

REPORT
OF THE
Commissioner of Public Health
for the year 1959

Presented to both Houses of Parliament

ADDENDUM

INDUSTRIAL CHEST DISEASE I.—EASTERN GOLDFIELDS

This report is concerned, mainly, with the results of the periodical examination of miners and ex-miners. Many more miners have voluntarily submitted themselves for a clinical examination, in addition to those who are under special supervision. Visits were made to Wittenoom and Norseman to examine workers clinically, in association with the Mines Mobile X-ray Unit.

The result of the examinations during 1959 are as follows:—

Total number of Chest X-ray examinations	7,269
Total number of cases of Silicosis	569
Total number of new cases of Silicosis	71
Total number of cases of Active Pulmonary Tuberculosis	10

The figures below are the results of the periodical examinations since their inception in 1925-26 to 1958. It should be borne in mind that not all miners are X-rayed annually, so that yearly totals are not an accurate reflection of the incidence of lung disease in any one year.

Table II
RESULTS OF PERIODICAL MEDICAL EXAMINATIONS OF MINE WORKERS
FROM INCEPTION OF EXAMINATION (1925) TO 31st DECEMBER, 1958

Year	Total number of examinations	Total number of silicosis cases diagnosed	New cases of silicosis (previously normal or linear markings only)	Silico-tuberculosis and Tuberculosis without Silicosis
1925-26	4,023	642	642	142
1927	3,728	474	41	138
1928	3,483	440	62	46
1929	2,588	420	102	48
1930	3,399	450	138	164
1931	3,012	399	94	83
1932	4,285	426	35	24
1933	3,377	439	58	18
1934	5,563	406	55	17
1935	4,808	364	36	7
1936	7,363	372	30	19
1937	7,852	352	15	13
1938	7,141	296	13	12
1939	6,975	292	18	13
1940	7,299	263	12	8
1941	7,141	294	32	7
1942	8,824	350	61	5
1943	4,298	367	63	9
1944	4,468	375	70	14
1945	3,334	258	54	7
1946	5,606	300	90	12
1947	6,450	396	101	33
1948	5,134	298	24	9
1949	5,489	314	24	12
1950	6,203	349	14	12
1951	5,721	305	13	12
1952	5,959	294	9	12
1953	5,312	356	80	3
1954	6,179	487	158	16
1955	5,506	497	70	5
1956	5,476	474	30	9
1957	4,811	483	34	10
1958	6,286	582	54	8
	174,093	12,837	2,331	947

These results are disappointing and it would appear that there has been little significant improvement in the incidence of industrial chest disease for many years, despite the great advances in medicine, engineering, ventilation and working conditions. The totals only refer to miners and ex-miners examined under the *Mine Workers' Relief Act* and some examined for re-admission certificates under the *Mines Regulation Act*. They do not, therefore, contain all the cases of silicosis and/or tuberculosis attributable to the mining industry. Although 10 new cases of active pulmonary tuberculosis are recorded as being diagnosed in 1959, there were actually 24 cases, if men who have left the industry and were outside the scope of the relevant Mining Acts are included. Some cases may also have been diagnosed elsewhere and not brought to our notice.

It is hoped that the intensive follow-up and investigation of miners with abnormal chest X-rays will reduce the incidence of tuberculosis soon. This is a pious wish which has probably been repeated annually since 1926. It would appear that one worker out of every 60 who entered the industry contracted pulmonary tuberculosis and even today the annual incidence is 2 per 1,000. Fortunately, with the modern treatments available, even in the presence of advanced silicosis, successful treatment of the tuberculosis is almost guaranteed.

In regard to silicosis, the function of the periodical examination of workers, as conducted here, is mainly diagnostic, prevention being regarded as an engineering problem. One worker in every 30 who entered the industry developed silicosis with an annual incidence of 10 per thousand. It is not possible to determine what percentage of silicotics, recorded, subsequently appear in the last column as having developed tuberculosis. A frequent criticism made to account for the variation in the number of new cases of silicosis diagnosed annually and the continued high rate is that the diagnostic criteria vary. There is some truth in this criticism. With minimal radiological silicosis a subjective error is present but this usually only results in anticipating or delaying the diagnosis for a few years and the effect is cancelled out. Chest X-rays dating back to 1936 and clinical record cards containing the diagnoses made at the time are available and reappraisal of the films does not suggest that the diagnostic standards have varied greatly. Improved X-ray technique, high definition screens, dual film reading and comparison with standard films from the Pneumoconiosis Research Council are probably enabling a diagnosis to be made at a slightly earlier stage than hitherto and this is most desirable. If the diagnosis is in doubt, complete investigative facilities, including lung function tests, are available at the Perth Chest Hospital and ever increasing use is being made of these facilities.

Statistical analysis of the results is most difficult, if not impossible, because miners and ex-miners are examined under several different Acts and because of the large number of itinerant and casual short-term workers in the industry. Australian Blue Asbestos at Wittenoom which has been in full production for a relatively short time presents a different problem and has been the subject of a special report.

A report on the health of miners would not be complete without introducing the question of the miners general susceptibility to respiratory infections, particularly chronic bronchitis. It is extremely difficult to apportion pulmonary disability between silicosis and chronic bronchitis. Chronic bronchitis almost invariably accompanies silicosis but occurs equally commonly without any evidence, whatever, of silicosis. It is a disease associated with heavy industry and air pollution of which silica dust forms only a part. Dust, fumes, artificial working conditions, lack of sunlight, changes in temperature and humidity, cigarettes, alcohol, all contribute. The system of ventilation, which at times direct a stream of cold air on a man perspiring from over exertion, may also contribute. At the pre-entry medical examination all applicants for initial medical certificates with evidence of bronchitis are excluded and miners who develop suggestive symptoms are strongly advised to leave the industry. Intensive research is being conducted in England into this particular problem and further developments will depend on the outcome.

