Chronic Obstructive Lung Disease (COPD)

1990
Addendum, 1993

Prepared by:

Dr. D. Linn Holness

Respirologist

With supplemental information provided by:

Dr. David C.F. Muir, Director
Occupational Health Program, McMaster University

This medical discussion paper will be useful to those seeking general information. It is intended to provide a broad and general overview of a topic and is written to be understood by lay individuals. Discussion papers are not peer reviewed and do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by the discussion paper in any particular case. It is always open to parties to an appeal to rely on or distinguish a medical discussion paper, or to challenge it with alternative evidence.

CHRONIC OBSTRUCTIVE LUNG DISEASE

The question of whether occupational exposure to dusts in a general sense, or specific types of dust can cause chronic obstructive pulmonary disease is controversial and has produced much debate within the medical community. Ideally, we would like detailed exposure information including the specific mineralogic data, the size of the dust particles and the level of exposure. On the health outcome side of the equation detailed lung function results and pathological information would be useful. Unfortunately, at this time the medical literature does not provide studies which address all of these issues and provide clear-cut answers. Rather information on which to consider the question comes from a variety of different types of studies of communities or working populations with have examined broad categories of work exposures and respiratory symptoms and/or lung function. There are also a number of studies examining exposure to specific types of dust and respiratory outcomes such as chronic obstructive lung disease. These include silica (gold mining), coal dust and asbestos or a mixture of various dusts. The studies have used a variety of outcome measures, usually symptom reporting and lung function results but some have also examined pathological material.

Following are brief summaries of some of the studies which have suggested an association. Also included in the bibliography are three editorials which have appeared over the past 4 years (Soutar, 1989, Lancet, 1990 and Seaton, 1991). All have a similar message, and while recognizing that our current picture isn't complete, all state that there is enough current evidence to suggest a relationship between such exposures and chronic obstructive lung disease.

Silica and Chronic Obstructive Lung Disease

There are several studies which examine silica exposed workers. These studies have the advantage that the exposure information is often based on actual measurements and detailed job histories and tends to be better than available in community studies. On the other hand, because these studies tend to be cross-sectional in nature, various selection biases may pose a greater problem than in community studies. Following is a brief description of the results of some these studies.

Wiles and Faure examined 2209 South African gold miners between the ages of 45 and 54 who had worked underground for at least 10 years. Dust exposure was quantified as the dust counts (respirable surface area) for the various job titles multiplied by the length of time in each job. They found that symptoms of chronic bronichitis increased as dust exposures increased. The effect was smaller than that associated with smoking. Lung function values decreased as dust exposure increased, this effect was reported to be similar in magnitude to that of smoking. There was no difference in chronic bronchitic symptoms and lung function between heavy and light (<15 cig per day for 25 years) smokers. There was a tendency for the values to level off in the very high exposure categories. A number of possible explanations were suggested, one being that those who were susceptible to the effects of dust exposure had developed the effects at a certain point whereas those who weren't susceptible continued to have exposure and ended up in the higher exposure groups without any adverse effects. They noted that those who had left employment had a somewhat higher rate of chronic bronchitis and lower lung function, suggesting that there was no improvement with removal from exposure.

Irwig and Rocks carried out a cross-sectional study of 1973 South African gold miners between 45 and 54 years of age. They compared the 134 workers with findings of silicosis (p/q/r greater than 1/0) with the 1839 who did not have findings of silicosis. Exposure was measured as the respirable dust level for each job (respirable surface area) multiplied by the length of time in each job. There was no difference in the reporting of chronic bronchitic symptoms between those with and without silicosis. There was a slight decrease in the FEV1 and flow rates in those with silicosis compared to those without but no difference in FVC. They found that there was no difference in smoking between the two groups but those with silicosis had higher dust exposures. When they selected 116 pairs matched for age, smoking and exposure they were no differences in lung function between the silicotics and non-silicotics. When they used the entire data set and performed analysis of co-variance (age, height, dust level) the only differenece between those with and without silicosis was a decrease in flow rates. They concluded that the difference was due to higher dust exposure in the silicotic group, there were a 0.4L and 0.6L/s difference in FEV1 and FEV25-75 respectively between those with high and low dust exposures.

