Constructing Naval Vessels, J. Indust Hyg & Tox 28.9 January 1946

   An excellent reference that shows how little the exposure patterns and trade methods have changed. The authors concluded that the number of diagnosed asbestosis cases reflected the hazards, which has since been found not to be true. Most PC&I personnel do not file claims.

2. Marr, W.T., Asbestos Exposure During Naval Vessel Overhaul, AIHA J 25:264 May 1964

   Marr's work better reflects the exposure patterns and the potential hazards from asbestos exposures.

3. Selikoff, I.J., et al, Asbestos Exposure and Neoplasia, JAMA April 6 1964

   Selikoff's article suggested that a high rate of cancer was related to asbestosis.

4. Balzer, J. Leroy, Industrial Hygiene for Insulation Workers, J Occup Med, Vol 10 #1, January 1968

   This University of California researcher found in the union insulators about the same rate of pulmonary abnormalities as found at PSNS.

5. Selikoff, I.J., et al, Asbestos Exposure Smoking and Neoplasia, JAMA April 18 Vol 204 #2, 1968

   Selikoff linked smoking to high rates of cancer among asbestos workers.

6. C.A. Mangold, R.R. Beckett, D.J. Bessmer, Asbestos Exposure and Pulmonary X-ray Changes to Pipe Coverers and Insulators at Puget Sound Naval Shipyard, Industrial Hygiene Division, PSNS, August 1968

   Asbestos workers at PSNS have an average of 21% pulmonary abnormalities, compared to all other work groups. The percentage of pulmonary abnormalities grouped by years in the trade are: (1) 15 to 20 yrs----20%; (2) 20 to 25 yrs----57%; 25 yrs + ----90%.

FILE COPY

Asbestos as an Environmental Hazard

IRVING R. TABERSHAW, M.D.

IN FEBRUARY OF THIS YEAR Cooper, in a paper entitled, "Asbestos as a Hazard to Health,"[1] commented as follows: "We often try to emphasize that part of the challenge of our field is that it is at the forefront of medicine and technology where environmental hazards can first be detected in view of the relative levels of exposure and the opportunities to study populations at risk. But when the relationships are actually suspected and gradually established, very real and practical consequences become apparent. So the speculations, call them fancies if you will, that are part of the normal processes of developing new knowledge in other fields become menacing and are misused and misunderstood."

You have heard today that in industries using asbestos as a basic material, that is mining, milling, fabrication and installation, that a relationship has been established between asbestos exposure and fibrosis of the lung, lung cancer, and possibly with an increase in the incidence of mesothelioma and gastrointestinal malignancies. The incidence and severity of the pulmonary fibrosis (asbestosis) are apparently dependent on dose. This dose-response relationship in asbestos workers will probably be proven in time to be true also for the neoplasia of the lung, pleura and gastrointestinal tract. Concern, however, has been expressed that asbestos in ambient air may constitute a real threat to the entire public and not just to those occupationally exposed.

This grave implication is summarized in an article by Thomson,[2] who raises the question of "whether a small amount of asbestos in the lung bases or in the adjacent pleura or peritoneum is an effective carcinogen or co-carcinogen in humans, if it is present there for 30 years or more." This concern is reiterated by Selikoff and his coworkers[3] in a lengthy editorial with the statement: "The neoplasms now encountered associated both with industry and environment are consequent upon inhalation of asbestos associated with the limited asbestos production of some 30 years ago. The neoplasms associated with current utilization and exposure to asbestos will not be evidenced until 1990."

If this is true, it changes the order of magnitude of the hazard and places asbestos in the forefront of public health problems. It is to this allegation that I wish to address myself. A good deal of the information on which this potential threat is postulated is in the realm of "speculation" and it is not always possible to separate the "fact" from the "fancy."

DEPOSITION EXHIBIT NO. 1 C.S.R., INC.

Dr. Tabershaw is Professor of Occupational Medicine, School of Public Health, University of California, Berkeley.

Presented at the Eleventh Annual Meeting of the Western Industrial Health Conference, San Francisco, September 29 and 30, 1967.

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PLAINTIFF EXHIBIT SOA 5504

The data which lead to this speculation is mostly epidemiologic in character. There is unequivocal evidence that fibrosis of the lung (asbestosis) occurs in asbestos workers. Pleuritis is a frequent sequela of asbestos exposure and a rare malignancy of the pleura, mesothelioma, shows a remarkable increase in asbestos workers and in individuals living near asbestos-producing mines and factories. Neoplasms of the lung occur more frequently in individuals who have asbestosis or who are occupationally exposed to asbestos.

