"B-READERS" AND ASBESTOS MEDICAL SURVEILLANCE

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ABSTRACT

Civilian "B-Readers" certified in ILO methodology interpreted randomly distributed asbestos medical surveillance X-rays of more than 105,000 U.S. Navy employees. Analysis of 23 participating observers demonstrated a three hundred-fold prevalence of perceived "definite" pulmonary parenchymal abnormalities. There was an evident geographic component to interpretation habits, with east and west coast observers more likely to interpret films as abnormal than observers from the midcontinent. The most expert observers, a group who instruct the course leading to NIOSH certification in ILO methodology, also perceived fewer abnormalities than other readers. Instructors still exhibited a seven-fold prevalence range of positive interpretation. Under usual surveillance conditions, the habits of "B-Readers" appear to have a major impact upon the X-ray interpretation of asbestosis. Certification in "B-Reading" should not be the only quality assurance for radiographic surveillance programs, medical decision-making, nor related legal activities. Epidemiologic comparison between populations should account for the apparently wide spectrum of interpretation habits.

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IS THE US COAL MINER CHEST X-RAY SURVEILLANCE PROGRAM SUCCEEDING IN CONTROLLING LUNG DISEASE?

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The 1969 Federal Coal Mine Health and Safety Act established a system of periodic chest X-ray examinations for underground coal miners in the United States.[1] The program, as operated by the National Institute for Occupational Safety and Health since 1970, has both surveillance and screening components.

The surveillance component of the program is directed toward observation of the incidence of coal workers' pneumoconiosis in the population of working miners. There is an expectation that the program can document decreasing disease incidence as exposure controls are put in place.

The purpose of medical screening is detection of asymptomatic disease in individuals at a point at which intervention will favorably affect disease outcome. It is a back-up mechanism to reduce impairment when environmental controls are insufficient to prevent disease in individual miners.

This paper explores whether the current surveillance and screening program is functioning adequately to contribute significantly to the reduction or elimination of lung diseases in underground coal miners. Both the surveillance and screening components depend upon the use of tests which can identify lung diseases of importance. They require high levels of participation by miners at greatest risk of disease and acceptance of preventive interventions. Ultimately, the measure of the success of the program is the extent to which the development of pulmonary impairment in coal miners is abated.[2]

PROGRAM STRUCTURE

The 1969 Coal Mine Health and Safety Act was the first legislation to establish a national program for medical surveillance. The X-ray program was continued under the Federal Mine Safety and Health Act of 1977 [MSHAct]. The MSHAct also contained an expanded mandate to utilize medical screening as a preventive strategy. When a determination is made that miners may suffer "material impairment of health or functional capacity" as a result of hazardous exposures, removal from exposure and reassignment must be offered.[3] To date, the periodic X-ray program is the only effort to fulfill the mandate for ongoing screening for dust diseases.

The MSHAct requires that all miners receive chest X-rays on entering the work force and after three years of work. Thereafter, periodic X-rays must be made available to coal miners at no less than five year intervals. The X-rays are offered at no cost to the miners and, according to regulation, at locations and times convenient to the miners. Acceptance of the later X-rays is voluntary.

The law provides for transfer of miners from areas of higher to lower dust exposure if they show signs of the development of pneumoconiosis on the basis of the X-rays or "other medical examinations." Alternatively, low dust levels can be achieved in the miner's current job through engineering controls. By regulation, exposure control for miners who participate in the program must be confirmed through frequent personal dust sampling.[4] Exercise of these transfer rights is at the discretion of the affected miner.

In practice, the right to transfer is offered only on the basis of X-rays read as positive for coal workers' pneumoconiosis utilizing the ILO method of X-ray interpretation.

TEST SELECTION

The MSHAct obligates the Secretary of Labor to develop mandatory health standards including, where appropriate, medical examinations to determine whether workplace exposures are adversely affecting a miner's health. The medical literature at the time the X-ray program was initiated concentrated on coal workers' pneumoconiosis as the characteristic and single important response of the lungs to inhalation of coal mine dust. Prevention of impairment from dust exposure was assumed to depend on elimination of Progressive Massive Fibrosis.[5] The transfer option is predicated on the assumption that PMF can be eliminated if the progression of simple CWP can be halted or slowed or through reduction of ongoing exposure in affected individuals.

The current literature is broader in its focus. Recent studies consistently demonstrate a range of pathological and physiological abnormalities in miners.[6] For example, pathologically confirmed emphysema is found more commonly in miners than non-miners even when the analysis controls for smoking status.[7,8,9] Symptoms of chronic bronchitis occur with increased frequency in both smoking and non-smoking miners as duration of coal mine dust exposure increases.[10] These symptoms may be associated with clinically significant impairment.[11,12] Miners with symptoms of chronic bronchitis tend to retire earlier with disability than those without these symptoms.[13]

Longitudinal studies in the US and UK demonstrate excess loss of FEV_1 in miners when compared to control populations.[14,15] This excess loss is related to dust exposure after the effects of cigarette smoking are considered. A subset of miners may develop severe pulmonary impairment in the absence of PMF.[11]

Further, both smoking and non-smoking miners have manifested abnormalities of gas exchange demonstrable on exercise testing.[16,17] Mortality studies of miners have consistently demonstrated that former coal miners die from chronic respiratory diseases at excess rates.[18]

People manifesting dust-related impairments do not necessarily have radiographically demonstrable CWP.[15,16] The chest X-ray appears to be neither sensitive nor specific for the identification of individuals with functional loss resulting from coal mine dust exposure. One cannot differentiate between miners with lung disease and those without through exclusive reliance on the chest X-ray.

PARTICIPATION

The periodic X-ray program has been plagued by low and diminishing participation by eligible miners. Administratively, program activities have been divided into four time periods or "rounds" thus far. If the compulsory films required of miners entering the work force are eliminated from consideration, approximately 32% of eligible miners participated in round three (1978–81), the latest round for which data is available. This is down from the 44% participation rate during round two (1973–8), and approximately 50% participation in the initial round (1970–3).[19]

The distribution of participants by mining experience is also significant. (see Table I) Approximately 35% of participants in round one had worked for twenty or more years in mining. In round two, only 12.4% of participants had worked this long. By round three, the percentage of participants with twenty or more years' experience was further reduced to 10.4%. In part this may reflect an evolution of the work force with older miners retiring and younger miners being hired. However, this hypothesis cannot be tested at this point; the necessary demographic data detailing the age and tenure distribution of the mining work force over time is lacking.[19]

A number of problems contribute to poor participation in the program. Some approved facilities are not, in fact, convenient for miners. Miners must take examinations during their off-work hours. Travel time can be as much as an hour from the mine site, and may be further from the miner's home. The facilities are selected by the employer and may be the same ones that provide pre-employment examinations as well as evaluative examinations used to contest workers' compensation claims. There is limited understanding of the nature and purpose of the program among coal miners, employers, and health care providers in the coal mining areas. Concerns about confidentiality and adverse impact on future employment are widespread.[20]

Miners who have worked longest on average have the greatest lifetime dust exposures. Low participation rates by the most experienced miners could distort understanding of disease patterns in the mining population and diminish the value of the screening function of the program.

TRANSFER ACCEPTANCE

The primary preventive intervention offered by the X-ray program is transfer with pay rate retention from a high to lower dust exposure job for individuals demonstrating CWP on chest X-ray. Miners are permitted to exercise this transfer option any time after notification of their eligibility status. Through the life of the program, 9138 miners have been eligible for transfer but only 2119 have exercised this option. The number of miners actually working who have exercised the option has declined from a total of 550 at the end of 1981 to 140 by the end of 1987.[21]

The consequences of delaying or failing to exercise transfer rights means that most miners who are identified through

Table I
Percentage Distribution of Participants in
Rounds 1, 2, and 3, by Tenure Group. 1970–1981

Years in Mining	Round 1 (1970-73)	Round 2 (1973-78)	Round 3 (1978-81)
0-4	42.1	68.7	51.9
5-9	9.8	11.4	24.8
10-19	13.1	7.5	12.9
20-29	18.4	6.5	4.6
30+	16.6	5.9	5.8

(reproduced from reference 19)

the screening program as having CWP continue to be exposed to higher levels of coal mine dust than necessary.

IMPAIRMENT DEVELOPMENT

Despite the mandate of the MSHAct to eliminate occupationally-induced health impairments, miners continue to develop dust related disease. The extent to which this is happening is not currently measured. However, data from the surveillance program indicates that miners continue to demonstrate CWP on X-ray.[21]

Indirect evidence from the Black Lung Benefits program supports concern that some miners are developing severe pulmonary impairment in part or in whole from their workplace exposures. In fact, the number of retired miners who are awarded benefits for permanent and total pulmonary disability arising from coal mine employment is greater than the number of active miners being offered transfer rights. (see Table II) Even with a significant tightening of eligibility standards in 1981, over five hundred awards of disability benefits have been made each year to miners who applied after March 1, 1978.[22]

DISCUSSION

The surveillance and screening program for US coal miners was designed almost twenty years ago with a narrow focus on coal workers' pneumoconiosis. With minor modification, the program regulations have remained constant since its inception. The success of the surveillance component of the program is limited, in large measure, by poor participation and incomplete data. Nevertheless, the program has developed an invaluable data base through effective use of limited resources and the strong commitment of involved researchers. However, the surveillance activity has not yet been tied to the institution of primary exposure control measures.[23]

With almost twenty years' experience, it may be time to modify the program. Additional demographic data on both participants and non-participants should be collected. Also, efforts should be made to develop improved exposure information and to tie the surveillance program more directly to exposure control. The causes of non-participation in the program merit serious study. Attempts to overcome these should be continued.

The screening component of the program is more troublesome. It has operated in the shadow of the CWP surveillance activity maintaining the same narrow focus. The overall legislative mandate to identify miners with material health impairment from their workplace exposures and aid them in exposure elimination has remained largely unfulfilled. The current screening program relies exclusively on X-ray-diagnosable abnormalities and excludes consideration of dust induced functional derangement. Miners at greatest risk for the development of asymptomatic disease are least frequently screened. Only a limited number of miners avail themselves of the preventive intervention that is offered. At the same time, the number of miners qualifying for total disability benefits far exceeds the number participating in preventive options.

The screening component of the activity would improve to some extent with expanded participation in the current X-ray program. Nevertheless, until the medical screening focus is broadened to include efforts at early identification of other dust diseases in miners, the impact on overall health status will be quite limited. Part or all of the screening activity should be disaggregated from the surveillance program and new regulations developed. These would require consideration of the range of lung diseases caused by coal mine dust exposure, the methods available to detect them, and interventions that would prevent their progression. Each area presents difficult scientific issues which must be resolved. Nevertheless, it is not too soon to begin to fulfill the promise of the Mine Safety and Health Act of 1977 to "assure that no miner will suffer material impairment of health or functional capacity even if such miner has regular exposure to the hazards for the period of his working life."

