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HISTORICAL PERSPECTIVES IN OCCUPATIONAL MEDICINE

Wittenoom, Western Australia: A Modern Industrial Disaster

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GEOGRAPHY

Wittenoom is remotely located in Western Australia's north west, approximately 1,600 Km by road from Perth, approximately 320 Km from the coast, and 720 Km from the railhead at Meekatharra.

The rocks exposed in Wittenoom Gorge consist predominantly of quartzites interbedded with siliceous or cherty ironstone, the deposits of crocidolite being associated with the ironstone. The bands of crocidolite occur approximately 25 feet apart, with the lower vein of crocidolite averaging 2 1/2 inches in thickness and the upper crocidolite vein occurring some 200 feet above the base of the cliffs.

DEPOSITS OF BLUE ASBESTOS

A considerable amount of fiber was available in the cliff faces and could be obtained without much difficulty at outcrops [Mines Department of W.A., 1938]. Initially it was considered doubtful that the narrow width of the veins would permit the deposits to be worked economically by underground methods.

In 1938, the State Mining Engineer reported [Mines Department of W.A., 1939] that the crocidolite deposits had been worked by "hand dressing" only along the main ribbon of material containing long fibers in the lower horizon. The ironstone

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amount of chrysotile and amosite imported from Canada and South Africa (Xu et al., 1985).

CROCIDOLITE PRODUCTION

The Production of Asbestos

In 1937, 14 tons of crocidolite were produced from the Wittenoom area with a total value of £816. In the following year, 55 tons valued at £3,170 were produced; but in 1939 the total fell to only 24 tons, valued at £1,900, and in the following two years just over one ton was produced. Production rapidly increased from 1943, when Australia Blue Asbestos Ltd. took over all the leases and confined their operations to Wittenoom Gorge. By 1952, 3,000 tons of crocidolite per annum were being produced and by 1957 this had increased to 11,000 tons per annum [Australia Blue Asbestos Limited, 1958]. This level of production was sustained until the industry closed in 1966.

Mining Practice

The initial mine and mill operated by Australian Blue Asbestos Ltd. [1948] were situated in Wittenoom Gorge. In 1957, a new mine and mill were opened in Colonial Gorge [ABP, 1958]. Initially, most of the ore was won from the upper seam, in order to allow this to advance far enough ahead of the lower seam for safe extraction of the latter. The ore was delivered into a bin and then crushed, first in a coarse crusher and next in a fine crusher, after which it passed to an oil fired rotary drier. The fiber was cleaned and packed into jute sacks in 100 lb. loads.

The whole 23 years of mining and milling was a process of trial and error. The mining technique was fixed after five or so years of experimentation by British and American managers. Milling of the ore never reached this point of stability. It was a simple dry mechanical process of crushing, grinding, and aspirating, but problems persisted in dust control, ventilation, adequate fiber purity, the extremely abrasive host rock which made maintenance a huge task, and in the company's mining inexperience. The old mill, which operated from 1943 to 1957, was poorly organized, while the new mill, which took over in 1958, was better planned, with a better system of dust control. However, dust control and fiber purity remained problems throughout the life of the works. In the mid-1950s, the company experimented with a wet extraction method, but nothing came of it.

Dust Collection and Measurement

In the new mill, roloclone dust collecting units were used for dust collection [Australia Blue Asbestos Limited, 1948; Major, 1968]. The dust was saturated with water in the rotoclones and the sludge discharged to a pump which delivered it to a thickening tank. The heavy sludge settled out and was pumped to waste, while the clear water overflowed to a tank, from which it was circulated back through the plant. The plant was designed on the company's own experience. Canadian and South African plants were examined, but the condition of the ore in the former and the labor conditions in the latter were so different that no direct application of this knowledge was possible. The most efficient types of machinery, based on performance in the old plant in Wittenoom Gorge, were installed in the new plant in Colonial Gorge.

