THE NEW YORK TIMES, TUESDAY, MAY 2, 1978

Warning to Workers Seen as Start of U.S. Effort to Put Focus on Asbestos

By JANE E. BRODY

Although in the eyes of some health experts it was too little and too late, the warning issued Wednesday to asbestos workers represents a Federal effort to begin to focus more public attention on a longstanding and widespread occupational health hazard.

News Analysis

Eight to 11 million Americans have been exposed to this persistent mineral through their work, however brief, in asbestos plants, shipyards, construction and elsewhere.

These workers, especially those exposed years ago when asbestos dust was rampant in the work environment, face greatly elevated risks of developing and dying of a wide range of cancers, especially lung cancer and an otherwise rare and, thus far invariably fatal cancer called mesothelioma, as well as the lung disease, asbestosis. As little as a month's occupational exposure to asbestos has induced cancer decades later.

The warning comes late because these dangers have been known for decades. The first report of asbestosis among American asbestos workers was made in 1930. In 1961, Dr. Irving J. Selikoff of Mount Sinai Medical Center in New York, a leading researcher into the effects of asbestos, first revealed the grave cancer risk faced by those who worked in a dusty asbestos plant in Paterson, N. J.

Hazards to Insulation Workers

In 1963, insulation workers who handled asbestos products were shown to be facing similar hazards. In 1971 Dr. Selikoff warned the United States Navy that shipyard workers, even those who worked only a month or two during the war and did not directly handle asbestos, risked developing mesothelioma 30 or 40 years later. Asbestos was the favored insulation material in ships, as well as in homes, factories and office buildings

The Navy stopped using asbestos a few years ago, but that does little to help the millions already exposed or those who will be exposed in the future while servicing old, asbestos-lined ships.

The warning issued last week by Joseph A. Califano Jr., Secretary of Health, Education and Welfare, was undoubtedly prompted in large part by the growing number of lawsuits filed against Federal agencies for illness and death caused by job-related asbestos exposure.

Thus far, the courts have held that once the Government knows of a hazard resulting from its activities, it must inform those who have been put at increased risk. If there is an opportunity for redress, the Government should also say that those harmed are entitled to compensation.

Worker's Right to Sue

According to Dr. Sidney Wolfe, head of the Health Research Group, a watchdog agency in Washington, D.C., Mr. Califano failed to tell workers of their right to sue the Government. He noted that some 500 asbestos workers had already collected $20 million in damages, including $5 million from the Federal Government

Dr. Wolfe said that the Government should also have organized a surveillance program, contacting and examining former Government workers to determine which ones might have been exposed to asbestos without knowing it.

As Dr. Selikoff has found, it sometimes takes detailed searching through the job histories of mesothelioma patients to find the time, perhaps only weeks long, when relatively large doses of asbestos were undoubtedly inhaled. Nearly all the shipyard workers who have developed mesothelioma never worked directly with the mineral, but every workday they inhaled asbestos-laden air.

The Secretary also did not address himself to the fact the mesothelioma has begun to show up in people who never worked with or near asbestos; rather, these cancer victims lived with asbestos workers or near where asbestos was used. Two were men who lived or worked across from the Brooklyn Navy Yard. Four cases of mesothelioma have occurred among the families of former Paterson asbestos workers.

Asbestos-Coated Clothes

In Quincy, Mass., a 35-year-old woman with mesothelioma is the daughter of an asbestos worker who died of mesothelioma, as did his wife. The young woman recalls her mother's practice of shaking out her husband's asbestos-coated clothing in the yard where the children played.

What good can come of Secretary Califano's warning, since asbestos-caused diseases are largely incurable? The chances of developing asbestos-induced lung cancer, which causes one in five deaths among asbestos workers, are practically nil if the worker does not smoke: Quitting smoking reduces the risk. Through early detection of asbestos-caused cancers, it may be possible to increase the chances of cure.

The chance of developing an asbestos-caused cancer is directly related to the amount of asbestos in the body, but it is not known how little asbestos it may take to produce disease. Through dust control and better work practices, asbestos workers today are far better protected than those of decades past. But is current protection good enough?

In addition, virtually all city-dwellers and many other Americans harbor asbestos particles in their lungs and other body tissues, the result of airborne particles that wear off brake linings, buildings and air-conditioning systems. Surveillance of present and former asbestos workers may help to delineate the hazards of low-level exposure and the role, if any, that asbestos may play in causing cancer in the general population.

AA-32

4C-10 FLEET OWNER
BI-MONTHLY 74,100

JAN 1978

Bus fleet praised for fighting asbestos hazard

For its "excellent performance in protecting district mechanics from a cancer-causing substance, asbestos in fiber form," California's Health Dept. has commended the Golden Gate Bridge, Highway and Transportation District.

Golden Gate Transit, operating a fleet of 248 buses, was praised for protecting the 42 district mechanics who work with asbestos lining while performing brake and clutch maintenance and repairs on buses.

California is the first state to require employers to report workplace use of asbestos. Asbestos is the most widely used of 18 cancer-causing chemicals and substances regulated in California as part of a comprehensive program to prevent job-related cancer.

Excessive exposure to asbestos fibers can cause lung cancer, a rarer form of cancer known as mesothelioma, and a lung ailment called asbestosis.

State Health Director Jerome A. Lackner, M.D., said, "State industrial hygienists are discovering in inspections that asbestos in bus, truck and auto brake-repair shops is a significant potential health hazard to California workers, but one which safe work practices can control.

"An estimated 74,000 mechanics and other workers in 22,000 auto- and brake-repair shops serving the public are potentially exposed to asbestos through brake-repair work. Several thousand more workers doing brake repairs for transit systems, truck and bus lines and utility-vehicle fleets potentially risk similar exposure. Several citations for unsafe use of asbestos have been issued.

"So it is encouragingly good news when we learn that a large fleet operator such as Golden Gate Transit is effectively protecting its mechanics against exposure to abestos fibers.

"The district's San Rafael brake repair facility is an outstanding example of safe asbestos use. It is well equipped to minimize dust. The district mechanics are using safe work practices."

The district mechanics are members of the International Association of Machinists & Aerospace Workers (IAM), Marin County Lodge No. 238. The Machinists union was represented, along with employer, government, and science representatives, on the State Health Dept. advisory committee, which early this year drafted the asbestos regulation now in effect.

Dr. Fred Ottoboni, Ph.D., head of the department's Occupational Cancer Control Unit, said the transit district followed these key procedures to maximize worker protection against asbestos fibers:

- ☐ New brake linings are "arced" (shaped to fit) with a low-speed lathe rather than a power sanding machine that could produce airborne fibers.
- ☐ Arcing is done in a room separate from other workers so that asbestos waste is isolated from other workers.
- ☐ The small amount of dust generated by this process is collected by an exhaust system.
- ☐ Waste material is put in labelled containers and disposed of in plastic bags.
- ☐ Cleanup of brakes prior to repair is done with a damp rag rather than with compressed air, thus eliminating a serious source of dust.

AA-33

Wanted: 'reasonable' cancer plan

The American Industrial Health Council, an *ad hoc* industry group formed to oppose the policy proposed by the Occupational Safety and Health Administration for defining and regulating carcinogens, launched its attack last week at OSHA's lengthy hearings in Washington, D.C. (*CW, May 24, p. 14*).

Paul Oreffice, president of Dow Chemical and AIHC chairman, stressed that the OSHA proposal needs "substantial modification" but the AIHC is taking a "positive and open stance" and is "seeking dialogue and cooperation." In laying the foundation for AIHC's 17 scientific witnesses who followed, Oreffice emphasized the "need for control and regulation of carcinogens in the workplace." He said OSHA's categorization plan—separating suspect chemicals according to the likelihood that they will cause cancer—can be made "practical and workable" if it is designed along the lines of AIHC's proposed version rather than OSHA's.

Robert E. Olson, a St. Louis University professor of medicine, criticized OSHA's policy for leaning "too heavily" on data from laboratory mice and for ignoring human evidence that shows no risk of cancer when animal tests indicate otherwise. When there is "vast human exposure to given chemicals without a detectable increase in cancer incidence," Olson said, "this evidence should be weighed heavily in interpreting mouse or rat test results."

He also took exception to the implication in OSHA's policy that there is an "epidemic of cancer." Olson claimed that tobacco, alcohol and rich diets are responsible for 75-79% of total cancer mortalities, and that the number of cases caused industrial carcinogens is "much less than 1%."

George Claus, a toxicologist with Rutgers Medical School, challenged much of the government's cancer plan. Specifically, it ignores the significance of the various routes of administering chemicals tested, permits use of only a single test species, and ignores negative findings in cases where a carcinogen has been tested on two or more species, he says.

Bernard L. Oser, a toxicological consultant, disputed OSHA's position that there is "no safe level" for humans for material that causes cancer in animals. He emphasized that "there is a wide judgmental gap between the findings in a group of animals and the conclusion that the substance is a potential carcinogen" to humans.