Table II

Numbers of Miners Awarded Total Pulmonary Disability Benefits for Claims Filed After March 1, 1978, and Miners Offered Transfer Option. 1981–1985

Year	Awarded Benefits	Transfer Option Offered
1981	5148	245
1982	1145	119
1983	763	94
1984	556	271
1985	570	79

(source: references 21,22)

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EPIDEMIOLOGIC SURVEILLANCE BY A STATE HEALTH DEPARTMENT USING THE ILO CLASSIFICATION SYSTEM FOR PNEUMOCONIOSES

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Since October, 1985, the Wisconsin Division of Health has provided radiologic consultation using the ILO classification system for pneumoconioses. X-ray interpretations have been provided to physicians and to workers with a significant history of occupational exposure to silica or asbestos. The X-ray interpretations when evaluated in association with their respective occupational histories, provide a data source to complement other methods of epidemiologic surveillance. This project has reviewed 1124 X-rays in 2 and 1/2 years. Of these, 663 or 59% were normal; 233 or 21% showed abnormalities consistent with pneumoconiosis; the remainder showed other abnormalities judged not related to dust exposure.

Data sources available for occupational disease surveillance include death certificates, workers' compensation cases, hospital discharges and third party liability lawsuits. These data sources which focus on end stage disease apparently underestimate the incidence of pneumoconiosis when compared with the results of the voluntary radiologic consultation program using the ILO classification. For example, during the two and 1/2 years of this program, 36 of the approximately 100,000 death certificates filed with the State Health Department listed pneumoconiosis as a cause of death, whereas our program, which is not population based and which employs selective criteria for participation identified 233 individuals with X-ray abnormalities consistent with pneumoconiosis. Thus, the active provision of radiologic consulting services can effectively supplement existing passive data sources for epidemiologic surveillance.

A strength of this program has been the ability to use medical surveillance to identify and remedy exposure hazards before the exposure has resulted in end stage disease. Impediments to the program have included the lack of occupational histories in most patients' medical files, physicians' difficulties in recognizing occupational disease and the unexpected finding that 24% of the films reviewed were of marginal quality for ILO Pneumoconiosis Classification. The experience of this program indicates a need for quality assurance programs to maintain film quality if the ILO Classification system and the B reader programs are to be used to their full potential.

INTRODUCTION

Wisconsin enjoys a well deserved reputation as a America's Dairyland. As of 1986, Wisconsin was the leading producer in the United States of milk, butter and cheese. Wisconsin is also the leading producer of green peas, beets, cabbage and sweet corn for processing. Although 41% of Wisconsin's land area is devoted to agricultural production, Wisconsin's population is more industrial than agricultural, with only 6% of the population engaged in agriculture, compared to 28% employed in manufacturing. Wisconsin's strength as an industrial producer is less well known, but we lead the nation in the production of small horsepower gas engines, outboard motors, power cranes and other mining and construction equipment. The state also leads in the production of writing paper, sanitary tissue products and laminated and coated process paper.

Wisconsin has had a progressive record in terms of recognizing the occupational health issues resulting from industrialization. In 1911 Wisconsin passed the first Workers' Compensation law in the U.S. Wisconsin also was the first state to recognize asbestosis disability outside of a manufacturing context when in 1932, a maintenance worker was compensated for disease arising from handling and using insulation materials.[1]

Despite Wisconsin's history of concern and the passage of the Federal Occupational Safety and Health Act in 1970, there is evidence that problems persist. Industrial hygiene evaluations of Wisconsin foundries in the mid 1970's measured 1270 air concentrations of silica and found 41% to be above the federal OSHA standard.[2] Other national studies have confirmed that silica as well as asbestos problems are widespread throughout the U.S. A 1980 U.S. Department of Labor Report to Congress predicted that 6% of all workers in silica exposed industries would develop silicosis.[3] In Wisconsin, with 25,000 silica exposed workers, this would mean, at a minimum, 50 new cases a year for the next 30 years.

NIOSH has identified the pneumoconioses as one of their top ten priority diseases for improved surveillance and prevention activities and the U.S. Public Health Service has set forth the goal, that "among workers newly exposed after 1985, there should be virtually no new cases of four preventable diseases, asbestosis, silicosis, byssinosis and Coal Workers Pneumoconiosis."[4]

Unfortunately it is difficult too for us to quantify progress toward these goals in the U.S., since with the exception of the coal miners' programs, there are no comprehensive national reporting systems in the U.S. for asbestosis, silicosis or other occupational diseases. Those systems which are used to estimate occupational disease incidence have severe limitations. A recent National Academy of Sciences report has concluded that occupational diseases are grossly under reported.[5] However, a number of states have established occupational disease surveillance systems. Since the 1930's, Wisconsin has had laws requiring occupational disease reporting, but compliance has been minimal.

PROGRAM DESCRIPTION

In 1985, with the support and cooperation of NIOSH, Wisconsin began to implement new surveillance efforts, including a review of existing databases. Currently in Wisconsin, for the surveillance of pneumoconiosis and other dust diseases, only three systems provide population based information, death certificates, the tumor registry and workers compensation files. These three systems all record cases of end stage disease. The hospital discharge and ambulatory care surveys provide a broad range of morbidity outcomes, but their usefulness is limited since these data sets are currently only small samples of the annual disease incidence. What all these systems have in common is that they require physician recognition and reporting of the occupational nature of disease. Physician resources in occupational health are limited. In Wisconsin there are only 8 board certified occupational physicians and 9 certified B readers. For the surveillance of occupational disease to be improved, occupational links must be noted at the point of entry into the medical system, when the patient first sees the physician. Also, if occupational disease recognition is to lead to intervention and preventive activity, then more cases of early stage diseases must be recognized. While end stage disease is easier to recognize, it has less utility for prevention. Early stage disease while harder to recognize, provides more opportunity for prevention. For these reasons, Wisconsin, in cooperation with NIOSH, decided to focus our surveillance activities in two directions, a continuous review of data from existing systems, combined with a new radiographic abnormality reporting and interpretation program designed to facilitate the detection of early stages of pneumoconiosis, thje "State pneumoconiosis Radiologic Consultative Program."

In reviewing the existing data, we found that there was no uniform radiographic description of pneumoconiosis used by Wisconsin radiologists with the exception of the B-readers using the ILO Classification system. Thus, in order to meet our surveillance objectives for prevention and to facilitate consistency and uniformity in reporting, we selected the ILO Classification System for a standard definition of radiographic abnormality. (Figure 1) For individuals with asbestos exposure, the case definition for abnormal consistent with pneumoconiosis requires a small opacity profusion of 1/0 or greater and/or pleural thickening or plaques. For individuals with silica exposure, the case definition involves a small opacity profusion of 1/0 or greater. Since few radiologists are familiar with or trained in the ILO system, it became necessary for the state to have a B reader interpret films of exposed workers so as to provide the required consistency of interpretation. From all participants we require that a chest X-ray and a brief occupational exposure history be submitted. In general, we limit participation to those whose first dust exposure occurred 15 or more years ago.

We have explored a variety of methods to promote the program's purposes and availability. These efforts have met with a variety of responses, ranging from indifference to enthusiasm. We have done mass mailings to 2000 physicians and to all AFL-CIO locals in the state and have found both efforts to be remarkably ineffective, generating less than ten requests for X-ray interpretation each. Face to face meetings with groups of exposed workers and physicians known to have an interest in occupational health have been more productive.

Another factor which may have influenced participation is the U.S. Department of Labor OSHA regulations concerning asbestos which were changed in June 1986 to require that physicians examining asbestos exposed workers have access to the standard X-rays prepared by the International Labor Office for the Classification of Pneumoconioses. It is difficult to assess the potential impact of these regulations on the utilization of our non-regulatory voluntary radiologic consultation program, but at a minimum, the 1986 OSHA regulations have increased the public awareness and the credibility of the ILO Classification System and the NIOSH B Reader certification program.

RESULTS

The X-rays received have been from diverse sources including employers, clinics, labor unions, individual workers, physicians and family members. From November, 1985 to July, 1988, multiple promotional activities have resulted in 1124 X-rays submitted. Of the 1124 X-rays submitted, 233 or 21% have shown abnormalities consistent with pneumoconiosis. (Figure 2)

It is interesting to compare the results of this targeted surveillance using the ILO system with the other, more traditional population-based epidemiological data sources available to us (Figure 3) Searching for both underlying and multiple contributing causes of death, silicosis was recorded as a cause of death on an average of 12 death certificates per year from 1981 to 1986. Asbestosis as a cause of death was recorded on an average of 4 death certificates per year during that time. Mesothelioma reports averaged 15 per year from 1981 to 1986 with increasing frequency. In 1987, 40 mesothelioma deaths occurred. In the workers compensation system slightly more silica disease and less asbestos disease has been recorded. From 1982 to 1986 an average of 17 silicosis and 6 asbestosis claims per year were closed. There were another 7 cases per year of other dust related diseases, such as mesothelioma. Our targeted X-ray surveillance system found an average of 86 new cases per year. In addition, our survey of other B readers in Wisconsin found that they read approximately 600 Wisconsin films per year of which approximately 65 or 11% are abnormal consistent with pneumoconiosis. Although the case numbers from our active program are larger than in the existing data sources, our cases are not sufficiently representative to allow a population based description of the total impact of past dust exposure on the population.

In reviewing the industries which have participated in the program, (Figure 4) the paper industry, food processing machinery manufacturing, foundries and construction have contributed more than 60% of the X-rays. These same industries have contributed a similar proportion of the X-rays showing abnormalities consistent with pneumoconiosis. Evaluating the data concerning participation by occupational groups (Figure 5) a similar pattern emerges, with the number of abnormalities found in various occupations reflecting the degree of participation by the exposed group, rather than an indication of the relative risk of dust exposure by industry or occupation such as one could derive from an analysis of population based data.

Serves Two Purposes:

1. **Epidemiological:**

   Provide Information About Pneumoconiosis Incidence

   * **Offers Gradation of extent of abnormality**
   * **Offers Consistent, Uniform Descriptive Method**
   * **Can Be Easily Performed Outside Clinical Context**

2. **Service:**

   Supports Diagnostic Evaluation by Physicians

Figure 1. The ILO Pneumoconiosis Classification System as an epidemiological surveillance instrument.

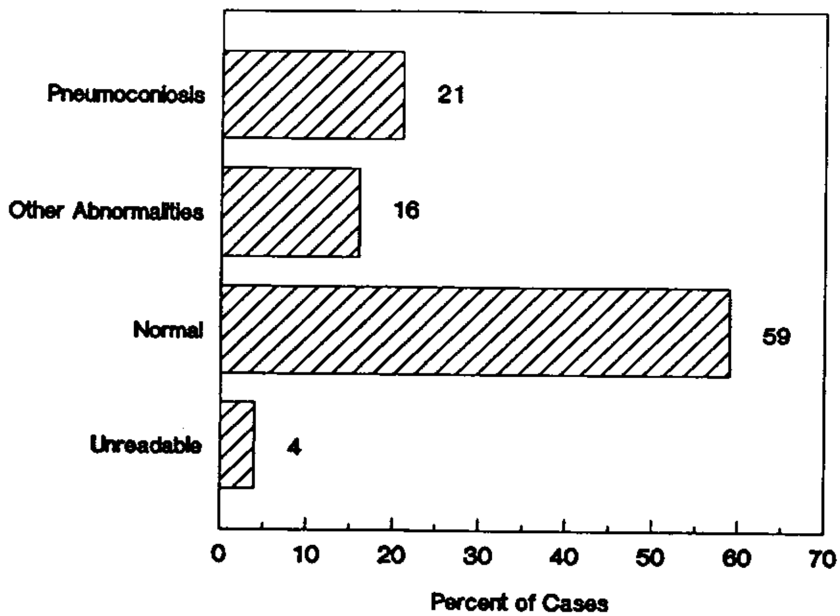

Figure 2. Summary of X-ray interpretations (1985–1988) using ILO Classification System—N: 1124.

