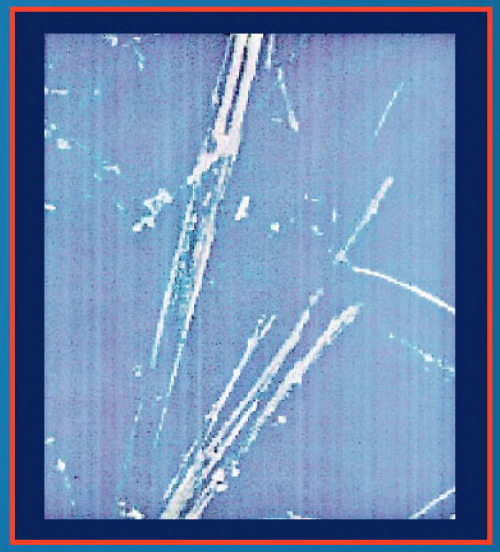
4121-EP564

Different Types of Asbestos

Amosite



Crocidolite

Chrysotile

Amphiboles

- long, thin fibers
- straight, rigid fibers

Serpentine

- curvy, wavy, flexible
- readily break down
- released from lung

Mr. Toole Was Exposed to Amosite Pipe Insulation – Probably the Single Most Common Cause of Mesothelioma

Q. (By Mr. Buck) Is it fair to say, Mr. Toole, that the pipe insulation shown in Exhibit 25 fairly and accurately represents the pipe insulation you saw being removed and installed by the North Brothers employees at Great Northern Paper?

A. Yes.

Mr. Toole Was Exposed to Amosite Asbestos

A. I was a state meat inspector for 11 years.
Q. And during what period of time was that?
A. 1971 through 1982.
Q. And what were your duties as a state meat inspector during that time period?
A. I was responsible for all operations as far as cleanliness, inspecting the meat that went on in about five different plants that I had under me.
Q. And what occasion would you -- would you have had as a state meat inspector to have worked with or around any asbestos-containing products?
A. Well, in packing houses, especially in your slaughter areas and in every department, they use steam to heat the water for purposes of sanitation and cleaning up, whether it be walls and floors or ceilings or the actual equipment they used, and all of these pipes had that type insulation on it.
Q. And when you are talking about those pipes and that type of insulation --
A. Right.
Q. -- you are talking about the insulation shown on Exhibit 25?
A. Yes.

Q. (By Mr. Buck) As far as the location of plants or the names of plants, can you identify for the record what plants and places you're talking about?

A. My home plant was in Bainbridge at Tillman Brothers Packing Company.

Q. And the type of insulation that you have described in your testimony, is that the type of insulation that was present there at Tillman Brothers?

A. Yes.

MR. WOOD: Objection, form, leading, vague, foundation.

Q. (By Mr. Buck) What -- what type of insulation did you observe at Tillman Brothers in Bainbridge between 1972 and -- I'm sorry, 1971 and 1982?

A. It looks just like this in Exhibit 25.

The Plaintiffs' Factual Evidence About Ford is Unreliable and Has Changed in Response to Information Provided by Ford

SWORN INFORMATION FORM OF JIMMY L. TOOLE

COMES NOW, JIMMY L. TOOLE, and to the best of his personal knowledge, as well as to the best of his belief based upon information provided to him by co-workers and other sources of information relating to product content, and provides the following information in support of his Complaint as contemplated by the provisions of O.C.G.A. § 51-14-7:

(1) Exposed person's name: **Jimmy L. Toole**

Address: **8236 Sealy Circle, Donalsonville, Georgia 39845**

Date of birth: **02/21/1948**

Social security number: **XXX-XX-6500**

Marital status: **Divorced**

Claimant expressly reserves the right to supplement the information provided in this document to the extent that exposure to additional asbestos containing products is established in the future.

(2) If the exposed person alleges exposure to asbestos or silica through the testimony of another person or other than by direct or bystander exposure to a product or products, the name, address, date of birth, social security number, and marital status for each person by which claimant alleges exposure, hereafter the 'index person,' and the claimant´s relationship to each such person: **N/A**

Index Person's Name:

Address:

Date of birth:

Social security number:

Marital status:

Claimant's Relationship to Index Person:

(3) The specific location of each alleged exposure:

Plaintiff has had various exposure locations in and around Seminole County,

SWORN INFORMATION FORM OF JIMMY L. TOOLE

COMES NOW, JIMMY L. TOOLE, and to the best of his personal knowledge, as well as

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(5) The beginning and ending dates of each alleged exposure as to each asbestos-containing product or silica-containing product for each location at which exposure allegedly took place for plaintiff and for each index person:

Manufacturer:	Product:	Dates of Use:
Georgia-Pacific, LLC and its predecessor entities	Ready Mix Joint Compound, All-Purpose Joint Compound	1967-Mid 1970's
ACDelco	Brakes	1963-2004
[redacted]	Arc Brake Grinder	1992-2004
Atlas	Brakes	1963-1989
Bendix Commercial Vehicle Systems, LLC	Brakes	
Bondex, Inc.	Dry Mix Joint Cement, Spackling	1967-Mid 1970's
[redacted]	Brakes	
Ford Motor Company	Transmission Parts, Clutches, Brakes	1979-2004
Ford Motorcraft	Brakes	1979-2004
General Motors Corporation	Brakes	1963-2004
Genuine Parts Company/NAPA	Transmission Parts, Clutches, Brakes	1963-2004
Mopar/Chrysler, LLC	Brakes	1963-2004
PneumoAbex	Brakes	1963-2004
[redacted]		
Western Auto Supply Co.	Transmission Parts, Clutches, Brakes	1963-2004

(6) The occupation and name of employer of the exposed person at the time of each alleged exposure:

Family Farmer, Self Employed 1963-1989;

Paper Mill Worker, Great Northern 1967-1968;

Meat Inspector, State of Georgia, 1974-1982;

Sales Manager, Don Kirksy GM, 1992-1999;

Manager, Kay Ford, 2000-2004.

Mr. Toole's Sworn Testimony in April / May 2009

- Customer exposures to grinding at GM and Ford dealerships on service visits with father
- One brake job on a 1966 Ford Galaxy 500 using "Motorcraft" brakes
- Two brake jobs on a 1968 Ford Mustang using "Motorcraft brakes
- Undefined number of brake jobs between 1980-1988 on a 1979 Ford F-150 truck using "Motorcraft" brakes
  - Annual changes of valve cover gaskets
- One brake job on 1984 Ford Ranger truck using "Motorcraft" brakes
- Bystander exposure as Salesman at Kay Ford 2000-2004

Mr. Toole's Sworn Testimony in April / May 2009

- Customer exposures to grinding at GM and Ford dealerships on service visits with father
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- Bystander exposure as Salesman at Kay Ford 2000-2004 [No "arc brake grinder" at dealership]