Dept. of Labor attorney Edward Klein and labor representatives questioned industry's policies rather than the scientific issues. Klein asked Oreffice whether companies differed in the degree of responsibility with which they treat their employees and, if so, whether more irresponsible firms had a competitive advantage. "That's why we support reasonable regulation," Oreffice replied. He likened it to the "reasonable" 55-mph. speed limit rather than a "unreasonable but undeniable safer" 10-mph. limit.

Oreffice concluded by saying that industry wants government to understand that "we are not trying to injure or kill people, we are trying to protect people as best we can."

No time to sit back

"The days when business people can sit back in the bushes and not participate in the public decision-making process are over," Dow Chemical President Paul Oreffice said last week in a wide-ranging interview with McGraw-Hill editors. That will be a key to developing a "rational" approach to government regulation of industry, he said.

Oreffice had just returned from Washington, where he led off industry's testimony on the Occupational Safety and Health Administration's proposed cancer policy as a chairman of the American Industrial Health Council (*see story, left*).

"Regulation is the single most inflationary thing" affecting Dow, he said. The company's health and environmental research has been growing at a rate of 20-30%/year and now amounts to about 15% of the over-all R&D budget. Calling it "defensive" research, he explained that the company is considering splitting regulation-inspired research into a separate category to assure that emphasis on basic product and process R&D is not diluted.

The company will spend $230 million on R&D this year, a gain of 12% over the $205 million spent in 1977. Oreffice said he is dedicated to keeping "offensive" R&D going "up and up and up."

Touching on other areas, he said:

- The company will soon announce an on-site co-generation project at Freeport, Tex. It recently withdrew from a project to supply lignite to Houston Lighting & Power (*CW, June 7, p. 22*).
- Unless Dow can reach an agreement with the Environmental Protection Agency over emissions from coal-fired power plants at Midland, Mich., it will temporarily shift some operations to other locations until the Consumer's Power nuclear plant comes on-line in 1982.
- Dow now buys "a lot of naphtha" from Russia, and company executives have met with Soviet Chemical Minister Leonid Kostandov to discuss possible future deals.

Thorium traces in pit

The Nuclear Regulatory Commission is reviewing AMAX's plans for cleaning up a thorium-contaminated pit at the Parkersburg, W.Va., plant formerly owned by the company. NRC says the highest levels of radioactivity found at the site—2 millirems/hour, measured aboveground—do not pose a short-term health problem to workers, but are unacceptable long-term levels. But cleanup efforts, normally a simple procedure, are being slowed by the presence of highly combustible zirconium also in the soil.

The problem arose when L.B Foster (Pittsburgh), which bought the plant last year from AMAX Specialty Metals Corp., was installing equipment for its planned $2-million piping plant. While digging a hole inside a building, the zirconium caused an explosion. In a later incident there, the tracks of a bulldozer were reportedly melted off during grading work.

Subsequent analysis of the soil showed traces of the highly combustible zirconium, and also of the thorium. NRC notes the thorium was contained in Nigerian zircon sand used to manufacture zirconium alloy rods, and that thorium was not used in the process itself.

NRC says the 20-30-ft.-deep pit also contains discarded machinery and assorted acids. NRC says the agency never gave any authority for thorium burials there, but it adds that agency provisions allow up to 12 burials/year of small amounts under certain conditions, including keeping records of burials. NRC is reviewing such records.

AMAX Speciality Metals says it did not bury any radioactive materials there at any time. It did speculate that there may have been some undetected spillage when the company removed 1,100 tons of Nigerian sands to a disposal site approved by the former nuclear agency—the Atomic Energy Commission—after AMAX bought Carborundum's half of the venture in 1967. It adds, however, that AEC inspected the operation at the time and was satisfied about the operation. Carborundum also denies any dumping of radioactive materials.

CURRICULUM VITAE

Hans Weill, M.D.

Education

Tulane University, 1951-54, B.A., June, 1955
Tulane University School of Medicine, 1954-58, M.D., June, 1958

Post-Graduate Medical Training

Intern, Mt. Sinai Hospital, New York City, 1958-59
Resident, Charity Hospital of Louisiana, New Orleans, Tulane Medical Unit, 1959-60
Research Fellow, Department of Medicine and Pulmonary Laboratory, Tulane University School of Medicine, 1960-61
Chief Resident, Charity Hospital of Louisiana, New Orleans, Tulane Medical Unit, 1961-62

Teaching and Research Appointments

Instructor in Medicine, Pulmonary Diseases Section and Pulmonary Laboratory, Tulane University School of Medicine, 1962-64
Assistant Professor of Medicine, Tulane University School of Medicine, 1964-67
Associate Professor of Medicine, Tulane University School of Medicine, 1967-71
Professor of Medicine, Tulane University School of Medicine, 1971-present
Director, Specialized Center of Research (SCOR) in Occupational Respiratory Disease, NHLI, 1972-

Hospital Appointments

Visiting Physician, Charity Hospital of Louisiana at New Orleans, Tulane Unit
Chief, Pulmonary Function Laboratory, Veterans Administration Hospital, New Orleans, 1963-present
Consultant in Pulmonary Diseases and Medicine, USPHS Hospital, New Orleans, 1964-present

Honors and Societies

Phi Beta Kappa, 1955
Alpha Omega Alpha, 1958
American Thoracic Society, 1962
Orleans Parish Medical Society, 1963
Diplomate, American Board of Internal Medicine, 1965
Fellow, American College of Chest Physicians, 1965; Governor for La., 1970
Fellow, American College of Physicians, 1967
Certified in Pulmonary Diseases, American Board of Internal Medicine, 1966
American Federation for Clinical Research, 1967
Southern Society for Clinical Investigation, 1969
Chairman, Post-Graduate Course, Pulmonary Function in Health and Disease, 1970-72
Fellow, Royal Society of Medicine, 1971
New York Academy of Sciences, 1971
Society for Occupational and Environmental Health, 1972
Councilor-at-large, American Thoracic Society, 1973 -
President-elect, American Thoracic Society, 1975

Consultant and Committee Assignments

Member, Task Force on Environmental Lung Diseases, National Heart and Lung Institute, NIH, 1972
Consultant, National Institute of Occupational Safety and Health, TDI Criteria Document, 1973, Phosgene Criteria Document, 1975
Planning Committee, Occupational Lung Disease post-graduate course, Annual Meeting of American Thoracic Society, 1973
Program Committee, American Lung Association, 1973
Faculty Advisory Committee, Tulane University School of Medicine, 1973 -
Secretary-Treasurer, New Orleans Academy of Internal Medicine, 1973 -

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COPY

17 November 1975

Memorandum For: John Marsh, Raybestos-Manhattan
John Riopelle, Bendix Corporation
E. H. Feierabend, Abex Corporation

Subject: Materials of interest concering friction materials

The enclosed material is forwarded as a matter of interest:

1. "Is Brake Lining Dust Harmful?" pamphlet, Nov., 1975 Asbestos Information Committee, U.K.

2. Extracts from two papers presented at International Conference, Dust and Gases in the Work-Place, June 18-20, 1975, Bonn, Germany

As you are aware the Association proposes to rewrite the pamphlet, "Asbestos and Brake Linings" (also enclosed). Your recommendations will be appreciated.

Bob

R. H. Mereness
Executive Director

cc: Stanford Christian, Bendix Corp.
Jim Armstrong, Bendix Corp.
Ike Weaver, Raybestos-Manhattan, Inc.
✓Ed Drislane, FMSI

Enclosures
RHM:vld

AA-36 (10)

Is brake lining dust harmful?

asbestos information committee

Is brake lining dust harmful?

The brake linings on which drivers of motor vehicles depend to bring them to a safe stop incorporate asbestos fibre. However, writers on this subject occasionally express the view that the braking of millions of vehicles emits particles of asbestos into the urban atmosphere in such quantities as to create a risk to the health of the general population. What are the facts?

What happens in braking?
Any examination of this theory must start with what happens when the brakes of a vehicle are applied. Vehicle brakes operate by converting the energy of the moving vehicle into heat which is then dissipated into the air. In drum brakes a friction lining is applied to a brake drum; in disc brakes friction pads grip a rotating disc on the wheel hub. Asbestos is an essential ingredient in friction linings and pads primarily because it acts as a reinforcing agent which maintains its strength and stability through a wide range of temperatures and pressures. For technical reasons only chrysotile (white) asbestos is employed in friction materials.

Little dust liberated
The friction between lining and drum or between pad and disc creates wear products, which look like a fine dust. This dust consists almost entirely of degraded resin, fillers and other brake lining additives, and products of wear from the metal brake drum or disc. Although the asbestos content of conventional brake linings may vary from 25–65 per cent by weight it is almost entirely converted by the considerable heat generated by the action of braking into other materials.

