DUST EXPOSURE AND COALMINERS' RESPIRATORY HEALTH

MICHAEL JACOBSEN, Ph.D.

Deputy Director, Institute of Occupational Medicine
8 Roxburgh Place, Edinburgh, Scotland EH8 9SU

During the last 20 years my colleagues and I have been publishing a series of reports describing the relationships between dust exposure and coalminers' respiratory health. Those reports were from the British Coal Board's Pneumoconiosis Field Research—probably the most comprehensive long-term epidemiological study of an industrial population ever undertaken. It began 35 years ago in the late 1960's. It provided interim estimates of risks of coalworkers' simple pneumoconiosis for different levels of dust exposure, and this information was used by the British authorities, and by our colleagues here in the USA as basis for fixing dust exposure limits in coal mines. Soon afterwards, the same data were used to show that exposure to coal mine dust also increased risks of having symptoms of chronic bronchitis and of suffering some reduction in breathing capacity, as measured by the forced expiratory volume.

Many other studies were completed subsequently, as the field research continued at 10 of the original 24 mines that had been selected originally, in 1953. They refined, elaborated, and eventually verified the reliability of the CWSP risk estimates that were first produced in the late 60's and early 70's; explored the effects of exposure to dust on coal miners' mortality; described how exposure to respirable dust increased miners' chances of having pathological determined emphysema, and more recently, showed how exposure to respirable dust was related quantitatively to risks of developing not only coal workers' simple pneumoconiosis, but also the more serious disabling, and often fatal condition, PMF—progressive massive fibrosis of the lung.

This afternoon I will describe results from some further analyses of some of the accumulated material which were aimed at expressing the long-term working-life risks of pneumoconiosis, and of other effects of dust exposure on respiratory health, on a common baseline: the different mean concentrations of respirable dust to which a miner might be exposed during his working life under different scenarios of exposure limits and his responses to them.

First, however, I need to remind you of the essence of the strategy used in the research which generated the information in the medical data; radiographic signs of pneumoconiosis, respiratory symptoms and lung function levels, were obtained at medical surveys at each of the 24 mines, representing, in each case, an approximate 10-year period of observation during the calendar years 1953 through 1968. Two further medical surveys, generating a further decade of observations were completed during 1978 at 10 of the mines that were still open.

The exposures to dust for each individual miner who worked at the pits during the 25-year period, were calculated by keeping careful records throughout about precisely where each man was working during each of the tens of thousands of shifts that he attended. The average concentrations for such "occupational groups" were multiplied by the number of shifts each worked in them; and those products of concentrations (n mg/m^3) and shifts (converted into hours) were to give estimates of each individual's exposure to dust. Exposures experienced before the research measurements began were estimated, from data acquired by interviews with each man about where, how long, and what kind of work he had been doing and the results from the dust sampling. Thus, each man's cumulative exposures to respirable dust, for the whole of his preceding working life was calculated, the units being products of concentrations of dust and time. The various disease risk estimates were therefore expressed as functions of exposures measured in those units.

If we assume that a miner starts exposure at 20 years of age, and continues working underground for 35 years, with 1630 working hours per year, then those exposures can be expressed as mean of concentrations of dusts. So we may ask from the data: What risks of disease may be expected at age (20 + 35) = 55 for different mean concentrations of respirable dust?

The pneumoconiosis risk estimates were calculated from the most comprehensive set of data, describing more than 5,000 to approximately 5-year man intervals of exposure during the whole 25-year period. This study involved more than 30,000 miners from all 24 mines.

The results from a study were first described briefly here in Pittsburgh 5 years ago. Risks of pneumoconiosis were higher, for any given level of exposure at pits with greater carbon content in the coal. This is illustrated for category 1, simple pneumoconiosis, for higher categories, 2 or 3, and also for PMF.

Don't be misled by the apparently widening gap between the two lines in this series of graphs—this is due to the changing scale on the vertical axes of the graphs. The effect of the higher (91b) carbon content is about 2 to 3 additional percentage concentration, and about 5 additional percentage probability units at 7 mg/m^3.

Note, however, that the PMF risk, at the end of a 35-year exposure period increases steadily and smoothly with the mean concentration of dust experienced. This is a reflection of two important factors:

First, a small, but statistically highly significant increase in PMF risks among man with *no* simple pneumoconiosis (i.e., category 0);

Second, a very much more substantial increase in risks for that small minority of miners who develop even category 1 simple pneumoconiosis—and increase very clearly with increasing exposure.