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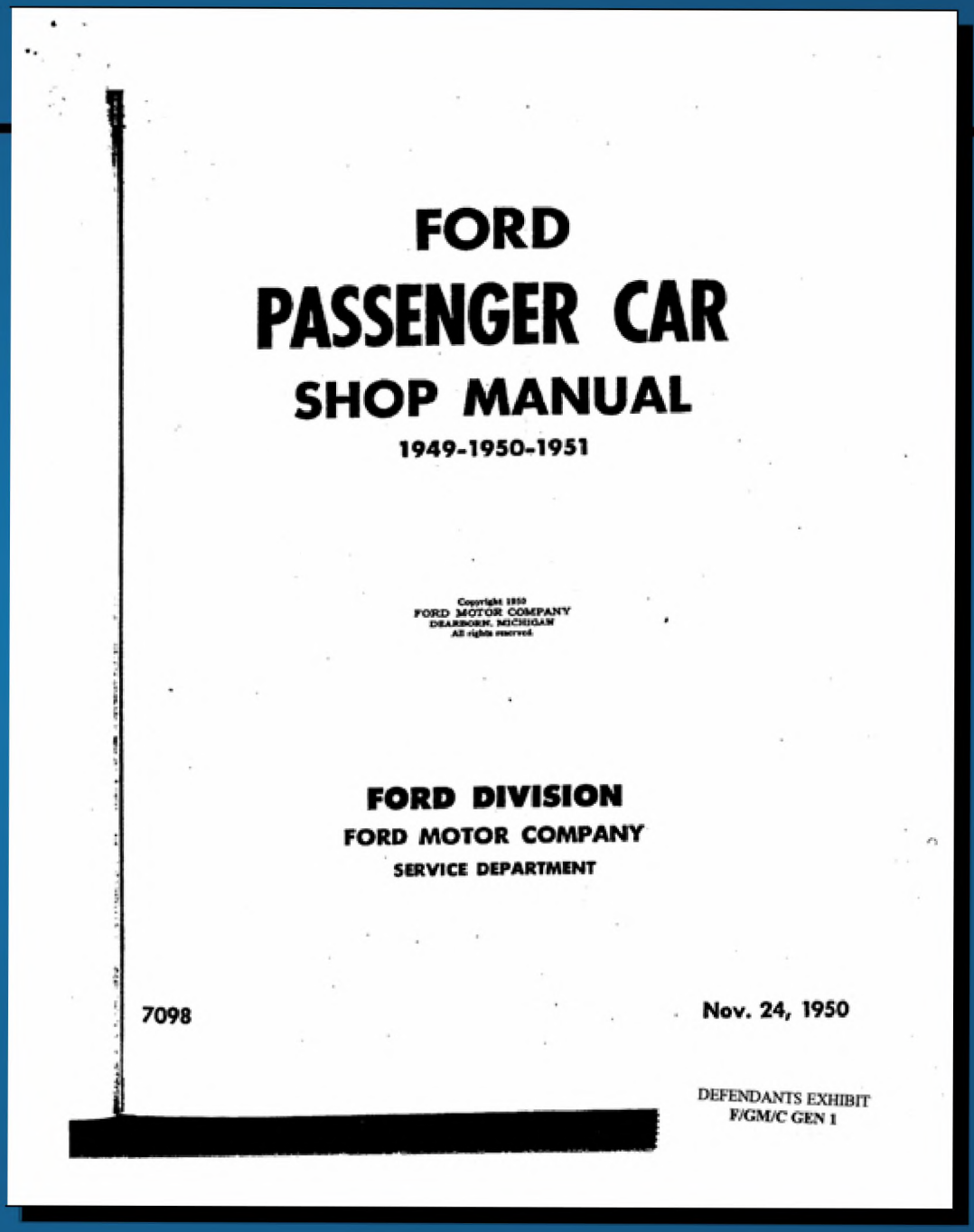


FORD
PASSENGER CAR
SHOP MANUAL
1949-1950-1951

FORD DIVISION
FORD MOTOR COMPANY
SERVICE DEPARTMENT

7098

Nov. 24, 1950

1950

FORD

PASSENGER CAR

SHOP MANUAL

burrs or high spots. Each kit contains primary linings, front and rear brakes. Each kit contains primary linings, secondary linings, and the necessary rivets. These linings are ground in production and do not require grinding after installation.

lining on the shoe, then install the

FORD MOTOR COMPANY

SERVICE DEPARTMENT

7098

Nov. 24, 1950

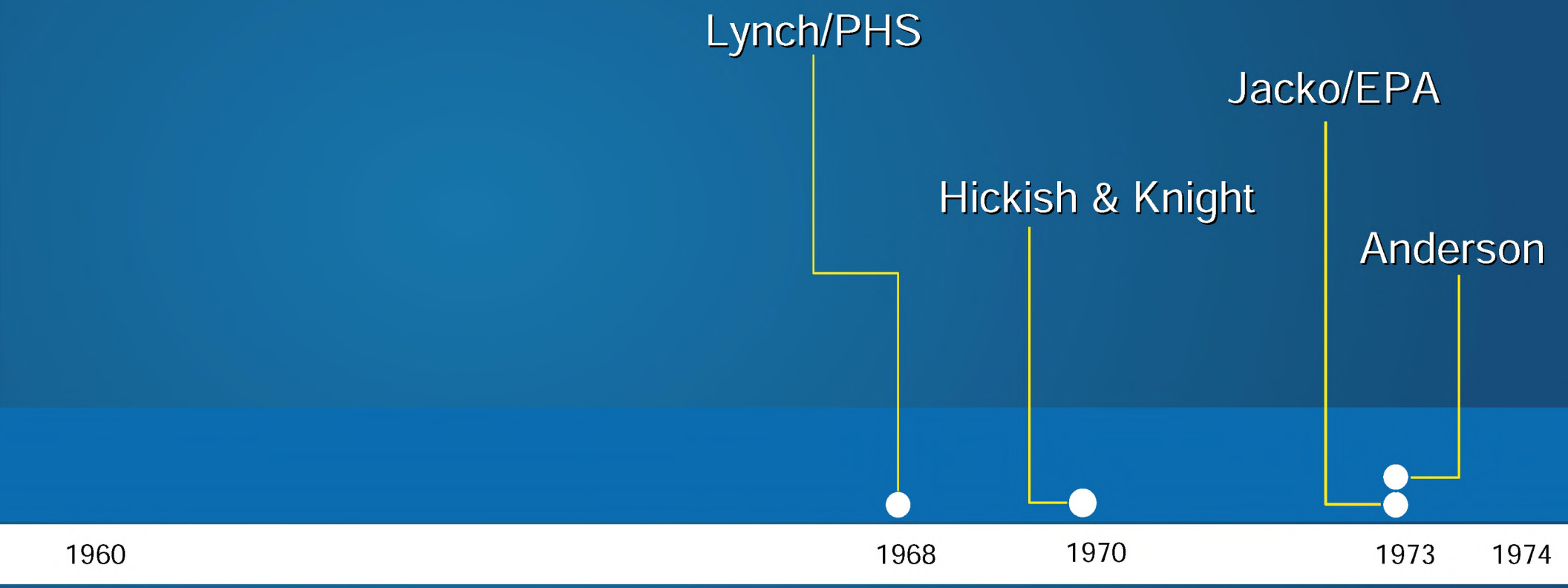
DEFENDANTS EXHIBIT
F/GM/C GEN 1

1950

Ford Acted Responsibly in Investigating Concerns Raised about Potential Risks from Friction Products

Brake Emission and Characterization Studies (1968-1973)