Source	Time	Disease	Annual Incidence
* Death Certificates	1981-1986	Silicosis	12
		Asbestosis	4
		Mesothelioma	15
* Workers Compensation	1982-1986	Silicosis	17
		Asbestosis	6
* Third Party Suits	1980-1986	Asbestos Diseases	6
* Targeted Surveillance Using ILO System	11/85-7/88	Pneumoconiosis	86

Figure 3. Wisconsin occupational disease surveillance.

SIC	Industry Name	Number	(%)
2621	Paper Mills (with pulp mills)	315	(28.4)
3551	Food Products Machinery	171	(15.4)
3321	Gray iron foundries	157	(14.1)
1711	Plumbing, heating and air conditioning	85	(07.7)
3011	Tire manufacturing	51	(04.6)
3731	Ship building and repairing	51	(04.6)
3462	Iron and steel forgings	41	(03.7)
4911	Electric Services	34	(03.1)
4931	Electric and other services combined	30	(02.7)
1742	Plastering, drywall, acoustical and insulation	23	(02.1)
	All Other	153	(13.8)

Figure 4. State of Wisconsin, Department of Health and Social Services, Pneumoconiosis Surveillance Program. Industry participation in pneumoconiosis radiologic consultation program ranked by number X-rays submitted per standard industrial classification, November 1985 through July 1988 (N = 1111).

Census Code	Occupation	Number X-Rays	Percent of Total
777	Miscellaneous Machine Operators	102	(9.3)
783	Welders and Cutters	57	(5.2)
596	Sheetmetal duct installers	54	(4.9)
593	Insulators	50	(4.6)
709	Grinding, abrading, buffing and polishing machine operators	49	(4.5)
	All Other	784	(71.5)

Figure 5. State of Wisconsin, Department of Health and Social Services, Pneumoconiosis Program. Participation by occupations in pneumoconiosis radiologic consultation program ranked by number X-rays submitted per occupation, using 1980 U.S. census occupational classification system, November 1985 through July 1988 (N = 1096).

DISCUSSION

Our surveillance program has demonstrated some potential for disease prevention. We have conducted several follow-back field investigations prompted by groups of identified cases in single work places. In one facility, for example, the identification and confirmation of a single case led to an expanded screening program which found more radiographic abnormalities. This prompted a careful industrial hygiene survey and led to the eventual recognition and control of a previously unknown asbestos exposure in a paper making industry.

Two significant programmatic problems have persisted which must be addressed before the program can become more effective. We have had some difficulty obtaining thorough occupational histories. This problem will require increased effort to educate physicians, employers and employees on the value and importance of occupational histories in disease detection and prevention. These efforts will be strengthened by current Wisconsin regulations requiring such histories for hospital inpatients and cancer patients.

The other problem we have had concerns film quality. (Figure 6) This problem will be more difficult to correct. Of the films reviewed, 4% have been unreadable, while 19.7% have been category three or marginal quality for ILO classification. We have distributed the ILO guidelines for equipment and technology to those submitting large numbers of poor quality films, but we feel that more effort is needed in this area on national or international level.

There has been much attention to the errors or variability which may be introduced into the X-ray interpretation by the persons who read the films. For example, we have, in the U.S., a national program to train, test and certify physicians who interpret X-rays for pneumoconiosis. There have also been numerous studies which have evaluated inter-reader variability issues.[6,7] However, the effect of varying film quality on radiographic interpretation has not received sufficient attention. This is unfortunate, since inaccurate X-ray interpretations may result. Indeed, it is likely that poor quality films may introduce systematic bias into X-ray interpretations. Furthermore, if films must be repeated because of quality problems, unnecessary radiation exposure may result.

Originally intended as a program to provide coal miners with accurate readings of their X-rays through physician training and certification, the B reader program in the United States has expanded to included training and certification for physicians who read films of persons exposed to silica and asbestos. Maintenance of X-ray interpretation quality for coal miners' films is assured both by the certification of the physician readers and by the certification of the X-ray machines. A program to ensure the taking of high quality films for workers exposed to asbestos and silica dusts has not yet been implemented in the United States. We recommend that Congress, through NIOSH, establish a quality assurance program for facilities which provide X-rays for workers exposed to silica and asbestos, either on a voluntary or mandatory basis. The program which currently certifies and evaluates film quality for coal miners' X-rays could serve as a model.

RECOMMENDATIONS

We recommend: (1) the initiation of studies to determine the impact of film quality on X-ray interpretation using the ILO classification; (2) the development and distribution of instructions to radiology technicians as to how to achieve better film quality on X-rays taken for the evaluation of pneumoconioses; and (3) the development of quality assur-

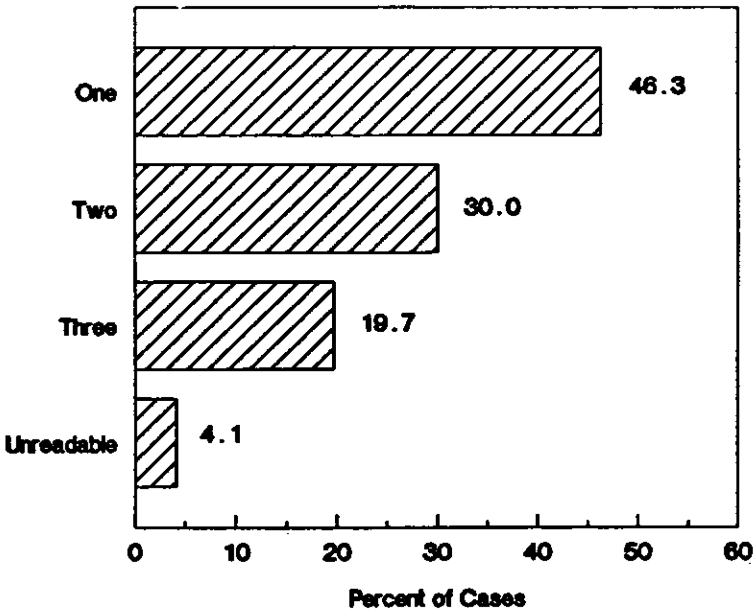

Figure 6. Film quality—total N: 1124 1985–1988.

ance programs for facilities providing X-ray services for the evaluation of workers exposed to silica and asbestos.

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UNIVERSITY PARTNERSHIP FOR WORKSITE MEDICAL PROGRAMS WITH INDUSTRY

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INTRODUCTION

Partnerships in the name of prevention between coal companies and university staff can be mutually beneficial, with worker well-being a positive outcome. Cyprus Minerals, a major American mining corporation based in the Denver, Colorado area, has joined with the Department of Preventive Medicine and Environmental Health of the University of Kentucky College of Medicine in Lexington to operate a preventive occupational medicine program at its coal mining operations in eastern Kentucky. Located in the heart of the Appalachian coal fields, Cyprus is one of the leading coal producing companies in the leading coal producing state. Operating both underground and surface mines, and having washing units, laboratories, and office staff, the approximately 1,000 employees in geographically dispersed facilities have access to, and are kept under medical surveillance by, a medical team from the University. This relationship is now four years old, having begun a year after the establishment of the Department of Preventive Medicine and Environmental Health at the College of Medicine, and continues as an ongoing program.

NEEDS AND SERVICES PROVIDED

There are a wide variety of services provided as part of the occupational medicine program. These begin with pre-employment examinations, which include standard medical evaluations with special attention being paid to pulmonary function results, chest X-ray, and a mandated drug screen. A program for yearly interim exams has been established for all "at-risk" individuals, defined as any person who spends significant or regular time at a mine site. These yearly interim exams are offered to all employees and are conducted on all three shifts at a location at the mine work sites. The focus of these examinations are on pulmonary function, hearing and vision testing, and a review of the general health status of the individual. Given the locations of the mine, washer, and office sites, there is a background of traditional Appalachian health problems.

These background problems include elevated rates of heart disease, diabetes, and other nutritional problems, as well as problems associated with either poverty or lack of education. Nutritional research specifically done on miners has demonstrated poor eating patterns and it does not appear as if working miners, with their adequate income, will pursue an improved diet with the money available to them.

In addition to the yearly interim exams, the company has established a program for periodic examinations of all employees, cycled by birthmonth, the periodicity depending on age, job category, and similar factors. These exams are offered on a voluntary basis and include a thorough medical review, physical examination, and appropriate laboratory tests.

In addition to these services, many of which serve a preventive function, there is care given for injury evaluations, non-occupational illness evaluations, and follow-ups for disabled mine employees, both those on short-term and long-term disability. University personnel assist employees with appropriate referrals for both work-related and non-work-related conditions, and the medical staff participates in special projects such as health education, some of which is done through miner re-training activities, and other medically-related activities, such as local blood drives.

PROBLEMS OF SPECIAL IMPORTANCE

Clearly, in any mining population attention to pneumoconioses is of special importance. As part of the establishment of this program a chest X-ray was obtained on all personnel, with a follow-up scheduled for the near future. The prevalence rate of pneumoconioses was small, and the patterns found on X-ray correlated well with specific occupational histories.

As might be expected, a small number of what appeared to be traditional coal workers' pneumoconiosis (CWP) cases were detected. These generally occurred in older miners, most of whom were still working underground. However, a few cases were noted in previous underground miners who were now working at surface operations. Other X-ray abnormalities were more compatible with silicosis, in workers such as drillers and driller helpers who had had a life-long career working in surface operations and who rarely were exposed to coal dust per se.

As might be expected in any general population, other X-ray abnormalities were detected, such as a high prevalence with what appeared to be old histoplasmosis, a common finding in Kentucky, and evidence of old tuberculous infections, as well as pulmonary abnormalities not directly related to mining activity. Each employee was sent a letter with the findings as noted, sharing with them the fact that their X-ray was either normal or abnormal, and a copy of the official report was sent with the suggestion to review matters with one's personal physician or to come to the medical department for further assistance. A significant number of employees who received letters noting a wide range of abnormalities availed themselves of visiting or calling the medical department. Ongoing attention is given to the matter of dust levels at the workplace. With modern mining techniques and ventillation, the dust levels in underground facilities are kept at low levels. At surface operations there is the use of dust-suppression techniques as well as a program for using respirators, as required.

