

**Final Performance Report
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Coal Dust Particle Size and Respiratory Disease

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The primary goals of this research project were to:

- a) characterize the particle-size distribution of dusts in underground coal mining operations based on personal exposure monitoring data,
- b) apply job-specific estimates of the tracheo-bronchial to respirable dust exposures to historical respirable dust exposure data in order to estimate cumulative tracheo-bronchial exposures for a cohort of previously-studied underground miners,
- c) consider the changes in exposure-response relations using tracheo-bronchial exposures in place of respirable exposures for obstructive lung disease, and
- d) develop models to integrate exposure data over time to improve the validity of the exposure metrics used for epidemiologic analyses.

Collection of particle size distribution data was accomplished at four underground Appalachian mines. The data included 180 valid samples obtained with the assistance of volunteer miners at each site using an 8-stage personal cascade impactor. The initial intention of the project was to collect data from a larger set of mines, however this effort was frustrated by the unfortunate timing of the release of information by the Mine Safety and Health Administration concerning the potentially fraudulent sampling practices observed at a significant number of coal mines and subsequent legal proceedings. The Bituminous Coal Mine Operators Association and many of the individual mines contacted were unable to support our efforts at the time that they were contacted. The four mines that did participate were mines with previous relationships with our collaborators at NIOSH and the Bureau of Mines.

Findings from this effort indicated little variability between occupations, or groups of occupations in the particle size distributions, or the ratio of respirable to tracheo-bronchial dust fractions. There was some difference observed between mines, which may have resulted from the use of diesel transportation equipment in one mine. The findings are somewhat at deviance to observations of previous researchers, however all previous data were obtained by area samples placed in positions which may have accentuated the different distributions. The results were transmitted to NIOSH to aid in the development of the Criteria for Recommended Exposure to Coal Mine Dust which is currently under development by the agency. The data collection and findings are detailed in our report which is currently submitted for publication and is attached (Seixas, Hewett, Robins and Haney, 1993). Combined with additional particle size data sets, the findings are also scheduled to be presented at the 1994 American Industrial Hygiene Conference and Exposition (Hewett and Seixas, 1994).

The findings of this proposal made application of particle size-specific fractions to historical data infeasible so the original intent of estimating exposure-response relations using cumulative tracheo-bronchial dust exposures was not possible. However, the results also suggest that data collected as respirable exposure fractions should be equally predictive of obstructive disease, at least for historical reconstruction purposes.

Additional analysis of the relationship between respirable dust exposures and obstructive disease, especially symptoms of bronchitis and pulmonary function variables, was conducted prospectively on the previously analyzed cohort of 977 underground miners attending Round 2 and Round 4 of the National Study of Coal Workers' Pneumoconiosis NSCWP). These findings suggest a steep exposure-response relationship between dust exposure and the incidence of bronchitis symptoms within a few years of onset of exposure. Further, the data suggest a strong relationship between bronchitis symptoms and loss of pulmonary function. The results are described in Appendix A.

A further exploration of the dimensions of cumulative exposure resulted in a proposal to integrate time of exposure, dust concentration and exposure duration into a single flexible dose metric. The proposed metric uses exponents of the concentration and time of exposure components to estimate the optimal contribution of these factors in the development of disease. The metric was tested in the previously described cohort from the NSCWP, and the result suggested that the square root of concentration and the first power of time since exposure might be an optimal expression for cumulative exposure for low level coal dust exposure and decrements in forced expiratory volume in one second. For the incidence of wheezing, concentration and time since exposure squared may represent an improvement in a dose metric over simple cumulative exposure. The model is seen as experimental and application of this, or similar models to other data sets will ultimately prove its utility. The model and these findings were published in the American Journal of Epidemiology (Seixas, Robins, Becker, 1993) and are attached.

Overall, it was disappointing that our results made an exploration of the potential significance of tracheo-bronchial dust estimates over respirable measurements infeasible. However, an important implication of this finding is that respirable dust measurements are probably a sufficient surrogate for tracheo-bronchial dust exposures. Additional exploration of the particle size-specific dust exposures will have to be conducted prospectively. The introduction of a novel method of accumulating exposure data over time should stimulate other researchers to question the usual simple cumulative exposure model and allow a more thorough exploration of dose-response modeling for coal dust and bronchitis as well as for other chronic occupational diseases.

References

Hewett P, Seixas NS (1994). The tail of the elephant or an analysis of 8-stage cascade impactor samples collected in underground coal mines. to be presented, Am Indus Hygiene Conference and Exposition, May 21-27, 1994, Anaheim, CA.

Seixas NS, Hewett P, Robins G and Haney R (1993). Variability of Particle size-specific fractions of personal coal mine dust exposures. Am Indus Hyg Assoc J (submitted).

Seixas NS, Robins TG, Becker M (1993). A novel approach to the characterization of cumulative exposure for the study of chronic occupational disease. Am J Epid 137:463-471.

Appendix A

A Longitudinal Evaluation of Coal Dust Exposure, Pulmonary Function and Respiratory Symptoms in New Miners.

Introduction

Chronic exposure to coal dust is a well characterized predictor of decrements in pulmonary function (both FVC and FEV1) and symptoms of chronic bronchitis, especially chronic productive cough and phlegm production (Becklake, 1985; Soutar, 1987). While these associations are now relatively well accepted, the relationship between respiratory symptomatology and pulmonary function decrements, in both coal mining and other populations, is inadequately understood. Cross-sectional studies have been reasonably consistent in demonstrating reduced pulmonary function in relationship to symptoms of chronic bronchitis (Brodkin and Rosenstock, 1993). For instance, in a population of asbestos exposed workers, Brodkin et al (1993) have demonstrated 2 to 8% reduction in FEV1 in association with cough or phlegm production and an 11 to 17% reduction in association with wheeze and dyspnea. However, in longitudinal studies in which the significance of symptom reports for subsequent pulmonary function declines can be assessed, the results have been less clear-cut. In a large study of working men Fletcher et al (1976) demonstrated that chronic phlegm production was predictive of lower levels of FEV1, but was unrelated to the subsequent rate of decline of function. While similar results have been observed in some other populations (Brinkman, 1972) these findings have not been consistent. For instance, Higgins et al (1982) found that bronchitis, wheeze and dyspnea predicted the development of FEV1 less than 65 percent of predicted over a 15 year follow-up period, after controlling for baseline FEV1. In a 5-year follow-up to the study of asbestos-exposed workers cited above (Brodkin et al, 1993), the presence of symptoms was unrelated to subsequent rate of decline of FVC or FEV1, although the development of symptoms from baseline to follow-up, (Cough or Dyspnea and especially Wheeze or Phlegm), was associated with substantially greater rate of decline of both parameters (Brodkin et al, 1992).