Measuring the Dust – Ford Takes the Lead

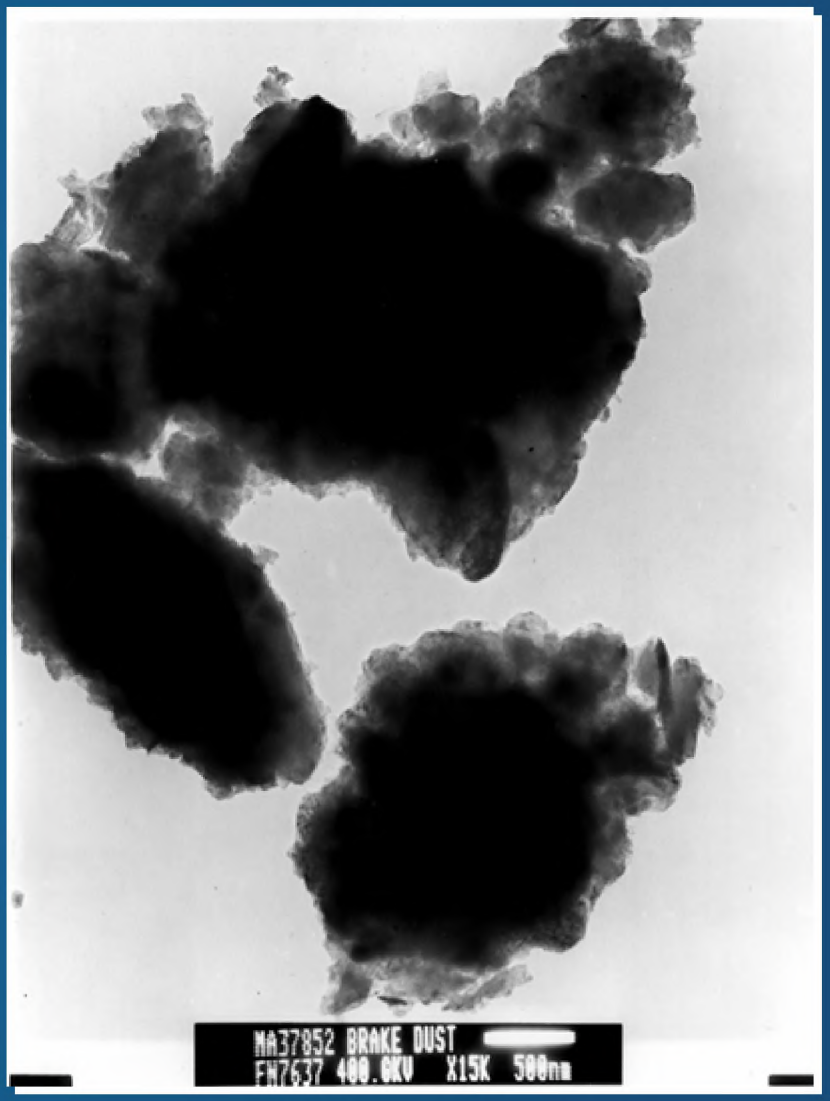


Asbestos in Brake Dust?

- Lynch 1968: less than 1%
- Hickish 1970: less than 1%
- Jacko et al. 1973: 0.23%
- Anderson et al. 1973: 0.05%

Forsterite Conversion

Chrysotile Fibers

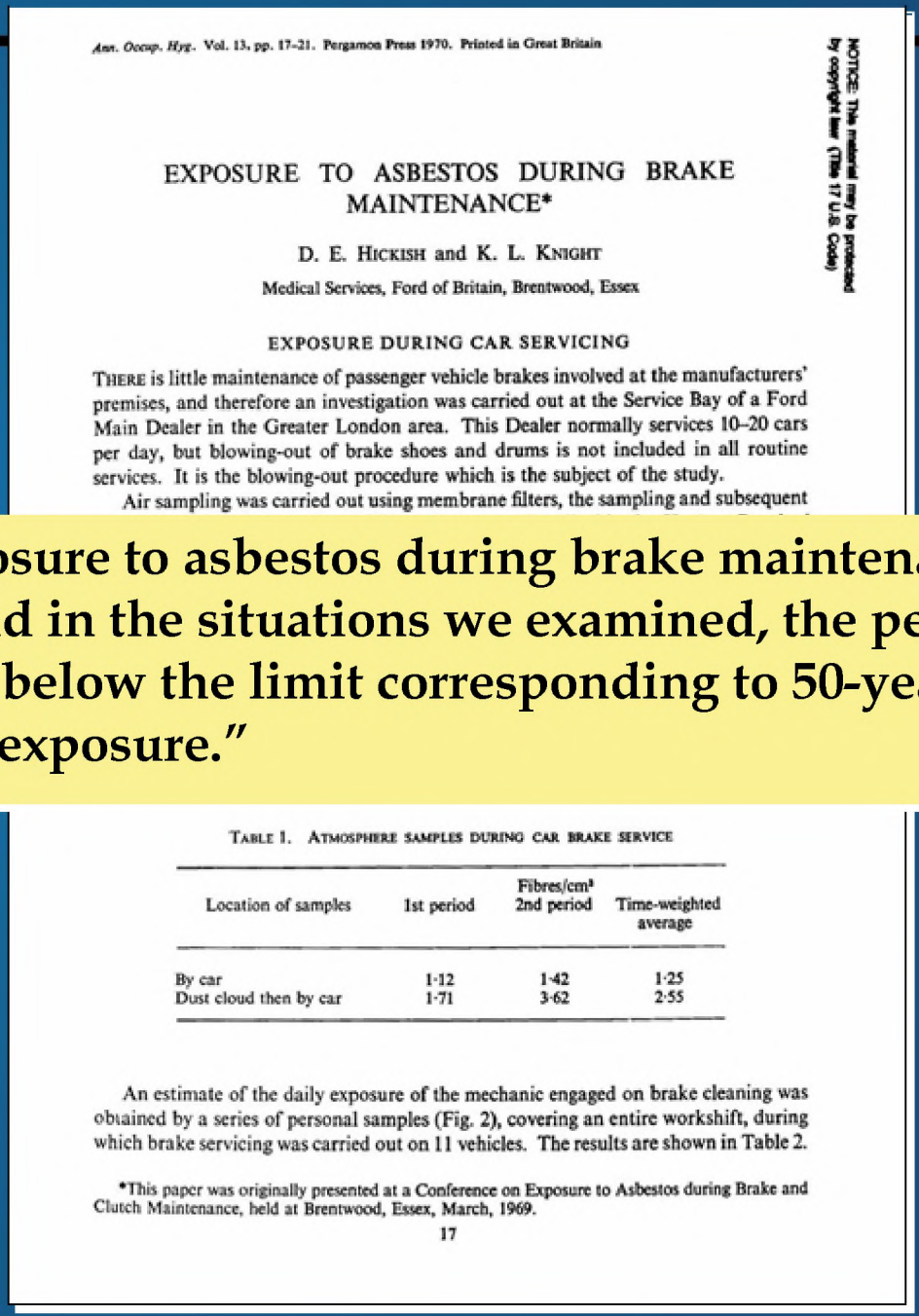


Brake Wear Debris

Source: McCrone Associates; brake dust sample provided by Arne Anderson

Hickish and Knight (1970)

Hickish, DE & KL Knight. 1970. Exposure To Asbestos During Brake Maintenance. Ann Occup Hyg 13:17-21.



Ann. Occup. Hyg. Vol. 13, pp. 17–21. Pergamon Press 1970. Printed in Great Britain

EXPOSURE TO ASBESTOS DURING BRAKE MAINTENANCE*

D. E. Hickish and K. L. Knight

Medical Services, Ford of Britain, Brentwood, Essex

EXPOSURE DURING CAR SERVICING

There is little maintenance of passenger vehicle brakes involved at the manufacturers' premises, and therefore an investigation was carried out at the Service Bay of a Ford Main Dealer in the Greater London area. This Dealer normally services 10–20 cars per day, but blowing-out of brake shoes and drums is not included in all routine services. It is the blowing-out procedure which is the subject of the study.

Air sampling was carried out using membrane filters, the sampling and subsequent

"Our investigations show that exposure to asbestos during brake maintenance is not as severe as was anticipated, and in the situations we examined, the personal exposure of the operators was below the limit corresponding to 50-year exposure."

Table 1. Atmosphere samples during car brake service

Location of samples	Fibres/cm³ 1st period	Fibres/cm³ 2nd period	Fibres/cm³ Time-weighted average
By car	1·12	1·42	1·25
Dust cloud then by car	1·71	3·62	2·55

An estimate of the daily exposure of the mechanic engaged on brake cleaning was obtained by a series of personal samples (Fig. 2), covering an entire workshift, during which brake servicing was carried out on 11 vehicles. The results are shown in Table 2.

*This paper was originally presented at a Conference on Exposure to Asbestos during Brake and Clutch Maintenance, held at Brentwood, Essex, March, 1969.

17

1970s - Ford Agrees to Fund Mt. Sinai School of Medicine to Investigate Garage Exposures in NYC

irregular or nodular interstitial opacities of limited extent and intensity on their films. Silica or asbestos exposure had occurred in 22% of the work force examined. Such exposures had occurred in various job categories in the titanium facility as well as in prior occupations. Eight of the 26 workers with abnormal X-rays had had silica or asbestos exposure. No clear pattern of restrictive disease in relation to X-ray findings could be seen.