INDUSTRIAL CHEST DISEASE II.—WITTENOOM

Periodical chest X-ray examination of workers in the Asbestos industry during 1959 disclosed no new cases of Asbestosis, although there are several men whose X-rays must be considered suspect. Those whose X-rays are considered suspicious of Asbestosis will be examined again in 1960.

The length of exposure of workers contracting Asbestosis or Silicosis is much shorter than comparably employed men in the gold-mining industry and the extent of disease is greater. The small numbers of workers and the extremely high labour turnover make this less obvious.

There are a number of puzzling and disquieting features. In several instances Asbestosis has been diagnosed in underground workers although the disease had been considered peculiar to the Mill. One member of the train crew underground, not usually a great dust risk, has what appears to be a mixture of silicosis, asbestosis and possibly tuberculosis. Another underground worker has what appears radiologically to be pure silicosis, but his sputum contains asbestos bodies.

The Mines Department has permitted me to inspect the results of their periodical dust counts. It is obvious that the new Mill is a great improvement on the old Mill and that conditions underground have also improved. It is too early to tell whether the new Mill will be responsible for fresh cases of industrial chest diseases or not.

The disquieting feature about the dust counts are two fold:—

- (1) The rock mined is apparently highly silicious and therefore particularly dangerous. The dust counts are on a par with the figures for the gold mines. A higher standard should be set in view of the high silica content and the additional Asbestosis risk.
- (2) The dust counts are of particles only; the same instrument is used for detecting silica particles and asbestos fibres. The dangerous particle size in Silicosis is less than 5 microns; in Asbestosis the dangerous fibre length is between 20 and 40 microns. The dust counts therefore are of little value unless they show the number of fibres per cc within that range. The Rotameter in use in this State is not regarded as an effective instrument for dust counting in the Asbestos industry in Canada. A impact-impinger is good. It is stressed that fibres over 10 microns in length are of particular importance and whilst the total dust concentration should be below 10 million particles per cubic foot of air, the maximum fibre count (i.e. over 10 microns in length) permitted, is 1 million fibres per cubic foot of air.

Many workers remain at Wittenoom for less than one year and less than 50 per cent. of the work force, at any one time, have been continuously employed for more than four years. A very rough estimate of the percentage of workers, with over four years exposure and suffering from industrial chest disease is 12 per cent. (estimated by expressing the number of workers, detailed below, as a percentage of the number of workers examined in 1959 whose initial mining certificate was issued prior to 1955).

pressure. For example, in 1958 there were no new cases of Silicosis in the 0-10 year group, 20 cases in the 10-20 year group and 29 cases in the 20-30 year group. Expressed as a percentage of the total number of mine workers, this gives an annual incidence of Silicosis of approximately 1 per cent. percentage incidences. These are not very accurate and are not really comparable, but they do illustrate the grave hazard which existed at Wittenoom compared with the general mining industry.

Since 1958, there have been one underground and five mill workers at Wittenoom definitely diagnosed as suffering from Asbestosis. All were advised to leave the industry but for various reasons have not done so. Five of the six were investigated in hospital. The average exposure to asbestos dust in these cases was four years only.

There are two other suspect cases and two suggesting they are affected with a combination of Silicosis and Asbestosis.

Apart from these, four others were diagnosed with Silicosis, three having had a previous history of coal mining abroad. These cases suggest that there is also an increased risk of Silicosis underground at the mine. The average exposure in these cases is 11 years (excluding previous mining). In two of these cases there is probable superadded tuberculous infection.

Some newly diagnosed cases of Silicosis are not included because of previous lengthy gold-mining history which could reasonably be held responsible.

There have also been a few cases of workers developing pulmonary tuberculosis without Silicosis or Asbestosis, but their number does not suggest any particular hazard at Wittenoom, other than that associated with mining in general.

Three workers with a mixed gold and asbestos mining history have developed silicosis and asbestosis. Four workers with a similar mining history have developed pure silicosis. Of these seven workers, the average exposure to dust including all mining was 12 years and excluding previous mining (which is not entirely justifiable) was 7 years.

Conclusion

The percentages quoted above (i.e., 12 per cent. of asbestos workers suffering from industrial chest disease with over 4 years exposure compared with 1 per cent. of gold miners with over 10 years exposure) are crude and probably paint too black a picture of the conditions which existed. With a shifting working population, it is almost impossible to work out accurate incidences. However, the very high labour turnover at Wittenoom is necessarily associated with a short average exposure per worker and many workers at risk were protected simply because their exposure time was too short. At least one of these short-term workers has been diagnosed as suffering from Asbestosis, many years after he left the industry.

The problem is essentially one of ventilation conducted with a proper appreciation of the relative importance and medical significance of the asbestos fibre as distinct from the silica particle. Despite the many marked improvements which have been effected at the Mine and Mill, I am not satisfied that the risk of industrial chest disease has been eradicated or even brought to par with the risk of Silicosis in the gold-mining industry. The prevention of industrial chest disease is a medico-engineering problem and requires close liaison between the Mines Department, who are responsible for ventilation and dust counts, and the medical officers who are responsible for the periodical clinical and chest X-ray examinations.

J. McNULTY, M.B., Ch.B., B.A.O.,
Chest Physician.