Experiments carried out by various Government and industrial laboratories throughout the world have shown that the free asbestos fibre in the small amount of dust liberated from brake linings rarely exceeds 1 per cent[1] of the total products of wear.

The Environmental Protection Agency of the US Government commissioned the Bendix Corporation to set up a test programme to measure brake and clutch emissions during actual vehicle operation. The asbestos content of the brake emissions ranged in two sets of tests from 1·65 per cent to 0·03 per cent; only in 3 out of 90 analyses was it over 1 per cent. In a third group of tests the range was from 0·22 per cent to 0·003 per cent[2].

Experiments with disc brakes have shown that, in their case, the free asbestos fibre is an even lower fraction

of the total products of wear. In tests carried out by research staff of the Ford Motor Company, Detroit, on a production disc brake[3], less than 0·02 per cent of the lining wear was released as asbestos fibres.

Amount not harmful

Asbestosis is a disease associated with the inhalation of high concentrations of asbestos fibres over many years. The British Occupational Hygiene Society has published a Hygiene Standard for Chrysotile Asbestos Dust which is designed to virtually eliminate the risk of developing the earliest recognisable clinical signs of asbestosis. This standard is the basis of those which apply under the Asbestos Regulations 1969, to work with asbestos and asbestos products. The minimisation of asbestosis has, at the same time, minimised any associated risk of lung cancer.

Tests made in the urban atmosphere have recorded the presence of asbestos in amounts at least 1,000 times below those regarded as the standard for chrysotile dust in occupational situations. There is no evidence that this trace amount of asbestos in the urban environment can cause damage to the lungs of the general public.[4]

Thus the risk of contracting asbestosis or lung cancer through the application of brakes on motor vehicles is negligible for the general public.

The risk of contracting mesothelioma (a rare tumour which has been mainly associated with crocidolite (blue) asbestos) can be virtually discounted as this type of asbestos is not used in the manufacture of brake linings. It is recognised that chrysotile asbestos is less likely to produce mesothelioma and where it has done so the dust levels to which the cases were exposed were greater than could occur in the urban air as the result of vehicles applying their brakes.

"Asbestos bodies" may be found in the lungs of people in random series of autopsies. The presence of asbestos bodies does not necessarily imply a connection with asbestos-associated disease nor is it an indication of it. Where large numbers of asbestos bodies have been found they have invariably been the result of occupational, not general environmental exposure.

Occupational exposure

The only possibility of a risk to health from brake and clutch linings arises in their manufacture and where regular or continuous machining and maintenance

work is carried out. Such work, however, is controlled by the 1969 Asbestos Regulations to ensure that those employed on such operations are not exposed to excessive amounts of dust likely to impair their health.

The Environmental Control Committee of the Asbestosis Research Council has produced a Control and Safety Guide[5] and a simple illustrated leaflet explaining the precautions which may be necessary for workers involved in these activities.

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2. Brake and Clutch Emissions generated during Vehicle Operation, M G Jacko, R T DuCharme and J H Somers, Proceedings of Automobile Engineering Meeting of the Society of Automotive Engineers, May 14th to 18th 1973.

3. Asbestos Emissions from Brake Dynamometer Tests by A E Anderson, R L Gealer, R C McCune and J W Sprys, Proceedings of Automobile Engineering Meeting of the Society of Automotive Engineers, May 14th to 18th 1973.

4. Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer (World Health Organisation), Lyon, October 1972.

5. Control & Safety Guide No. 8, Asbestos-based Friction Materials and Asbestos-reinforced Resinous Moulded Materials ; Environmental Control Committee, Asbestosis Research Council, P.O. Box 18, Cleckheaton, West Yorkshire, BD19 3UJ.

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November, 1975

Stäube und Gase am Arbeitsplatz

International Conference

Dust and Gases in the Work-Place

Colloque International

Poussières et Gaz au Poste de Travail

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Bonn - Bad Godesberg

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Zusammenfassungen

Abstracts

Résumés

Staubforschungsinstitut des Hauptverbandes der
gewerblichen Berufsgenossenschaften e.V.
B o n n

15. G. Kühnen, G. Heidermanns, A. Schütz, Bonn
und
R. Prochazka, München (BRD)

Dust Concentration During Production of Friction Linings and their Treatment in Garages

In connection with the Accident Preventing Regulations i.e. "Protection against Hazardous Mineral Dusts" (VBG 119) measurements on dust concentration and of the asbestos content were taken during the production of friction linings (final treatment) as well as in the brake service of garages.

The aim of these technical investigations which were made within a research plan of the "Employer's Liability Insurance" together with medical investigations, was the identification of eventual hazards to health of the employees occupied with these labours.

Friction linings contain 20 to 60 % of chrysotile asbestos depending on the utilization purpose. These values have been figured out during measurements of the dust concentration at working sites during mechanical treatment of friction linings as well as during manufacturing and at the brake service. The evident dust concentration depends on the way and extent of preventive dust protection actions. During manufacturing the concentration is about the TRK value for chrysotile, at the brake services above the TRK value, however, for short exposition times only.

During blowing out of brake drums a short termed high dust concentration occurs, however, asbestos was not proved to be evident. Laboratory and test stand experiments issued that asbestos is transformed morphologically by the temperatures produced within the friction area. Under extreme conditions

.../2

16. Woitowitz, H.-J. und Valentin, H. Erlangen (BRD)

Occupational Medicine Investigations on Hazards to Health by Dusts Containing Asbestos

In Germany in 1973 extensive regulations for the protection of asbestos exposed persons became effective (VBG 119). Hereby, the question arose whether workmen exposed to dusts of brake linings containing asbestos can be generally considered as endangered in respect of fibrogenic effects via inhalation.

In regard to this operating method problem there has been performed on a selective universe a casual cross section study with retrospective onset. 210 male and female workers with at least ten years brake lining dust exposition and of three operating methods out of about 50 different plants were involved. The data for each person comprised 200 details - together more than 40.000 - i.e. concerning the anamnesis, under special consideration of profession and working site, and the diagnosis. The thorough examinations were led by occupational physicians and internists who applied physical, in-vitro-clinical, electro-cardiographic, radiological and latest lung functional analysis procedures.

The occupational medical evaluation of diagnoses of an asbestos exposed worker, nowadays has to be multidimensional, principally. The extensive data, therefore, after formation of syndrome groups had to be reduced to 5 types of diagnoses with defined degrees of diagnosed security:

Type I	AIF eventually
Type II	AIF possible
Type III	AIF not presumable
Type IV	AIF competible diagnosis
Type V	AIF normal diagnosis

the transformation into Forsterit has been proved. The transformation, probably, succeeds via an amorphe phase.

Due to these results, besides dust protection, we regard prophylactic examinations in respect to occupational health of all employees in the final manufacturing as necessary. At the brake services all employees occupied with the mechanical treatment of friction linings should be controlled by the occupational physicians in case the weekly exposition time to asbestos dusts exceeds 3 hours. The employees occupied with blowing out do not have to be controlled unless the result of the medical examination does make it necessary.

The judgement of risks is mainly based on diagnoses in respect to eventual AIF (Type I). Suspected diagnoses in respect to a beginning AIF (Type II) were not disregarded. The prevalent diagnoses of type I and II amounted to:

a) 51 males who produced brake linings or handled unused brake linings 5,9 % resp. 15,7 %
43 females of the same operating method 0,0 % resp. 7,0 %

b) 63 males in the truck service who treat unused brake linings and blow out brake drums 1,5 % resp. 14,5 %

c) 53 males in the car brake service who blow out or brush out brake drums 0,0 % resp. 5,6 %

The considerations in respect of occupational health resulting from this will be discussed according to the specific risks of operating methods.

Occupational Cancer: Government Challenged in Beryllium Proceeding

The beryllium industry and the government have been locked for months in an intense struggle over the question of whether beryllium should be declared a carcinogen. At issue are not only the merits of an epidemiological study conducted by the chief government agency involved, the National Institute for Occupational Safety and Health (NIOSH), but questions of fair play in government regulation. Even the conduct of the director of NIOSH, John F. Finklea, a respected but controversial federal official, has come under question, because he engaged in a telephone conversation that industry says was a naked threat to intimidate industry witnesses.

The focus of the controversy is a study done by NIOSH which concludes that, among the beryllium workers employed since the 1940's at a plant in Reading, Pennsylvania, the incidence of fatal lung cancer was unusually high. Industry has accused NIOSH of "gerrymandering" its data to produce this result. Joseph K. Wagoner, who is a principal author of the study and is now a special assistant to the director of the Occupational Safety and Health Administration (OSHA), which must decide on beryllium's carcinogenicity, defends the study, saying the deficiencies are due to industry's poor records.