In coal miners, most of the available studies have considered cross-sectionally the effect of long-term exposure to relatively high levels of coal dust on respiratory outcomes. In a study of long-term miners in Great Britain, Rogan et al (1973) observed an adverse effect of chronic bronchitis (cough and phlegm for 3 months in a year) on level of FEV1 and a larger effect among bronchitics who also reported breathlessness. In a large follow-up of this cohort, Soutar and Hurley (1986) observed that miners reporting chronic bronchitis had lower FVC, FEV1 and FEV1/FVC. They further demonstrated that miners with bronchitis had a greater effect of cumulative dust exposure on FEV1. A sub-group of this cohort, miners who reported chronic bronchitis and had left the industry for other jobs, had an especially large effect of exposure on loss of FEV1. Although longitudinal analyses of the effect of coal dust exposure on pulmonary function have been reported (Love and Miller, 1982; Attfield, 1985), these analyses have not considered the impact of respiratory symptoms.

The current analysis addresses the relationship between the development of respiratory symptomatology, decrements in pulmonary function and their relationship to quantitatively assessed exposure to respirable coal mine dust in a longitudinal study design. In addition, these effects are considered in a group of miners with relatively low level and short term coal dust exposures.

The analyses are conducted on a sub-cohort of underground miners studied by the U.S. National Institute for Occupational Safety and Health as part of the National Study of Coal Workers'

Pneumoconiosis (NSCWP). The NSCWP was conducted as a series of rounds beginning in the early 1970s. The data developed for the current study were collected as Rounds 2 (R2) and 4 (R4) of the study, conducted in 1972-1975 and 1985-1988, respectively. The sub-group used for the current analyses was defined as participants in R2 and R4 who began work in 1970 or later, and who were therefore exposed only to the relatively low concentrations in U.S. coal mines present since regulations under the Coal Mine Health and Safety Act of 1969 were in effect.

Previous analyses of dust exposure and pulmonary function parameters in this cohort have been completed (Seixas, et al 1991; Seixas et al 1993). In cross sectional analyses of pulmonary function 3-5 years after the start of exposure (at R2), a steep exposure-related reduction was observed for both FVC and FEV1 (Seixas et al 1993). After about 18 years of exposure history (at R4), there continued to be an exposure-related decline in FEV1, while no statistically significant effect was observed on the FVC. The magnitude of the dust-related change was much smaller at the later time. There was no statistically significant association between exposure and change in pulmonary function parameters between R2 and R4. These analyses suggested that there were initial rapid dust-related decrements in pulmonary function over the first 3-5 years that may be interpreted as consistent with an inflammatory response of the small airways. Subsequent to this response, there was less effect of dust exposure, and possibly some recovery of function.

The purpose of the current analysis is to consider the potential impact of this relatively short term and low level coal dust exposure on respiratory symptoms, and to explore the relationship between dust exposure, the development of respiratory symptoms and their impact on pulmonary function in this cohort of coal miners.

Methods

The cohort studied in this analysis includes miners who were enrolled in Round Four of the NSCWP (1985-1988) and who also had participated in Round Two (1972-1975). The cohort was further restricted to men who had pulmonary function studies at both rounds, and who began working in mines after January 1, 1970. Pulmonary function was measured using comparable methods at both rounds, using equipment and procedures meeting the then current American Thoracic Society recommendations (ATS, 1979; 1987). At least three forced expiratory maneuvers meeting ATS acceptability requirements were conducted at each round. Round 4 timed flows were recalculated using the flow threshold method to make the data comparable between rounds. Consistent with current ATS recommendations (ATS, 1987), no reproducibility criteria were used, however an analysis of the effect of reproducibility in this cohort has previously been published (Seixas, 1991). For some analyses, pulmonary function was expressed as a percent predicted based on age, height and race using the prediction equations of Crapo et al (1981).

Respiratory symptoms were elicited with a standard British Medical Research Council (BMRC) questionnaire and defined as follows:

- Cough: Cough first thing in the morning or during the day for most days at least 3 months per year.
- Phlegm: Produce phlegm in the morning or during the day for most days at least 3 months per year.
- Breathlessness: Short of breath walking with people of your own age on level ground.
- Wheeze: Chest sounds wheezing or whistling on most days or nights.

A composite symptom variable (Any) was defined as having a positive response to any of the four primary symptoms.

Exposure to respirable coal mine dust was quantified on the basis of MSHA air sampling data in combination with verbal work histories taken at R4 as previously described (Seixas et al, 1991). Cumulative exposure was calculated up to R2 and up to R4 and used in the appropriate analyses.

Analyses relied primarily on logistic and linear regression. All regression models included covariates age, height, smoking status, pack-years, race, pre-existing asthma and cumulative exposure. For cross-sectional analyses, smoking status was entered as two dummy variables indicating current or ex (v.s never) smokers. For longitudinal analyses, change in pulmonary function was standardized to the rate of change in liters per year $((R4PFT - R2PFT) / (R4date - R2date))$. Symptom reports for the longitudinal analyses were categorized into four groups as positive or negative at each of the two rounds (R2+R4+, R2-R4+, R2+R4-, R2-R4-). Smoking status over the period was combined into a four level variable; 1) R2 Never/R4 Never, 2) R2 Current/R4 Current, 3) R2 Current/R4 Ex and 4) all other combinations. For some of these analyses, exposure was partitioned at R2 and expressed as cumulative exposure up to Round 2 (mg/m³-years) and the average exposure between R2 and R4 (mg/m³). Model fit for linear regression was evaluated by examination of residual plots.

Results

Nine hundred and seventy-seven miners met the definition of the cohort and are described in Table I by smoking status at R2. On entry into the study, the cohort was relatively young (average age 27 years) and mostly white (95%). At Round 4, most were current or ex smokers (39% and 37%, respectively). Of 557 current smokers at R2, only 358 (65%) continued to smoke at R4. Pulmonary function parameters decline from R2 to R4, as one would expect based only on an effect of aging with or without a separate coal dust effect (Table II). Similarly, an increased prevalence of each of the symptoms was observed at R4, and was generally most prevalent among current smokers and least among R2 never smokers.