Only one adequate survey of dustiness was performed at Wittenoom. This was

undertaken in 1966, in the final year of operation of the plant by an industrial hygienist [Major, 1968]. Fiber levels higher than 100 f/ml were measured in many parts of the plant, and subsequently permitted estimates of individual exposure of the workforce to be made [Armstrong et al., 1988]. More recently, some of the original dust filters used by Major in his 1966 survey have been re-examined [Rogers, 1990], and the measurements were said to have underestimated the degree of exposure by "orders of magnitude." Prior to these dust measurements, crude konimeter readings had been carried out by the industry and the Mines Department of Western Australia. These readings usually exceeded measurement limits of 1,000 particles per cc. In 1951, Western Australia having adopted a safe dust limit of 176 particles per cc, the company was warned repeatedly and the Commissioner of Public Health wrote to the Under Secretary for Mines that the "hazard from asbestos is considerably greater than that from silica . . . we have reason to believe that attention to this aspect of mining operations at Wittenoom has been inadequate in the past." In 1955, the Mines Inspector threatened to close the mine. However, the works were not closed until the company itself took the decision in December 1966.

Economics of the Industry

The Government reserved all ground around the ABA leases from selection for crocidolite mining by others and provided shipping subsidies to the company, but the asbestos industry was always economically marginal because of its inability to compete with large scale producers in South Africa and Canada. The company made a profit for only a few years between 1955 and 1960, when it had a five-year export contract with Johns Manville in the U.S.A. By the last year of its operation, it had accumulated losses of $2.5 million. Labor shortages, especially of skilled Australian miners and tradesmen, were continuous especially in the 1960s, when the nearby iron ore industry attracted many workers by offering better working conditions. Capital costs also rose, but market prices did not rise as anticipated.

TOWNSHIP OF WITTENOOM

The original settlement for the mine and mill in Wittenoom Gorge was in the gorge itself, about one mile from the mine. As the workforce grew, the township of Wittenoom was laid out and built at the entrance to Wittenoom Gorge, about 12 km. north of the mine. In 1961, the population of the division, which included Wittenoom as the town where the majority of people resided, consisted of 671 males and 309 females. At the time of closure of the mine in 1966, there were 1,468 males and 421 females [Bureau of Census Statistics, 1971]. The township in 1958 contained 151 houses, a general store, cafe, library, cinema, billiard room, hotel, tennis courts, race course, cricket and football grounds, post office, police station, hospital, State school, Catholic school, and kindergarten. A weekly baby clinic operated and a doctor resided in the town. Single men were accommodated in two-roomed huts, four men to each hut, and a central cafeteria was provided for their use. Later, dormitory accommodation was also provided. Housing, school, hospital, police station, water supply, and post office were provided by the Government, and all other buildings and amenities were company projects. There was a Roman Catholic church in the township, and Church of England and Methodist clergy visited once a month. The township enjoyed a regular air service by the MacRobertson Miller Airline Company each

TABLE I. Duration of Employment of Australia Blue Asbestos Company, 1943–1966

Period of employment (months)	% of workforce
< 3	45
3–6	15
6–9	12
9–12	5
> 12	22

Tuesday, Thursday, and Saturday, and by a Dove Station Service aircraft each Wednesday, Thursday, and Friday. Layman [1983] has portrayed the town as stereotypical of the usual mining town in only one respect: its isolation. Its community institutions were few and fragile, its trade unionism characterized by apathy and inactivity, evidence of class conflict was infrequent. Virtually no challenge existed to company dominance in a company town. The workforce was not militant and did not display much solidarity or overt class struggle. It was transient; less than one-quarter stayed for longer than one year (Table I). Many of the workers were refugees from a depressed postwar Europe and were there only to earn sufficient money to get a start elsewhere. As a result, all organized activities, such as trade unionism, political activism, and community organization, were weak and sporadic. Despite the poor working conditions, no major strikes occurred. Collective activism to remedy grievances was rare; it was not until 1965 that men living in the single men's quarters petitioned the Health Department to remedy their sub-standard living conditions. The one community organization which did survive successfully was the Catholic church and convent school.