"There is absolutely no doubt in my mind that beryllium is a human carcinogen," Wagoner told *Science*. But Wagoner and other officials stress that sound epidemiology is hard to do because industry may be tampering with or destorying worker records. The beryllium companies deny doing any improper meddling. They would like to see the study redone, or another study made of a separate cohort of beryllium workers. Wagoner, however, says another study "will mean more delay and increased risk to the health of the American worker."

The study, known as Bayliss III because it is the third study of beryllium workers by NIOSH bench scientist David L. Bayliss, is crucial to the determination of a new standard for beryllium. NIOSH proposed a new standard on the basis of animal data in 1975. The industry has argued that the animal data are an insufficient base for the new standard because the animal data are negative for beryllium copper, the form to which some 70 percent of all the industry's workers are exposed. The government's case, based only on animal data, therefore looks iffy.

But Bayliss III, claiming on epidemiological grounds that human beings exposed to beryllium incur a lung cancer risk, has strengthened the government's case. If the study is considered acceptable, it will increase the likelihood that the Administrator of OSHA, which is part of the Department of Labor, will approve a new, stricter standard and that the courts would uphold a subsequent appeal. But by the same token the study has caused the industry great alarm.

Aside from the questions of scientific merit and fair play, the NIOSH beryllium controversy also involves the issue of when animal data alone can be the basis of federal standard setting, and what level of human epidemiological evidence should serve as allowable in federal regulation. These questions are all the more important now because the OSHA's new director, Eula Bingham, is a former cancer scientist, who has announced a new, get-tough stance on regulating carcinogens in the workplace (*Science*, 21 October).

Toxicity of Beryllium

The beryllium industry came of age in the 1940's, when there was increased demand for the tough, lightweight, heat-resistant metal during the Second World War. Since then, beryllium has been widely used in electronics and missile parts, and in other applications. But from the start it was known that beryllium is toxic to humans; it causes a disease known as berylliosis. Since the 1940's then, the industry has had to limit exposures to 2 micrograms per cubic meter for workers.

Although animal data linking beryllium to tumors in some animals have been around for years, NIOSH only recently (in 1972) issued a "criteria" document laying out the case against the metal as carcinogenic. In 1975, NIOSH proposed that the 2 microgram per cubic meter standard be lowered to 0.5 microgram per cubic meter. Industry has responded that such a change is unwarranted by the scientific evidence and that the lower standard is technically impossible.

It seems that in the early 1970's few people paid much attention to the possible carcinogenic potential of beryllium to humans, particularly since the only two well-known studies of the subject, known as Bayliss I and Bayliss II (published in 1971 and 1972) examined large cohorts of beryllium workers at several plants and found no unusual incidence of lung cancer. But at some point, Bayliss and Wagoner decided to restudy the life histories of workers at a single plant. They picked one at Reading, owned by Kawecki Berylco Industries, Inc. (KBI). In early 1977, OSHA scheduled a hearing on the proposed new standard; the two major producers of beryllium, KBI and Brush Wellman, Inc., who knew of the existence of a new study partly because it had been reported in a Cleveland, Ohio, newspaper, began asking NIOSH for the data it was examining. In March, Brush hired a Washington consulting firm, Equitable Environmental Health Corp., to handle the human epidemiological issues relating to the proposed standard.

At this time, there began a series of events through which each side has become embittered, with each accusing the other of harassment, obstructionism, and bad faith. The entire controversy, and many of the associated emotions, were aired on the public record in August and September during the hearing.

The industry charges, first, that NIOSH did not make a good faith effort to turn over its data on the more than 3000 Reading workers it was using as the basis of Bayliss III, so that industry could check NIOSH's calculations and conclusions. Brush's Vice President, Martin B. Powers, testified that "throughout 1976" the company made both informal and formal Freedom of Information Act requests to obtain the data. Although NIOSH kept giving the industry other information and documents, it did not hand over the Bayliss II paper or accompanying backup until 1 July 1977, shortly after officials met with OSHA's Bingham, and threatened to sue if the material was not forthcoming. Indeed, it seems that, throughout the hearing NIOSH continued to hand over information at the last minute, and then only in response to formal, freedom of information act requests—in some instances 72 hours, or 48 hours, before industry was to present expert testimony on the material.

NIOSH's version of these events is that the repeated freedom of information

requests were intended to paralyze NIOSH's attempts to get its beryllium study finished for the hearing. Said Peter Infante of the NIOSH Cincinnati staff, where he and Bayliss were working on it, "They strap you down with all those requests. They had the whole staff battened down to the point where we couldn't get our own work done . . . We could have met the [hearing] deadline easily if we hadn't had those requests."

In any event, industry received a copy of the Bayliss III paper—which turned out to be a first version, a second was submitted at the hearing—on 1 July, and turned it over to Equitable, when another incident occurred. Industry charges that Finklea, through a telephone threat, caused Equitable to cease working with Brush and to prevent one of its scientists, Michael Utidjian, from testifying on the deficiencies in the NIOSH study.

According to the hearing transcript, on 12 August, 4 days before the hearing was to begin, Finklea telephoned William Malloy, the executive vice president of Equitable, and "suggested that there might be a conflict of interest on Dr. Utidjian's part if he participated in the hearing on Brush's behalf." Finklea was referring to the fact that Utidjian was also working on Equitable's NIOSH contracts to prepare criteria documents on other substances. The following day Malloy ordered Utidjian off the Brush contract and told Brush that Equitable would not participate further.

Brush officials say the call was tantamount to a threat of the loss of NIOSH business if Equitable did not stop working for the beryllium industry. Brush may sue Equitable for breach of contract, while another Equitable official, a beryllium expert, who objected to Malloy's decision is leaving the company.

Finklea was abroad for several weeks and could not be reached for comment on this incident. Both Wagoner and Howard Walderman, a lawyer who works on NIOSH matters, declined to comment on Finklea's actions and what he really intended to accomplish. At the hearing, Finklea defended himself by saying that he wanted "Mr. Malloy to look at the contract he had with the Federal government, which had a clause in which people were seeking to avoid the appearance as well as any actual conflict of interest. I expressed concern about that and . . . just called that to his attention."

But Edward J. Baier, deputy director of NIOSH, told *Science* that Finklea had been under the impression that Utidjian was working full time on NIOSH work, and became alarmed when he saw Utidjian's name on the industry witness list. But in Finklea's absence, Baier declined comment on why Finklea made his concern known by a phone call to Utidjian's boss, instead of by a more conventional route.

Industry is also charging foul play because NIOSH did not produce the principal author of the paper, Bayliss, at the hearing. By all accounts, on 16 August when the hearing began, Bayliss was on leave from his NIOSH job in Cincinnati and was registering as a doctoral student in the department of epidemiology at the University of North Carolina.

Industry believes that Bayliss was deliberately kept away because his testimony would have admitted the weaknesses in the case against beryllium.

NIOSH's Wagoner, who was Bayliss' superior at the time, told *Science*, "Bayliss didn't want to testify. We asked him and he said he didn't want to come."

Science located Bayliss in North Carolina, where he gave his version of these events. Bayliss says he received a phone call from his boss's boss, Finklea, the Friday before he was to register. "He said he wanted it to be known that an invitation stands if I cared to testify. He indicated that Wagoner could handle the whole thing though, so I said I didn't see any need for me to go." He says NIOSH has barely contacted him since, and that he learned of the "Where's Bayliss" controversy through "a third party who had access to an OSHA newsletter."

Study Under Fire

Besides the fair play issues, the Bayliss II study has become the major focus of the controversy. Brush's president and chief executive officer, Robert W. Biggs, claims that the study is "slanted" and that the cohort of workers has been "gerrymandered" to come to the conclusion that beryllium workers have an increased risk of getting lung cancer. NIOSH counters that any errors that have been found in the paper are "insignificant." Wagoner told *Science* that the Bayliss study, and another one produced by Thomas Mancuso on the first day of the hearings showing an increased risk, "converges" with the animal data presenting a total case that is "irrefutable."

Bayliss III examined the histories of 3070 workers, who worked at the Reading KBI plant, between January 1942 and December, 1967. The study calculated the expected number of deaths from lung cancer for the group at 33. The conclusion that beryllium is linked to lung cancer hinged on the fact that the observed number of lung cancer deaths was larger, namely 46.

At the hearing, Brush's statistical consultant H. Daniel Roth, testified that the results were impossible to verify because the tables that he received in July on 3070 individuals showed no birth dates for 70 percent of the cohort, or everyone listed as alive. Since the ages of the majority of the cohort were unknown, it was impossible to replicate NIOSH's life table analysis or verify its expected lung cancer death rates.