Summary and Conclusions

Clinically significant or symptomatic pulmonary disease was infrequent in a survey of 207 currently employed production workers in a plant producing titanium dioxide from ilmenite ore. Evidence of airways obstruction was found in 47% of all workers (including 38% of workers who had never smoked regularly). This abnormality was not frequently accompanied by disabling shortness of breath. Despite the fact that approximately 90% of the group of workers examined had worked for 20 years or more, radiological changes consistent with pneumoconiosis were relatively few, and unrelated to the respiratory abnormalities observed.

We conclude (with the caveats inherent in a prevalence study in which only half of the eligible long-term workers were examined) that occupational exposure associated with titanium production by the sulphate process may commonly cause undesirable irritation of the upper and lower respiratory tract and functional abnormalities of the lung, but does not result in an important incidence of serious occupational lung disease. Individual workers may, however, suffer unwanted abnormalities. These findings, of course, do not speak to the presence or absence of increased risk of malignant pulmonary disease. This is being separately studied.

Acknowledgment: This research study was supported in part by National Institute for Occupational Safety and Health Contract CDC-99-74-91.

REFERENCE

Miller A, Chuang M & Selikoff I J
(1976) *American Review of Respiratory Diseases* 113, Suppl., p 89 (abstract)

Asbestos Content of Dust Encountered in Brake Maintenance and Repair

by A N Rohl PhD, A M Langer PhD, R Klimentidis BA, M S Wolff PhD and I J Selikoff MD
(*Environmental Sciences Laboratory, Mount Sinai School of Medicine of the City University of New York, New York, NY 10029*)

Asbestos in Brake Linings

The composition of automotive brake linings includes chrysotile asbestos fibre which comprises about 50% of the friction material. The exposure of garage workers to asbestos during brake lining maintenance and repair has recently been investigated (Rohl *et al.* 1976). This important issue was studied because a large labour force is potentially exposed (over one million people in the United States alone). Consequently it was thought essential to determine if chrysotile fibre survives braking, and to measure the amounts liberated as an aerosol during maintenance and repair operations.

Investigators in the past have expressed doubt as to whether chrysotile fibres can survive the high temperatures generated during braking (Lynch 1968, Hickish & Knight 1970, Hatch 1970). Chrysotile is alleged to be subjected to temperatures in excess of 800°C, which would cause its thermal transformation to forsterite or to an amorphous magnesium silicate phase. While 'hot spots' up to 1000°C may be attained (Carroll 1962), the heat distribution is nonuniform, and other processes, in addition to thermal wear, contribute to degradation of brake linings. For example, abrasion and macroshear may also cause physical breakdown (Burwell 1957, Mizutani *et al.* 1973). Accordingly, brake lining disintegration by these mechanisms may liberate partially altered or even unaltered chrysotile fibres.

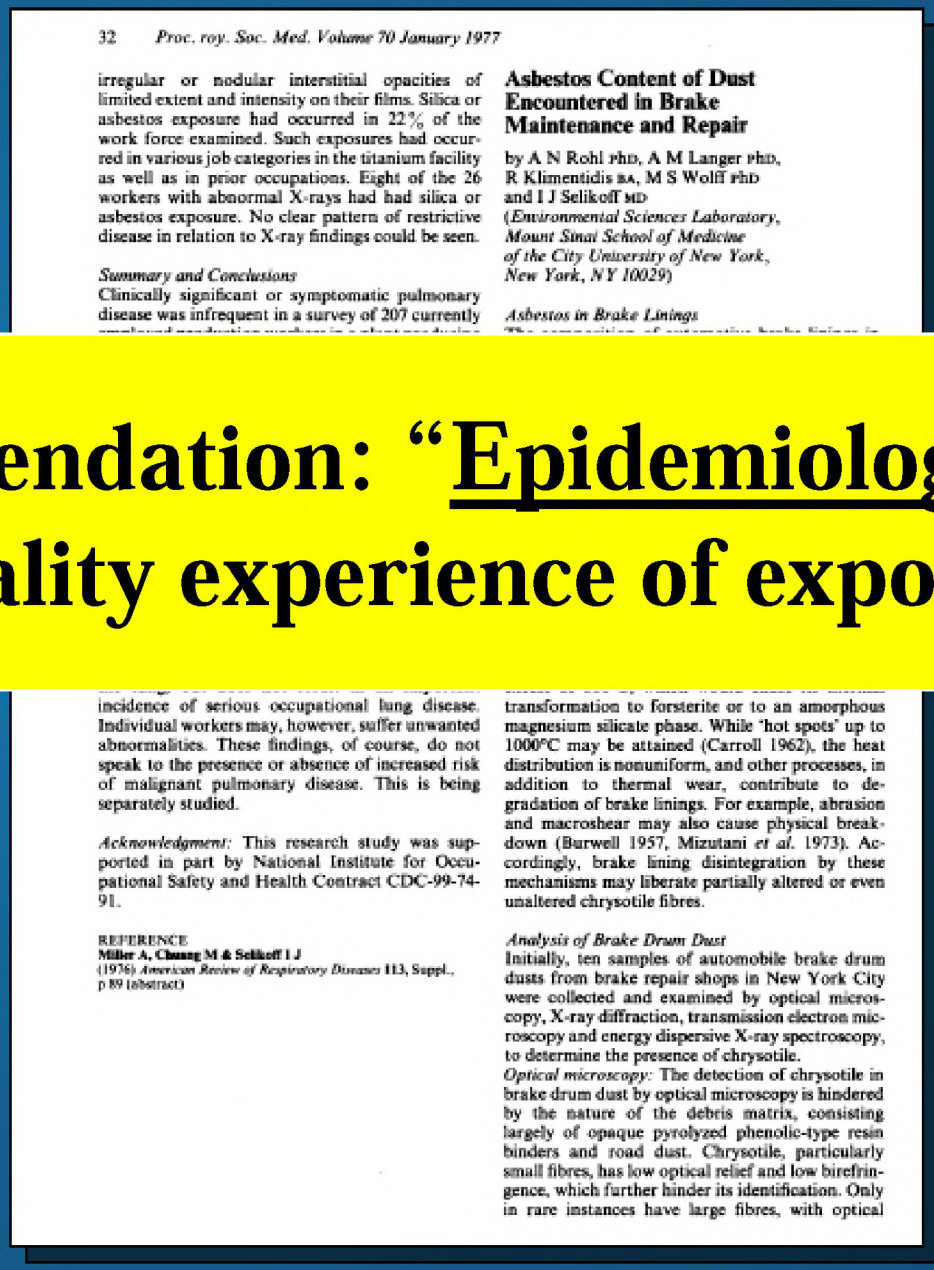
Analysis of Brake Drum Dust

Initially, ten samples of automobile brake drum dusts from brake repair shops in New York City were collected and examined by optical microscopy, X-ray diffraction, transmission electron microscopy and energy dispersive X-ray spectroscopy, to determine the presence of chrysotile.

