



Industrial Dust Exposure and Chronic Obstructive Airways Disease

Discussion paper prepared for

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

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INDUSTRIAL DUST EXPOSURE AND CHRONIC OBSTRUCTIVE AIRWAYS DISEASE: AN OVERVIEW OF CURRENT OPINION

The major "event" that has dominated the legal and scientific dispute on the relationship of dust exposure to chronic obstructive airways disease has been the massive litigation in the U.K. between British Coal and a selected number of underground miners. This has been one of the longest and costliest personal injury court cases ever to take place in Britain. Mr. Justice Turner gave judgement on January 23, 1998. The importance of the trial relates to the dominant size of the industry in previous years and the detailed scientific research which was carried out to investigate the cause of lung disease in underground miners.

Both sides employed well known physicians and epidemiologists to present their side of the case and these were subjected to intense cross-examination by lawyers representing the litigants. It may be relevant to question whether teams of experts witnesses being cross-examined in court is the most appropriate way to determine scientific truth. Most recently a new approach has been indicated by Alabama Justice Sam Pointer in the litigation over the possible health effects of silicone breast implants. Justice Pointer appointed an independent panel of experts to review all the evidence and to report to the court their best estimate of scientific facts. The National Science Panel (or Pointer panel) included one well-known Canadian epidemiologist. This idea of a totally independent panel has obvious advantages and this may stimulate further developments.

The British Coal litigation was summarized in an Editorial in *Throax*, 1998, 53; 337-340. In the same journal there was a further general review of coal mining and chronic obstructive diseases. (*Thorax*, May 1998, 53, 398-407). The following points are drawn from those two documents. It should be noted that the author of the Editorial (Dr. R. Rudd) was one of the key experts appearing for the plaintiff as was one of the two authors of the review article (Professor D. Coggen).

Much of the litigation focused on the interpretation of epidemiological data derived by the Coal Industry itself. In general it was accepted by both parties that heavy dust exposure is associated with measurable decrements in spirometry (FEV1). There are two main schools of thought. One associated largely with Dr WKC Morgan (*Amer.Rev.Resp.Dis.* 1986, 134, 639-41) is that there is a small average fall in FEV1 affecting most miners, and is due to

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sputum in the airways. Such sputum with associated small decrements in FEV1 and is not likely to cause significant disability. The alternative view (Marien, et al. Amer.Rev.Resp.Dis. 1988, 137, 106-12) is that the small average decrement in FEV1 in the population of miners includes a number of men who are more seriously affected and are, in fact significantly disabled as a result of their exposure to underground dust.

The final judgement (summarized by Dr. Rudd in the Thorax editorial) made the following 6 points:

1. Coal mine dust (coal or stone) is a cause of centri-acinar emphysema.
2. Such emphysema may, and usually does, lead to a loss of ventilatory capacity most easily demonstrated by the loss of FEV1.
3. Tobacco smoke has similar effects as (1) and (2) above.
4. It is probable, but not certain, that there is a common causal pathway to both cigarette and mine dust induced emphysema which usually gives rise to breathlessness.
5. Whether (4) is established or not, the effects are generally the same in that there is a spectrum of effect which is not clinically detectable in the majority of cases but in the minority does produce a range of effects from simple impairment, frank disability and, occasionally death.
6. In the individual smoker it is not possible to attribute the cause of breathlessness either to the one insult or the other; this is so whether or not there is a common pathway.

The court recognized that dust exposure and cigarette smoking had similar effects and that, in the individual it was possible to assign the proportion of causality according to the level of dust exposure and cigarette smoking. Compensation was then awarded on a proportional basis. This approach emphasizes the point that it is not valid to suggest that, in a dust exposed cigarette smoker, the disability can be treated as if it was due entirely to one cause or the other. It makes no scientific sense to ask a physician which is "the most likely cause of the disability" or whether one or the other made a "significant contribution." The appropriate scientific method is to estimate, as far as possible, the contribution by each cause.

The general review of the evidence (Coggan and Newman Taylor, Thorax, 1998, 53, 398-407) also summarized their conclusions in concise form.

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1. The balance of evidence points overwhelmingly to impairment of lung function from exposure to coal mine dust.
2. There is strong evidence that coal mine dust can have a critical influence on health in an important number of people.
3. The exact nature of the pathology underlying the loss of lung function in miners is uncertain.
4. The best estimate at present of the average loss of lung function due to dust exposure in coal miners is 0.76 ml of FEV1 per mghm-3 (note mghm-3 is the calculated exposure to respirable dust multiplied by the duration of the exposure.)

In a comment on the comparative effects of smoking and the levels of dust exposure that were prevalent in the U.K. Coal mining industry Rudd (Thorax Editorial) quoted unpublished analyses from the evidence presented during the litigation. This analysis showed that cigarette smoking and high dust exposure in U.K. coal mining industry had effects of comparable magnitude on lung function.