The NIOSH paper presented blocks showing that the largest number of lung cancer deaths occurred among workers employed at the Reading plant 5 years or less. But examining the 46 cases of lung cancer death, name by name, Roth found that 4 who had been classified as being there for approximately 20 years actually worked there a year or less. Moreover, Roth found of the 46, some 30 had been there fewer than 1 year, 24 had worked there fewer than 6 months, and 17 had worked there fewer than 3 months. Both Roth and other expert witnesses testified they knew of no theory of cancer where the risk increased as the length of time of exposure shortened. (In an interview, Wagoner admitted he knew that a large number of the deaths were among people who had been employed less than a year. Asked why this fact was not mentioned in the paper, Wagoner replied, "I don't know what it would have meant.")

Roth claimed that the "excess" of 46 lung cancer deaths as compared to the expected 33 is explained by correcting for smoking and the plant's location. The Bayliss III paper used the assumption that smoking habits of the cohort were similar to those of U.S. males in 1964. Although the paper acknowledged that a 1968 plant survey showed smoking at the plant to be higher than this figure, it was discounted on the grounds that the county in which the plant is located was below national average.

Factoring in the actual 1968 smoking data instead of assumed 1964 data, Roth calculated the expected lung cancer deaths among the cohort to be 38 to 44—numbers which eliminate the statistical significance of the observed value of 46.

But Roth pointed out that the lung cancer death rate in Reading itself—an old industrial town—was much higher than both the surrounding county and the U.S. as a whole. In fact, applying the Reading rate to the KBI plant population, Roth says, the expected lung cancer deaths became even more than those actually observed.

Wagoner, interviewed about the specifics of the study, admitted that there were deficiencies in the information available on the cohort of workers. "We

had some people whom we weren't sure whether they were alive or dead," he said, "so we assumed they were alive." He explained the shifting size of the cohort, which at different times numbered 3070, 3201, and 3055, was because NIOSH was trying to square its information with that offered by Mancuso in his study, which included some Reading workers. Industry's position on the Mancuso study is that it should be stricken from the record, since the paper seems not to mention smoking at all, and since Mancuso has said he would give his backup data to the industry but has failed to do so.

Interestingly, the authors of the Bayliss study differ on how strong a case their paper makes. Bayliss told *Science*, "I thought there was a pretty, reasonably strong case, but of course that's a judgmental matter." Wagoner, in a separate interview, maintained that the evidence was all "converging" and the case "irrefutable." Infante said: "The results and the interpretation don't change. We had to keep stating, defining, who the cohort was . . . We know what the meal is, but we have to go out and buy the meat and vegetables."

The hearing ended in September, but the administrative judge before whom it was held, has allowed until mid-December for posthearing submissions. On the NIOSH side, these include a fourth Bayliss paper. Industry will submit the most comprehensive rebuttal yet to the Bayliss and Mancuso studies.

Warfare Continues

But the warfare continues. Brush's vice president, Powers, questions whether the hearing record will make any difference, since Wagoner, one of the most ardent supporters of a new beryllium standard, recently moved to OSHA to be Eula Bingham's special assistant. Industry believes that Wagoner should not participate in Bingham's decision-making on beryllium, and is seeking a written reply from Bingham on Wagoner's role.

Brian MacMahon, professor of epidemiology at Harvard, has gone over the latest cohort tape which has 3055 workers and 47 lung cancer deaths. The added lung cancer death is that of a man who was hired and terminated on the same day. *Science* asked Wagoner whether he knew of the man's brief employment. "I guess we didn't have that information" he replied.

So the parties to the controversy seem locked in an epidemiological treadmill, with NIOSH blaming the problems in its cohort data on industry, and industry blaming the "slanted" data on NIOSH.

In the long run, the controversy's importance lies not so much in whatever the beryllium industry ultimately knuckles under to a new tighter, standard. It is more important as a precedent, for beryllium is among the first of many alleged carcinogens on which OSHA's Bingham will have to rule.

These decisions will have their political element; that is, Bingham will want to not only protect American workers but to give the appearance of protecting American workers. But she will also have to judge whether the scientific evidence in each case ultimately supports or erodes those political decisions.

—DEBORAH SHAPLEY

National Laboratories: Focused Goals and Field Work Hinted Under DOE

Even before the federal energy agency underwent two face-liftings, people were saying that the national laboratories were declining in importance and were in need of new missions. Their old roles—as practitioners of basic research, nuclear reactor development and weapons design—proved to be embarrassingly narrow when the Energy Research and Development Administration (ERDA) inherited the labs from the Atomic Energy Commission in 1975. Although ERDA expanded the breadth of energy research at many of the individual laboratories, it never quite determined what should be the laboratories' role in the national energy program.

In the 2 months since the Department of Energy inherited all of ERDA's former programs, officials of the new energy agency have been crisscrossing the airways to inspect some of their 25 laboratories and research centers. The new undersecretary of the department has visited three laboratories in the west, Sandia, Los Alamos, and Livermore. The man who had primary responsibility for getting the new department running, Tom Reed, has visited a number of east coast labs. The major laboratory directors have also met with the undersecretary as a group. The message in these meetings has been that no abrupt changes will occur, but the past roles of the labs are being analyzed carefully and their future roles may gradually change.

Soundings taken in Washington when the energy department was inaugurated in early fall raised a number of problems. The laboratories had accumulated a multitude of new programs to spearhead ERDA's acceleration of energy research. Some critics said that the labs spend too much money on projects that are not put up for bids and that their expenditures would be more productive if brought under zero-based budgeting. The various laboratories have enjoyed considerable autonomy during most of their history.

On paper there are reasons for the laboratories to worry about losing their independence. The reorganization that accompanied the formation of the energy department created two new vehicles for monitoring the work of the various laboratories at the highest levels of the agency. For their institutional needs, the laboratories will no longer report to regional operations offices but will report to an administrator at the rank of assistant secretary in the department. In addition, the laboratories will be regularly scrutinized by a newly created council composed of all the line administrators of the department. The council will be chaired by the same man who has responsibility for day-to-day coordination of all the department's energy research activities, Undersecretary Dale D. Meyers, and that may be an indication of how closely the laboratories' efforts will eventually be interwoven into the whole research and development fabric.

Whatever develops in the new department's relations with the field, the possibility that the changes pose a threat to the traditional independence of the laboratories is taken seriously in some quarters. Two weeks ago the House Science and Technology Committee called in the directors of eight major labs to testify in a hastily arranged hearing that had no apparent routine purpose. Some observers thought that the committee, which has

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Clinical Reviews

THE BIOLOGICAL EFFECTS OF ASBESTOS

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Recent developments are reviewed. The terms pulmonary asbestosis and pleural asbestosis should be introduced to avoid confusion between the two. Pulmonary compliance is the single most valuable test of lung function in the detection of pulmonary asbestosis, and a method has been described recently which may make this measurement practicable in the routine surveillance of an asbestos exposed workforce. Pleural asbestosis may mimic restrictive lung disease but only if the pleural changes are very extensive. There is little evidence linking these essentially benign changes with mesothelioma. It is now thought that the carcinogenicity of asbestos and of other fibres is a function of their physical rather than of their chemical structure. Lung cancer in an asbestos worker can be attributed to asbestos exposure only if there is also evidence, albeit perhaps only histological, of pulmonary asbestosis. Accumulating evidence continues to emphasize the importance of cigarette smoking in the lung cancer of asbestos workers. Current standards of workplace hygiene are reviewed and conflicting opinions about methods of medical surveillance compared. In the general urban atmosphere, the amount of asbestos is between one hundred thousandth and one millionth of that held to be safe in the workplace. Motor-car brake linings release little, if any, asbestos in their normal road use. The amount of asbestos in water and beverages is likewise negligible.

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THE biological effects of asbestos are increasingly engaging the interest of scientific workers, and the annual number of papers on the subject published each year continues to increase exponentially (Figure 1).

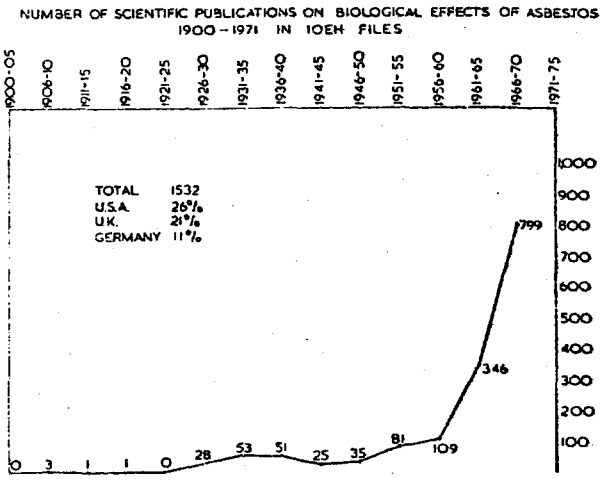

FIGURE 1: Number of scientific publications on biological effects of asbestos 1900 to 1971.

Recently a working group met at Lyons, in France, to review progress in this field. It is largely on the papers delivered at this meeting, and on subsequent work, that the remarks in this paper are based. Earlier work is

referred to only in so far as it is relevant to recent developments.