Optical microscopy: The detection of chrysotile in brake drum dust by optical microscopy is hindered by the nature of the debris matrix, consisting largely of opaque pyrolyzed phenolic-type resin binders and road dust. Chrysotile, particularly small fibres, has low optical relief and low birefringence, which further hinder its identification. Only in rare instances have large fibres, with optical

Rohl et al. (1977)

Mt. Sinai School of Medicine Investigation of New York City Garage Exposures in 1977



32 *Proc. roy. Soc. Med. Volume 70 January 1977*

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Asbestos in Brake Linings

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Recommendation: "<u>Epidemiologic studies</u> of the mortality experience of exposed workers"

Rohl et al. (1977)

Epidemiologic Studies of Mesothelioma in Auto Mechanics: Research Affiliations and Funding Sources

	Research Conducted By:	Research Funded By:
McDonald and McDonald (1980)	St. Mary's Hospital Medical School, London	Asbestos Mining Association
Petersen and Milham (1980)	San Francisco Bay Area Resource for Cancer Epidemiology and Washington State Department of Social and Health Services	State of California Department of Health and National Cancer Institute
Teta et al. (1983)	Yale School of Epidemiology and Public Health	Yale University and National Cancer Institute
Olsen and Jensen (1987)	Danish Cancer Society	Danish Cancer Society
Jarvholm and Brisman (1988)	Department of Occupational Medicine, Sahlgren Hospital	Swedish Metalworkers Union and Swedish Work Environment Fund
Hansen (1989)	Institute of Community Medicine University of Odense	The Danish National Anti-Cancer League
Gustavsson et al. (1990)	Karolinska Hospital and National Institute of Occupational Health	Folksan Insurance Group
Spirtas et al. (1985; 1994)	National Cancer Institute	National Cancer Institute
Woitowitz and Rodelsperger (1994)	Justus-Liebig University Germany	German Federal Ministry of Science and Technology
Coggon et al. (1995)	University of Southampton	Health and Safety Executive (U.K.)

= Research = Medical = Government = University = Industry

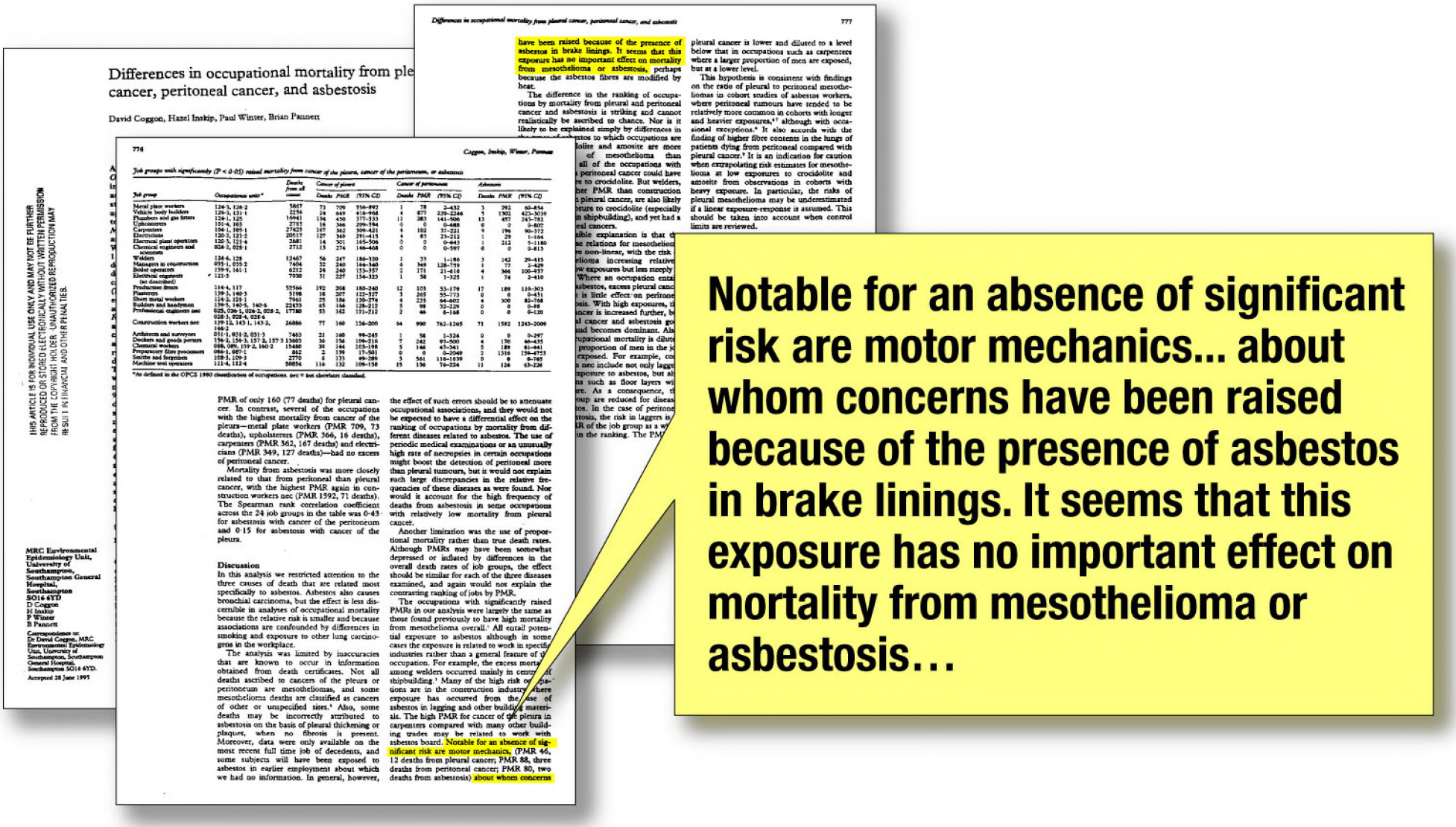
Epidemiologic Studies of Mesothelioma in Auto Mechanics: Research Affiliations and Funding Sources

	Research Conducted By:	Research Funded By:
Teschke et al. (1997)	University of British Columbia	British Columbia Health Research Foundation
Agudo et al. (2000)	Institute of Epidemiology and Clinical Research	Health Research Fund of the Spanish Ministry of Health
Milham and Ossiander (2001)	Washington State Department of Health	Washington State Department of Labor and Industries
National Institute for Occupational Safety and Health (2002)	NIOSH	U.S. Department of Health and Human Services
Hansen and Meersohn (2003)	Danish Cancer Society	Danish Cancer Society
Hessel et al. (2004)	Exponent	Ford Motor Co., Daimler Chrysler Corp., General Motors Corp.
McElvenny et al. (2005)	Health and Safety Executive (U.K.)	Health and Safety Executive (U.K.)
Rolland et al. (2005)	Institut de Veille Sanitaire and Universite Victor Segalen	Institut de Veille Sanitaire
Peto, Rake et al. (2009)	Health and Safety Executive, Institute of Cancer Research, London School of Hygiene and Tropical Medicine	Health and Safety Executive and Cancer Research U.K.

= Research = Medical = Government = University = Industry

Human Epidemiological Study	Risk of Meso From Garage Exposures?	Risk of Meso From Pipe Insulation Exposures?
McDonald (1980)	No	Yes
Petersen & Milham (1980)	No	Not Researched
Teta et al. (1983)	No	Yes
Olsen & Jensen (1987)	No	Yes
Jarvholm & Brisman (1988)	No	Not Researched
Hansen (1989)	No	Not Researched
Gustavsson (1990)	No	Not Researched
Woitowitz & Rodelsperger (1994)	No	Not Researched
Coggon (1995)	No	Yes
Teschke, Checkoway et al. (1997)	No	Yes
Agudo (2000)	No	Not Reported
Milham & Ossiander (2001)	No	Yes
NIOSH (2002)	No	Yes
Hessell et al. (2004) / Spirtas (1985)	No	Yes
Rolland et al. (2005)	No	Yes
McElvenny et al. (2005)	No	Yes
Peto, Rake et al. (2009)	No	Yes

Coggon et al. (1995)



Differences in occupational mortality from ple
cancer, peritoneal cancer, and asbestosis

David Coggon, Hazel Inskip, Paul Winter, Brian Pannett

MRC Environmental Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton SO16 6YD
D Coggon
H Inskip
P Winter
B Pannett

Correspondence to:
Dr David Coggon, MRC Environmental Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton SO16 6YD.