Cowie and Mabena studied 1197 South African gold miners who had a mean time underground of 25 years, 857 with silicosis and 340 without. Exposure categories were defined on a dust level and amount of physical work. They found that the duration of exposure underground was associated with decreases in FEV1 and flows and the size of the effect was similar to

that from smoking. Lung function results varied with degree of silicosis (profusion), dust exposure (years) and smoking (pack-years). They suggested that all miners were at risk of developing chronic airflow limitation whereas only a minority developed silicosis. Kreiss et al carried out a community-based prevalence study in a "one-industry town" (molybdenum mine (20-35% silica)) in Colorado where individuals were categorized as to whether or not they were miners. Cumulative dust exposure was calculated by using the historical respirable dust measurements for each job title multiplied by the length of time in each job. Increasing cumulative dust exposure was associated with decreasing flow rates when controlled for age, height and smoking. They found that for non-smokers, as the dust exposure increased the lung volumes decreased and the flow rates and diffusing capacity decreased consistent with obstructive disease. The mine had been closed for at least five months and therefore the effects observed were not thought to be acute in nature.

Manfreda et al examined several groups of workers including 95 underground hard rock miners and compared them to 382 controls in a cross-sectional study. For the miners, duration of employment was inversely related the lung function values suggestive of obstructive changes. They conducted a follow-up study 4 years later. They found that FEV1 and FEV1/FVC decreased significantly more in smokers compared to non-smokers and FEV1/FVC decreased significantly more in mining industry workers (underground and smelter) than in controls.

Three studies have examined pathological material. Becklake et al carried out a case-control study of autopsy specimens from 44 gold miners with emphysema and 42 without emphysema between the ages of 51 and 71. Exposure was based on classifying exposures as high, moderate or low. The presence and grade of silicosis and bronchitis were similar between the two groups. The predictors of emphysema were years worked in high dust exposure (Odds Ration 13, p=0.0004), age (Odds Ration 27, p=0.01) and number of cigarettes smoked per day (Odds Ratio 30, p=0.00001). These effects were independent.

Hnizdo et al studied types of emphysema in miners. They found that miners with a 20 year history of high dust exposure had 3.5 risk of having a significant degree of emphysema at autopsy than a miner not in a dusty occupation. They suggested that smoking may potentiate the effect of silica on emphysema and that in the absence of smoking emphysema in miners was unusual.

Churg et al examined pathological material from 53 workers who had been hard rock miners, or who had worked in the asbestos, construction or shipbuilding industry and compared them to 121 workers without dust exposure. They found changes which they characterized as mineral dust airways disease in 13 of the 53 exposed workers compared to only 1 of the 121 controls. These changes were associated with obstructive pulmonary function results. The authors concluded that abnormal small airways are present in some workers exposed to mineral dusts and these changes are associated with abnormalities of lung function greater than that induced by cigarette smoking alone. They also noted that the presence of the changes in only a proportion of workers could explain why other studies find conflicting results regarding obstructive lung disease and dust exposure.

Wright et al carried out an experimental study exposing rats to iron or silica (high and low) or no dust. There were no pathological differences between those with no dust or iron dust exposure. The rats exposed to silica dust had airflow limitation which was greater in the high exposure group. The airflow limitation correlated with morphometric findings of emphysema and thickening of airway walls. These effects were increased in the high exposure group.

In summary, these studies examining the effect of silica dust exposure on lung function have suggested that chronic obstructive lung disease and emphysema can develop in silica exposed workers, particularly in smokers. The question of individual susceptibility has been raised, namely that a small group of exposed workers appear to be particularly susceptible to this effect. When large population based studies are carried out, the group of susceptibles with a significant effect are diluted by those who do not develop problems, and therefore an overall adverse effect may not be detected. The relationship appears to have dose-response characteristics and those with higher exposures appear to be at greater risk. Because of the differences in dust sampling technique it is difficult to directly compare the results or provide a definite answer regarding the magnitude of effect at any specific level of exposure.

Community or Population Based Studies

There are several studies examining the health of communities which also examine the possible association between workplace dust exposure and lung disease.