Another special problem related to workers engaged in mining activity is the matter of noise exposure. Baseline audiograms on all employees and all new hires, show that some level of hearing deficit is a common finding in this population. Complicating matters, but amenable to study, is the fact that many employees have other significant non-occupational noise exposures such as hunting and/or trap shooting, motorcycle use, exposure to loud music, use of chainsaws, and other noisy activities. The majority of newly hired individuals also demonstrate hearing loss of some degree, but as part of the preventive health program for company employees a hearing conservation program has been developed, as well as hearing protection being made available to workers.

As noted above, there are special problems in this group of workers related to the background of disease in this area, factors not related to employment.

PROBLEMS IN ESTABLISHING A PROGRAM

Although there has been an excellent four-year cooperative effort between Cyprus Minerals and the Department of Preventive Medicine and Environmental Health, this program was established only after some education on both sides. From a corporate perspective, there was the need to educate both central and local administrators as to the role of an organized medicine program, especially one utilizing the services of a university based 125 to 150 miles from the actual operations. There was some initial resistance by the existing local medical community to the idea of university-based personnel developing and operating a medical program. Another logistical problem was to staff facilities, attempting to utilize as much as possible, local personnel. Because of the relative small size of the medical communities and the small number of well-trained personnel, this in particular has proven difficult.

SOLUTIONS TO PROBLEMS

Needless to say, most real or potential problems were successfully dealt with, which allowed this program to become established and continue to do well over the years. University personnel met with corporate and local managerial staff and there was a mutual exchange of views. The possibility for potential conflict in operating a program was minimized, since corporate policies, passed on from the corporate medical director in Denver, guide the program. The on-site medical staff always remembers that the best interests of the patient comes first and fortunately, corporate and patient interests most often coincide. It was clear that the university staff had much to learn about the day-to-day operation of coal company facilities, and there was a period of education of the members of the Department so that they could better understand the working realities of coal operations. Much of this was done by sharing information. Initially there were many field trips made to mine facilities, including visiting drag line operations, work in active mining pits, washing facilities, laboratory facilities, and underground mines. One continuing aspect of the program is a series of on-going, on-site visits by medical personnel on a regular basis to all operations. This not only continues to educate and reinforce the experience of the medical staff, but allows on-site interaction between the medical staff and miners at times other than those related directly to medical examinations.

The staffing for the occupational medicine program comes from the Department of Preventive Medicine and Environmental Health, based in Lexington. At the most distant facility at the southeastern corner of the state in Middlesboro, there is the main medical department office where all health records are maintained. A medical assistant on the staff is available daily to interact with patients, management, and others as necessary. Traveling from Lexington each week is an occupational physician assistant who spends one day at the medical office in the Hazard, Kentucky area and then travels on to Middlesboro the second day, performing the necessary hands-on medical evaluations required. A board-certified specialist in occupational medicine regularly goes to both sites and is responsible for all patient management questions and oversight of all patient records. Twenty-four hour communications is maintained to mine management for any routine or special problems that may arise.

Recognizing that there is a need for on-site medical evaluations that may occur on days when the Lexington staff are not present, arrangements have been made in both communities served by this program to have local physicians act as surrogates. These local practitioners serve at the request of the program director in Lexington and communicate with him regarding any substantive matters requiring decision making. By having such backup, no time is lost in processing new hires, evaluating injured workers, or in any other way in taking care of the company's needs.

Another aspect of the program which facilitates corporate/university interactions has been the establishment of a joint occupational health committee, which includes the corporate medical director, the director of the university program, safety personnel, and senior human resources management personnel. These individuals act frequently on an informal basis and come together formally to review the status of the program, discuss on-going or potential future difficulties, and to provide general oversight and direction for the program. It is through this mechanism that arrangements can be made to minimize disruption of production time when yearly interim exams are scheduled, which as noted above are done on all three shifts at the mine sites. For such examinations no more than two hours, and often less, is required to have the individual report for their exam, complete the procedures, and return to their regularly assigned duties.

Another aspect of the program is active involvement and participation with members of the local medical communities. Although based at a University hospital in Lexington, the program director holds courtesy appointments at the two major community hospitals in Hazard and Middledboro, and meets from time to time with individual physicians, medical boards, and hospital administrators. Initial apprehensions that a program such as this would diminish the patient activities of the local practitioners was quickly dispelled as these yearly examinations uncovered many previously unknown conditions, including diabetes, hypertension, heart disease, and other similar problems, which were then referred back to the local practitioners in the community.

ADDITIONAL UNIVERSITY RELATIONSHIPS

In addition to the medical program described above, the interaction between the corporation and the University has been fruitful in other areas. The University has the BRASH (Behavioral Research Aspects of Safety and Health) working group, a research and service organization comprised of individuals from many disciplines. Principle members of this group include educational psychologists, professors in public administration, community health nursing, and behavioral science, as well as the occupational physician who oversees the Cyprus-sponsored medical program. In addition, other associate members of the group include epidemiologists, nutritionists in both home economics and medical anthropology, and many other professional staff from the University. The presence of this group and the excellent working relationship with the coal company have fostered research activities in several areas. The ergonomics group at the University have used miners from Cyprus facilities for simulated mine activities and to better understand oxygen demand and other factors related to mine work. Nutritional work, including detailed surveys by a medical anthropologist, have been carried out by mutual agreement with Cyprus. The offices of the coal company and the activities of the medical program are utilized as a training site for residents in occupational medicine, and these physicians are made welcome at the mine site to learn first-hand about the mining industry. Free access to information has even allowed students in the University's Master of Science in Public Health degree program to utilize corporate medical records for analytical purposes, such as relating hearing loss with particular pieces of equipment and non-occupational causes of hearing loss among the mine employees. As a spinoff from this research, other activities are facilitated and the BRASH group has been especially successful in securing research funds from the United States Bureau of Mines, the NIH, and other similar agencies.

CONCLUSION

The success of this program, focusing as it does on the particular health problems of miners but also providing a wide range of preventively-oriented services, demonstrates that a university medical staff in the field of preventive medicine with its traditional population-oriented approach can successfully cooperate with a centrally-located corporate medical department and local mine management to provide health care services to miners. These mutually agreed to activities make resources available that go beyond the scope of any single practitioner and eliminate many logistical problems for the coal company. This successful university/corporate partnership demonstrates how mutual interactions can be beneficial to both parties.

HEALTH EFFECTS OF TREMOLITE, ACTINOLITE, AND ANTHOPHYLLITE

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ABSTRACT

On June 20, 1986, the Occupational Safety and Health Administration promulgated revised asbestos standards for general industry and construction. The standards regulate occupational exposure to asbestos and to all types of tremolite, actinolite, and anthophyllite, including nonasbestiform varieties.

We have examined the scientific basis for regulating, in the same manner as asbestos, exposure to nonasbestiform varieties of tremolite, actinolite, and anthophyllite. From our review of several hundred articles, we found strong evidence that pneumoconiosis and malignancies in both experimental animals and humans are associated with asbestos forms of these minerals. We found no experimental or epidemiologic evidence to indicate such pathogenic effects from exposure to nonasbestiform varieties of tremolite, actinolite, and anthophyllite.

Although scientific studies of the health effects of exposure to nonasbestiform varieties of tremolite, actinolite, and anthophyllite are limited, nonasbestiform tremolite and actinolite did not cause pulmonary fibrosis or excess tumors in three animal studies. We lack relevant human data to examine the health effects of exposure to nonasbestiform types because the several occupational cohorts reported in the literature were all exposed to a mixture of asbestiform and nonasbestiform minerals.

We conclude that the literature supports the regulation of the asbestiform variety of tremolite, actinolite, and anthophyllite. However, it does not support the regulation of the nonasbestos forms of these minerals in the same manner as asbestos.

No Paper provided.

EFFECTS OF TOXIC GAS INHALATION ON RESPIRATORY SYSTEM IN BHOPAL GAS VICTIMS

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INTRODUCTION

Toxic gas leak at about mid-night from Union Carbide pesticide plant affected majority of the citizens of Bhopal, on 3rd December, 1984. Since it was a comparatively cold and humid night the gas formed almost an aerosol and settled on the adjoining area in the shape of a mushroom; engulfing the population in the affected area and then gradually spreading to the neighboring area after reaching the ground level. It resulted in immediate death of approximately 2500 victims and a large number of those who survived developed irreparable damage to lungs and other systems which has crippled them for their life. The present report summarizes the effects of toxic gas inhalation on respiratory system including clinical, radiological and physiological abnormality, which was studied in depth, soon after the disaster.

MATERIAL AND METHODS

Selected cohort of severely affected individuals, as well as a control population is being followed for studying the long term effects of toxic gas inhalation. 978 patients were admitted in Medical College Hospital,[1] out of which 458 (46.8%) were males and 520 (53.2%) were females. Age and sex distribution of 544 victims admitted on first day is shown in Table I.

OBSERVATIONS

Of 978 victims analysed 733 hailed from areas within 1 km. of Union Carbide Factory, 127 were within 1–2 Km. and rest 117 were from areas situated more than 2 Kms. from factory. Thus all of them were exposed to gas in a sufficient concentration. All the hospitalized patients had respiratory complaints. They described that toxic gas had a curious odour, unfamiliar smell that they had never experienced. Soon after inhalation they developed an extreme degree of irritation in nose and throat, almost resulting in a sense of suffocation. They found it extremely difficult to breathe and some of them died in the same state almost instantaneously.

Main respiratory symptoms noted in a series of 544 patients were breathlessness, cough with scanty expectoration, presence of pink froth, irritation in throat and a choking sensation, pain in chest, haemoptysis and hoarseness of voice. Symptoms are illustrated in Table II.

Most of the victims had tachycardia, pulse rate above 100/mt. and almost all of them were afebrile (except 2%), which is remarkable, as with so much of pulmonary congestion to oedema in most of the cases, they did not develop frank infection, which possibly is due to lethal action of the toxic gas on the microbials. Almost all of them had tachypnoea. Ronchi and crepitations were present in 452 (83.08%) cases, pleural rub was recorded in 7 cases. When compared with radiological picture physical signs appeared to be much less.

Symptoms and physical signs decreased with passage of time but a few victims developed paroxysmal dyspnea of considerable severity. Dyspnea was of considerable severity soon after the episode but with passage of time both the severity and frequency of episode of dyspnea decreased.

At the end of three months and six months follow up respiratory symptoms mainly cough and pain in chest had decreased, clinical score had gone down; but breathlessness persisted in all patients almost with the same severity.

Radiological Abnormalities

There were X-Ray abnormalities in almost all cases. Most of them had diffuse non-homogenous opacities, mostly in mid and lower zones; in some of the severely exposed cases almost whole of the lung fields were opacified with ground-glass like appearance. The opacities in lungs started decreasing at the end of first week and there was considerable radiological clearing by the end of second week.

X-Rays of 500 patients who were symptomatic were taken sequentially to study the changes with the passage of time.

Various types of radiological lesions observed were:

1. Interstitial lesions.
2. Combined interstitial and alveolar lesions.
3. Destructive lesions.
4. Parenchymal opacities:linear, punctate, nodular—micro and macro-nodular, and reticular, alone or in combination.
5. Evidence of pre-existing lung disease was detected in few X-Rays, along with fresh changes due to toxic gas inhalation.