Accepted 28 June 1995

776 Coggon, Inskip, Winter, Pannett

Job groups with significantly (P < 0·05) raised mortality from cancer of the pleura, cancer of the peritoneum, or asbestosis

Job group	Occupational units*	Deaths from all causes	Cancer of pleura: Deaths	PMR	(95% CI)	Cancer of peritoneum: Deaths	PMR	(95% CI)	Asbestosis: Deaths	PMR	(95% CI)
Metal plate workers	124-3, 126-2	5867	73	709	556–892	1	78	2–432	3	291	60–854
Vehicle body builders	129-3, 131-1	2256	24	649	416–968	4	877	239–2246	5	1302	423–3039
Plumbers and gas fitters	124-1, 125	16941	134	450	377–533	11	383	141–506	13	457	243–782
Upholsterers	101-4, 103	2757	16	366	209–594	0	0	0–488	0	0	0–807
Carpenters	104-1, 105-1	27425	167	362	309–421	8	102	37–221	9	196	90–372
Electricians	120-2, 121-2	20517	127	340	281–415	4	83	23–212	1	29	1–164
Electrical plant operators	120-3, 121-4	2681	14	301	165–506	0	0	0–643	1	212	5–1180
Chemical engineers and scientists	024-3, 028-1	2712	13	274	146–468	0	0	0–597	0	0	0–815
Welders	124-6, 128	12467	56	247	186–320	1	33	1–186	3	142	29–415
Managers in construction	035-1, 035-2	7404	32	240	164–340	6	349	128–759	1	77	2–429
Boiler operators	159-9, 161-1	6212	24	240	153–357	2	171	21–618	4	366	100–937
Electrical engineers (not described)	121-3	7930	31	227	154–323	1	58	1–325	1	74	2–410
Production fitters	114-4, 117	52566	192	208	180–240	12	103	53–179	17	189	110–303
Plasterers	139-3, 140-3	5198	18	207	123–327	3	265	55–773	0	0	0–431
Sheet metal workers	124-2, 125-1	7981	25	186	120–274	4	225	64–602	4	300	82–768
Builders and handymen	139-5, 140-5, 140-6	22433	65	166	128–212	5	98	32–229	0	0	0–98
Professional engineers nec	025, 026-1, 026-2, 028-2, 028-3, 028-4, 028-6	17780	53	162	121–212	2	46	6–168	0	0	0–120
Construction workers nec	139-12, 143-1, 143-2, 160-2	26886	77	160	126–200	64	990	762–1265	71	1592	1243–2009
Architects and surveyors	031-1, 031-2, 031-3	7463	21	160	99–245	1	58	1–324	0	0	0–297
Dockers and goods porters	156-2, 156-3, 157-2, 157-3	13003	36	156	109–216	7	242	97–500	4	170	46–435
Chemical workers	088, 089, 159-2, 160-2	15480	39	144	103–198	5	146	47–341	5	189	61–441
Preparatory fibre processors	086-1, 087-1	882	2	139	17–501	0	0	0–2049	2	1316	159–4753
Smiths and forgemen	108-3, 109-3	2770	6	133	49–289	3	561	116–1639	0	0	0–765
Machine tool operators	111-4, 112-4	50854	116	132	109–158	15	138	76–224	11	124	63–226

*As defined in the OPCS 1980 classification of occupations. nec = not elsewhere classified.

PMR of only 160 (77 deaths) for pleural cancer. In contrast, several of the occupations with the highest mortality from cancer of the pleura—metal plate workers (PMR 709, 73 deaths), upholsterers (PMR 366, 16 deaths), carpenters (PMR 362, 167 deaths) and electricians (PMR 349, 127 deaths)—had no excess of peritoneal cancer.

Mortality from asbestosis was more closely related to that from peritoneal than pleural cancer, with the highest PMR again in construction workers nec (PMR 1592, 71 deaths). The Spearman rank correlation coefficient across the 24 job groups in the table was 0·43 for asbestosis with cancer of the peritoneum and 0·15 for asbestosis with cancer of the pleura.

Discussion

In this analysis we restricted attention to the three causes of death that are related most specifically to asbestos. Asbestos also causes bronchial carcinoma, but the effect is less discernible in analyses of occupational mortality because the relative risk is smaller and because associations are confounded by differences in smoking and exposure to other lung carcinogens in the workplace.

The analysis was limited by inaccuracies that are known to occur in information obtained from death certificates. Not all deaths ascribed to cancers of the pleura or peritoneum are mesotheliomas, and some mesothelioma deaths are classified as cancers of other or unspecified sites.[1] Also, some deaths may be incorrectly attributed to asbestosis on the basis of pleural thickening or plaques, when no fibrosis is present. Moreover, data were only available on the most recent full time job of decedents, and some subjects will have been exposed to asbestos in earlier employment about which we had no information. In general, however, the effect of such errors should be to attenuate occupational associations, and they would not be expected to have a differential effect on the ranking of occupations by mortality from different diseases related to asbestos. The use of periodic medical examinations or an unusually high rate of necropsies in certain occupations might boost the detection of peritoneal more than pleural tumours, but it would not explain such large discrepancies in the relative frequencies of these diseases as were found. Nor would it account for the high frequency of deaths from asbestosis in some occupations with relatively low mortality from pleural cancer.

Another limitation was the use of proportional mortality rather than true death rates. Although PMRs may have been somewhat depressed or inflated by differences in the overall death rates of job groups, the effect should be similar for each of the three diseases examined, and again would not explain the contrasting ranking of jobs by PMR.

The occupations with significantly raised PMRs in our analysis were largely the same as those found previously to have high mortality from mesothelioma overall.[1] All entail potential exposure to asbestos although in some cases the exposure is related to work in specific industries rather than a general feature of [the] occupation. For example, the excess mortality among welders occurred mainly in centres of shipbuilding.[3] Many of the high risk occupations are in the construction industry, where exposure has occurred from the use of asbestos in lagging and other building materials. The high PMR for cancer of the pleura in carpenters compared with many other building trades may be related to work with asbestos board. Notable for an absence of significant risk are motor mechanics, (PMR 46, 12 deaths from pleural cancer; PMR 88, three deaths from peritoneal cancer; PMR 80, two deaths from asbestosis) about whom concerns

Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis 777

have been raised because of the presence of asbestos in brake linings. It seems that this exposure has no important effect on mortality from mesothelioma or asbestosis, perhaps because the asbestos fibres are modified by heat.

The difference in the ranking of occupations by mortality from pleural and peritoneal cancer and asbestosis is striking and cannot realistically be ascribed to chance. Nor is it likely to be explained simply by differences in [the ex]posure to which occupations are [simi]lar to crocidolite and amosite are more [likely] of mesothelioma than [for] all of the occupations with [a] peritoneal cancer could have [been] exposure to crocidolite. But welders, [whose] PMR than construction [and] pleural cancer, are also likely [to] exposure to crocidolite (especially [in] shipbuilding), and yet had a [typical] cancers.

[A pos]sible explanation is that [the] exposure relations for mesothelioma [are] non-linear, with the risk [of] mesothelioma increasing relatively [to] exposures but less steeply [at] Where an occupation entails [exposure] to asbestos, excess pleural cancer [risk] is little effect on peritoneal [cancer] this. With high exposures, [the] cancer is increased further, but [pleural] cancer and asbestosis go [hand] and becomes dominant. Also, [the] proportional mortality is diluted [as the] proportion of men in the job [are] exposed. For example, [workers] nec include not only laggers [exposed] to asbestos, but also [others] such as floor layers who [are]. As a consequence, the [group] are reduced for diseases [related] to. In the case of peritoneal [cancer], the risk in laggers is [similar] to the ranking. The PMR

pleural cancer is lower and diluted to a level below that in occupations such as carpenters where a larger proportion of men are exposed, but at a lower level.

This hypothesis is consistent with findings on the ratio of pleural to peritoneal mesotheliomas in cohort studies of asbestos workers, where peritoneal tumours have tended to be relatively more common in cohorts with longer and heavier exposures,[1,7] although with occasional exceptions.[4] It also accords with the finding of higher fibre contents in the lungs of patients dying from peritoneal compared with pleural cancer.[3] It is an indication for caution when extrapolating risk estimates for mesothelioma at low exposures to crocidolite and amosite from observations in cohorts with heavy exposure. In particular, the risks of pleural mesothelioma may be underestimated if a linear exposure-response is assumed. This should be taken into account when control limits are reviewed.