There are a number of community based studies which had been carried out over 20 years ago. They are summarized in an article by Higgins who reviewed the results of studies carried out over a 20-year period ending in the late 1960's. He noted that the results of the studies were fairly consistent and demonstrated a higher prevalence of bronchitis and a lower mean ventilatory capacity among workers exposed to dust. He noted that the differences could not be explained solely on the basis of difference in smoking habit and that there were variable results regarding exposure effect relationships.

Bakke et al studied a random sample of Norwegians to examine the relationship between occupational airborne exposures and respiratory symptoms and asthma. They found that a history of occupational exposure to quartz dust was associated with a number of respiratory symptoms with adjusted relative odds ratios between 1.7 and 3.3 for the various respiratory symptoms.

Leibowitz reported on the results of general community study. Over 50% of the working males had had some exposure to potentially harmful substances. He noted that individuals who had had high exposure to silica had a higher rate of symptomology and lung function impairment than those who did not.

Krzyzanowski et al conducted a 13-year follow-up study of residents of Krakow. The relationship between rate of decline of FEV1 and occupational exposure was assessed. They found that dust did have some effect on lung function decline, particularly in certain occupational sub-groups. They estimated these effects to be similar in magnitude to those of tobacco smoking, and that the effects of smoking and occupational exposures were additive in effect.

Jedrychowski conducted a five-year follow-up study of workers in Krakow. They noted that levels of airborne dust exposure and fluorides were related to the prevalence of chronic bronchitis and decreases in FEV1 over the period of time.

Pham et al examined a group of 196 steel workers over a five-year period. The control population consisted of workers in a clean environment. They noted that steel workers had a significant deterioration in all of their lung function values, whereas the unexposed workers had decreases only in the diffusing capacity and the RV/TLC ration. The changes over the five years were greater for the steel workers.

Kjuus et al carried out a case reference study to examine the association between emphysema and occupational exposure to industrial pollutants. They analyzed 36 workers with emphysema and 72 controls matched for age and smoking habits. The estimated risk ration for those exposed (greater than ten years in polluted workplaces) for developing emphysema vs those non-exposed (less than ten years exposure) was 3. They were not able to define any further dose response effect.

Kauffmann et al conducted a follow-up study of over 500 men in Paris. They noted that FEV1 decline was related independently to occupational exposures in addition to smoking. They found that the slope of decline was greater in those with increased intensity of exposure to dust. Those who had changed jobs to a lesser exposure had a decrease in their decline.

Korn et al reported the results of occupational exposures on the respiratory status of 8,515 adult residents of 6 cities in the United States. Occupational information was recorded and total years of exposure to dust, gases and fumes was calculated for each subject. 31% of the population reported occupational dust exposure. Those individuals with dust exposure had increased odds of having respiratory symptoms and the frequency of symptoms increased with increasing length of exposure to dust. COPD was defined as a FEV1/FVC less than 0.6. Individuals exposed to dust had an odds ratio of 1.68 for COPD compared to non-exposed individuals (1.87 for current exposure, 1.61 for past exposure only). There was no difference in risk attributed in length of exposure between the different smoking categories and no significant interaction in multiple logistic regression analysis.

Coal Dust Exposure and Chronic Obstructive Lung Disease

There have been a large number of studies carried out looking at the relationships between exposure to coal dust and respiratory status. Some of these are briefly described below.

Marine et al studied over 3,000 British coal miners to assess the independent contribution of smoking and exposure to respirable dust to significant changes in lung function. Coal dust exposure was monitored over a ten-year period. They found that dust exposure could be related to an increase in prevalence of the various respiratory abnormalities they defined. There was no evidence that smoking potentiated the effect. They found that

with intermediate and high dust exposure levels, respiratory dysfunction in non-smokers was similar to that in smokers with no dust exposure. The concluded that both smoking and dust exposure can cause clinically important respiratory dysfunction.

Soutar and Hurley examined a group of men in the British coal-working industry who had been followed for over 22 years. They found that exposure to respirable dust could cause severe respiratory impairment in some of the workers. They found that in non-smokers the pattern tended to be one which suggested a restrictive defect, whereas in the smokers a more classic obstructive pattern was observed. Hurley and Soutar selected a subgroup of 199 miners who had left the industry and who they thought would be at greater risk. They found that this subgroup had a much larger decrease in lung function, demonstrating that some miners could have significant impairment.