The Government of Western Australia assisted in setting up the industry by providing the houses, as well as transport subsidies. It encouraged people to go to Wittenoom by projecting the town as modern and a model for future development in remote areas. Wittenoom was presented as the largest inland town in the north of State, with a stable and law-abiding community and a long and prosperous future.

Tailings from the mill, rich in residual crocidolite, were found to be ideal for use about the township for paving the roads, parking areas, and the like, and for reducing the dust and mud in the back-yards of the township's houses. From the middle 1960s, the Health Department of Western Australia tried to stop the company from using these tailings and even proposed to introduce a regulation under the Health Act banning their use. The Minister for Mines preferred a course of obtaining the company's co-operation, but the use of tailings was not stopped until the mid-1960s, the only exception being a permitted use when incorporated in concrete and in the construction of a police station. There was a major mishap when, in the late 1960s or early 1970s, the then Government Department of Civil Aviation, without consulting anybody, used massive amounts of tailings on the airport tarmac. A Public Health Department Inspector noted in 1973: "Streets are sealed, but in all except one or two cases have been made with mine tailings. Evidence of unsealed tailings may be noted on the sides of the roads. In the early days, it is obvious that people used tailings to make driveways for their houses and some even used them to cover garden areas; large areas covered by the tailings include the front of the Wittenoom Club, the car park for the Club, the convent playground, the vehicle parking site near the State

school, and the tennis courts. The race course is completely covered by tailings and it is interesting how the fibres have become pulverized and separated with rain to form concentrations in the hollows made by hoof marks."

THE WORKFORCE

Between 1943 and 1966, some 6,500 males and 500 females worked for Australian Blue Asbestos Ltd. at Wittenoom. The maximum workforce at any one time in the 1960s was about 500. The mine was run mostly on contract rates, rather than wages. Despite this, the company was unable to recruit skilled miners in Western Australia because Australian miners were not prepared to work continuously in 42 inch stopes. As a consequence, the company turned to overseas recruitment of miners. It recruited largely young and unskilled migrant labor, mostly from Italy, and brought them to Wittenoom on two-year contracts. Others were recruited directly off migrant ships in Fremantle (the port of Perth) on six-month contracts. These men had no tradition of mine work or any idea of the conditions they faced. Many were dismayed when they saw the situation, but they were relatively powerless; they had no money, no recognized skills, no common tradition with which to defend themselves. Of the local Australians who worked at Wittenoom, many were itinerant and quickly moved on. Work in the mill was also deemed unskilled. It was heavy, noisy, dirty, and hot, and few worked there for long. Many left for home as soon as their contracts had been fulfilled and some left sooner. Those who were forced to stay in Wittenoom moved to the mine at the first opportunity. Most of the mill workers had heavy laboring jobs at different points in the milling process where they provided a back-up for an inadequate mechanical process. At the end of the milling process, the baggers were doing the job widely regarded as the most intolerable in the mill. Workers responded to these oppressive working conditions by leaving the job. Turnover in the mill was continuous. What was found to be most intolerable was the amount of dust.

Dr. I.C. McNulty became the Mines Medical Officer in 1957 and has described his experience visiting Wittenoom personally: "On arrival I was met by a company official at the airport and taken on a tour of the mine and mill. I felt at the time that it was the dirtiest mine and mill I had ever seen, although I had no occupational health training. But by that time I had visited many surface and underground workings in the eastern goldfields...There were clumps of asbestos all over the floor and surfaces of the mill and one's clothing was rapidly soiled by contact with any surface. Sunbeams showed heavy dust particles and visibility was reduced in some areas. After crushing, at the exit of the underground mine high up on the face of the gorge, the ore on a moving belt entered the top story of the mill. The fiber was heavily admixed with rock and "pickers" stooped over the belt, manually removing the larger pieces of rock. This was a very dusty area. As the belt moved down through the various levels of the mill, smaller rocks were removed, fiber was freed, and then discharged from a hopper and bagged. Every operation in the mill was associated with dust, even the housekeeping. Sweeping up the floors was done dry and raised a great deal of dust. The men at the bagging plant simply stood over open bags, which were filled from a chute. The floor was heavily contaminated with clumps of asbestos and you could see it in the men's clothing, hair, everywhere. At some stage, either this year or subsequently when I was present, the baggers wore respirators, but these were of a very