Notable for an absence of significant risk are motor mechanics... about whom concerns have been raised because of the presence of asbestos in brake linings. It seems that this exposure has no important effect on mortality from mesothelioma or asbestosis…

Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis, Coggon et al., 1995, *Occupational and Environmental Medicine* 52:775-777, p. 777

4121-EP031(mk)

Comparison of Large Danish and Swedish Cohort Studies of Auto Mechanics with Large American Study of Insulation Workers

Study	Number of Workers	Years of Follow-up	Number of Meso Cases
Jarvholm (Car Mechanics)	21,905	19	1
Hansen (Car Mechanics)	21,800	10	1
Selikoff (Insulators)	17,800	20	181

Summary

- There is *consistent evidence of no increased risk of mesothelioma for auto mechanics* from the numerous epidemiology studies that have examined this issue
- It follows that *bystanders or persons that perform brake work non-occupationally are also not at an increased risk of mesothelioma*, since their potential exposure is far less than that of a vehicle mechanic

Car Mechanics Have Increased Risks. . . Of Accidents

- *Which Causes of Death Occur Disproportionately Among Car Mechanics?*
  - "Struck by Falling Object"
  - "Accidents Caused by Fire and Flames Other Than Private Dwellings"
  - "Motor Vehicle Non-Traffic Accidents"

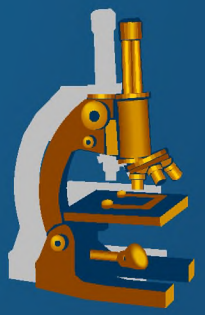
NIOSH PMR DATA 2002

Diverse Scientific Disciplines Agree – Brake Work Does Not Cause Disease

Toxicology

Industrial Hygiene

Pathology



Epidemiology

Toxicology

Doses of Common Substances

	Normal Daily Dose	Lethal Dose
Water 	1½ quarts	7½ gallons
Sugar 	2 ounces	5 pounds
Salt 	⅓ ounce	7 ounces
Coffee 	2 cups	75 cups
Aspirin 	2 tablets	90 tablets

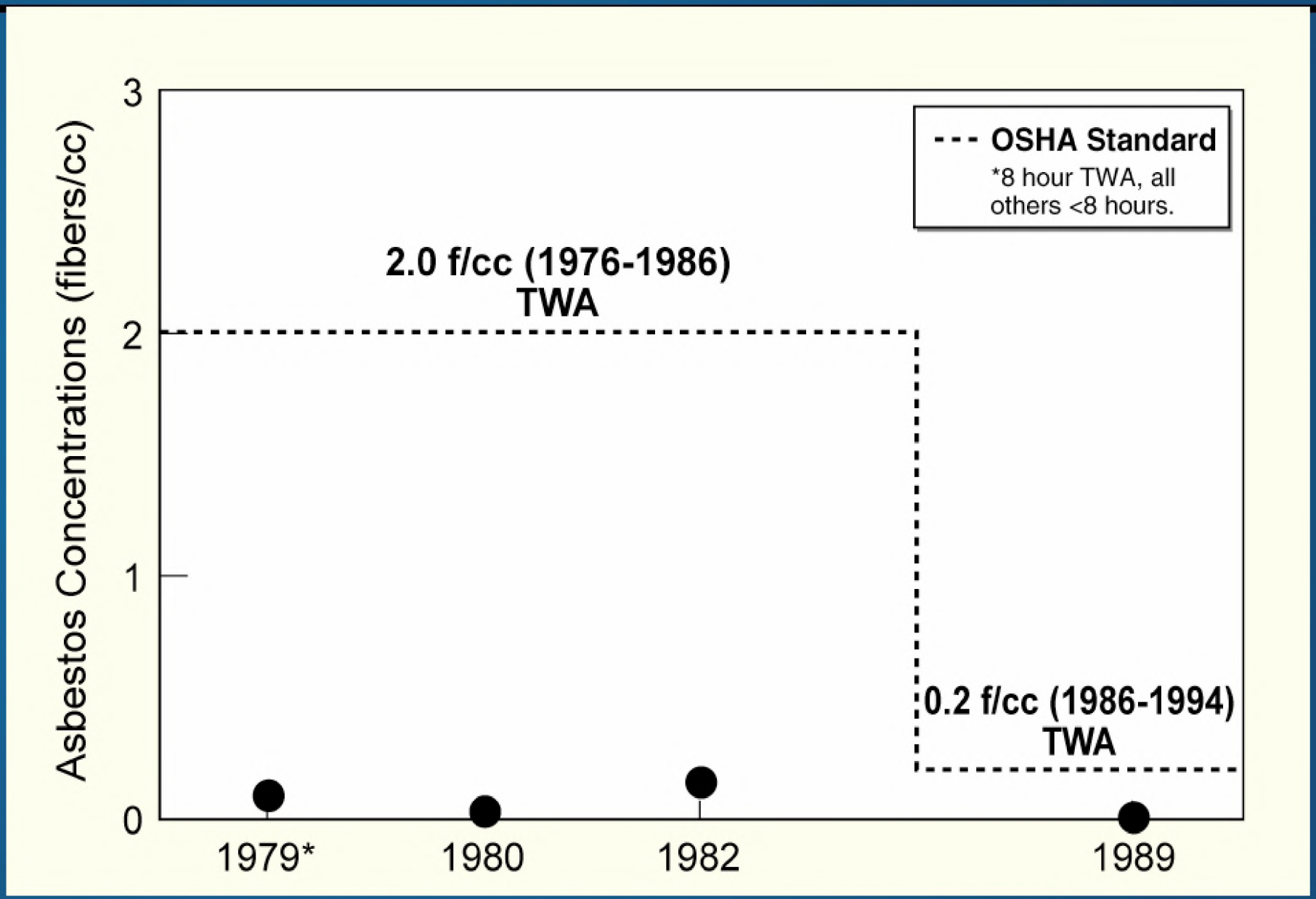
Source: Medical Toxicology by Ellenhorn & Barcelous, Elsevier, 1988, et al.

Drum Brakes

Disk Brakes

Industrial Hygiene

NIOSH Surveys of Asbestos Exposure Concentrations for Auto Brake Mechanics [Long-Term (TWA) - Mean Values]



Source: Johnson et al. 1979 (n = 26); Roberts 1980a and b (n = 3); Roberts and Zumwalde 1982 (n = 13); Sheehy et al. 1989 (n = 44)

Brake Pads

- Chrysotile in brake pad is embedded in resin
- Heat of braking causes most of chrysotile to change to non fibrous form
- Over 99% of the chrysotile fibers that remain in brake wear debris are < 5µm
- Chrysotile loses biological potency with heating