PULMONARY ASBESTOSIS

Confusion is arising over this term (McCullagh, 1969), since to some the word "asbestosis" means pulmonary asbestosis, while to others it also includes the pleural reaction and the two do not necessarily go together. My opinion is that the specific term "pulmonary asbestosis" should be adopted.

In the 1960s it was generally held that emphysema was not a feature of pulmonary asbestosis. This view has, in recent years, been losing ground, and Fournier-Massey has lately reported that emphysema was found in one-third of the asbestotics in the recent and most important Quebec study (M. R. Becklake, 1972, personal communication).

The former ILO radiographic classification of the pneumoconioses has been reviewed and a new classification, more suited to the radiographic changes of asbestosis, has been prepared (Bohlig *et alii*, 1970) and validated (Rossiter, 1972). Standard films illustrating this classification may now be obtained from the International Labour Organization in Geneva.

One of the most important recent developments is the study reported by Jodoin *et alii* (1971). A group of 24 men was studied; all had had normal chest radiographs but 13 had had a substantially greater exposure to chrysotile asbestos than the other 11. A battery of lung function tests was administered to them, and static pulmonary compliance was outstandingly the most reliable of these in separating one group from the other ($P < 0{\cdot}005$). The early detection of pulmonary response to asbestos exposure is of particular importance in the routine, periodical medical examination of asbestos-exposed workforces. Heretofore we have had to rely primarily on the chest radiograph but it now appears that a much more sensitive screening test may be available. The late Dr McKerrow and his colleagues (Bevan *et alii*, 1971) have devised a technique which eliminates the need for the balloon; they report that, except in the grossly obese and those whose respiratory disability necessitates the use of their sternomastoid muscles in quiet breathing, "early results suggest a good correlation with the inspiratory static compliance using the oesophageal balloon in normal subjects and in patients with pulmonary fibrosis and emphysema". The suitability of this method for the medical surveillance of an asbestos-exposed workforce is currently being investigated by us in association with Professor Bryan Gandevia and his colleagues of the University of New South Wales. Diffusing capacity, incidentally, is of little value in the early diagnosis (Harries, 1971; Becklake *et alii*, 1972).

PLEURAL ASBESTOSIS

Since 1927, thickening of the pleura has been associated with exposure to asbestos (Cooke, 1927) and, more recently, several other substances (Smith, 1952). These changes have generally been held to be of no more consequence than the callusses on the hand of a smithy. At Lyons, Jones (1972) emphatically held to this view. On the other hand Harries (1971) has reported substantial decrements of lung function in men who had only pleural, and no pulmonary asbestosis. Becklake *et alii* (1970) have expressed the view that such decrements only occur with very extensive pleural thickening.

Eisenstadt (1965*a* and 1965*b*) has reported four cases of mesothelioma following apparently benign pleurisy with effusion. Some clinicians have inferred, unjustly I think, that the changes we have referred to here as pleural asbestosis may on occasion give rise to pleural mesothelioma. It seems improbable that pleural asbestosis, which is known to be parietal (Thomson, 1970*a*), should be the precursor of mesothelioma, which is believed to be a tumour of the visceral pleura (Thomson, 1970*b*). It is also to be noted that while exposure to the asbestos anthophyllite commonly leads to pleural thickening (Raunio, 1966), it has never been known to lead to mesothelioma (Raunio, 1966; International Agency for Research on Cancer, 1972).

In Canada, G. W. Gibbs (1972, personal communication) has expressed the view that "pleural asbestosis" is more likely to be due to talc or mica, and indeed, in the formal report of the Lyons meeting it is recorded that "pleural plaques have been associated with past exposure to all commercial types of asbestos. But additional factors, other than asbestos itself, are involved" (International Agency for Research on Cancer, 1972).

It has been suggested (Kiviluoto, 1965) that the incidence of pleural asbestosis might be used as an epidemiological tool. In the study of those known to be substantially exposed to asbestos this may be so, but in the general urban community it is not so. Gilson (1969) found pleural thickening in 187 out of 3,860 routine chest X-ray films taken in the Midlands. The asbestos exposure of 113 of these subjects was compared with that of 113 age and sex matched controls. He found only "a slight but unimpressive excess of positive histories of exposure to asbestos among the cases".

THE CARCINOGENICITY OF FIBRES

Evidence is increasingly accumulating to show that the carcinogenicity of asbestos is a function of its physical, not its chemical, characteristics and that this ability is shared by other fibres having similar physical characteristics (Stanton, 1972; Timbrell, 1972). We have all long wondered why the mesotheliomogenic potency of crocidolite should be so much greater than that of other asbestoses, among which is included the particular form of crocidolite found in the Transvaal (Sluis-Cremer, 1965). Timbrell and his colleagues (1971) have produced very good evidence to show that it is the fine straight fibre of most crocidolites, including that formerly mined at Wittenoom in Western Australia, which enables them to be more readily and deeply inhaled and thus it is these fine crocidolites, much more than any other asbestoses, which give rise to mesothelioma. It is to be noted, however, that Cottrell and Holt (1972) have failed to confirm this work; the matter is thus *sub judice*.

The view that lung cancer associated with asbestos exposure is, in fact, due not to asbestos but to carcinogenic oils (Harrington and Roe, 1965; Gibbs, 1969) or to some associated trace metal or benzpyrene (Dixon *et alii*, 1970) is not now generally held.

THE ROLE OF TOBACCO SMOKING

The very important role of cigarette smoking in asbestos-associated lung cancers was first pointed out by Selikoff

and his colleagues (Selikoff *et alii*, 1968). In the continuing study both of Hammond and Selikoff's group of insulators in New York (Hammond and Selikoff, 1972) and by Muriel Newhouse and her colleagues (1972), of a group of asbestos textile workers in Britain, there has been no excess of lung cancer among asbestos workers who did not smoke cigarettes. There is no evidence that tobacco smoking plays any role in the development of mesotheliomata.

RESPIRATORY CANCER

This term is used here since the international classification of causes of death, on which most records, and therefore most epidemiological studies, are based, does not distinguish between lung cancer and pleural mesothelioma.

The only important recent development here is that reported by Newhouse. She has followed, up to 1970, a group of male asbestos textile workers first employed after the introduction of the British Asbestos Regulations of 1933, but before 1964. These she divided into those who had experienced high, moderate and low exposures. She had earlier reported (Newhouse, 1969) an excess of respiratory cancer among those who had experienced a high exposure, but she has now reported (Newhouse, 1972) that such an excess is beginning to appear among those whose exposures had been only low to moderate, though not if they had been exposed for less than two years.

On the other hand things are perhaps not quite as grave as Hammond and Selikoff's (1972) data would suggest. Among their 370 insulation workers, there have been 42 deaths from lung cancer, when only 4·75 were to be expected on the basis of age-specific male white United States death rates. The comparison would have been based better on local standardized mortality rates; but this objection does not substantially affect the conclusion to be drawn from the data.

Lung cancer, attributable to asbestos exposure, "occurs, so far as is known, only in workers whose lungs show signs of pneumoconiosis" (International Labour Office, 1972). However, this does not necessarily imply clinical signs of pulmonary asbestosis; the evidence may lie in the parenchymal histological findings.

MESOTHELIOMA

It is now generally accepted (Wagner *et alii*, 1971) that some 85% to 90% of mesotheliomata are attributable to asbestos exposure while, in the remaining 10% to 15%, the cause is unknown. Likewise, it is generally accepted (International Agency for Research on Cancer, 1972) that all commercially used forms of asbestos, with the exception of anthophyllite, may give rise to mesothelioma, though the overwhelming majority of cases of this still uncommon tumour are attributed to the fine-fibred crocidolites. Throughout the world, the asbestos chrysotile accounts for more than 90% of total usage.

Davis (1972), of the Institute of Occupational Medicine in Edinburgh, has produced "mesotheliomata" in mice and rats using intraperitoneal emplacement of crocidolite and fibreglass. He has shown that, at least initially, the abnormal cell reproduction is sub-mesothelial and takes place under a continuous layer of undisturbed mesothelium which only later disintegrates. This observation raises the interesting, if academic, question: "Are mesotheliomata really mesotheliomata or are they something else?"

OTHER CANCERS

Excessive numbers of cases of gastrointestinal cancer among those exposed to asbestos continue to be reported (McDonald, 1972; Meurman *et alii*, 1972; Newhouse, 1972; Selikoff and Hammond, 1972) but in no case has the excess been statistically significant. A similar excess of cases of laryngeal carcinoma has been claimed (Stell and McGill, 1973), though the claim has been criticized (Holmes, 1973).

The claim that exposure to asbestos was associated with ovarian tumours has not been supported by the first large mortality survey of women previously exposed to asbestos (International Agency for Research on Cancer, 1972).