simple, half-face cartridge type. During my visit, the maximum temperature rarely dropped below 100°F and it was obviously impossible to do reasonably heavy work 8–10 hours a day wearing such a respirator. They were obviously ill-fitting and ineffective anyway. The dust was extracted from the mill and simply poured out above roof level to flow back to the mill and staff offices. A mines inspector reported dust levels in the air measured on the lawn outside the staff offices would be unacceptable in an underground mine. The dust emerged at the same level as the main entrance to the underground workings, and dust in the air entering had been measured at between 200 and 300 particles per cubic centimeter, a level which was above what would be accepted in the gold mining industry. The underground workings were also fairly poorly ventilated and humid. The rock was particularly hard, had a high free silica content, water was not very evident, and there was obviously a serious silicosis risk. Apart from a general shortage of water, management had difficulty with damp fiber and, in fact, limited the use of water and this contributed to the general dustiness of the mine and mill. Outside the mill, the waste material, the tailings, and the material collected in the dust extractor system were simply dumped from hoppers onto the back of trucks in a cloud of dust and the trucks then driven up to the gorge over roadways laid with tailings to dump the ore. Vehicle movement over the roads raised a great deal of dust. Airline pilots claimed they homed in on Wittenoom from considerable distances by identifying the blue haze on the horizon."

Exposure Standards and Regulations

In the United Kingdom, official action to control exposure to asbestos was initiated in 1930, based on a 1928–1929 study of Merewether and Price (Merewether, 1930), which showed that asbestosis was an industrial disease and concluded that there was no substitute for dust control at source. The Asbestos Regulations (1931) were introduced and a scheme for compensation of workers developing asbestosis was instituted.

The first recommendations for asbestos exposure control in the United States were made in 1938 (Dreessen, 1938), following a study of 543 employees in four asbestos textile plants. The recommended air concentration of 5 mppcf, as determined by the impinger technique and counted by light microscopy, was established. In 1946, the American Conference of Governmental Industrial Hygienists adopted 5 mppcf as a threshold limit value. In New South Wales, the "Dreessen Standard" from the U.S. had been adopted by the Health Department in the measurement of asbestos in industry by 1938. The same standard was adopted in regulations in Victoria in 1945. In 1956, the asbestos industry was declared a dangerous trade under the Health Act in Victoria. Hence, in the responsible instrumentalities in Western Australia, knowledge of the harmful effects of asbestos and the need to control exposure existed during the time that the Wittenoom industry was operating.

The National Health and Medical Research Council in Australia recommended an exposure level of 5 mppcf (30 f/ml) for all asbestos in 1964, reduced this to 4 mppcf (12 f/ml) in 1969, 4 f/ml in 1973, and 2 f/ml for chrysotile and amosite and 0.1 f/ml for crocidolite in 1974. However, Western Australia did not legislate on asbestos until the Factories and Shops Act in 1978. Responsibility for health and safety in the mining industry was the statutory responsibility of the Mines Department, and it was jealous of the interference by the Public Health Department. In relation to the industry at Wittenoom, therefore, no legislative restrictions were ever imposed. None of the

published descriptions or information available during the 1950s was considered very relevant to what was taking place at Wittenoom. The literature discussed asbestosis as a disease which occurred after very lengthy exposure and not the very rapid onset and progression that occurred after the very short but intense exposures at Wittenoom.