Logistic models for the association of cumulative exposure with symptom outcomes are provided in Tables III (R2) and IV (R4). At R2, cumulative exposure was a statistically significant predictor for Phlegm, Breathlessness, Wheeze, and the combined variable, Any. The estimated odds ratios for an increase of ten units of exposure (10 mg/m³-years) were 2.2, 2.8, 3.7, and 2.2, for these four outcomes at R2, respectively. At R4, statistically significant associations were observed only for Phlegm and Any with odds ratios for 10 mg/m³-years of exposure, 1.3 and 1.4, respectively.

To determine the association of the presence of symptoms at R2 on outcomes at R4, the cohort was stratified on the presence of Any symptom at R2, and the R4 logistic models (including covariates) were re-run (Table V). It should be noted that for the stratum that had symptoms at R2, the model considers the association between exposure and the loss of symptoms by R4 in such a way that a positive exposure coefficient within this stratum means that individuals with lower exposures were more likely to become asymptomatic by R4. Similarly, for the stratum without symptoms at R2, a positive exposure coefficient means that individuals with higher exposures were more likely to become symptomatic by R4.

Within the asymptomatic stratum, there was an association of borderline significance between cumulative exposure and phlegm production ($p=0.078$). No other outcomes were statistically significantly related to cumulative exposure in the R2 symptomatic stratum. For the group that did

have symptoms at R2, associations were observed between cumulative exposure and phlegm ($p=.043$), breathlessness ($p=0.008$) and any symptom ($p<0.001$).

These analyses were rerun with cumulative exposure partitioned into pre-R2 cumulative exposure and the average exposure between R2 and R4 and both aspects of the exposure history were included in the regression models. As might be expected, the primary aspect of exposure giving rise to the observed symptoms at R4 among R2 asymptomatics was the exposure received between R2 and R4 (results not shown).

In summary, it appears that exposure to coal dust during the first 3 to 5 years of employment (e.g., at R2) had a dose-related association with respiratory symptoms including phlegm production, breathlessness and wheeze, but notably not chronic cough. About 12 years later (at R4), phlegm production was still associated with cumulative exposure; phlegm and breathlessness were particularly associated with exposure in miners with no symptoms at R2. Those individuals with symptoms at R2 showed no exposure-related effects on symptom prevalence at R4. These results suggest that an exposure-related increase in prevalence occurred in the cohort in general, but that some miners experienced the symptoms at lower doses, or earlier periods of time than others.

The association of the presence of symptoms at R2 with pulmonary function is addressed in Tables VI through X. Percent predicted values of FEV1 and FVC at R2 and R4, and their difference, are given in Table VI stratified by symptoms reported at R2. At both R2 and R4 miners reporting symptoms had lower age- and height-adjusted levels of FEV1 and FVC than miners reporting no symptoms. Pulmonary function levels were particularly low for miners reporting wheeze, with mean levels of FEV1 at R4 of 82% of predicted as compared to 94% of predicted at R4 for those with no symptoms at R2. Furthermore, the decline of adjusted pulmonary function between R2 and R4 was greater in symptomatic than in asymptomatic miners. The greatest declines (almost 10% of predicted values) were observed among miners reporting either wheeze or breathlessness at R2. It is noteworthy that even in asymptomatic miners, the percent predicted pulmonary function declined between R2 and R4 (about 6%). Although these values are age and height adjusted on the basis of a reference population, the suitability of the reference group used to develop the predictive equations (Crapo, et al) to this study population is open to question. Nevertheless, comparison of predicted values between subgroups of the study population should be valid.

These data were further stratified to consider the significance of leaving employment as a miner (for any unspecified reason) during the interval between R2 and R4. Changes in the percent predicted FEV1 and FVC, stratified by R4 mining status and R2 symptom reports are given in Table VII. Miners with breathlessness or wheeze who continued mining had faster rate of loss than those with other symptoms or no symptoms. Among miners who discontinued mining, any symptom was associated with a small increase in the rate of lung function loss in comparison to those with no symptoms at R2. The largest rate of decline was observed among the small group of miners who reported wheeze at R2 but continued to work in mining through R4. Among these miners, percent predicted FEV1 and FVC declined over 12% over this period. Interestingly, miners with wheeze who did leave mining appear to have lost function at a slower rate comparable to the other symptom categories.

A further exploration of the significance of symptoms to the decline in pulmonary function over the study period was conducted by constructing linear regression models with the change in FEV1, or FVC as the dependent variable and including variables for symptom status. Covariates included in the models were age, height, race, asthma, smoking status (dummy variables representing R2Cur/R4Cur, R2Cur/R4Ex, All Others except R2Never/R4Never which was the baseline category)

and R4pkys. An alternative set of models were run considering significance of persistent, developing or resolving symptoms over the R2 to R4 interval. A set of three dummy variables representing the four symptom response categories (R2-R4-, R2+R4-, R2-R4+, R2+R4+; baseline category R2-R4-) were included in the regression models. The results of these analyses are given in Tables VIII (for change in FEV1) and IX (for change in FVC).

The presence of breathlessness or wheeze at R2 was associated with a more rapid rate of decline for both FEV1 and FVC over the period R2 to R4 of about 10 to 12 ml per year. These findings are consistent with the observation in Table VI that miners reporting wheeze or breathlessness at R2 had a greater loss of function over the study period than miners in other symptom categories. With symptom response at both surveys in the analysis (Model 2), the results indicate that miners either with any symptom or cough or phlegm at R2 and R4 (R2+R4+), or developing symptoms between R2 and R4 (R2-R4+) had a faster rate of decline of pulmonary function than miners who were asymptomatic at both times. For wheezing and breathlessness, presence of symptoms in R2 and/or R4 in any combination was associated with a faster rate of decline.

To consider the potential impact of dust exposure on the change in pulmonary function, especially in symptomatic individuals, the cohort was stratified on the basis of presence of individual symptoms at R2 and the regression models for change in FEV1 or FVC were again run, including the variable cumulative exposure. The regression coefficients for cumulative exposure from these models are given in Table X. While no effect of dust was observed in miners with or without cough or phlegm, miners reporting wheeze, or to a lesser extent, breathlessness at R2 had adverse effects of dust exposure on changes in FEV1. For change in FVC, miners reporting breathlessness or phlegm at R2 had a small and not statistically significant decline in association with cumulative exposure.

Discussion

On the basis of the cross sectional analyses at R2, taking place less than 5 years since initial employment in coal mining, a strong association was observed between cumulative dust exposure and the development of respiratory symptoms, especially mucous hypersecretion (phlegm production) and wheeze, and to a lesser degree, reported breathlessness. It is noteworthy that cough, which is frequently thought of as an early symptom of reversible airways effects was not associated with exposure. These associations parallel earlier findings in the same cohort indicating a rapid loss of lung function (both FEV1 and FVC) in association with dust exposure (Seixas, 1993).