Rogan et al studied a group of British coal miners over a long-term prospective study. They noticed a progressive reduction in FEV1, with increase in cumulative exposure to airborne dust. There was no difference between those with and without pneumoconiosis.

Hankinson et al studie over 6,000 coal-miners in the United States. They found that age and cigarette smoking had significant effect on flows at all lung volumes. They noted that prolonged underground exposure did have an effect on flow rates at high lung volumes and that the effects were particularly noticeable in non-smokers.

Cockcroft et al carried out a post-mortem study of emphysema in coal workers and non-coal workers. They found excessive emphysema in coal workers compared to non-coal workers, even after correction for age and smoking. There was also an association between smoking and emphysema. When only smokers were considered, there was still an excess of emphysema in coal workers. Coal workers with PMF tended to have higher risk of emphysema, but even in those without the odds ratio was still over 5 for coal workers compared to non-coal workers. There was further a greater number of workers who had high dust scores in the group with a greater degree of emphysema.

Ruckley et al studied the lungs of 450 British coal workers. Cumulative respirable dust exposures were available for the majority of the workers. They demonstrated a significant relationship between the cumulative dust exposure and the presence of emphysema in workers with progressive massive fibrosis and a trend to such a relationship in workers with simple pneumoconiosis.

References for Chronic Lung Disease and Dust Exposure

Bakke P, Eide GE, Hanoa R, Gulsvik A. Occupational dust or gas exposure and prevalence of respiratory symptoms and asthma in a general population. Eur Respir J 1991; 4:273-278.

Becklake MR. Chronic airflow limitation: its relationship to work in dusty occupations. Chest 1985; 88:608-617.

Becklake MR, Irwig L, Kielkowski D, Webster I deBeer M, Landau S. The predictors of emphysema in South African gold miners. Am Rev Respir Dis 1987; 135:1234-1421.

Brinkman GL, Block DL, Cress C. Effects of bronchitis and occupation on pulmonary ventilation over an 11-year period. J Occup Med 1972; 14:615-620.

Churg A, Wright JL, Wiggs B, Pare PD, Lazar N. Small airways disease and mineral dust exposure prevalence, structure and function. Am Rev Respir Dis 1985; 131:139-143.

Cockcroft A, Wagner JC, Ryder R, Seal RM, Lyons JP, Andersson N. Post-mortem study of emphysema in coalworkers and non-coalworkers. Lancet 1982;600-603.

Collins HPR, Dick JA, Bennett JG, et al. Irregularly shaped small shadows on chest radiographs, dust exposure and lung function in coalworkers' pneumoconiosis. Brit J Ind Med 1988;45:43-55.

Cowie RL, Mabena SK. Silicosis, chronic airflow limitation, and chronic bronchitis in South African gold miners. Am Rev Respir Dis 1991; 143:80-84.

Editorial. Coal dust and compensation. Lancet 1990; 335:322-324.

Elmes PC. Relative importance of cigarette smoking in occupational lung disease. Brit J Ind Med 1981; 38:1-13.

Hankinson JL, Reger RB, Fairman RP, Lapp NL, Morgan WKC. Factors influencing expiratory flow rates in coal miners. In: Walton WH. Inhaled Particles IV, Pergamon Press, Oxford, 1977, pp 737-755.

Higgins ITT. The epidemiology of chronic respiratory disease. Prev Med 1973; 2:14-33.

Hnizdo E, Sluis-Cremer K, Abramowitz JA. Emphysema type in relation to silica dust exposure in South African gold miners. Am Rev Respir Dis 1991; 143:1241-1247.

Hurley JF, Soutar CA. Can exposure to coalmine dust cause a severe impairment of lung function? Brit J Ind Med 1986; 43:250-257.

Irwig LM, Rocks P. Lung function and respiratory symptoms in silicotic and nonsilicotic gold miners. Am Rev Respir Dis 1978;117:429-435.

Jacobsen M, Buirns J, Attfield MD. Smoking and coalworkers' simple pneumoconiosis. In: Walton WH. Inhaled Particles IV, Pergamon Press, Oxford, 1977, pp 759-772.

Jain BL, Patrick JM. Ventilatory function in Nigerian coal miners. Brit J Ind Med 1981;38:275-280.