These findings, per se, would not lead to the sweeping speculations made by Thomson, Selikoff and others were it not for the additional finding that a large percentage of individuals over the entire world show "asbestos bodies" in their lungs at autopsy even though most have had no known occupational exposure to asbestos (Table I). Since "asbestos bodies" are specific for past exposure to asbestos or similar fibers, the speculation that all of us are under some increased threat of lung cancer due to the presence of ambient asbestos is valid.

Assessment of the magnitude of this threat is the crucial question. The dynamics of the situation will not permit us to wait until 1990 for a definitive answer. Forces such as the increased use of asbestos products and the general soiling of ambient air in our industrial technological society demand that we make some judgment now. If we cannot make this determination, we must direct our research into those channels which will be most likely to give us early and definitive proof.

Epidemiological studies are of prime importance and in fact present the most convincing evidence that asbestos may be dangerous to the working population, but the likelihood of proving that there is an increased risk to the public from asbestos in ambient air, by using epidemiologic methods, is very small. Even if all the factors which could influence its evaluation are known and a prospective study devised to demonstrate an effect by 1990, any conclusions from such a study would be subject to severe doubt because of the many variables which could not be controlled satisfactorily. For instance, it would be almost impossible to select a proper cohort which could be followed over the many years; the selection of matched controls would be almost insurmountable; the increasing use of asbestos and the differences in composition and possible exposure in various localities, and the complications introduced by cocarcinogens in our environment further becloud the issues and make it very unlikely that an equivocal answer could ever be forthcoming from this type of evidence alone.

TABLE I

Percentage of Asbestos Bodies in Lung Smears*

Thomson	(1963)	Cape Towne	20.4%
Thomson	(1965)	Miami	2.2%
Coune et a	(1965)	Pittsburgh	41.0%
Webster	(1965)	Johannesburg	39.2%
Meurman	(1966)	Finland**	57.6%
Anivel et al.	(1966)	Montreal	48.0%
Cooper & Tabershaw	(1966)	San Francisco	45.0%

* A consecutive unselected autopsy series.
** Thick sections

Adapted from Cooper W. Clark, Asbestos as a Hazard to Health—Fact and Speculation, Arch. Environ. Health 15:283 (September) 1967

The experimental approach can provide only tentative answers since it has been demonstrated that, while asbestos will produce irritation, plaques and malignancies in serous cavities, it is relatively inert as an over-all irritant to biologic materials. Furthermore, the translation of effect from any species of animals to man is difficult at best and is made even more questionable by marked variations in respiratory tract response by different animal species. The animal selected must most nearly resemble the human lung in order for the experiments to be meaningful. Difficulty is evidenced, for instance, in the fact that some animals, e.g. the cat and the guinea pig, will produce fibrosis and relatively few asbestos bodies, and others such as the mouse will produce relatively few asbestos bodies but no fibrosis.[4] This is probably related to the variations in the evolution of alveolar macrophages which Collet et al.[5] have demonstrated to differ among humans, cats, rabbits, and guinea pigs.

Exposure factors are not completely understood. Asbestos is said to be indestructible, but the term is a relative one; high heat, chemicals and body fluids produce changes in its physical and chemical properties in time. There is undoubtedly a difference in effect from a heavy, brief or continuous exposure as occurs in industry to a sustained minor exposure which may occur from ambient air pollution. Selikoff[6] has iterated many times that there is an increasing use of asbestos (Table II) and surmises that we are presently seeing lung malignancies "consequent upon inhalation of asbestos associated with the limited asbestos of some 30 years ago." The implication is clear that if this relationship (tons of asbestos produced to incidence of lung malignancy) is true, the multifold increase in its use has started an upward trend which will be reflected for the next 30 years and more. This extrapolation is unwarranted.