While there was relatively little association observed between dust exposure and symptoms after about 18 years of exposure (R4), the association remained for the subset of miners who were symptom free at R2. This observation suggests that miners who were symptomatic at R2, may represent a sub-group of miners who were particularly sensitive to the early effects of the dust exposure. In fact, the group of miners who were symptomatic at R2, particularly with wheeze or breathlessness had lower lung function at R2 and R4, a faster rate of lung function loss over the period, and the loss of function (FEV1) was exposure-related; asymptomatic miners had reduced lung function as a percent of predicted, but the decrements was not exposure-related. Furthermore, the loss of function was particularly significant among miners with wheeze who remained in the industry over the period.

This study corroborates earlier findings that suggest that respiratory symptoms of chronic phlegm production, wheeze and breathlessness are valid indicators of changes in the respiratory tract which also cause airways obstruction. There was no evidence here that cough alone was indicative of obstructive changes, however.

Earlier studies of coal miners have addressed primarily long term miners exposed to dust concentrations much higher than the current cohort. The evidence presented here suggests that even exposure levels generally below 2 mg/m³ may be related to increased incidence of respiratory symptoms, as well as airways changes resulting in decrements in pulmonary function. Only among the subgroup of miners with wheeze at R2 was a dust-related effect on changes in lung function between R2 and R4 observed.

The levels of dust exposure experienced by this cohort were almost certainly substantially lower than previously-studied coal mining cohorts, the precise levels of exposure are debatable. The dust exposure estimates were developed from an unusually large set of quantitative measurements, however they were collected under a regulatory compliance program. As a result of this context, numerous problems with validity have been raised. In particular, recent revelations about submission of fraudulent samples to the regulatory agency suggests that exposures may have been substantially higher than those reported (BNA, 1991; Weeks, 1992). In the process of developing the exposure estimates, account was taken for the observed over-representation of low concentration samples in mine-operator collected data (Seixas et al, 1990). Nevertheless, the actual concentrations present in the mines must be taken only as estimates. Furthermore, in considering the potential effect of such biases in the analyses presented here, it is likely that errors in the exposure estimates would be relatively randomly distributed with respect to the outcomes considered. Such random distribution of errors would tend to bias the observed exposure-response relationships toward the null value, suggesting that the effects observed are under-estimates of the true dust effect in this population.

In a closely related cross-sectional analysis of R4 symptoms (Seixas, 1991) we observed statistically significant relationships between cumulative exposure and phlegm, breathlessness and wheeze whereas in the current analysis we only observed significant relationships for miners reporting phlegm. The primary difference between the two analyses was the sample size (1185 in the former and 977 in the current) and the procedures used to build the regression models. In the earlier paper, we included only variables that had p-values less than 0.2, while we forced all potentially significant variables into the current models. By forcing all potential co-variates into the model we may "over control" for some variables, however this procedure also ensures that all models presented are comparable. Nevertheless, the primary cause of the apparent discrepancy was the smaller sample size in the current analysis.

These findings are generally in concert with recent explanations of the "Dutch Hypothesis" that obstructive airways results from a combination of intrinsic factors in concert with extrinsic insults to the respiratory system (Sluiter, et al, 1991). Our results may be interpreted to suggest that dust exposure causes an inflammatory response in the lower airways that may be responsible for both the development of respiratory symptoms including phlegm production, wheeze and breathlessness and for obstruction of airflow. It also appears, however, that some individuals are more sensitive to these effects than others. In particular, it has been suggested that individuals with bronchial hyper-responsiveness may develop obstruction more rapidly than others. In our population, it was particularly miners who displayed wheeze at an early time point had the greatest decline in pulmonary function and it was only this group that had an exposure-related decline. Furthermore, those symptomatic miners who remained exposed to dust had the largest change among our sub-groups. Additional follow-up of this cohort will help elucidate the possible long-term implications of the early responses to dust exposure observed here.

References

- ATS (1979). Standardization of spirometry. (American Thoracic Society). Am Rev Respir Dis 119:831-838.
- ATS (1987). Standardization of spirometry - 1987 Update. (American Thoracic Society). Am Rev Respir Dis 136:1285-1298.
- Attfield MD (1985). Longitudinal decline in FEV1 in United States coalminers. Thorax 40:132-137.
- BNA, 1991. Companies agree to plead guilty to submitting false coal dust samples. Occupational Safety and Health Reporter, Bureau of National Affairs, Washington DC, Oct 23, 1991. p 573.
- Becklake,MR (1985). Chronic airflow limitation: its relationship to work in dusty occupations. Chest 88:608-614.
- Brinkman G, Block D, Cress C (1972). Effects of bronchitis and occupation on pulmonary ventilation over an 11-year period. J Occup Med 14:615-620.
- Brodkin, CA, Barnhart S, Anderson G, et al (1992). Correlation between respiratory symptoms and accelerated loss of ventilatory function in asbestos-exposed workers: A longitudinal study, presented at the 9th International Symposium on Epidemiology in Occupational Health. Cincinnati, OH, Sept. 23-25, 1992.
- Brodkin CA, Barnhart S, Anderson G, et al (1993). Correlation between respiratory symptoms and pulmonary function in asbestos-exposed workers. Am Rev Resp Dis, in press.
- Brodkin CA, L Rosenstock (1993). The relation between chronic respiratory symptoms and ventilatory capacity in adults. Spirometry: State of the Art Reviews, Occupational Medicine. pp363-374
- Crapo RO, Morris AH, Gardner RM (1981). Reference spirometric values for spirometry using techniques and equipment that meets ATS recommendations. Am Rev Respir Dis 123:659-664.
- Fletcher C, Peto R, Tinker C, Speizer FE. (1976). The natural history of chronic bronchitis and emphysema: An eight-year study of early chronic obstructive lung disease in working men in London. Oxford, Oxford University Press.
- Higgins MW, Keller J, Becher M, et al (1982). An index of risk for obstructive airways disease. Am Rev Respir Dis 125:144-151.
- Love RG, Miller BG (1982). Longitudinal study of lung function in coal-miners. Thorax 37:193-197.
- Rogan JM, Attfield MD, Jacobsen M, et al (1973). Role of dust in the working environment in development of chronic bronchitis in British coalminers. Br J Ind Med 30:217-226.
- Seixas NS, Moulton LH, Robins TG et al (1991). Estimation of cumulative exposures for the National Study of Coal Workers' Pneumoconiosis. App Occup Environ Hyg 6:1032-1041).