Interstitial lesions:

In 207 victims out of 500 interstitial pulmonary lesion was detected leading to pulmonary oedema of various grades, most of them having involvement of both mid and lower zones and in some whole of the lung was opacified. It appears that inhalation of toxic gas caused exudation of large amount of fluid in the interstitium not capable of being drained by lymphatic channels. This resulted in an increas-

Table I
Showing Age and Sex Distribution of Victims Studied

Age Group	Male	Female
Under 15 years	45	42
Between 15-30 years	100	154
Between 31-45 years	66	60
Between 46-60 years	43	16
Above 60 years	10	8

Table II
Showing Symptoms of the Victims in Early Phase

SYMPTOMS (n=544)	No.of patients	Percentage
Breathlessness	538	98.89
Cough	516	94.84
Presence of pink froth	283	52.00
Irritation in throat/choking	250	45.95
Pain in chest	136	25.00
Expectoration	87	15.99
Haemoptysis	66	12.60
Hoarseness of voice	11	2.00

ing streaky shadows developing and miliary shadows developing in the interstitium of the lung.

Alveolar lesions:

Alveolar pulmonary oedema was seen in 203 out of 500 victims whose serial X-Rays were analysed. 184 cases had bilateral involvement. In 94 cases all the six zones were effected, in 90 there was involvement of lower zones only. 19 victims had unilateral involvement only. Confluent fluffy shadows indicated alveolar lesions. Alveolar lesions could have resulted from higher dose of toxic gas than was inhaled by those patients who has only interstitial lesions, where exudation took place only in interstitial spaces. With increased amount of exudate it poured into alveolar spaces, resulting in alveolar oedema as well.

Destructive lesions:

In 40 victims lesions such as surgical emphysema, pneumomediastinum and pneumothorax were seen, in addition to alveolar lesions. These abnormalities indicate that a higher dose of the toxic gas led to break down of lung tissue resulting in leaking of air into soft tissues and mediastinum. A few cases had pleural effusion obliterating costophrenic sinus, which tended to disappear with passage of time, resulting from inflammation of underlying pulmonary tissue.

Pre-existing lesions:

Evidence of pulmonary tuberculosis and emphysema was found in 36 cases. Evidence of chronic obstructive airway disease was found in many X-Rays, mostly in smokers. All of them had evidence of lesions resulting from inhalation of toxic gas, described above, besides evidence of pre-existing disease. In many cases hyperinflation was observed due to air-trapping resulting from involvement of airways limiting airflow following inhalation of toxic gas.

In another study of radiological changes in 113 victims,[2] X-rays were taken soon after disaster and after three months. 2 victims showed normal picture. Emphysema was seen in 15%, pleural scars in 21%, consolidation in 4%. In lung parenchyma interstitial deposits were seen in almost all cases, 82% had linear and 37% punctate deposits. These deposits tended to clear with the passage of time. 36% had infiltrates in 2 zones, 40% in 3–4 zones and 24% in more than 4 zones. Evaluation of X-Rays taken three months later 38% showed some improvement, while some deterioration was seen in 16%.

Some victims developed episodes of paroxysmal dyspnea following toxic gas inhalation. Radiological studies of these subjects showed evidence of air-trapping indicated by hyperinflation in upper zones with evidence of infiltrates in lower zones mostly on right side, perhaps because right bronchus is direct continuation of trachea. A few of these subjects showed evidence of hypersensitivity pneumonitis and evidence of patchy consolidation in others.

Pulmonary Functions

Pulmonary functions were estimated in the victims to observe the abnormality that the toxic gas inhalation produced, immediately after the episode and are being followed in a longitudinal fashion to find out the long term changes in the victims with a control group.

224 gas exposed victims were studied,[3] soon after the episode. For the purpose of analysis of results they were divided into four groups on the basis of FVC and FEV_1 values, as shown in Table III.

All the victims showed involvement of peripheral (small) airways thus it appears that main brunt of the toxic gas inhalation was borne by the small airways. Almost half of the gas victims showed normal pulmonary functions as far as FVC and FEV_1 values are concerned, rest were almost equally distributed amongst those having obstructive (24), restrictive (38) and combined (36) abnormality.

Blood Gases

Arterial blood gases and pH were studied in the gas victims after the acute episode. PCO-2 values were slightly on the hypocapnoeic side, indicating that these victims were hyperventilating. There was slight hypoxaemia in a few subjects but most of them had a value above 70 m.m., only 7 out of 46 had values of PO-2 below 70 m.m. and only 2 out of these 7 had values below 60 m.m. All of them had normal pH.

COHb was studied in 70 cases, it was raised in 94.3%; in 11.4% it was higher than 6%.[4] Repeat study after 3 months showed that COHb values had returned to within normal limits in almost all cases. MetHb. was estimated in 111 subjects, and in 83% it was raised but declined to almost within normal range after 3 months.

Table III

Showing Distribution of Victims as per Pulmonary Function Abnormality (n=224)

Group	Pulmonary function abnormality	No.of cases
I.Normal	Normal FVC and FEV-1	126
II.Combined	Reduced FVC and FEV-1	36
III.Obstructive	FVC normal FEV-1 reduced	24
IV.Restrictive	FVC reduced FEV-1 normal	38

Oxygen uptake studies revealed that oxygen uptake had improved after 3 months, which was low initially but the improvement was not significant. Values of VO_2 being 1122.6 ± 280.4 initially and 1157.0 ± 402.6 after 3 months.

In an attempt to correlate pulmonary functions with clinical status of the gas victims, it was found that initial FVC and FEV_1 before bronchodilator and FEV_1 after bronchodilator were inversely proportional to symptoms score ($r=0.76$ for FVC and -0.79 and -0.85 in FEV_1 before and after bronchodilator therapy). There was no correlation of pulmonary functions with oxygen uptake at rest; and of pulmonary function and radiological abnormalities.

Flow Volume Loop Studies

There were four types of flow volume loops:

1. Doming: inability to sustain peak flow indicating fixed central obstruction.
2. Hesitation: poor starting flow with fluctuations due to incoordination of inspiration.
3. Saw tooth: incoordination of inspiratory movement indicating variable intra or extra-thoracic central obstruction.
4. Concavity: indicating small airway obstruction.

No correlation was found between these patterns and the initial severity of symptoms. Changes did not seem to improve significantly at the end of 3 months period. Bronchodilator therapy produced an improvement only upto 15% in small percentage of cases but most of them did not respond to bronchodilators, suggesting irreversible airway obstruction.

Bronchoalveolar Lavage:

Bronchoalveolar lavage was carried out in 12 subjects soon after the episode. 4 out 8 victims who were studied in first 4 weeks showed swelling of tracheo-bronchial mucosa and distortion of lumen. There was patchy congestion in 3, ulceration in 2, and suspected lymphoid hyperplastic follicles in 3 cases. B.A.L. showed a rise in total cell count indicating continuing inflammatory status even after 4 weeks of the acute episode. Mean cell count of 344 m./c.mm. (normal upto 150 m./c.mm.). In nine samples neutrophils were raised (more than 3%; total mean 14 ± 25%, range 1 to 93), in two macrophages were raised (above 94%), while in five they were lower than normal. In one case there were eosinophilia (14%). In another case there was 11% lymphocytosis.

Lung Histo-Pathology

Lung biopsies were performed in 3 patients by open biopsy technique and adequate tissue could be obtained.

Histology showed pleural fibrosis with focal mesothelial proliferation thickened inter-alveolar septa, mono-nuclear infiltration in bronchial and peri-bronchial tissues, with patchy evidence of peri-bronchial and peri-vascular fibrosis, with destruction of bronchial wall and epithelium. No desquamation was seen. Muscular arteries and arterioles showed intimal hyaline thickening in one case suggestive of hypersensitivity. In another patient who had a severe exposure to gas besides changes described, the bronchioles were full of inflammatory exudate obliterating the lumen completely with round cell infiltration around, a typical picture of Bronchiolitis Obliterans; which seems to be a feature of toxic gas inhalation.

DISCUSSION

Methyl iso-cyanate is highly irritant to respiratory mucosa and can produce irreversible damage, as has been proved by experimental studies, data which was not available at the time of the episode, which was a main handicap in dealing with the patients; as well as lack of its toxicological data and availability of antidote, presented enormous problem in the management of the patients. Toxicity to human beings was reported in a study from U.K. due to accidental release of TDI in 35 fireman, toxicity of phenyl isocyanate, hexyl isocyanate and hexyl di-isocyanate has also been reported, but there was no documented study on MIC at the time of acute episode. Toxicity and pulmonary irritation described in these studies is very close to observed effects in gas victims in lungs. Exposure to TDI has led to development of hypersensitivity 'isocyanate asthma'. A few patients in our series had paroxysmal dyspnea following gas exposure perhaps due to sensitisation with single large exposure to the gas (R.A.D.S.).

All victims who had a severe exposure to the gas developed pulmonary infiltrates and many of them developed pulmonary oedema, which cleared with the passage of time, leaving evidence of either radiological or functional pulmonary abnormality. All the subjects were symptomatic. Patients who were managed with high dose steroids (Methyl prednisolone 1 gm I.V. repeated after 12–24 hrs., if necessary) showed much better response than those who did not receive it.

Although after the acute episode there was slight improvement in the condition of the victims but most of them continued to be symptomatic, many showed radiological shadows, and had abnormal pulmonary functions, indicating continuing inflammatory process, supported by histopathological finding. A long term follow up of a cohort is being conducted to study the ill-effects of toxic gas inhalation.

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A STUDY OF SPANISH SEPIOLITE WORKERS

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ABSTRACT

Sepiolite is a naturally occurring fibrous clay which has a wide variety of commercial applications. It can occur as long thin lathe-like crystals which have similar dimensions to asbestos fibres. This has prompted concern that both materials may have similar biological properties. Sepiolite has been processed at a site close to Madrid for the past 30 years. We report here a cross-sectional study of this workforce and mortality data for the total work population.

All 218 current workers provided personal, occupational and smoking histories and all had a full-size chest X-ray. Data from previous environmental sampling was available to help derive measures of total exposure. Our study shows significant relationships between age and small opacities on the chest X-ray (as expected) but no relationship between years worked and chest X-ray appearance. There is a relationship between types of occupation and small opacities on the chest X-ray but this was less than the relationship with age.

There appears to be no excess mortality in this population from lung cancer or other disease and no cases of mesothelioma have been reported. This supports the contention that exposure to sepiolite dust does not present a hazard.

No Paper provided.

CHEST RADIOGRAPHIC FINDINGS AMONG TIRE MANUFACTURING WORKERS —INITIAL RESULTS FROM A CROSS-SECTIONAL SURVEY

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ABSTRACT

Tire and rubber manufacturing has been associated with a wide spectrum of occupational hazards. Cancer risk has been suggested by some investigators.

Because of potential for exposure to inorganic dusts in these industrial processes, a cross-sectional chest roentgenologic survey was conducted of 475 individuals with long-term employment in tire manufacturing.