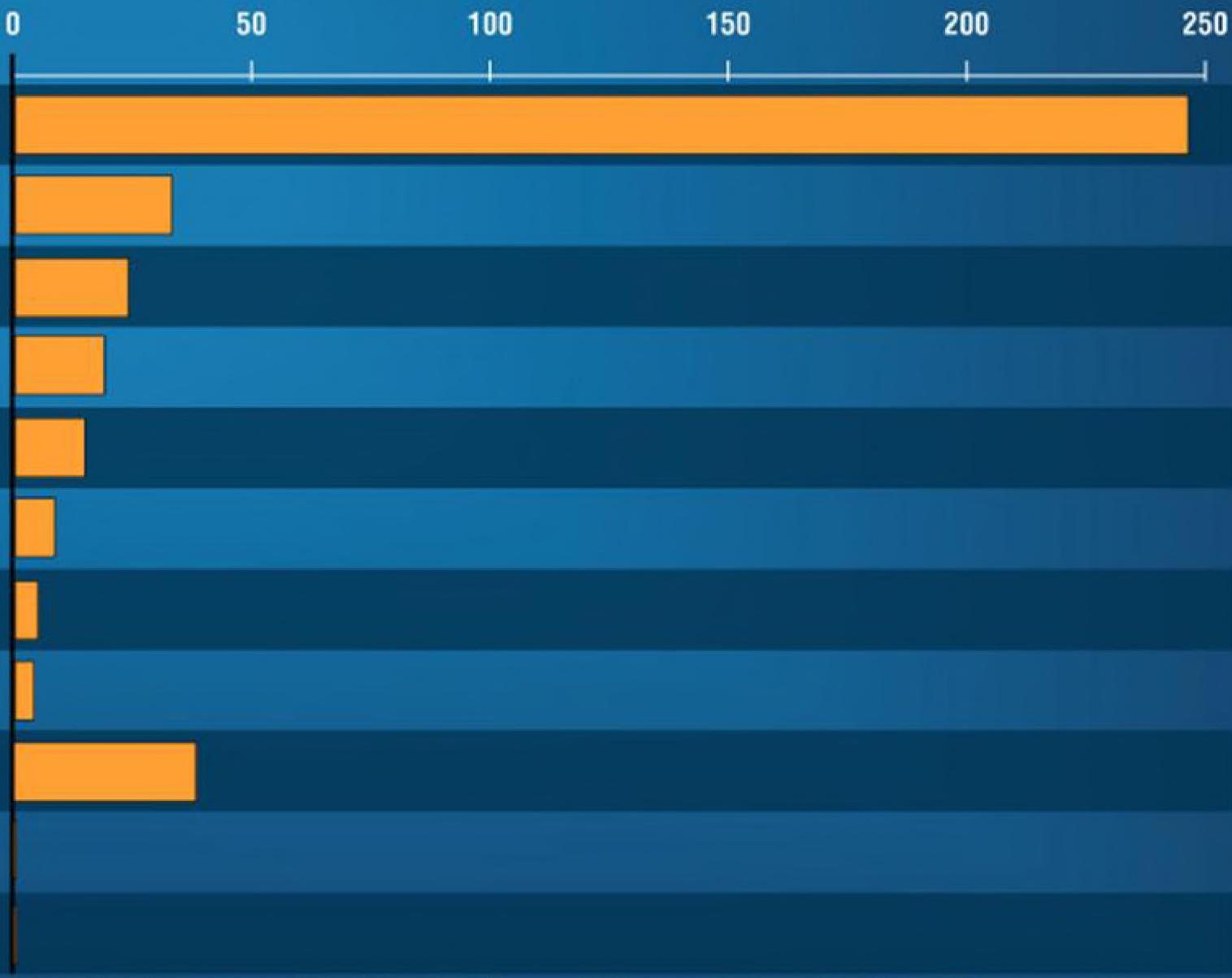
4121-PA003a

Lung Fiber Burden Analysis

AC[b]

268 Mesothelioma Cases

Commercial amphiboles (amosite + crocidolite)



*Based on mesothelioma cases whose only potential asbestos exposure is from brake dust. Fiber concentration expressed as number of fibers x 1000 per gram wet tissue.

Roggli et al. 2002 Malignant mesothelioma and occupational exposure to asbestos: A clinicopathological correlation of 1445 cases. Ultrastructural Pathology 26:55-65

Butnor et al. 2003. Ann. Occup. Hyg. 47(4):325. Exposure to brake dust and malignant mesothelioma: A study of 10 cases with mineral fiber analyses.

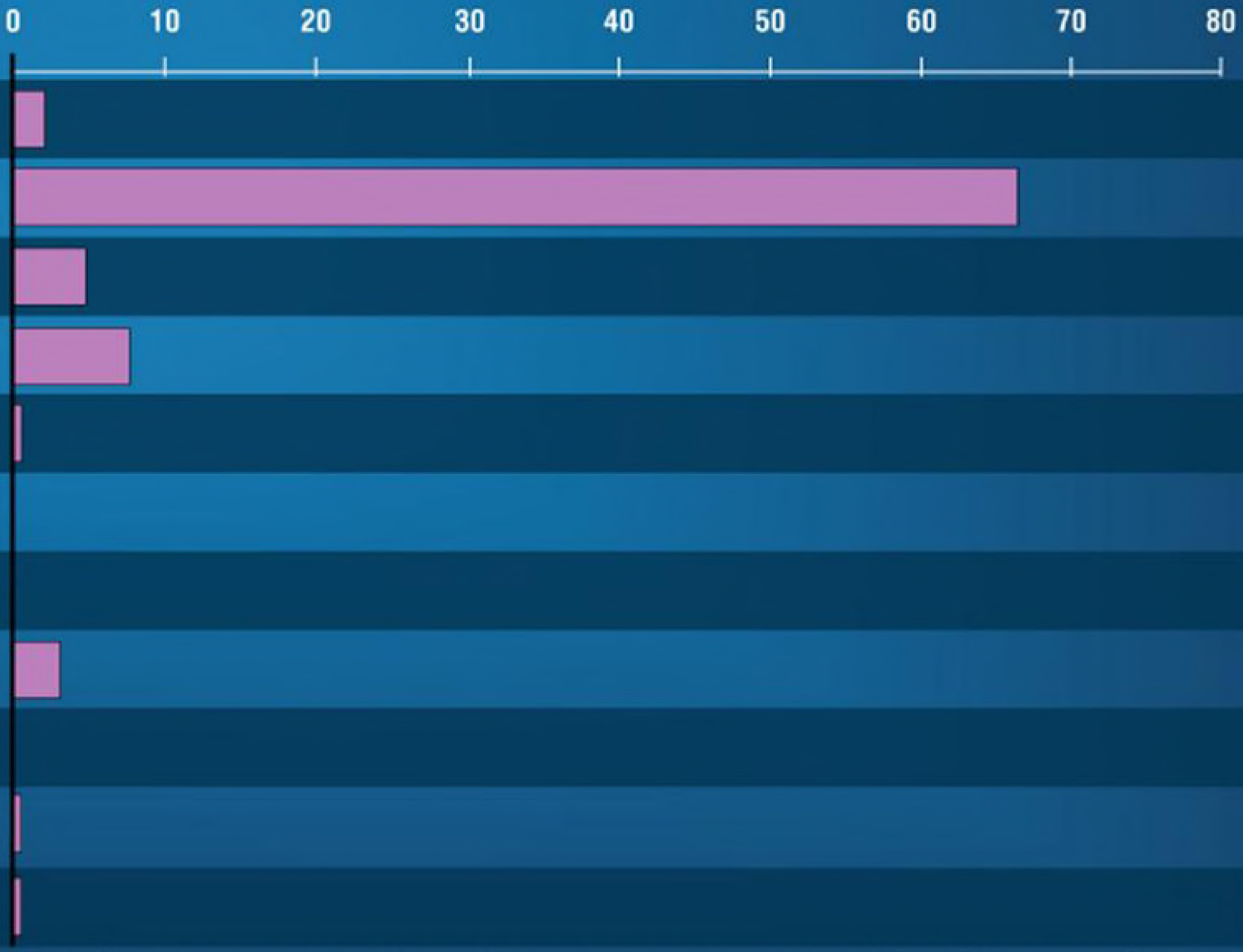
4121-PA005a

Lung Fiber Burden Analysis

Chrys[b]

Chrysotile

268 Mesothelioma Cases



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What This Case is About

- Case is not about "asbestos"
- Case is not about government policies to err on the side of protection
- Case is about determining whether Ford products caused Mr. Toole's cancer
  - Likely exposures to amphibole asbestos fibers from pipe insulation at paper mill where he worked and other plants he inspected
  - Little or no exposures to chrysotile fibers from Ford cars
  - Exposure to amphibole fibers increase the risk of disease; garage exposures do not