Seixas NS, Robins TG, Attfield MD, Moulton LH (1992). Exposure-response relationships for coal mine dust and obstructive lung disease following enactment of the Federal Coal Mine Health and Safety Act of 1969. *Am J Ind Med* 21:715-734.

Seixas NS, Robins TG, Attfield MD, Moulton LH (1993). Longitudinal and cross-sectional analyses of coal mine dust exposure and pulmonary function in new miners. *Brit J Ind Med*, (in press)

Seixas NS, Robins TG, Rice CH, Moulton LH (1990). Assessment of potential biases in the application of MSHA respirable coal mine dust data to an epidemiologic study. *Am Ind Hyg Assoc J* 51:531-540.

Sluiter HJ, Koeter GH, de Monchy JGR, et al (1991). The Dutch hypotheses (chronic non-specific lung disease) revisited. *Eur Respir J* 4:479-489.

Soutar, CA (1987). Occupational bronchitis. in *Recent Advances in Occupational Health* 3. Harrington M (ed.), Churchill Livingstone, London. pp285-302.

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

Weeks JL. (1991). Tampering with dust samples in coal mines - again. *Am J Ind Med* 20:141-144. PEnd

Variability of Particle Size-Specific Fractions of Personal Coal Mine Dust Exposures

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Abstract

Particle size-specific sampling may be used to increase the specificity of a measured exposure to a particular disease outcome being studied. Although respirable dust is a reasonable quantity to measure for the assessment of risk of pneumoconiosis among coal miners, the tracheo-bronchial or thoracic fraction may be a more appropriate quantity for addressing obstructive lung disease. The primary purpose of this study was to estimate the ratio of the tracheo-bronchial dust fraction to the fraction collected by a respirable dust sampler for a variety of job classifications found in conventional, continuous and longwall coal mining sections. The ratios could then be applied in epidemiologic studies to existing respirable dust measurements to estimate thoracic mass concentrations for evaluation of the relative importance of the respirable and thoracic dust fractions to obstructive lung disease. The data collected for this study include particle size distributions collected in four U.S. underground coal mines using 8-stage personal cascade impactors. A total of 180 valid samples were obtained and examined by mine, occupation and occupations grouped by proximity to the face, and by mining technology. From these particle size distributions several fractions were estimated: the fraction collected by the 10 mm nylon cyclone, the ACGIH respirable and thoracic particulate mass fractions and the estimated alveolar and tracheo-bronchial deposition fractions. These fractions were not significantly different when grouped by occupation, by proximity of work to the mine face, or by the type of mining technology in use. The distributions from one mine were somewhat different than the others, perhaps as a result of their use of diesel equipment in the haulage ways which contributed to the fine aerosol fractions. The results suggest that although the tracheo-bronchial dust fraction may contribute to the development of

obstructive lung disease, there is little to be gained in calculating occupation-specific tracheo-bronchial dust fractions since they are not likely to produce stronger exposure-response estimates for obstructive lung disease among coal miners than the historically-collected respirable dust concentrations.

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Introduction

The primary basis for dust exposure evaluation in underground coal mines is personal measurements of exposure to respirable coal mine dust.⁽¹⁾ The rationale for controlling exposure to respirable coal mine dust is the importance of Coal Workers' Pneumoconiosis (CWP) as a disabling lung disease.⁽²⁻⁴⁾ Because CWP is a disease of the deep lung tissues, control of dust reaching this anatomical region is likely to help prevent its occurrence. Respirable dust is generally defined as the fraction of dust capable of penetrating to the deep portions of the lung, where gas exchange takes place and beyond the reach of the muco-ciliary transport system.⁽⁵⁻⁷⁾ In Britain respirable dust is measured with a horizontal elutriator and in the U.S., with a 10 mm cyclone. The two instruments measure closely related dust fractions designed to approximate the dust penetrating to the gas exchange region.⁽⁸⁾ As a result, almost all of the historical exposure data available for coal mines is based on these measures of respirable dust exposure.

In recent years, several studies have demonstrated an association between dust exposure in coal mines and chronic obstructive pulmonary disease (COPD), including emphysema and chronic bronchitis.⁽⁹⁻¹²⁾ Because COPD may involve changes in the larger airways, respirable dust might be an inappropriate target for measurement and control. Rather, dust depositing in the airways (i.e., the tracheo-bronchial fraction) might be more suited to the evaluation of risk of bronchitis from coal dust exposure. Although the theoretical advantage of using this measure has been cited repeatedly in the epidemiologic literature concerning bronchitis in miners, no epidemiologic studies apply estimates of tracheo-bronchial dust to respiratory outcomes in coal miners owing to the lack of such historical

measurements.

Two studies in U.S. underground mines have observed substantial differences in particle size distributions between specific areas of mines and between longwall and continuous mining technologies (13, 14). On the basis of these observations, Potts et al (13) suggested that controlling respirable dust may be inadequate for control of hazards related to dust depositing in the thoracic region. The potential importance of these observations lead to this effort to better characterize the personal particle-size distributions of exposures received by underground coal miners, and if possible, to test the idea that tracheo-bronchial exposures might be important to the development of chronic obstructive lung disease in miners.

Since almost all historical exposure measurements have been made as respirable concentrations, a method to estimate job-specific tracheo-bronchial exposures from the respirable dust concentrations would be required to conduct such an investigation using the existing historical data. The current study was designed to determine the relationship between tracheo-bronchial and respirable dust exposures in coal miners. To accomplish this goal, particle size distributions in underground mining jobs at four coal mines were assessed so that the job-specific respirable and tracheo-bronchial dust fractions could be estimated and used to convert the historical respirable dust measurements into estimates of tracheo-bronchial dust exposures. The intention was to then examine the relationship between newly estimated tracheo-bronchial dust exposures and respiratory outcomes in coal miners in order to see whether predictive power was increased over models using only respirable dust estimates.

Materials and Methods

Surveys were undertaken at four bituminous mines in Western Pennsylvania and West Virginia that volunteered to participate. Three of the four (A, B and C) were in reasonable proximity to the National Institute for Occupational Safety and Health (NIOSH) laboratory in Morgantown and used both continuous and longwall mining technology. The fourth mine (D) utilized conventional drilling and blasting techniques. One of the mines (C) used diesel equipment for transporting personnel and equipment while the others used only electric machinery. Surveys were conducted at each site for three or four days. The occupations targeted for evaluation were those highly represented in the work history data of the epidemiologic cohort that was intended to be used for this project (12). Miners in the selected job titles, on both face jobs (in areas in which coal is actively being extracted) and non-face occupations (i.e., jobs away from the face including transportation and mine maintenance activities) were identified before entering the mine. For non-face occupations, in which the total dust concentrations were expected to be relatively low, miners donned the sampler at the portal and returned with it at the end of the day. For face occupations, in which dust concentrations are higher and sampler overloading could occur, the samplers were carried into the mine by the investigators, donned by the miners and worn for a variable period, depending on the expected dust loading.