Increased production cannot be equated with increased hazard, especially in a substance like

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TABLE II
World Production of Asbestos*

YEAR	NO. TONS
1880	500
1900	35 000
1920	200 000
1930	450 000
1950	1 300 000
1960	2 300 000
1963	2 [illegible] 000
1964	3 540 000
1965	3 600 000

*Adapted from Various Sources

asbestos which is used for the most part in products which are unlikely to become airborne. The largest amount—one can only guess at the percentage—goes into asbestos products which, once applied do not disintegrate, such as building materials and floor tile. A fair estimate might be that some 10 to 15% of the asbestos in the approximately 3,000 end-products containing this mineral, e.g. brake lining and asbestos textiles, may by deterioration, weathering, heating, etc., become airborne. However, there are no studies of asbestos concentrations in ambient air. Indications that asbestos may be carried considerable distances in the airstream[7] are based upon: (1) Analysis of deposits on the surface of the earth, snow, etc., (2) evidence that significant numbers of people with mesothelioma worked or lived near asbestos mines or plants; and (3) evidence that a significant population has asbestos bodies in their lungs at autopsy. But as yet the type, amount and particle size of ambient airborne asbestos has not lent itself to systematic detection and identification. It should also be borne in mind that asbestos deposits have mineral outcrops and hence there is a strong possibility that asbestos in ambient air may be a natural phenomenon and not only a result of increased industrial activity.

Clinical experience indicates that the appearance of asbestosis (pulmonary fibrosis) is dose-dependent. Children have died of asbestosis and cor pulmonale after a few years of an overwhelming exposure "within large shipping bags, trampling down fluffy amosite asbestos which all day long came cascading down over their heads."[8] Sluis-Cremer[9] gives an average in one factory in South Africa of 6 years at work before asbestosis was manifest. Jacob and Anspach,[10] in Dresden, determined an over-all average of 30 years before the asbestosis became disabling. X-ray changes are induced in most cases after 20 years' employment as an insulator (Selikoff, p. 147). One of our cases showed evidence of the fibrotic process on lung biopsy some 22 years after a heavy exposure of less than 2 years' duration.

TABLE III
Workers Exposed to Asbestos in the United States
(1963 Census of Manufacturers—Dept. of Commerce)

RAW PRODUCT MANUFACTURING	NO. WORKERS
Asbestos Products (1963)	19 516
Asbestos Paper Products (1963)	11,874
Asbestos Mines & Mills	497
Cement*	[illegible]
USE OF MANUFACTURED PRODUCTS	
Insulation Workers (1965)	18 000
Approximate Total U.S. workers exposed	50 000+

While long latency is usual and progression of the fibrotic process may continue long after the exposure has ceased, there is little information forthcoming regarding quantitative data on the dose which each individual was exposed to. Lung clearance studies indicate a marked difference in the effects of short, heavy dust exposures as compared to minor, low exposures. Heavy dust inhalation incites an acute flammatory reaction and stasis in the respiratory bronchiole and adjoining alveolar ducts and may provide a nidus for a continuing effect.

That asbestosis, i.e. the pneumoconiosis, does not present a major problem for the future is mostly due to the fact that the occupational population at risk, which is difficult to determine exactly, is still quite small and can never reach large numbers (Table III).

While members of the construction industry, for example plasterers and some automobile workers, e.g. brake repairmen, may have greater exposures than insulators working directly with asbestos products, it is safe to say that the kinds of exposure experienced in industry will never affect any sizable group in the general or working population.

The threat from asbestos, however, lies in its possible association with lung cancer; and the expectation that there will be no increase in the mortality from this disease among exposed workers is not so sanguine. Cancer of the lung occurs more frequently in those with occupational exposure, whether or not they develop fibrosis, and this evidence is amply substantiated (Table IV). Two aspects are not known, the pathogenic factors and the dose of asbestos which produce this increased susceptibility. We do not know whether asbestos has properties of primary carcinogenicity or whether its effect is entirely synergistic. That other factors must potentiate the asbestos exposure has been postulated by Selikoff and Hammond[11] who show that asbestos insulators who smoke have about 90 times the incidence of lung

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TABLE IV

Epidemiologic Studies of Asbestos Exposures and Lung Cancer*

PLACE	AUTHOR	YEARS FOLLOWED	NO. WITH LUNG CANCER	COMPARISON Ob/Expected
United Kingdom	Doll	31	11/39 deaths	+0.8 expected
Quebec	Braun & Truan	5	9/67 deaths	−0.0 expected
Pennsylvania	Mancuso & Coulter	20	9/1.6 deaths	−5.6 expected
New York & N. Jersey	Selikoff et al	30	45/235 deaths	−0.1 expected
California	Dunn & Wade	7.1	10/41 deaths	−2.8 expected
Dresden	Jacob & Ansbach	39	24/150 deaths	−11.9 expected
U.S.A.	Enterline deaths	24/286	12	−11.9 expected