The Marine analysis (Am.Rev.Resp.Dis. 1988, 137, 106-12) showed data on the probability of developing clinically important decrements in lung function in association with increasing levels of cumulative respiratory dust exposure. From the table it is possible to assign the aetiologic fraction in an individual miner, ie, how much of the observed lung function abnormality was caused by smoking and how much by dust exposure. A more "user friendly" presentation of the same information, based on Marine's results was published by Muir, (Appl.Occup.Environ.Hyg.13 (8) 1998, 606-607)

This is reproduced below:

Respirable Dust (mg/m3)	Exposure (Years)			
	10	20	30	40
2	10	19	27	33
3	14	27	37	46
4	19	33	46	57
5	23	41	54	64
6	27	46	60	70

Percentage Abnormality Attributable to Dust Exposure in a Cigarette Smoker with FEV₁ < 65% predicted

Dusts other than coal mine dust

The foregoing discussion summarizes the present situation on coal mine dust. An overview of all available information on the effects of dust on the development of obstructive airway disease was published by Oxman, et al (Amer.Rev.Resp.Dis. 148, 34-48, 1993).

The purpose of the overview was to identify all available information on the effect of occupational dust exposure on chronic obstructive pulmonary disease (COPD). Studies were only included if dust exposure was measured quantitatively and outcomes of interest (COPD, lung function) were controlled for smoking. Thirteen reports from four cohorts met the inclusion criteria. Three of these cohorts were from coal miners and one was from gold miners. (Hnizdo, et al. Scan.J.Work.Envir.Health. 1990, 16, 411-22). The effect of a given level of cumulative dust exposure in the South African Gold mines appeared to be substantially greater than the same amount of dust in U.K. coal mines. The risk of clinically important loss of lung function attributable to dust among non-smoking gold miners was estimated to be three times as large as for coal miners at less than one fifth of the cumulative respirable dust exposure (21.3 mg/m³, the maximal exposure observed among the cohort of gold miners.) The reasons for this remarkable difference were discussed in detail. One possibility might be the difference in the quartz content of the respirable dust. In gold mines of South Africa about 30% of the respirable dust is silica, with an absolute respirable silica concentration of 0.05 to 0.84 mg/m³. On the other hand, in collieries in the U.K. coal industry surveys the quartz content of the respirable dust was about 4%, giving an absolute concentration of about 0.16 mg/m³. If the quartz content of the respirable dust is an important factor in the causation of C.O.P.D. then it is difficult to explain why the U.K. coal mine data show no effect when stratified for different collieries, i.e., different silica content.

As far as the comparative effects of cumulative respirable dust exposure and cigarette smoking are concerned Hznido, et al found an interactive effect between dust and smoking. Although the effect of dust exposure per mg of dust was so high it happened that the measured levels of dust in the South African gold mines were low. As a result Hznido et al concluded that 90% of the C.O.P.D. in the gold miners would be prevented if the miners stopped smoking. No such interaction between smoking and the effects of dust exposure was observed in U.K. coal miners where the two appeared to have a simple additive effect.

It will be evident from this overview that there are remarkably few cohorts where the effects of dust exposure and smoking have been analyzed in sufficient detail to provide quantitative risk estimates. Most authors appear to

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accept that dust exposure can cause significant decrements in lung function and that these cannot be distinguished from the effects of cigarette smoking. The contribution from each source (smoking, dust exposure) depends on the magnitude of each and this provides a legitimate method of assigning proportional causality in the individual worker. There are obvious and real uncertainties in this method but it appears to be fairly robust and to have clinical utility.

For dusts having high quartz contents, close to that of the South African gold mines further consideration is required. Possibly some simple multiplication factor based on the fractional silica content of the dust is appropriate. However this is speculative and is not apparently supported by subanalysis within the U.K. coal industry itself. For the general type of mixed dust in industry when the quartz content is low, the best (in fact the only) approach may be to use the U.K. coal mining data which is described above. Historically the levels of dust in coal mines has been high much higher than generally found in surface industries or in hard rock mines. For this reason, the overall contribution of industrial dust to the causation of clinically important C.O.P.D. may turn out to be fairly low in comparison with the effects of cigarette smoking. This applies not only to the population of dust exposed workers but also to the individual clinical cases.

References from "*Industrial Dust Exposure & Chronic Obstructive Airways Disease*" a Discussion Paper prepared by Dr. David Charles F. Muir

Locations given are those in the Ontario Workplace Tribunals Library.

British Coal litigation. Summary of Justice Turner's judgement.
LOCATION: JUR 1211

Coal mining and chronic obstructive pulmonary disease: a review of the evidence.
Coggon, D Newman-Taylor A
Thorax 1998 v.53 n.5 p.398-407.
LOCATION: VF 9051

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Combined effect of silica dust exposure and tobacco smoking on the prevalence of respiratory impairments among gold miners.
Hnizdo, E et al.
Scandinavian journal of work, environment & health. 1990 v.16(6) p.411-422.
LOCATION: Source

Clinically important respiratory effects of dust exposure and smoking in British coal miners.
Marine, William M Gurr, Derek Jacobsen, Michael
American review of respiratory disease. 1987 v.137 p.106-112.
LOCATION: MF 2063

On dust disability and death.
Morgan W C K
American review of respiratory disease. 1986 v.134 p.639-641
LOCATION: MF 903

Occupational dust exposure and chronic obstructive pulmonary disease.
Muir, David C F
Applied occupational and environmental hygiene. 1988 v.13(8) p.606-607.
LOCATION: VF 9301

Occupational dust exposure and chronic obstructive pulmonary disease.
Oxman, A D Muir, D C F Shannon, H S Stock, E H Lange, H J
American review of respiratory diseases. 1993 v.148 p.38-48.
LOCATION: VF 8996

Coal miners' respiratory disease litigation [editorial]
Rudd, R
Thorax. 1998 v.53 p.337-340
LOCATION: VF 9311