Particulate samples were collected with an 8-stage personal cascade impactor (Sierra Instruments; henceforth designated M298) using a flow rate of 2 liters per minute (Lpm).⁽¹⁵⁾ The nominal cut-off diameters and sampler efficiency correction factors for the stages are given in Table 1. These values are refinements of the

manufacturer's suggested numbers, as developed by Rader et al.⁽¹⁶⁾ The lower cutoff for the filter (0.26 μm) was adopted by convention as one half the lower cut point for the last stage.⁽¹⁷⁾ The upper cutoff for the first stage (42 μm) was chosen because the inlet efficiency curve for the impactor approaches zero at this point.⁽¹⁶⁾ The inhalation (penetration or deposition) factors for each stage given in Table 1 were calculated by dividing the stage interval, expressed in log units, into 20 equal segments, calculating the expected fraction at each endpoint of each segment (using the functions that define each of the inhalation fraction definitions), integrating across the stage interval width with the trapezoidal rule, and then dividing by the interval width.

Each impaction plate held a Mylar substrate which was prepared using a spray application of Apiezon grease (Apiezon Products Limited, London), equilibrated for at least twenty-four hours to control for weight changes due to off-gassing, and then weighed. After sampling, the impactors were disassembled and the impaction plates reweighed. Any sample with visibly loose dust was rejected on the basis of unknown particulate loss. Further, any sample with weight losses greater than 0.02 mg on any one stage was rejected.

The filter weight change was first corrected for internal losses by dividing the observed weight change by the correction factors given in Table 1. The size-specific mass fractions were calculated by multiplying the mass fraction on each stage times the appropriate factor specified in Table 1 (for each type of dust fraction) and summing across the eight impaction stages and the final filter:⁽¹⁷⁾

$$\text{MF}(X)_j = \sum_{i=1}^9 \text{MF}(X)_i \times \text{MF}_{ij}$$

where $MF(X)_j$ is the mass fraction according to size definition X measured on sample j, $MF(X)_i$ is the factor for stage i (see Table I) calculated using one of the size-specific definitions described below, and MF_{ij} is the efficiency corrected mass fraction measured on stage i for sample j.

The penetration and deposition factors calculated include:

10 mm Nylon Cyclone (Cyc): This quantity estimates the dust fraction actually sampled by the cyclone operated at 2.0 Lpm.⁽¹⁸⁾ This apparatus and flow rate is mandated by the MSHA sampling program⁽¹⁾ and is the basis of the respirable dust estimates used for previous analyses.⁽¹⁹⁾ Those data represent the concentration of dust measured by the cyclone and multiplied by 1.38 to convert to an British Medical Research Council (MRE) equivalent concentration.

ACGIH Respirable (RPM) and Thoracic (TPM) Penetration: These factors estimate the fractions of airborne dust on each impactor stage that will penetrate to, but not necessarily deposit in, the gas exchange region (respirable) and beyond the larynx (thoracic). They are defined by the American Conference of Governmental Hygienists as a cumulative lognormal curve with geometric mean of 3.5 (respirable) or 10 (thoracic) microns and standard deviations of 1.5.⁽⁶⁾ Changes in these definitions were adopted in 1993 to bring the American and European definitions into concordance (20). The most significant change in the new definitions is that the 50% cut diameter for the respirable dust fraction is

4.0 microns instead of 3.5 as used in this analysis and historically for analyses in the US.

Stahlhofen Alveolar (DE_a), Tracheobronchial (DE_b) and Thoracic (DE_{ba})

Deposition: These factors estimate the fraction of inhaled dust that will penetrate to and deposit in the gas exchange (alveolar), airways (tracheobronchial) and thoracic (sum of tracheobronchial and alveolar) regions using a lung deposition model reported by Stahlhofen.⁽⁵⁾ The Stahlhofen factors given in Table 1 represent the stage-specific fractions according to the models multiplied by the fraction of total particulate which is inhalable according to the ACGIH inhalable (formerly inspirable) particulate curve.⁽⁶⁾ This step was necessary only for the Stahlhofen deposition fractions since they are defined on the basis of inhalable particulate rather than total airborne particulate.

In order to convert the historically collected respirable exposure data (collected with the cyclone) to estimates of historical thoracic or tracheobronchial dust exposures, the ratios of the ACGIH and Stahlhofen mass fractions to the fraction represented by the cyclone were calculated. Only the mass fraction that would be collected by a cyclone (Cyc) is given in Tables II through IV. The mass fractions by the ACGIH or Stahlhofen definitions can be easily derived by multiplying the cyclone mass fraction by the ratio given in the tables.

After calculation of the mass fractions and their ratios, the data were compared by stratifying by mine and occupation. Both individual occupations and grouped occupations were considered. Occupation groups were defined by proximity to the

face (face and non-face jobs) and by mining technology (jobs specific to continuous, longwall and conventional mining technologies). Histogram particle size distributions averaged across the various strata were constructed using a log scale for the aerodynamic diameter and the mass fraction divided by the log interval of each stage for the bar heights.⁽¹⁷⁾ Analysis of variance was used to further consider the significance of mine and occupation as determinants of the deposition or penetration parameters.

Results

Of the 208 samples collected, a total of 28 were rejected because of overloading or negative weights, yielding a total of 180 samples available for analysis. Of these, 142 were on face occupations and 38 in non-face jobs. The calculated cyclone-collected mass fraction and ratios of the other fractions (specified above) to the cyclone fraction are given in Table II. As would be expected, the ratio of the mass fraction representing the ACGIH respirable dust definition to the mass fraction collected by the cyclone was very close to unity. The comparable ratio for the Stahlhofen respirable fraction ratio was lower, as a result of the low deposition predicted at very small particle sizes compared to the fraction collected by the cyclone. The tracheo-bronchial deposition fractions were greater than the alveolar fractions in all cases.

No substantial differences between face and non-face distributions (Fig 1), or their mass fraction ratios (Table II) were observed. Grouping of face occupations by the type of coal mining technology used (Table II and Fig. 2), indicated little variation in the size distribution parameters or calculated mass fractions between technologies.