A high prevalence of chest radiographic abnormalities consistent with effects of inorganic dust was found. One hundred thirty one (28%) had parenchymal changes graded as $\geq 1/0$ according to the ILO Classification of Radiographs of Pneumoconioses, 1980. They appeared primarily as small, irregular opacities. Bilateral pleural thickening was present in 142 (30%) of the examined workers, and 72 additional workers had unilateral pleural abnormalities. Sixty four percent of those with abnormal parenchymal findings also had signs of pleural thickening. These findings are consistent with effects of significant exposure to airborne asbestos. Potential sources of such exposure in this trade will be discussed.

No Paper provided.

DOSE-RESPONSE RELATIONSHIPS FOR CAUSE-SPECIFIC MORTALITY AND CANCER MORBIDITY AMONG ASBESTOS-CEMENT WORKERS

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ABSTRACT

Cause-specific mortality (1952–1986) and cancer morbidity (1958–1985) were studied among male, Swedish asbestos-cement workers (N=1,929) employed for at least 3 months 1907–77. Individual dose-estimates were calculated from dust-measurements performed 1956–1977, data on technical changes, and work histories (N=1,503). The median estimated intensity of exposure was 1.2 fibers/ml and the median cumulated dose 2.3 fiber-years/ml. The mineral-fiber content in lung tissue was analyzed in 76 dead workers, and tissue review was performed for respiratory and gastrointestinal cancers.

Chrysotile was the major fiber type in lung tissue, which is in accordance with the asbestos used in the factory (>95% chrysotile). A remarkably increased risk was found for death from pleural mesothelioma (13 cases out of 560 deaths in workers with a latency time of at least 20 years). Three of the mesotheliomas were epithelial, seven mixed, and three fibrous. Material for immunohistochemistry was available for 12 of these, which all showed a staining pattern consistent with mesothelioma. The mesothelioma cases had a much higher crocidolite content in tissue, than the non-cases. There was also an increased risk for cancer in the colon and rectum (SMR=1.6; 95% confidence level; CI=1.1–2.4; histopathological review revealed no peritoneal mesothelioma). The dose-response relationships (duration of employment and cumulated dose) were steeper than usually reported. Totally mortality (SMR=1.2, CI=1.1–1.3), mortality from non-malignant respiratory disease (SMR=1.7, CI=1.3–2.3), and lung cancer mortality (1969–1986; SMR=1.7, CI=1.2–2.4; after histopathological review) were also increased, but with no dose-response relationships.

INTRODUCTION

We have earlier presented the cause-specific mortality and cancer morbidity in a cohort of asbestos-cement workers and a referent cohort.[1] This study includes a further six years of follow-up of the same cohorts, and a more detailed analysis of dose-response relationships. Additionally, the mineral-fiber content in lung tissue among asbestos-cement workers, with and without mesothelioma, has been analyzed.

EXPOSURE

The plant operated 1907–1977, producing asbestos-cement products from mainly chrysotile asbestos (>95%). Before 1966, some crocidolite was also used, and amosite was used for a few years during the 1950's. All asbestos was disintegrated before mixing with the cement. The cement had a low content of crystalline silica.

Dust measurements were performed from 1956, but before 1969 only Impinger or gravimetric counts exist. The individual cumulated dose was estimated in fiber-years/ml (f-y/ml) from data on dust measurements, changes in the production, and dust control.

MATERIAL

Lists were made including all male, Swedish workers who had been employed at least three months (N=1,929). Work histories were available for 1,503 (78%) of these. The median estimated intensity of exposure was 1.2 (range 0–10)f/ml, and the median cumulated dose 2.3 (0–420) f-y/ml. Only 57 persons were assigned a dose of 50 f-y/ml or more.

A referent cohort (N=726) was set up, including industrial workers from the same region, fulfilling the same requirements as the asbestos-cement workers, but without known exposure to asbestos.

METHODS

Follow-Up

Vital status was determined for the two cohorts up to December 31, 1986. Loss in follow-up was 1.5% in the exposed cohort and 0.3% in the referent cohort. Death certificates were obtained and recoded according to the International Classification of Disease (8th revision). Both cohorts were matched with the national (1958–1983) and regional (1958–1985) cancer registries.

The respiratory and gastro-intestinal cancers in the exposed cohort were reviewed. The pleural tumours were also classified as to type of mesothelioma, according to the 1982 WHO Classification, using light microscopy and stained for immunohistochemical analysis.

The mineral-fiber content in lung tissue in 76 of the asbestos-cement workers (7 with and 69 without mesothelioma) was determined by transmission electron microscopy.

Statistics

The mortality (1952–1986) and cancer morbidity (1958–1985) in the two cohorts were compared with the general population in the county, using annual rates (1958–1985), grouped in five-year age intervals. SMRs (standardized mortality/morbidity ratios) and 95% confidence intervals (CI), were calculated. Due to coding problems for the period before 1969, reliable rates for lung cancer mortality are available only since then.

Since the distribution of dose, even within each exposure category was skew, the median was used as a co-variate in the calculation of the dose-response relationships. These were analyzed by testing for trends in SMR.[2] The rate in the referent cohort was included in the analysis as a crude correction for the bias introduced by deriving the expected values from the general population. To evaluate the potential bias introduced by unequal age-distribution in different strata of exposure, an age-standardized SMR (SSMR) was calculated.[3] A minimum induction/latency time of 20 years since onset of employment was employed in all analyses. All tests are two-tailed.

RESULTS

Comparison with the General Population

When compared with the general population in the county, the *exposed cohort displayed* an increased overall (Observed (O)=560, SMR=1.17, CI=1.08–1.27), as well as cause-specific mortality from respiratory cancer (O=46, SMR=1.98, CI=1.46–2.66), non-malignant respiratory disease (O=54, SMR=1.73, CI=1.31–2.27), gastro-intestinal cancer (O=47, SMR=1.46, CI=1.08–1.95), and cancer of the colon and rectum (O=26, SMR=1.64, CI=1.09–2.44). The SMR for stomach cancer was slightly above one (SMR=1.35, CI=0.82–2.16).

The overall *cancer morbidity* was also increased (O=231, SMR=1.29, CI=1.13–1.47), as well as the malignant tumours in the gastrointestinal tract (O=58, SMR=1.39, CI=1.06–1.81), due to an equally raised rate in the upper and lower part. The risk estimate for the total respiratory cancer morbidity was slightly lower than for the corresponding mortality (O=39, SMR=1,55, CI=1.11–2.14), especially when only lung cancer was considered (O=30, SMR=1.39, CI=0.95–2.01)

The referent cohort showed no significant deviation from the general population but tended to have a raised rate of deaths from stomach cancer (O=9, SMR=1.90, CI=0.87–3.61). As to cancer morbidity, only the risk for stomach cancer (O=10, SMR=2.11, CI=1.01–3.88) was statistically significantly increased.

Tissue Review

For nine of the respiratory cancers, the diagnosis of mesothelioma, or pleural tumour, was given on the death certificate. An additional four cases were diagnosed when the histopathology of the respiratory tumours was reviewed. Three were epithelial, seven mixed and three fibrous. Material for immunohistochemical staining was available for all but one of the cases. These were all CEA negative.

When the result of the review of the respiratory cancers was accounted for in the mortality analysis, the rate of lung cancer was still increased (SMR=1.7, CI=1.2–2.4; 1969–1986).

Thirty-two of the totally 44 cancers in the stomach, colon and rectum (73%) were available for histopathological review. The diagnosis was not changed for any of these cancers; no peritoneal mesothelioma was found.

Pulmonary Mineral-Fiber Content

Among the 69 asbestos-cement workers without mesothelioma, chrysotile was the major asbestos type (median count 41 of totally 50×10^6 total asbestos fibers per mg dry weight), and the crocidolite content was low (median 1.8×10^6). For the mesothelioma cases, however, chrysotile was still the major asbestos type (62×10^6), but the total amount of asbestos fiber was higher (189×10^6), and especially so the crocidolite content (54×10^6). The tremolite count also differed between the two groups, but to a much lesser degree.

Dose-Response Relationships

A significant relationship was found between mortality from respiratory cancer and duration of employment (p=0.0016), as well as cumulated dose (Table I). More than half of the respiratory cancer deaths were due to mesothelioma (using the information from the histopathological review) in the two highest exposure strata, corresponding to 5–6% of all deaths. The remaining lung cancers displayed no dose-response relationship. The same applied for the cancer morbidity from these causes.

Mortality from gastro-intestinal cancer displayed no significant relationship with duration of employment, but an association was found with cumulated exposure. This was due to a dose-response relationship for cancer in the colon and rectum, while stomach cancer displayed no such pattern (Table I). The cancer morbidity for the corresponding diagnoses showed no statistically significant dose-response relationships, but a tendency towards such a relationship was found between cumulated dose and cancer in the colon and rectum (p=0.11).

No dose-response relationships were found for non-malignant respiratory disease, nor overall mortality, neither with duration of employment nor cumulated dose.

DISCUSSION

The estimated cumulative exposure in the cohort was low; only about 3% were assigned a dose of 50 f-y/ml or more. This offers a possibility to assess risks directly at rather low doses, but provides low power for detection of moderately increased risks at intermediate to high exposure.

Table I
SMR for Cause-Specific Mortality, Stratified by Cumulative Exposure, Among Asbestos-Cement Workers and Referents

		Asbestos-cement workers Cumulative exposure (fiber-years/ml)					
Cancer site	Referents	≤1	1-4.99	5-14.99	15-39.99	>40	P-value [1]
Respiratory							
Observed	5	7	9	4	12	5 [2]	
SMR	0.67	1.64	1.92	1.32	3.30	2.61	0.028
CI [3]	0.22-1.6	0.66-3.3	0.88-3.6	0.36-3.4	1.7-5.7	0.85-6.1	
SSMR [4]	0.64	1.51	2.01	1.40	3.78	2.61	
(Mesothelioma [5]							
Observed	0	1 (1)	0 (0)	0 (1)	5 (6)	2 (4)	-)
Gastro-intestinal							
Observed	14	8	5	7	9	8	
SMR	1.22	1.30	0.76	1.72	1.76	2.47	0.042
CI	0.67-2.1	0.56-2.6	0.25-1.8	0.69-3.5	0.8-3.3	1.1-4.9	
SSMR	1.25	1.30	0.99	1.67	1.57	2.47	
Colon, rectum							
Observed [6]	4	6 (4)	2 (1)	4 (3)	3 (2)	6 (5)	
SMR	0.70	2.00	0.63	2.00	1.20	3.88	0.009
CI	0.19-1.8	0.74-4.4	0.08-2.3	0.54-5.1	0.25-3.5	1.4-8.5	
SSMR	0.73	1.96	0.90	2.35	1.11	3.88	
(Dose; median)	(0)	(0.5)	(2.0)	(8.3)	(24.7)	(67.0)	
(Person-years)	(7,677)	(4,287)	(4,264)	(2,687)	(2,719)	(1,501)	

1. Test for trend across exposure strata, including referents; two-tailed tests.
2. One mesothelioma mis-classified as ICD 215 not included here,but among the mesotheliomas.
3. 95% confidence intervals.
4. SMR standardized to age distribution in the highest exposure category.
5. Diagnosis stated on death certificate (diagnosis according to tissue review within brackets).
6. Cases verified by review of histopathology within brackets.