THE INCIDENCE OF DISEASE

The youth of the workforce (57% were less than 30 years of age when first employed and only 16% were aged 40 or more) has had an important effect on the subsequent incidence of asbestos-related diseases, as many have survived to experience the onset of diseases with a long latent period. In 1946, the Wittenoom Mine Manager wrote to his head office describing the first known asbestosis case—a man called Dignam [Hills, 1989]. In the same year, the Mines Department Inspector described the dust conditions at Wittenoom as "terrific." Two years later, Dr. Eric Saint, the flying doctor for the Pilbara, but formerly from Newcastle-on-Tyne, where he had acquired a knowledge of occupational lung diseases, informed the Wittenoom Mine management that asbestos was extremely dangerous and that men exposed could contract chest disease within six months. He wrote to the Public Health Department in Perth predicting that the Wittenoom Mine would produce "the richest and most lethal cases of asbestosis in the world's literature."

In 1961, the first cases of mesothelioma was detected among ex-Wittenoom workers [McNulty, 1962]. By 1967, numerous cases of asbestosis had been described among Wittenoom workers [Elder, 1967] and the local council was also warned that the tons of asbestos tailings being spread around the township to control the red dust and mud could threaten even tourists.

Mortality in the workforce of Wittenoom has been studied subsequently [Hobbs et al., 1980; Armstrong et al., 1988]. Based on two different assumptions as to the vital status of subjects lost to follow up standardized mortality ratios (SMR) have been calculated. The first assumed all those lost to follow up were still alive up to age 85, whereas the second censored subjects at the date they were last known to be alive. Among the 6,505 men who were employed in the mining and milling of crocidolite at Wittenoom between 1943 and 1966, the SMR for all causes of death as of December 1980 was 0.96–1.53. There was a statistically significant excess number of deaths from cancer, neoplasms of the trachea, bronchus, and lung (SMR 1.60–2.64), and neoplasms of the stomach (SMR 1.16–1.90); 32 deaths were noted from malignant mesothelioma. Excess mortality also occurred from respiratory diseases, particularly pneumoconiosis (SMR 15.1–25.5); infections, particularly tuberculosis (SMR 2.72–4.09), mental disorder, particularly alcoholism (SMR 3.16–4.87); digestive diseases, particularly peptic ulceration (SMR 1.58–2.46) and cirrhosis of the liver (SMR 2.53–3.94); and injuries and poisonings, particularly non-transport accidents (SMR 1.62–2.36). The mortality from pneumoconiosis and from malignant mesothelioma and the excess from respiratory cancers, but not from stomach neoplasms, was dependent on time since first exposure to crocidolite and on cumulative exposure. There was no increase in mortality from laryngeal cancer (SMR 0.68–1.09) or other neoplasms.

As a result of the crocidolite industry in Western Australia, the incidence of malignant mesothelioma has been greater in this state than in all other states of Australia since the mid-1960s [Musk et al., 1989] and is one of the highest in the

world [de Klerk and Armstrong, 1991]. Pleural mesotheliomas have accounted for nearly 90% of all cases of mesothelioma in Australia. Up to the end of 1986, 12 of 94 cases of malignant mesothelioma have been described as peritoneal in the Wittenoom workforce cohort [de Klerk et al., 1989a].

As in other studies, lung cancer incidence has appeared to increase linearly with cumulative exposure to crocidolite [de Klerk et al., 1989b]. Smoking and crocidolite exposure have acted independently and hence, multiplicatively, in the production of lung cancer in a subgroup of the Wittenoom cohort in whom smoking information was obtained by mailed questionnaire [de Klerk et al., 1991].

MORBIDITY FROM ASBESTOS-RELATED DISEASES

A study of the prevalence of radiographic abnormality in the Wittenoom workforce as of 1980 showed that undiagnosed radiographic changes compatible with the presence of asbestosis (small irregular shadows of profusion 1/0 or greater on the ILO scale) were present in approximately 15% of the workforce, especially those more heavily exposed to crocidolite [Cookson et al., 1986a].