*Adapted from W. Clark Cooper—Asbestos as a Hazard to Health—Fact and Speculation. Arch. Environ. Health, 15:285 (September) 1967.

malignancy of smokers who are not exposed to asbestos. The metal complexes in the asbestos fibers (nickel, chromium, etc.), benzpyrene and others carcinogenic agents absorbed on the asbestos, allergic response which produces altered immunity, and the increased burden from urban populations or smoking are now postulated to be the major co-factors which make asbestos carcinogenic.[12]

A dose too small to produce fibrosis, i.e. asbestosis, may, nevertheless lead to lung cancer. A dose large enough to produce asbestosis may cause malignancy if the worker does not die first from his pneumoconiosis. However, many men, after a lifetime of work with asbestos products—specifically insulators—show no evidence of any untoward pulmonary pathology. Again, in all of these, the amount of asbestos inhaled is unknown. When the dose is known to be related to the incidence and severity of a disease (and it grossly appears to be the case in asbestosis), it is safer to postulate that a non-threshold relationship exists. From a practical standpoint, however, asbestos would be an unusual disease agent if it did not have such a threshold. "Proof," however, will always be a speculation, as it is for example in determining the carcinogenic level of radiation. There is no way of estimating this threshold level, which at present needs more industrial hygiene studies.

Our findings support those of other investigators who state that pleuritis occurs in about 25% of insulation asbestos workers. The incidence of mesothelioma in this group is extremely large, considering the rarity of the tumor. Selikoff reporting on a series of 124 deaths from neoplasia in insulators found 10 with mesothelioma.[6] This malignancy has also occurred in increased incidence in some populations which did not work directly with asbestos. In a very large proportion in South Africa and in a considerable number of the the victims of this malignancy in the United States, environmental exposure is postulated as the cause since they lived close to asbestos plants using especially amosite or crocidolite.[13] The evidence is strong that the source of the agent was inhaled airborne from the asbestos factories or mines, even though many of these individuals did not show an underlying asbestotic fibrosis. However, the ambient concentration or indeed the specific type of asbestos to which the person was exposed and which could be responsible for the disease has never been determined, either in the neighborhood or in the working environment.

An increased incidence of gastrointestinal malignancy is still being investigated, but the statistical inference is strong that there is an increased susceptibility in those exposed to asbestos. This will probably be clarified in time. Emphysema or chronic bronchitis, if it does occur as a result of inhalation of asbestos dust, would always be obscured by the underlying pneumoconiosis; and the diagnosis of these conditions would always be considered a secondary rather than a primary disease.

A link and perhaps the key to the problems lie in understanding the role of the "asbestos body" in the pathogenesis of the different tissue responses. Our experience supports the statement of Gough:[14] "Asbestos is diagnosed histologically by the association of asbestos bodies and asbestos fibers with fibrosis." However, what the "asbestos body" means in individuals who apparently have never been exposed to asbestos is not clear. If this can be explained, inferences then could be made with some certainty regarding the potential environmental hazard.

The presence of "asbestos bodies" in the lung is a biological indicator of *exposure* to asbestos. Whether or not other mineral fibers can produce an "asbestos body" is not pertinent in an environmental sense. The highest concentration of mineral fiber in ambient air is overwhelmingly asbestos and hence one can assume that "asbestos bodies are almost entirely derived from asbestos fibers." "Asbestos bodies" probably can be found in every lung at autopsy if searched for long and hard enough and an occasional one is adventitious and simply indicates that mineral fibers make up a part of the dust in our environment.

The fibrosis of asbestosis is nonspecific and has no special structural characteristics except for the presence of "asbestos bodies." Hence, interstitial

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fibrosis without the presence of "asbestos bodies" cannot be called asbestosis. Conversely, however, "asbestos bodies" in sputum or lung tissue without fibrosis is not asbestosis but simply an evidence of exposure. Phagocytosis by macrophages is a major cleansing mechanism of the lung; they engulf the offending mineral fiber producing the typical "asbestos body." No relationship has been established between the number, type, or size of "asbestos bodies" and the time of exposure, fiber size, concentration, or form of asbestos

Individuals who show "asbestos bodies" on lung smears at autopsy do not seem to have an increased incidence of any specific pulmonary disease. "Asbestos bodies" may be found in lung smears and not in the correlating tissue. Cauna et al.[15] found that only 4% of lungs showed "asbestos bodies" on histological section, whereas smears of the same lungs showed "asbestos bodies" in 41% Our findings are similar—lung smears in consecutive autopsies without known exposure to asbestos revealed an incidence of "asbestos bodies" in 45% while search of the matched lung tissue yielded only 3%. This may be a function of the amount of material available for study since the lung *smear* represents a much greater anatomic area than the lung tissue studied in histological section. Meurman found a considerably higher correlation of lung smears to lung sections but he used special techniques to demonstrate "asbestos bodies."