An increased mortality and morbidity from respiratory cancer was found, with a dose-response relationship with duration of employment, as well as with cumulated dose. Several deaths were attributed to pleural mesothelioma on the death certificates, a finding which was confirmed and amplified by tissue review. The life-time risks in the highest exposure categories (>20 years of employment or >15f-y/ml) are very high, and compatible only with the experience from cohorts of insulators,[4] factory workers,[5] and one of the cohorts of asbestos-cement workers,[6] but considerably higher than reported from other asbestos-cement plants.[7-9] The analysis of the mineral-fiber content in lung tissue among workers without mesothelioma confirms, that the cohort has mainly been exposed to chrysotile asbestos, since this was the major asbestos type observed in the tissue, in spite of the much higher persistence of amphibole fibers. The comparatively much higher amphibole (especially crocidolite) content among the workers with mesothelioma, is in agreement with former observations that these fibers are especially liable to produce mesothelioma. No dose-response relationships were found for lung cancer, but our data are not incompatible with previous estimates of such relationships, since the present median exposure was low.

An important finding was the increase of cancers in colon and rectum. This has formerly been observed among workers in chrysotile mines,[10] but the dose-response relationship in our study is much steeper. This is not likely to be due to misclassified peritoneal mesotheliomas, since most of the cancers could be verified by tissue review, and all but one in the highest exposure category. We found no evidence for a dose-response relationship for stomach cancer at these levels of exposure.

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EPIDEMIOLOGICAL INVESTIGATIONS OF THE FIBRE CEMENT INDUSTRY IN THE FEDERAL REPUBLIC OF GERMANY (1981-1986)

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INTRODUCTION

The present study is based on an analysis of death causes in 9 plants of the Association of the Fibre Cement Industries in the Federal Republic of Germany. These represent the entire West-German fibre cement industry. It is a continuation of the epidemiological investigations conducted in 1976–1980.[1,2,3] In order to extend the conclusions drawn from the results of the first study phase (1976–1980) it was followed by a second phase (1981–1986) for comparison and crosschecking;[4] in this case the registered death cases were already considered following a minimal exposure period of 5 years as compared to 10 years in the first study. Of the workers exposed for at least 5 years, 325 cases of the death were registered by the plants of the Association of the Fibre Cement Industries between January 1, 1981, and December 31, 1986. A total of 290 cases were recorded. The remaining 35 cases could not be included in the investigation due to the lacking consent of the next of kin until the beginning of the evaluation. Thus a response rate of 89% was obtained. Furthermore, not all criteria of interest could be ascertained leading to different sample sizes for the individual criteria. As there was only one female worker among the deceased, as 4 cases were already reported in 1980 and as the duration of exposure was less than 5 years in 5 cases, only 280 cases could be included in the evaluation.

METHODS

In the present communication it was attempted to analyse the proportional mortality rates (SPMR) with regard to the ICD classification of the observed death causes in order to derive indications concerning the health hazards due to asbestos exposure of the employees in the asbestos industry. In addition this allows a comparison with the results of the study executed in 1976–1980. The SPMR was determined according to the method of Rao and Marsh.[8] The mortality of the total male population (German Federal Office of Statistics) was adjusted according to the age of the test population. For the calculation of the probability that the various diagnoses or diagnostic groups occurred at the observed rate or at more extreme frequencies, it was assumed that the observed instances followed a Poisson distribution.

RESULTS

Of the registered 280 death cases 160 died in hospital and 118 at home, of 2 cases the location was unknown. The dates were taken from the following documents or records: 280 death certificates, 156 medical records (56%), 73 pathological reports (26%) and 81 histological reports (34%); an occupational disease was recorded in only 10 cases (3.6%). The average age at entry into employment of the employees of the first phase was 50 years and of the second phase $x = 42.5$ years (median = 44 years).

The average age at death is $x = 69.5$ with a median of 72 years; the exposure time $x = 18.7$, median = 17 years, and the survival time (time from start of exposure to death) $x = 27.2$, median = 27 years. The employment and survival times of the registered deceased correspond approximately to earlier data on the actual exposure time and also confirm the results of the first phase of our study. With regard to the distribution of the death causes according to the ICD classification the diseases of the cardiovascular system (ICD 460–519) are in the foreground, followed by malignant neoplasms of all organs (ICD 140–239) and the diseases of the respiratory organs (ICD 460–519). Firm distinctions between the diseases of the circulatory and respiratory systems cannot be drawn. This corresponds to expectation and does not differ essentially from the most frequent diseases of the total male population of the FRG and thus confirms the results of the first study period as well.[1,2,3]

Malignant neoplasms of the respiratory system (ICD 160165) were registered in 29 deceased, among them pleural mesotheliomas (ICD 163) in 7 and malignancies of the digestive tract and peritoneum (ICD 150159) in 9 cases. In 21 of the deceased workers asbestosis was diagnosed. This agrees with previous experience concerning the incidence of this disease in the asbestos cement industry during the time without sufficient dust protection measures.

For the evaluation of the incidence of the observed death causes the SPMR was calculated. A clear excess incidence (SPMR 1.7, $p < 0.01$) compared to the general population was noted with regard to respiratory diseases excluding cancer (ICD 460–519). The incidence of bronchitis, bronchial asthma and lung emphysema (ICD 490–493) is even higher (SPMR 2.1, $p < 0.001$). This is typical of industries with high dust concentrations (Table I). Malignancies of the respiratory organs (ICD 160–165) are more frequent in the exposed group than in the general male population (SPMR 1.36, $p = 0.09$). Less frequent are the total malignant neoplasms (ICD 140–239) in asbestos workers; this lesser incidence leads to a SPMR of 0.77 and $p = 0.05$. Similarly there is a clearly less frequent incidence of cancer of the

Table I

Analysis of the Proportional Mortality (n=262) 1981-1986

ICD-Classification	Observed*	Expected	SPMR	(chi)²	p
390-459 Diseases of the cardiovascular system	103	125.2	0.82	3.9	0.048
140-239 Total malignant neoplasms	55	71.5	0.77	3.8	0.050
160-165 Malignant neoplasms of the respiratory organs	29	21.3	1.36	2.8	0.095
160-162, 164-165 Malignant neoplasms of the respiratory organs without mesotheliomas	22				
163 Pleural mesotheliomas	7				
150-159 Malignant neoplasms of the digestive tract and peritoneum	9	23.1	0.39	8.6	0.004
150-157, 159 Malignant neoplasms of the digestive tract	9				
158 Peritoneal mesotheliomas	-				
460-519 Diseases of the respiratory organs without malignant neoplasms	33	18.7	1.7	10.9	<0.001
490-493 Bronchitis, emphysema, asthma bronchiale	25	11.9	2.1	14.4	<0.001
501 Asbestosis	5				
800-999 Accidents	4				
303, 570-577 Alcoholism, hepatic cirrhosis	12	46.6	1.52	12.7	<0.001
Other death causes	55				

The calculation of the expected values is based on the distribution of age at death in 1981 - 1986. * 18 cases are excluded due to lacking data concerning age at death or year of death; p is stated in a two-tailed manner.

digestive organs and the peritoneum (ICD 150–159; SPMR 0.39, p = 0.48). The same behaviour can be observed in diseases of the circulatory system (ICD 390–459) with a SPMR of 0.82, p = 0.48.

DISCUSSION

The average age at death (72 years) of the deceased employees in our study does not essentially differ from the average age at death of the general population of the FRG. The mean age at entry into employment of the group of the first study period was 50 years, 50% of these had an age of 40–55 years. It was 42.5 years in the employees of the second phase of the study with a 50% interquantile of 36–50 years.[4] Why the mean age at entry of the employees in the asbestos cement industry is relatively high, especially in the first phase, could not be elucidated. There was a critical discussion that this elevated age at entry impairs the significance of the results of the first phase in the sense that the tumour expectancy would be significantly greater with an earlier age at entry. However, the age correlated expected value has been used for the calculation of the SPMR. In addition, this argument can be countered with results of other authors. In the study by Neuberger et al.[7] the age at the beginning of employment was higher in asbestos-associated cancer death cases than in the not asbestos-associated death cases. In their evaluation of the results of 11 international epidemiological studies of lung cancer mortality in employees of the asbestos cement industry, Gardner and Powell[5] stated latency periods from first exposure to tumour manifestation of at least 5 and maximally 20 years. This is in accordance with the concept of the present study where a minimal exposure time of 5 years has been used as criteria of inclusion and in which an exposure time of 20 years could be observed.

The present investigations of the first as well as the second study phase confirm essentially the results of Lacquet et al.[6] and of Weill et al.[11] Thomas et al.[10] examined a cohort of comparable size of deceased employees of the British asbestos cement industry and obtained results largely in accordance with those of the present study, particularly with regard to the expected and observed cases of all malignant neoplasms and of cancer of the respiratory organs and the digestive system. The studies by Ohlson and Hogstedt[8] and Gardner and Powell[5] are also in agreement.

Neuberger et al.[7] conducted a recent investigation of ca. 2800 employees of the Austrian asbestos cement industry using the general population as controls just as in the present study. Based on official death certificates death from lung cancer was diagnosed in 535 cases exceeding the mortality of the age and sex adjusted general population by 1.7. This excess mortality was explained by the authors as due to the higher tobacco consumption of the employees as compared to the general population. In the same study 5 cases of mesothelioma were demonstrated and attributed to the earlier use of crocidolite in the production of asbestos cement pipes. The findings of the present study also point to a possible correlation between mixed dust exposure with crocidolite and chrysotile and the incidence of 7 pleural mesotheliomas (p = 0.08). In the review by Gardner and Powell[5] cited above this is also stressed with regard to the SPMR of lung cancer which lies in the range of 5.2–0.8 in plants with previous mixed dust exposure (chrysotile and crocidolite) and 1.5–0.9 in those with pure chrysotile exposure, while the SPMR of the present investigation is 1.4. Of the three mesotheliomas listed in the review two are attributed to mixed exposure with chrysotile and crocidolite and one to exposure with chrysotile and amosite.