The Wittenoom cohort has also permitted an examination of the natural history of asbestosis and of benign pleural disease to be conducted. The course of pulmonary asbestosis and its determinants were examined in 280 applicants for compensation from the Wittenoom workforce. Serial chest X-rays, accrued over more than three decades, were graded separately by two observers according to the 1980 ILO Classification of Radiographs of Pneumoconioses without knowledge of exposure histories or compensation details. In 136 subjects whose median duration of exposure was 37 months, radiographic asbestosis appeared between 1–34 years after initial exposure and, usually, progressed continuously. Total exposure to asbestos and time from first exposure to the appearance of definite radiographic asbestosis were significant determinants of the rate of progression in profusion of small opacities. Asbestosis in these subjects usually appeared to be progressive, even 30 years after exposure had ended [Cookson et al., 1986b]. No effect of smoking on the incidence of asbestosis (as measured by a successful claim for compensation) can be demonstrated in the Wittenoom workforce [de Klerk et al., 1991].

Minor benign pleural thickening, as observed in plain chest radiographs of the Wittenoom workers, has been shown to frequently progress in extent along the lateral chest wall, but progression beyond 5 mm in thickness is less common [de Klerk et al., 1989c]. Pleural plaques were not seen to progress beyond their initial thickness or extent. The onset of pleural thickening in the Wittenoom workforce increased continually from the time of first exposure and also increased slightly with age. There was evidence that the level of total cumulative exposure to crocidolite was directly related to the rate of onset of pleural thickening in the period between 5 and 15 years after exposure to crocidolite. The rate of progression of established pleural thickening was greatest in subjects who first developed thickening soon after first exposure, and the relative rate of progression decreased slowly with time from first signs of thickening, there being no evidence of any progression more than 15 years after onset. In the Wittenoom workforce, the rate of diffuse pleural thickening in relation to pleural plaques has been greater than in other asbestos-exposed cohorts where the incidence of malignant mesotheliomas is lower [Hillerdal and Mark, 1990].

Asbestos-related diseases, especially malignant mesothelioma, have also been

observed in workers involved in the transport of Wittenoom crocidolite by road to Point Sampson or Port Hedland, and by sea to Fremantle and beyond (Armstrong et al., 1984).

PREDICTIONS FOR FUTURE DISEASE

Predictions have been made of the future incidence of malignant mesothelioma, bronchogenic carcinoma, and asbestosis in the Wittenoom workforce using statistical models to fit the incidence of these diseases to December 1986 to age, duration and intensity of exposure, time since first exposure, and other exposure-related variables [de Klerk et al., 1989a]. The number of new cases of malignant mesothelioma in the cohort of 6,505 males and 410 females is expected to rise to a peak of over 20 cases per year by the year 2010, with an expected total number of up to 700 cases between 1987 and 2020. A total of about 3,000 deaths from all causes is expected during the same period. New cases of lung cancer and asbestosis are expected to continue at their current rates of about 8 and 17 cases per year, respectively, before declining after the year 2000, leading to totals of around 200 cases of lung cancer and 450 cases of asbestosis by the year 2020.

COMPENSATION AND LITIGATION

As a result of its heavy dependence on the mining industry, initially gold mining, the State of Western Australia has had a long experience with the occurrence of pneumoconiosis and the need for compensation of affected workers. This need was first addressed in the Workers Compensation Act in 1925, which was subsequently revised to include mesothelioma in 1970 and lung cancer with heavy asbestos exposure in 1984. To date, 389 cases of asbestosis, 116 cases of mesothelioma, and 159 cases of lung cancer attributed to asbestos exposure among workers formerly employed by Australian Blue Asbestos Ltd. have filed. Modeled on the British system of compensation, a non-adversarial decision on eligibility is made by a medical panel which, according to legislation, consists of the Mines Medical Officer for the State, a physician from the Perth Chest Clinic, and a respiratory physician, who sit jointly and examine all applicants to determine the presence of a dust-related disease and the degree of disability which results from this disease. The decision of the panel is binding on the workers' compensation insurers.

Successful civil litigation has been much more slowly achieved. The first mesothelioma case to sue the holding company of ABA occurred in 1977, but the patient died before his case came to court. The second attempt was made by a 51-year-old woman who had been employed in an office on the mine site and who had developed malignant pleural mesothelioma in 1979. The judge in this case considered that negligence had not occurred, but "sad misadventure." The patient died a few hours after her appeal was to be heard and the case did not proceed further.