Our own interpretation is that while the differences in amount of material which is available for study may account for some of the disparity, there is evidence that mineral fibers are cleansed through the macrophage system and hence found in the alveoli and bronchial lumina but do not enter into the interstitial tissue. Only overwhelming exposure or perhaps some other factors which permit fibers to penetrate with formation of interstitial "asbestos bodies" lead to lung malignancy, pleuritis and mesothelioma. "Asbestos bodies" are not seen frequently in routine histologic study of lung pathology. This is not an oversight by the pathologist since sections of lung especially stained and studied for "asbestos bodies" do not reveal an increased incidence (only about 3%).

Our present view is that asbestos fibers are phagocytized in the airways and excreted in the sputum. In some instances the fibers penetrate the bronchial tree, giving rise to fibrosis, or into the pleura producing pleuritis. Co-factors, which are not completely understood at present, operating with the asbestos cause an increase in lung cancer susceptibility and in the appearance of mesothelioma. As the role of the "asbestos body" becomes clearer and more data becomes available on the distribution, type, fiber size, and concentration of airborne asbestos and the role of co-factors, the better we will be able to assess the potential environmental hazard from this ubiquitous mineral

In summary, if one accepts the assumptions (1) that asbestos minerals increase the risk of lung cancer in occupational groups, (2) that they lead to an unusual risk of mesothelioma of the pleura and peritoneum in occupational groups and those living near asbestos plants, (3) that such malignancies usually result from exposures 30 to 50 years earlier, (4) that the "asbestos bodies" found in from 25 to 50% of lung smears from routine autopsies are probably due to asbestos in most cases, (5) that these "asbestos bodies" may result from recent exposures as well as those many years earlier, (6) that world production and use of asbestos has increased from 500,000 tons to 3,500,000 tons in 30 years, it is important to consider whether or not asbestos is a major threat to public health. One is not yet justified in such a conclusion, in view of the fact that much of the world tonnage goes into uses that do not lead to air contamination; asbestos is not actually indestructible, that the effects are dose-dependent, and that low doses probably lead to lower rates and longer latency.

Nevertheless, there is need for epidemiologic studies directed to groups with intermediate exposures and for evaluation of the beneficial effects of cessation of smoking. Another need is for experimental and industrial hygiene studies to determine the nature and degree of exposure. Biologic studies centered on the meaning of the "asbestos body" are crucial to understanding the clinicopathologic response. Strict control of industrial and neighborhood environments is essential, but it is premature to extrapolate from the effects of heavy exposures to minor and low level exposures.

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The Urban Environment

Natural resources have always been defined as those parts of the natural environment that have value to mankind because the demand for them exceeds the limited supply. It is in this sense that "new" resources, other than the traditional ones of agricultural land, water, and minerals, have increased in value.

In this light *urban land* is particularly valuable. Just as we had to learn to use agricultural land with special regard for its qualities and the demands made on it, so we will have to change our view of urban land. Today its use is haphazard and largely unthinking. We have not yet learned to ask ourselves, for example, whether we want to use a third, a half, or two thirds of our urban land for the movement and storage of cars. How much are streets worth to us—underground, on the surface, above ground?

Equally important, or possibly even more important, we now have to conceive of yet another critical resource: the third dimension of the city—the space over the urban land. This is used even more haphazardly than the land itself. Just on the horizon is a host of problems caused by elements competing for this limited resource—high-rise buildings, interbuilding walkways, high-level and air-cushion highways, and private rooftops. This space over the city is an asset that must be conserved, developed, and used with the greatest care. Two-dimensional land-use planning is out-of-date, and we urgently need to learn how to prepare three-dimensional plans.

—*From "Modernizing Urban Development" by Harvey S. Perloff in Daedalus—Summer, 1967.*

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