When the data were further stratified by mine (Table III and Fig. 3), differences were observed. In particular, larger proportions of the dust from Mine C were observed at lower particle sizes, yielding consistently higher cyclone mass fractions and lower ratios on both face and non-face jobs compared to the other mines. However, there was no consistent pattern between face or non-face jobs across mines. For instance, the ACGIH thoracic to cyclone ratio (RPM/Cyc) was higher for face jobs in Mine A and lower for face jobs in Mines B and C. Distributions for a few selected face and non-face occupations for which there were at least 10 samples (Table IV) show little variability.

As a check on the degree of variability among occupations and mines, analysis of variance models were constructed using the ratio of the Stahlhofen tracheo-bronchial fraction to the cyclone fraction (DE_b/Cyc) as the dependent variable and mine and occupation, or occupation groups, as the independent variables. No significant differences were observed between occupations or occupations grouped by proximity to the face or by mining technology. The models consistently indicated a difference between Mine C and the other three mines but not among Mines A, B and D.

Discussion

A useful exposure assessment for occupational epidemiology or risk evaluation requires that the measured quantity is an accurate predictor of the relevant dose to the target organ for the specific outcome being considered.⁽²¹⁾ For the case of particulate exposures and respiratory disease, particle size-specific sampling should help make the measured quantity specific to the underlying disease process being

studied.⁽²²⁾ Respirable dust exposure estimates for the epidemiology of diseases of the lung, especially the pneumoconioses, have worked well. However, for diseases of the airways, especially chronic bronchitis, it would appear that the fraction of dust depositing in the large airways would be a more specific measure.

Because the existing historical data for exposure assessment in coal mines is based on respirable dust fractions, a research program to assess the impact of tracheo-bronchial dust fractions on disease would require the conversion of the respirable dust concentrations to tracheo-bronchial concentrations. This conversion would be conducted using the ratio of the tracheo-bronchial to cyclone fraction by specific identifiable categories of mine work. Note that if using the existing MSHA exposure data, one would first divide the MSHA concentrations (or the ratios) by 1.38 to obtain the specified fraction concentrations, accounting for the conversion of the cyclone-collected dust to the MRE-equivalent respirable dust concentration⁽¹⁾.

However, in order for conversion of respirable dust concentrations to tracheo-bronchial dust concentrations to be achievable and worthwhile, three conditions must hold. First, there would have to be significant variation in the distributions across specific occupations, or alternatively, groups of occupations assigned on the basis of their proximity to different dust generating activities or control technologies. In this analysis, groups were formed on an a priori basis to examine differences between face vs. non-face occupations, and between face occupations that were specific to mining technologies. The current data showed no significant differences in the ratio of cyclone to tracheobronchial deposition fractions for specific occupations or the selected occupation groups. As a result, these data do not support the ability to distinguish these fractions between categories of mining jobs.

Second, there would have to be no significant differences in the distributions between mines, while adjusting for the technology, occupation or other easily measured parameters. Specifically, the variability in the ratio would have to be greater between job, location or mining method than between mines. The data presented here suggest that some significant differences occur between mines with significantly smaller aerodynamic diameter particulate found in Mine C as compared to the other three mines. It is suspected that this difference may be due to the use of diesel equipment to haul personnel and supplies to the working sections in Mine C. However, because only four mines were included in this sample, it would be premature to suggest modeling the distributions on the basis of use of diesel equipment. A much larger study comparing diesel to electric equipment mines would be required to allow such a conclusion.

Finally, particle size distributions would have to be invariant across time. Because historical particulate distribution data are unavailable, it is impossible to determine this with any certainty. However, it appears that this may be a reasonable assumption given the relative lack of change of technology (within continuous, longwall or conventional techniques) over the past 20 years.

In summary, the results indicate that it is not possible to distinguish the particle size distributions between specific occupations, or groups of occupations and it appears that the variability in distributions may be greater between mines than between occupations. The cause of this cross-mine variability cannot be assessed with the current limited data. Therefore, even assuming that the particle-size distributions were invariant over time, it does not appear possible to historically reconstruct

particle size-specific distributions for a large cohort of miners including work in multiple mines without extremely extensive particulate distribution sampling in these mines. It should be noted however, that because conversion of historically-collected respirable dust measurements to estimates of thoracic exposures is infeasible, this does not mean that measurement of thoracic fraction exposures is equivalent to respirable measurements and is without merit.

These findings appear to be in contrast to earlier reports of particle-size distributions in mines.^(13, 14, 23, 24) To a variable extent, these earlier reports have found different particle size distributions in longwall compared to continuous mining and between locations in different proximity to the mining machines within a particular technology. However, most of these reports are limited by the use of static (area) samplers rather than the personal samplers used in the current research.

Potts, et al found ratios of thoracic to respirable dust (using ACGIH definitions) ranging from 1.8 in the intake airway to 6.7 downwind of support movement in longwall operations and 1.5 in the intake airway to 4.1 on a continuous mining machine.⁽¹³⁾ He concluded that control of respirable dust would not equally limit the exposures of miners to thoracic fraction dust. The measurements were made by sampling at locations defined by their proximity to the dust generating machinery. Particle size distributions reported for longwall shearer and downwind of roof support movement (his Figure 3), and for a continuous mine roof bolter (his Figure 6) are in general agreement with the distributions we observed. The extreme values obtained by Potts, in intake air (small particle sizes) and downwind of support movement (large particle sizes), do not reflect personal exposures. It is not surprising that intake air, being made up primarily of suspended fine particles, had a

very low thoracic to respirable ratio. However, when a miner works in intake air, he would be expected to reentrain settled dust by his activities and thus raise the thoracic to respirable ratio. Dust sampled just downwind of support movement would similarly be unlikely to reflect true exposure distributions since miners would only spend a fraction of their time in this location.

Burkhardt et al presented particle size distributions for a continuous miner, on a haulage road and at the feeder/breaker (his Figures 3, 4 and 5, respectively) which were very similar to those observed here⁽¹⁴⁾. Rubow and Marple examined underground coal dust particle size distributions using the Microorifice uniform deposit impactor (MOUDI)⁽²³⁾. In their study, the samplers were located several blocks distant from the coal cutting operations (a block is a unit of coal cutting operations and is generally about 40 feet long). As a result of the sampler locations, the larger particles would have had sufficient time to settle before reaching the sampler and the particle size distributions are therefore probably unrepresentative of personal exposures to mining equipment operators.