No further civil litigation was undertaken for another ten years. During this time, the previously loose social network of ex-Wittenoom people became cohesively organized as the Asbestos Diseases Society of Western Australia. The first asbestosis case was mounted by an inter-State law firm which specialized in personal injuries. The plaintiff in this case was a man who had worked at Wittenoom for six months. He had had an open lung biopsy performed for diagnostic purposes ten years earlier

which had confirmed the presence of diffuse interstitial pulmonary fibrosis. His was the longest running civil litigation case in the history of the Western Australian Supreme Court to that time. It was heard by a single judge who did not appear to comprehend the complexities of medical evidence and found that the plaintiff did not have an asbestos-caused disease. The defense had questioned every piece of exposure history and medical evidence, even that which was not important to establishing that diffuse pulmonary fibrosis was not present (since this was conclusively shown by the open biopsy). The judge then concluded that the condition was instead "idiopathic pulmonary fibrosis," persuaded by the paucity of asbestos bodies in the lung tissue and despite the exposure history. The so-called "four dog defense" (Brodeur, 1986) was used and almost succeeded. The exposure history was questioned (we don't own a dog). The cause of the man's disability was disputed; he had airway disease from smoking (we own a dog, but another dog bit him). The plaintiff should have worn a mask (he knew the dog was dangerous). If he had disease, he was not disabled (the dog bit him, but didn't hurt him). Subsequent appeal on this case, however, held the man to have asbestosis, and the company to have been negligent. During these proceedings, a further case of mesothelioma was heard by judge and jury in the State of Victoria. The plaintiff was awarded Australian $426,000 by way of compensation and Australian $25,000 punitive damages against his ex-employer for "reckless indifference to the safety of its workers." In Western Australia, the next case kept the court sitting for 131 days when two patients with mesothelioma (one of whom died during the proceedings) were ultimately successful with their claims. Since then, the employer has endeavored to settle cases out of court. Up to June 30, 1990, a total of 336 cases had been settled. No case of lung cancer has yet been concluded in the court.

THE TOWNSPEOPLE

It has been estimated that at least a further 5,000 people lived in the township of Wittenoom (many as children), but did not work in the mining, milling, or transport of asbestos. These people are currently the subjects of a further formal cohort mortality study (Hansen et al., 1991), as at least 15 cases of malignant mesothelioma have been recorded among them in the Western Australian Cancer Registry.

FUTURE INTERVENTIONS

The previous workforce of Wittenoom has been shown to be a group which is at increased risk of developing asbestos-induced malignancies, particularly lung cancer and malignant pleural mesothelioma. Because of this risk, intervention in the form of dietary supplementation with vitamin A preparations has been initiated in the ex-workers and ex-residents of the township. This project will explore two questions arising from epidemiological and laboratory evidence of the value of beta carotene or retinol in preventing the onset of malignancy. In the workforce cohort, beta carotene 30 mg daily will be compared with retinol 25,000 units daily to determine if one agent is superior to the other. As a result of knowledge of the outcome of subjects who do not take part in the project, a comparison group of subjects who receive supplementary vitamin A in neither form will be available to serve as a comparison group. It is estimated that sufficient events will have occurred after five years to demonstrate if one form of vitamin A is more effective than the other, and if neither form is better

than no intervention. In the cohort of ex-residents, beta carotene 30 mg is being compared with beta carotene 0.75 mg to determine whether or not deficiency is causative or whether large amounts are preventive.

CONCLUSION

Wittenoom has been described as Australia's greatest-ever industrial disaster. Controversy still surrounds the future of the township of Wittenoom and attention has turned to the environmental risks to people exposed to crocidolite who have lived there or who have encountered Wittenoom crocidolite elsewhere. There still remains great concern for the future of the people who worked in Wittenoom. This concern extends to current workers in the iron ore industry in the Pilbara region of the State in whom inadvertent exposure to crocidolite deposits appears possible during mining operations, and certainly for workers engaged in the removal and disposal of the vast quantities of building products containing Wittenoom crocidolite.

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