THE WORKPLACE HYGIENE STANDARD

Briefly put, in the United Kingdom the standard is a time-weighted average of two fibres per cubic centimetre (f./cm³) (British Asbestos Regulations, 1969; (British) Department of Employment and Productivity, 1970); at the moment in the United States it is 5 f./cm³, but will become 2 f./cm³ on July 1, 1974 ((United States of America) Department of Labour, 1972) though it is to be noted that the Threshold Limit Value Committee of the American Conference of Governmental Industrial Hygienists stands firm in its view that the standard should remain at 5 f./cm³ (Stokinger, Chairman of the TLV Committee, 1972, personal communication). In Australia the standard, though it has at present no statutory authority, is a time weighted average of 4 f./cm³ (National Health and Medical Research Council, 1970).

The British Standard is based on the study of some 300 asbestos textile workers (British Occupational Hygiene Society, 1968). The recent study of 908 men in New Orleans who were employed in manufacturing asbestos cement products (Weill *et alii*, 1973) suggests that the standard should probably lie somewhere between 4 and 5 f./cm³. One cannot say that any of these standards has a sound scientific foundation, but in the American study exposures appear to have been more accurately established.

It is, I think, unanimously agreed that the most accurate method of measuring workplace asbestos-in-air levels is the counting of fibres on a membrane filter on which they have been collected from a known volume of air (a procedure which, however, entails much tedious microscopy). Recent provisions for the surveillance of workplace asbestos-in-air levels in the United States ((United States of America) Department of Labor, 1972), and in Germany (Shultz, 1972, personal communication) impose such a burden by this tedious microscopy that in both countries (Holmes, 1972; Shultz, 1972, personal communications) there is an inclination to return to gravimetric sampling. However, the accuracy of this method leaves very much indeed to be desired. A happier solution would be the further development of a device known as the Quantimet-720 which, it is hoped, will prove capable of automatically counting one complete membrane filter every four minutes.

MEDICAL SURVEILLANCE OF THE WORKFORCE

The usual practice, when a workforce is exposed to some substance known to be hazardous, is to require a periodical medical examination, as is now the case in America ((United States of America) Department of Labor, 1972). No such provision is made in the British regulations. When these regulations were drawn up it was decided that it would be far better to study, with great care, the men working for the major employers, whose exposure levels were measured and known and the progress of whose health could be watched by a factory doctor with special knowledge and understanding of the problem, with H.M. Factory Inspectorate supervising and coordinating the study. Proceeding thus it is to be expected that our knowledge and understanding of the biological effects of asbestos will progressively increase and that the adequacy of the hygiene standard will be more readily and promptly assessed.

I am in no doubt that this philosophy is the correct one and very much hope that any regulations that may be adopted in Australia will be so framed as to enable this approach.

I should add that the accurate counting of asbestos fibres on a membrane filter cannot be done by the inexperienced, and two laboratories counting the same filters have been known to disagree by as much as a factor of 15 (Beckett, 1972, personal communication). Laboratories in the United Kingdom, the United States and Australia are now collaborating to reduce these differences and to establish interlaboratory correlation. Unless they achieve these goals it will remain impossible to measure the dose-response relationship in man.

THE GENERAL URBAN ENVIRONMENT

Asbestos levels in the general atmosphere are of the order of nanograms per cubic metre (10^{-9} gm/m^3) and can only be measured by electron microscopy. In 16 such measurements in and about the town of Rochdale in England, the level in seven cases was of the order of 10^{-9} gm/m^3 and in nine cases of the order of 10^{-10} gm/m^3. Unexpectedly the level on the moors was found to be higher than in the Rochdale town centre. The British workplace hygiene standard of 2 f./cm^3 is equivalent to 0·1 mg/m^3 or 10^{-4} gm/m^3 ((British) Department of Employment and Productivity, 1970). Thus the level of asbestos in the urban air is about one hundred thousandth to one millionth of the hygiene standard. Similar studies in the United States have reported levels of the order of 10^{-9} gm/m^3. Even "in lower Manhattan about construction sites where extensive spraying of asbestos containing fire-proofing material was taking place", asbestos in air levels one-eighth to one-quarter of a mile (some 200 to 400 metres) away averaged 60 × 10^{-9} gm/m^3, the highest count being 375 × 10^{-9} gm/m^3 (Nicholson and Pundsack, 1972; Selikoff *et alii*, 1972). There is no evidence to suggest that urban asbestos-in-air levels are anything other than harmless.

Something should be said about the much-maligned brake lining. Every application of the brake, it is widely believed, releases a shower of fibrous asbestos into the atmosphere. This was never a very promising hypothesis since it is most unlikely that any asbestos would survive the temperatures generated at the braking interface. Lynch (1968) of the United States Public Health Service, having studied the matter, concluded that "only a very small fraction of the asbestos escaped as free fibre while the remaining was transformed into some other non-fibrous material. A significant release of free fibre occurred only under conditions extreme enough to produce brake failure". There have been several other studies but none that I know of has led to a contrary conclusion. Selikoff (1970) summed the matter up: "Brake linings do not constitute a hazard".

Since asbestos occurs commonly in nature, though only infrequently in commercially worthwhile amounts, it has no doubt been present in many of the world's drinking waters since time immemorial. Its presence in North American waters (Cunningham and Pontefract, 1971; Nicholson and Pundsack, 1972) and in British beer (Biles and Emerson, 1968) has lately been reported. It has been estimated by the Asbestos Information Committee in London that the total annual British output of beer, of over one thousand million gallons, contains only two thousandths of an ounce of asbestos, or some 10^{-9} gm/m^3.

CONCLUSION

The industry is well aware of the hazards of asbestos, and having briefly reviewed these I think we should also remember that, if we consider no more than its fire-retardant properties and its use in brake linings, asbestos has saved far more lives than it has claimed. With the great improvement of standards of industrial hygiene over the last decade this credit balance, if I may so call it, will increasingly grow more favourable.

ACKNOWLEDGEMENT

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In the Forum

Is Short-Fibered Asbestos Dust a Biological Hazard?

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Contrary to the determination that the finer the quartz dust, the greater its pathogenicity; the pathogenicity of the finest asbestos dust has been shown to be negligible.

It has been the finding of research laboratories in Germany, England, South Africa, and the United States that short-fibered asbestos, dust, ie, less than 5μ in length, is incapable of causing fibrosis or cancer. This finding, in conjunction with the failure of different laboratories in the United Kingdom and in this country to discern abnormalities following prolonged asbestos feeding to rats, should lead to the abandonment of the present concept that maintains that mesotheliomas and gastrointestinal cancers arise from the ingestion of asbestos dust cleared from the lungs.

These negative results should also allay the alarm that has been raised as a result of the finding of ultramicroscopic mineral fibers in certain beverages and drinking water.

By short-fibered asbestos dust is meant that which has a fiber length of less than 5μ. Although fibers of this size usually constitute a very small fraction of the weight of a dust cloud, their numerical preponderance over the larger fibers may be manifold.

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Inasmuch as asbestos fibers smaller than 5μ tend to remain airborne longer than the larger ones, they have a greater chance of being inhaled. Furthermore, although the anatomy of the respiratory tract tends to prevent the intrusion into the airspaces of all but a few of the larger suspended particles, this deterrence does not extend to the smallest particles. The latter very readily enter the airspaces with the inspired air. Some of the short asbestos fibers may settle on the alveolar surface by sedimentation, whereas the smallest fibers, behaving almost like gas molecules, contact the alveolar membrane by diffusion.

What is the potential of these extremely fine submicronic fibers to produce disease? Is their potential greater than that of optically visible fibers? Is the behavior of submicronic asbestos fibers as opposed to that of larger fibers similar to that of the fibrogenic effect of very fine quartz dust as compared with that of the same weight of coarser quartz particles?[1,2]

These questions take on added importance in view of the commonly held hypothesis that mesotheliomas of the pleura and peritoneum arise by the transmigration of fibers to the pleura and peritoneum, respectively. In the case of abdominal mesotheliomas, it is assumed that the asbestos fibers cleared from the lungs are swallowed and then migrate through the intact intestinal wall to the peritoneum, there to initiate the development of mesotheliomas. So far as ability to penetrate into and transmigrate across the intact intestinal wall is concerned, once again it would appear that the submicronic fibers would be better able to accomplish this feat than would the coarser fibers.

Originally, the question of the pathogenicity of the short-fibered asbestos dust had relevance only to people occupationally exposed to asbestos; but more recently short asbestos fibers have been found in certain beverages and city water, in ambient community air, and in the lungs of city dwellers.[3,4] Consequently, the relevance of the above question must now extend to entire urban populations. However, lest undue alarm be raised by the last statement, it should be pointed out that in city dwellers no disease has been found that could be attributed to the presence of submicronic asbestos fibers in the pulmonary tissues. Neither has there been documentation of an increase in abdominal cancers in the general population, in spite of the fact that in many cities and smaller communities drinking water has been and is now transported in asbestos-cement pipes.