According to these results and those of the authors cited above a distinction should be made between crocidolite and chrysotile asbestos with respect to their carcinogenic effects. Thus inhalable crocidolite fibres possess a stronger oncogenic potential than the corresponding chrysotile fibres. The higher durability of crocidolite in the reacting tissue may explain its pathogenetic importance for the development of pleural or peritoneal mesothelioma. Under present-day working conditions and with the considerably restricted use of crocidolite only for exceptional technical products (pipes) a reduction of the increased mesothelioma hazard appears possible. With the sole exposure to chrysotile the relative risk of mesothelioma is expected to be low (see also Neuberger et al.)[7]

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LUNG CANCER AND NNRD MORTALITY SIMILARITIES OF VERMONT AND NEW YORK STATE TALC WORKERS

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ABSTRACT

The 741 employees of a New York talc plant observed from 1947 to 1978 had statistically significant excesses of lung cancer mortality among short-term workers and of non-infectious, non-malignant respiratory disease (NNRD) among workers with at least one year of employment. Occupational exposures to elongated particulates were greater in the mill than in the mine. However, the excess lung cancer risk was limited to the miners. Some analysts have asserted that there was asbestos in the talc at this New York plant, in contrast to the talc at the Vermont talc plants. However, the only difference in pulmonary mortality observed between the New York and the Vermont talc workers was a greater risk of NNRD among the Vermont talc millers than among the New York state talc millers. Miners but not millers showed a statistically significantly increased risk of lung cancer in both the Vermont and the New York plants. Millers but not miners showed a statistically significantly increased risk of NNRD only in the Vermont plants but not in the New York plant. Thus, epidemiologic assessment of the mortality of the NY talc workers does not support the hypothesis that they have been exposed to asbestos.

No Paper provided.

EPIDEMIOLOGIC STUDIES OF MINING POPULATIONS EXPOSED TO NONASBESTIFORM AMPHIBOLES

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INTRODUCTION

Current standards for the control of asbestos exposures have resulted from proven and serious health effects caused by commercial asbestos, principally chrysotile, amosite and crocidolite. There is more limited information from asbestiform tremolite, actinolite, and anthophyllite. The latter amphibole minerals are also commonly found in nonasbestiform habits. It has become a matter of considerable scientific, social, and economic importance to define precisely which elongated and asbestos-like particles are hazardous to health. Of particular interest is to determine whether or not there is any need to regulate exposures to acicular particles, such as cleavage fragments of nonasbestiform tremolite, actinolite, and anthophyllite as well as the nonasbestiform varieties of other amphiboles. Particles of these minerals found in the environment may meet the currently accepted 3:1 length-to-width (aspect) ratio used to define fibers, but not be asbestiform. The mining populations to be discussed in this report supply information on the health effects of such exposures.

Although not addressed in detail, important related questions pertinent to the biologic effects of elongated particles are: (1) What should be the minimal particle length subject to control, i.e. should fibers shorter than 5 micrometers be included? (2) Since there is considerable doubt as to whether particles with aspect-ratios below 1:10 or 20:1 are hazardous, is it proper to use an aspect-ratio of 3:1? These important questions are discussed in detail in mineralogic references cited in the bibliography.[5,6,17,24,33,38,39,41,42,43,44,45,46]

MINING POPULATIONS THAT WERE STUDIED

Four areas were identified where there have been health studies of mining populations known to have been exposed to nonasbestiform amphiboles. In each there had been concern about the potential for asbestos-like effects. These were (1) taconite mines in the eastern Mesabi Range in Minnesota; (2) the Homestake gold mine in western South Dakota; (3) the Sydvaranger iron mine in northern Norway; (4) iron mines in southwestern Labrador.

MINNESOTA TACONITE OPERATIONS

Minnesota taconite mines came under active study after the discovery in 1972 of asbestos-like particles in the water supply of Duluth.[23,27] These were attributed to the discharge into Lake Superior of tailings from the Reserve mining operation located at the eastern end of the Mesabi Range. The mining of taconite as a major source of iron had begun in this area in the early 1950's with pilot operations starting about 1947. The consensus of mineralogic and environmental studies[2,3,15,32,35,36,39,40,45] is that (a) ore bodies in the eastern portion of the range contain amphiboles, principally cummingtonite, grunerite, actinolite and hornblende; (b) there are many cleavage fragments that meet the regulatory definition of fibers by having 3:1 aspect-ratios; and (c) asbestiform particles are rare, although a small amount of asbestiform ferroactinolite was found in one area.[9,10] Dust exposures in some locations were sufficient to cause concern as to possible silicosis. Langer in 1981[22] summarized the major issue in the Minnesota taconite mines as being "the biological activity of acicular (needle-like) cleavage fragments of grunerite..."

Clinical studies of Reserve miners and millers by Clark et al.[8] and Higgins et al.[18] showed no evidence of asbestos-like effects, but there was radiographic evidence of possible early silicosis. An analysis of mortality in 5,751 Reserve workers who had been employed for one or more years in the period 1952–1976 showed 15 lung cancer deaths with 17.5 expected for an SMR of 84.[19] During the observation period 15 or more years after hire, the SMR was 102, based on 8 deaths from lung cancer.

Cooper et al.[11] analyzed mortality in 3,444 taconite miners and millers employed by the Erie Mining Company or by U.S. Steel for 3 months or more between 1947 and 1958. There were 801 deaths, with 41 due to respiratory tract cancer, 61% of the number expected using U.S. death rates or 85% of expected using Minnesota rates. There were small but not statistically significant excesses in deaths from kidney and lymphatic cancers. There was one death from pleural mesothelioma, which was not attributable to mine exposures, since it occurred only 11 years after hire and there had been probable pre-employment asbestos exposures.

One can conclude that there is no evidence of asbestos-related disease associated with employment in the Reserve, Erie and U.S. Steel iron mines in Minnesota, where there have been opportunities for exposure to nonasbestiform amphiboles in the taconite ore deposits.

HOMESTAKE GOLD MINE

In 1974 it was recognized that the ore body of the Homestake gold mine in western South Dakota contained cummingtonite-grunerite similar to that which was of concern in Minnesota. Since Homestake mining operations had begun in 1876 and past dust exposures had been relatively high, it was realized that epidemiologic studies could yield valuable information.

There have been a number of mineralogic and environmental studies.[1,4,12,29,30,39] There is general agreement that cummingtonite-grunerite, tremolite-actinolite and hornblende are present, that acicular fragments are common, and that there are few if any asbestiform particles. Current exposures to acicular fragments corresponding to 3 f/ml were reported, with estimates of time-weighted average exposures to such particles ranging from 0.25 to 1.72 per ml.[47]

There have been three published epidemiologic studies of Homestake miners. The first, by Gillam et al. in 1976[14] reported 10 deaths from respiratory tract cancer with 2.7 expected as well as 8 deaths from non-malignant respiratory disease with 3.2 expected. The authors attributed the latter to asbestos. This study was seriously flawed, and later analyses of mortality in larger cohorts have not confirmed the authors' conclusions. McDonald et al.[28] in 1,321 Homestake workers employed for 21 years or more found no excess lung cancer deaths (17 with 16.5 expected) but there were 37 deaths from pneumoconiosis and 39 deaths from tuberculosis (SMR = 1,038, or over 10 times the number expected). A single mesothelioma death was observed, in a surface worker who during a relevant time period (22 to 26 years before death) had worked in machine maintenance with probable exposures to asbestos. In a more recent study sponsored by NIOSH, Brown et al.[4]) similarly found no excess lung cancer deaths in a population of 3,328 underground Homestake miners (43 with 42.9 expected). There were 53 deaths from nonmalignant respiratory disease observed with 19 expected, due to a large number of deaths from silicosis and silicotuberculosis.

In describing the exposures of workers in their study, Brown et al.[4] stated that cummingtonite-grunerite, silica, arsenopyrite, and radon were possible hazards. Their results were consistent with the conclusion that silica was of major importance. With respect to amphibole exposures, they stated that the mean time-weighted-average exposures of all miners to C-G particles longer than 5 micrometers was 0.44 such particles per cubic centimeter, based on a 1977 survey. Early exposures had probably been greater.

One can conclude that despite dust exposures sufficient to cause severe and often fatal silicosis, with concurrent exposures to nonasbestiform amphiboles, there was no evidence of excess deaths attributable to asbestos, i.e., lung cancer, mesothelioma, or asbestosis.

SYDVARANGER IRON MINE

Iron mining began near Kirkenes in the northernmost county of Norway in 1907. The ore body resembles that in Minnesota, containing cummingtonite-grunerite, actinolite, and hornblende. These amphiboles occur in elongated fragments, many of which are over 5 μm in length with aspect-ratios as high as 11:1.[16] Amphibole bodies have been found in the lungs of deceased miners by Gylseth et al.[16] There are no reports to indicate that any of the elongated particles are asbestiform, nor is there any evidence to date to indicate asbestos-related disease. Gylseth et al.[16] stated that an analysis of deaths during the period 1949 through 1963 showed no excess lung cancers. Saugstad in 1980[34] studied deaths from lung cancer in Finnmark County where the mine is located. While the county had a higher lung cancer incidence than Norway as a whole, this did not appear to be related to working in or living near the iron mine. Data from the Norwegian Cancer Registry have not shown any excess mesothelioma deaths in the area.

LABRADOR IRON MINES

During the early 1970's it was discovered that iron ore deposits in the Wabush Range in southwestern Labrador contained amphibole minerals, including cummingtonite-grunerite. The two iron mines in the area, one operated by the Iron Ore Company of Canada (IOCC) and the other by the Erie Mining Company, had begun operations in 1962 and 1965 respectively. They had been alerted to dust hazards because radiographic changes consistent with pneumoconiosis had been found during surveillance programs required because of potential quartz exposures. Because of concern that there might be asbestos-related disease, a major study was started in 1979. Financed by industry, it was coordinated and supervised by the provincial government and a distinguished scientific committee selected by industry, government, and labor. The actual investigation, the Labrador West Study, was carried out by the Labrador Institute of Northern Studies, based in the University of Newfoundland.

The results of the Labrador West Study were made available in 1982.[21] They confirmed the presence of cummingtonite-grunerite and other amphiboles, but the concentrations of fibrous particles (i.e. those with aspect-ratios 3:1 or more) were relatively low and 98.5% were shorter than 5 micrometers. Very few were considered to be asbestiform. Lee and Fisher[25] and Lee et al.[26] reported more detailed mineralogic findings in the Labrador mine which were in essential agreement.

Medical studies[7,13,21] have confirmed the presence of a mixed-dust pneumoconiosis, presumably due to combined exposures to iron oxides and silica. Although the original detailed report[21] contained a section which suggested that there was evidence of pleural thickening in some of the chest films, this was not mentioned in the published report of the the radiographic findings.[7] Review of films reported as positive for pneumoconiosis by Cooper and Sargent (personal observation) showed no changes suggestive of asbestos effects.

The Labrador populations have not been the subject of a cohort mortality analysis. They are reaching a time period, 23 to 26 years after the start of operations, where meaningful results might be obtained.

SUMMARY AND CONCLUSIONS

In four mining areas where exposures to elongated but nonasbestiform amphibole particles have been confirmed, there has been no evidence of asbestos-like effects. The negative evidence is strongest in the Homestake gold mine, because of the proven high dust exposures in the past as shown by the high silicosis incidence, and the opportunity for observation after long latency. The evidence is convincing in the Minnesota taconite miners. Although the exposures were lower, the populations are large and there has been ample time for latent disease to appear. The information from the Sydvaranger iron mine and the Labrador mines is also reassuring, but it would be of value to have historical-prospective cohort studies of mortality to augment the existing negative evidence.

Cumulatively, these studies do not indicate that there is any reason to control nonasbestiform amphiboles in a manner comparable to that required for commercial asbestos.

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