Jedrychowski W. A consideration of risk factors and development of chronic bronchitis in a five-year follow-up study of an industrial population. J Epidemiol Commun Health 1979;33:210-214.

Kauffmann F, Drouet D, Lellouch J, Brille D. Occupational exposure and 12-year spirometric changes among Paris area workers. Brit J Ind Med 1982;39:221-232.

Kjuus H, Irstad H, Langard S. Emphysema and occupational exposure to industrial pollutants. Scan J Work Environ Health 1981;7:290-297.

Korn RJ, Dockery DW, Speizer FE, Ware JH, Ferris BG. Occupational exposures and chronic respiratory symptoms a population-based study. Am Rev Respir Dis 1987;136:298-304.

Kreiss K, Greenberg LM, Kogut SJH, Lezote DC, Irvin CG, Cherniack RM. Hard-rock mining exposures affect smokers and nonsmokers differently. AM Rev Respir Dis 1989;139:1487-1493.

Krzyzanowski M, Jedrychowski W, Wysocki M. Occupational exposures and changes in pulmonary function over 13 years among residents of Crakow. Brit J Ind Med 1988;45:747-754.

Lebowitz MD. Occupational exposures in relation to symptomatology and lung function in a community population. Environ Res 1977;14:59-67.

Leigh J, Outhred KG, McKenzie HI, Wiles AN. Multiple regression analysis of quantified aetiological, clinical and post-mortem pathological variables related to respiratory disease in coal workers. Ann Occup Hyg 1982;26:383-400.

Love RG, Miller BG. Longitudinal study of lung function in coal-miners. Thorax 1982;37:193-197.

Manfreda J, Sidwall G, Maini K, West P, Cherniack RM. Respiratory abnormalities in employees of the hard rock mining industry. Am Rev Respir Dis 1982;126:629-639.

Manfreda J, Johnston B, Cherniack RM. Longitudinal change of lung function: comparison of employees of hard rock mining industry and general population. Am Rev Respir Dis 1984;129:A142.

Marine WM, Gurr D, Jacobsen M. Clinically important respiratory effects of dust exposure and smoking in British coalminers. Am Rev Respir Dis 1988;137:106-112.

Morgan WKC. Industrial bronchitis. Brit J Ind Med 1978;35:285-291.

Pham QT, Mastrangelo G, Chau N, Haluszka J. Five year longitudinal comparison of respiratory symptoms and function in steelworkers and unexposed workers. Bull Europ Physiopath Resp 1979;15:469-480.

Rogan JM, Attfield MD, Jacobsen M, Rae S, Walker DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. Brit J Ind Med 1973;30:217-226.

Ron WN, Kanner RE, Renzetti AD et al. Respiratory disease in Utah coal miners. Am Rev Respir Dis 1981; 123:372-377.

Ruckley VA, Gould SJ, Chapman JS, et al. Emphysema and dust exposure in a group of coal workers. Am Rev Respir Dis 1984;129:528-532.

Saric M, Palaic S. The prevalence of respiratory symptoms in a group of miners and the relationship between the symptoms and some functional parameters. In: Walton WH. Inhaled Particles III, Unwin Brothers, Old Woking, 1971, pp 863-871.

Seaton A. Coalmining, emphysema, and compensation. Brit J Ind Med 1990;47:433-435.

Sharp JT, Paul O, McKean H, Best WR. A longitudinal study of bronchitic symptoms and spirometry in a middle-aged, male, industrial population. Am Rev Respir Dis 1973;108:1066-1077.

Smidt U. Dust exposure, chronic bronchitis and emphysema. Eur J Respir Dis 1982;63:Supp118:59-67.

Soutar CA. Update on lung disease in coalminers. Brit J Ind Med 1987;44:145-148.

Soutar CA, Hurley JF. Relation between dust exposure and lung function in miners and ex-miners. Brit J Ind Med 1986;43:307-320.

Wiles FJ, Faure MH. Chronic obstructive lung disease in gold miners. In: Walton WH. Inhaled Partiles IV, Pergamon Press, Oxford, 1977, pp 727-735.