At the International Conference on the Biological Effects of Asbestos held in Dresden in 1968, Klosterkötter[5] found that both chrysotile and crocidolite, ground to an average fiber length of less than 5μ when injected intratracheally or intraabdominally, produced no fibrosis. The

pulmonary response consisted only of a macrophage reaction. In contrast, longer fibers of the same asbestos resulted in fibrosis in both regions. At the same conference Timbrell and Skidmore[6] reported the results obtained in rats and guinea pigs exposed to equal concentrations (by weight) of short-fibered amosite (90% of the fibers <4μ long) and long-fibered asbestos (45% of the fibers >4μ long). They concluded, "minimal reaction has been observed to short fibres but a marked reaction has been observed to the longer fibres." In the following year, Webster[7] reported that monkeys inhaling finely ground crocidolite (fiber length <5μ) also showed merely a macrophage reaction in the lungs. In 1970, Hilscher et al[8] showed the chrysotile or crocidolite, when ground to a fiber length of <3μ with a microtome and injected intraabdominally, produced no fibrous adhesions; whereas the same asbestos with greater fiber length did cause dense fibrous adhesions.

In 1971, the Johns-Manville Research Laboratory prepared for us chrysotile asbestos ground to a fiber length of <5μ. We injected this dust intratracheally into 10 rats and were able to confirm that such short-fibered asbestos could induce no more than a macrophage reaction (unpublished study). In 1973 Smith et al[9] reported that hamsters injected intrapleurally with chrysotile ground to a fiber length of <1μ developed no pleural cancer, whereas hamsters injected intrapleurally with longer chrysotile fibers did develop such cancers. Recently Wright (private communication) disclosed that in his laboratory short-fibered asbestos injected intratracheally also failed to elicit a fibrotic reaction. Maroudas et al[10] have concluded that, "Particles (mineral fibers) smaller than 20μ in length induce neither growth in vitro nor mesothelioma in vivo."

Thus, these reports from different laboratories are unanimous in finding asbestos that has an average length of <5μ is devoid of pathogenic potential. This included not only the fibrogenic potential[6-8,10] but also the cancerogenic potential.[9,10]

It may be argued that when asbestos is ground to a very small fiber size, either in a ball mill or a hammermill, much of the energy is converted into heat and the heat may change the chemical structure of the fibers. To continue this argument: since, strictly speaking, the fibers so altered may no longer be asbestos, the biologic "inertness" of such "altered" asbestos need not necessarily apply to fine asbestos dust that has not been heated to a high temperature.

This argument is rendered void by the following facts:

1. The short-fibered asbestos of Hilscher et al[8] was found to have maintained its fibrous structure after the grinding process.

2. Smith et al[9] prepared short-fibered asbestos as an aqueous slurry. This obviated excessive heat.

3. It has been concluded that the chemical structure of asbestos does not determine its pathogenicity since synthetic chrysotile is devoid of pathogenicity.[11] The latter has the same chemical and crystalline structure as the natural product. Therefore, the mere process of grinding with the associated heat production does not account for the lack of pathogenicity of the finely ground asbestos. However, by fitting together some newly derived experimental findings, a theory has recently been formulated regarding the locus of pathogenicity of asbestos dust that does offer a reasonable explanation for this lack of pathogenicity.[12]

There is, however, one laboratory that reported that short-fibered asbestos is tumorigenic. Pott and Friedrichs[13] and later, Pott et al[14] maintained that 100 mg of chrysotile with a fiber length <3μ injected into the abdomen of rats caused the development of cancers. Nearly 80% of the tumors were sarcomas—mostly fibrosarcomas.

The character of the tumors produced by this technique should have given the authors pause for reflection; not only because rats will produce fibrosarcomas secondary to injected or imbedded materials known to be biologically inert, but also because subcutaneous fibrosarcomas are very common spontaneous tumors in aging rats. An indication of the ease and nonspecificity of such tumor production in rats is demonstrated in the first[13] of the two above-mentioned papers when the authors list a better-than 60% tumor production with magnesium hydroxide and a 55% tumor production with fibrous glass! In contrast, they reported only a 40% tumor production with chrysotile. This lower tumor production was doubtlessly related to a high mortality caused, in turn, by the exceedingly high dosage of materials injected (100 mg).

The employment of unrealistic dosage, of inappropriate routes of administration, and of inappropriate animal species (all three "sins" were committed by the above authors) to achieve positive results has recently been adequately discussed by Dr. H. E. Stokinger.[15]

When asbestos is ingested, it is the ultramicroscopically-sized asbestos fibers that are assumed to be responsible for the development of mesothelioma by virtue of their alleged penetration and transmigration through the intact intestinal wall. The failure of short-fibered asbestos to induce mesotheliomas when injected intrapleurally[9,10] makes the above assumption highly questionable. Unpublished data from different laboratories (David B. Clayson, University of Leeds; L. M. Swinburne, St. James's Infirmary, Leeds, England; and John M. G. Davis, Institute of Occupational Medicine, Edinburgh) in which rats were fed asbestos intimately mixed in their food, indicate complete failure to induce tumors or any other kind of abnormality by these regimens. (A joint paper describing these investigations from the different laboratories is in preparation.)

As one example, the following experiment may be cited: ten weanling male rats were placed on a finely ground basal diet containing 5% by weight of chrysotile asbestos. Five litter mates were pair-fed with the same weight of food as the experimental rats had consumed on the previous day. This regimen was continued for 21 months. At the end of this time, the weight curve of the asbestos-fed animals was not significantly different from that of the pair-

fed controls. The animals were killed 21 months after the initiation of the feeding period. At autopsy, no gross abnormality was found in either group of animals and microscopically no tumor or other gastrointestinal lesion was observed.[16] It is to be noted that in previous studies, the first asbestotic lung cancer death occurred 16 months after the initiation of the dust exposure[17] and the first asbestotic pleural cancer death in rats occurred 17 months after the intrapleural injection of asbestos dust.[12]

It is of interest in this connection that the dose of fibers in the intestinal tract of the asbestos-fed rats was astronomical compared with the dose of fibers that is likely to be swallowed daily by a person occupationally exposed to asbestos dust—and he, in turn, would have an astronomically greater dose of fibers than the dose of fibers ingested daily by an urban dweller drinking a beverage or water containing mineral fibers.

The uniformly negative asbestos-feeding results should cast some doubts on the tenability of the concept that peritoneal mesotheliomas and an increased prevalence of gastrointestinal cancers arise in occupationally asbestos-exposed people from the ingestion of asbestos fibers cleared from their lungs. There must, of necessity, be some other explanation!

Although not an asbestos-feeding study, a recent report purports to demonstrate that the presence of asbestos in the intestinal lumen results in the penetration of asbestos fibers into the blood stream and organs throughout the body, inclusive of the brain.[18] The writers injected the asbestos into the stomach by means of a syringe and needle, thereby ignoring the probable opening of vessels in the path of the needle track and the presence of injection!

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Rebuttal

In looking through "Biological Effects of Asbestos" (*Ann NY Acad Sci* 136:87, 1965) I find one paper by Holt, Mills, and Young that says very small asbestos particles do cause fibrosis in the guinea pig lung. In the published discussion, no one challenges this result; one discussant, Ian Webster from South Africa agrees with it, and Gilson quotes it approvingly in his final wrap-up. At least in 1965, ultramicroscopic asbestos particles were believed to have fibrogenic potential for guinea pigs.

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I reply that Paul Holt used the same make hammermill to comminute his asbestos as I used. I fully agree with his statement that a high proportion of the particles to which he exposed his guinea pigs was too small to be seen by the light microscope.

The more important aspect of Holt's statement is the *long fibers were present* in the dust cloud. Having seen his set-up, I was impressed by the high density of the dust concentration (unmeasured!) to which his guinea pigs were exposed. The dosage of long-fibered (optically visible) particles must have been enormous whereas the dosage of submicronic fibers (those visible only with the electron microscope) must have been astronomical.

Holt's finding of many optically visible fibers in the lung sections of his animals as pictured in his illustrations and of many asbestos bodies attest to the plentiful dosage of long fibers. This undermines his claim that "Fine dust particles, too small to be seen under the light microscope, will produce asbestosis in the guinea pig."

The determinant(s) of asbestos toxicity is not known and I make no claim to such knowledge. However, in this article, I point to one aspect of asbestos dust which is not associated with pathogenicity.

By "submicronic" is meant something invisible with the light microscope but visible with the electron microscope. This generally means a particle $<0.25\mu$ in thickness. Although the vast bulk of fibers that have been ground to a length $<5\mu$ are submicronic, some would be thicker than 0.25μ and therefore, optically visible. Perhaps, it would be best not to specify "submicroscopic" and speak only of "short" fibers as defined in the opening sentence of the report.

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