The very dramatic increase in PMF risks after they develop simple pneumoconiosis is illustrated, which shows what happens if a man develops category 1, or even category 2, during the first 10 years of his 35-year exposure. At low (2 mg/m^3) concentrations, there is an approximate ten-fold increase in the risk if a man has category 1 already at the age of 35, and a 20-fold increase if he progresses during those first ten years to category 2. These results demonstrate dramatically reduction in developing PMF by reducing dust levels. This will effectively eliminate, or at least reduce, the higher risks associated with the early presence of simple pneumoconiosis, and will also protect the majority of miners, with no pneumoconiosis, from the small but finite, PMF risks at higher concentrations.

Risks of ending the 35-year exposure periods with respiratory symptoms are taken from a study of a sample of miners from 10 of the mines, who all attended the first four of the medical surveys. The sample was selected to provide an approximate uniform distribution of exposures to dust and to maximize the statistical precision of estimates of exposure-response relationships.

At hypothetical zero dust levels, cigarette smokers in this population stood a more than 3-fold higher chance than lifelong non-smokers of reporting chronic cough and sputum production. The just over 7% probability at zero exposure for non-smokers reflects the effects of all other causes of chronic bronchitis. Nevertheless, the risk more than doubles, at about 18%, for non-smokers who are consistently exposed to high (i.e., 7 mg/m^3) concentrations of dust.

A similar picture is found when considering breathlessness, that is men who acknowledge that they are breathless when they walk at their own pace on level ground: an approximate three-fold increase for cigarette smokers (as compared with non-smokers) at low dust concentrations, and also an approximate three-fold increase in the risk for non-smokers who are exposed to concentrations of dust as high as 7 mg/m^3.

Do these dust-related increases in bronchitic symptom risks imply corresponding increases in lung function impairments? A recent re-analysis of data first published in 1973, shows that they do.

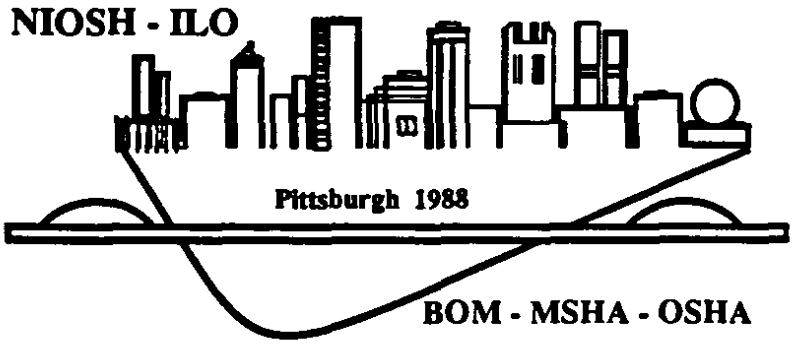
If the dust-associated decrements in FEV_1, small or average, are expressed as risks of having an FEV_1 less than 80% of that expected in non-smoking miners with no exposure to dust, then the risk of having this deficiency increases steadily with increasing exposure, both among cigarette smokers and non-smokers; and what is more, at approximately the same rates. Note again, that the risk for a non-smoker who has been exposed to high (7 mg/m^3) concentrations of dust, is comparable to that attributable to cigarette smoking in the absence of dust exposure.

FEV_1 65% of that predicted, also shows the relationship with exposure; and again, the effect attributable to smoking, at zero exposures, is similar in magnitude to that associated with a high, 7 mg/m^3, exposure over 35 years in non-smokers.

Slides not provided.

Proceedings of the VIIth International Pneumoconioses Conference | *Part*
Transactions de la VIIe Conférence Internationale sur les Pneumoconioses | *Tome*
Transaciones de la VIIa Conferencia Internacional sobre las Neumoconiosis | *Parte*

I



Pittsburgh, Pennsylvania, USA—August 23–26, 1988
Pittsburgh, Pennsylvanie, Etats-Unis—23–26 aout 1988
Pittsburgh, Pennsylvania EE. UU—23–26 de agosto de 1988

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

CDC
CENTERS FOR DISEASE CONTROL

Sponsors

International Labour Office (ILO)
National Institute for Occupational Safety and Health (NIOSH)
Mine Safety and Health Administration (MSHA)
Occupational Safety and Health Administration (OSHA)
Bureau of Mines (BOM)

September 1990

DISCLAIMER

Sponsorship of this conference and these proceedings by the sponsoring organizations does not constitute endorsement of the views expressed or recommendation for the use of any commercial product, commodity, or service mentioned.

The opinions and conclusions expressed herein are those of the authors and not the sponsoring organizations.

DHHS (NIOSH) Publication No. 90-108 Part I