Wright JL, Harrison N, Wiggs B, Churg A. Quartz but not iron oxide causes air-flow obstruction, emphysema, and small airways lesion in the rat. Am Rev Respir Dis 1988;138:129-135.

Occupational Health Program
McMaster University
1200 Main Street West
Hamilton, Ontario, L8N 3Z5
Telephone: Area Code 416 525-9140

August 19, 1993

Ms Marie Makinson
Manager
Medical Liaison Office
Workers' Compensation Appeals Tribunal
505 University Avenue, 7th Floor
Toronto, ON M5G 1X4

Attention: Marie Makinson

Dear Marie:

RE: Discussion Paper on COPD

I enclose replies in relation to the agreed list of questions. I have left this fairly brief and in note form for reasons of convenience.

Yours sincerely,
(Signature)
David C.F. Muir, M.D.
Director

DCFM:py

(1) What is COPD and how does it differ from chronic bronchitis?

C.O.P.D. (Chronic Obstructive Lung Disease) is a narrowing of the small airways of the lung which causes the patient to have difficulty in breathing. Chronic bronchitis, strictly speaking, refers to a chronic productive cough with sputum. It may or may not be combined with C.O.P.D. in the patient. The two names C.O.P.D. and Chronic Bronchitis are often used interchangeably to refer to narrowing of the small airways.

(2) What is the natural history of C.O.P.D.?

The condition usually deteriorates when the cause continues (generally cigarette smoking). On stopping exposure it continues unchanged - the patient does not get worse but, unfortunately, does not get better. There is good evidence of this in the case of cigarette smokers. It may be that the same is true of industrial dust exposure although there is no documented evidence.

(3) What is the underlying physiological mechanism of COPD?

Constant irritation of the small airways leads to scarring (fibrosis) or even complete blocking of many small air passages. A separate mechanism is due to loss of elasticity of the lung in the condition of emphysema.

(4) Is there an individual sensitivity of develop COPD?

It appears that only certain individuals develop C.O.P.D. The reason for this is not known. In the case of emphysema there is a subgroup in the population who are at special risk. This tendency can be detected by a blood test (a antitrypsin). It is very uncommon and is not really relevant to the present discussion.

(5) What is the cause of COPD?

The overwhelming cause is cigarette smoking. Dust exposure if heavy enough and for a prolonged period of time can have the same effect. Some unfortunate patients develop C.O.P.D. in the absence of dust exposure or of cigarette smoking.

(6) What is the general medical view on the relationship between COPD and dust exposure?

This has been a bitterly disputed subject. Unfortunately both sides have exaggerated the situation. A McMaster paper summarizing our views has been published in the American Review of Respiratory Disease. *

(7) Is there a dose/response relationship between COPD and cigarette smoking and dust exposure?

Yes.

(8) Does the type of exposure to tobacco smoke matter: ie, cigarettes, pipe, cigars.

Cigarettes are the main culprit. Pipe and cigars are thought to be less harmful. I have not done a formal literature search on this issue but will do so if it is relevant.

(9) Does the composition and particle size of the dust exposure matter?

The particles must be of a size which can be inhaled during breathing. For calculation of the effects of exposure it is essential that the appropriate particle sizes and concentrations are measured. The particles cannot be seen in the air because of their small size. Comments about the apparent dustiness of the air or dust on clothing are not usually relevant. The concentrations of silica (quartz) may be important. *

(10) Is it known how irritants such as smoking and dust exposure cause COPD?

No.

(11) Does the type of COPD cause by dust exposure differ from that caused by cigarette smoking?

The two cannot be distinguished.

(12) Is there any interaction between the effects of cigarette smoking and of dust exposure?

Probably not. This would require a very detailed perusal of the literature. I will do this if requested.

(13) Is it possible to apportion the degree of severity of COPD and exposure to dust or cigarette smoking?

Yes. What is required is an estimate of duration and concentration of dust exposure on the one hand and, if possible, the duration and intensity of smoking on the other.

* Occupational Dust Exposure and chronic obstructive pulmonary disease, a systematic overview of the evidence. Andrew D Oxman, David CF Muir, Harry S Shannon, Susan R Stock, Eva Hnizdo, HG Lange; American Review of Respiratory Disease 1993 V 148:38-48. **Paper available from the WCAT Library**