Marks, et al, developed particle-size fraction estimates using an improved personal cascade impactor, the Personal Inspirable Dust Spectrometer (PIDS), and compared inspirable dust concentrations with respirable and tracheo-bronchial sub-fraction concentrations in three British coal mines.⁽²⁴⁾ This study found a high correlation between the various sub-fraction concentrations. However the relationships between the fractions (e.g., respirable to inspirable ratios) varied across job groups. Nevertheless, on the basis of the high correlations between respirable and other particulate sub-fractions of dust exposures, the report concluded that respirable dust estimates should be equally predictive of obstructive lung disease as other fractions.

It is not possible to directly compare the current results to those of Mark, et al, since his were reported as the ratio of the various sub-fractions to inspirable mass fraction instead of the cyclone-collected fraction, as provided here. Since our data was obtained with the M298 which has poorly defined inlet characteristics and high internal losses for large particles sizes, it is not possible to calculate the inspirable fraction from the current data. However, an approximate thoracic to respirable fraction ratio can be derived from his data (as reported in his Table 20, page 96). The results suggest that there is little difference between these fractions across the job groups considered (range 2.0 to 3.4) and support our observation that there is little substantial difference in the particle size distributions between coal mining job categories, at least in the thoracic and respirable particle size range.

Despite the fact that miners are assigned relatively specific occupations, they frequently trade-off jobs or help each other conduct specific tasks. For instance, the continuous miner operator and helper generally share the tasks of operating the machinery and doing the support work for about equal time. Utility or general laborers frequently help operate machinery when needed. Thus, these findings which show little variation in particle size distributions across jobs, may be a result of the averaging of activity-specific size distributions across various types of activities in which miners are engaged over the work day.

In this analysis samples collected both historically and for this analysis have been referred to as "personal." This generally means that the sampling apparatus was worn by the individual during the whole work shift. For certain occupations, most importantly the continuous miner operator, MSHA defines a personal sample as

being placed on the machine within 36 inches inby (toward the coal cutting face) the operator.⁽¹⁾ When not operating the machinery, or for miners in more mobile occupations, the sampler would be worn by the miner. Therefore the samples were not all strictly "personal," but would be highly representative of the individual miner's dust distribution.

The definitions for ACGIH respirable and thoracic dust fractions used in this analysis refer to the long-standing definitions which have recently been changed.⁽²⁰⁾ The most significant change in definitions was the increase in the 50% collection efficiency size for respirable aerosols from 3.5 to 4.0 microns. This change would make relatively little difference to most measured concentrations⁽²⁵⁾, and would make almost no difference in the ability to distinguish the ratios of various fractions as was done for this analysis. Nevertheless, for future analyses, the authors support the change to the new international definitions.

The particle-size distributions measured by the M298 sampler may not accurately reflect the inhaled dust as a result of the poor inlet collection efficiency and internal losses prior to entering the orifices of the upper stage. The inlet efficiency of the M298 falls off rapidly for particle sizes greater than about 15 μm .^(15, 16) The accuracy of the correction factors used for the first two stages are therefore particularly crucial for estimating the true particle size distribution. While this problem makes the observed distributions inaccurate, and the calculation of the mean aerodynamic diameter and geometric standard deviation impossible, it should not greatly affect calculation of ratios of respirable or tracheo-bronchial mass fractions to cyclone mass fractions. Less than one percent of the dust deposited on the first stage and about eight percent deposited on the second contribute to the ACGIH thoracic fraction.

The percentages for the other fractions on the first two stages are even lower. Thus, large diameter dust makes very little contribution to these calculated fractions.

These results indicate that, while there are some differences across the four mines sampled, it is not possible to distinguish mining occupations on the basis of their particle size distributions measured with personal impactors. Therefore, unless these four mines were to prove to be quite unrepresentative, we conclude that occupation-specific tracheo-bronchial dust exposures would be highly correlated with historically collected respirable exposures and exposure-response analyses using the two measures would be similar.

References

1. Mine Safety and Health Administration: Mineral Resources. In: U.S. Code of Regulations, Title 30 Part 70 Subpart C - Sampling Procedures, 446-451 (1985).
2. Jacobsen, M.: The Relation Between Pneumoconiosis and Dust-Exposure in British Coal Mines. In Inhaled Particles III. pp 903-919, Walton, W.H., Ed. Old Woking, Surrey. (1971)
3. Attfield, M.D.; Morring, K.: An Investigation Into The Relationship Between Coal Workers' Pneumoconiosis and Dust Exposure In US Coal Miners. Am. Ind. Hyg. Assoc. J. 53:486-492 (1992).
4. Attfield, M.D.; Seixas, N.S.: Prevalence Of Pneumoconiosis and Its Relationship To Dust Exposure In A Cohort Of US Coal Miners And Ex-Miners. in preparation (1992).} **not yet released by NIOSH for citation**
5. Stahlhofen, W.; Rudolf, G.; James, A.C.: Intercomparison of Experimental Aerosol Deposition Data. J. Aerosol. Med. 2:285-308 (1989).
6. American Conference of Governmental Industrial Hygienists: Particle Size-Selective Sampling in the Workplace. Report of the ACGIH Technical Committee on Air Sampling Procedures. American Conference of Governmental Industrial Hygienists. Cincinnati (1985).
7. Lippman M.: Size-Selective Health Hazard Sampling. In: Air Sampling

Instruments for Evaluation of Atmospheric Contaminants, pp. 163-198. S.V. Hering, Ed. American Conference of Governmental Industrial Hygienists, Cincinnati, OH (1989).

8. Treaftis, H.N.; Gero, A.J.; Kacsmar, P.M.; Tomb, T.F.: Comparison Of Mass Concentrations Determined With Personal Respirable Coal Mine Dust Samplers Operating At 1.2 Liters Per Minute And The Casella 113A Gravimetric Sampler (MRE). *Am. Indus. Hyg. Assoc. J.* 45:826-832 (1984).
9. Rogan, J.M.; Attfield, M.D.; Jacobsen, M.; et al.: Role Of Dust In The Working Environment In Development Of Chronic Bronchitis In British Coalminers. *Br. J. Indus. Med.* 30:217-226 (1973).
10. Soutar, C.A.; Hurley, J.F.: Relation Between Dust Exposure And Lung Function In Miners And Ex-Miners. *Br. J. Indus. Med.* 43:307-320 (1986).
11. Marine, W.M.; Gurr, D.; Jacobsen, M.: Clinically Important Respiratory Effects Of Dust Exposure And Smoking In British Coal Miners. *Am. Rev. Respir. Dis.* 137:106-112 (1988).
12. Seixas, N.S.; Robins, T.G.; Attfield, M.D.; Moulton, L.H.: Exposure-Response Relationships For Coal Mine Dust And Obstructive Lung Disease Following Enactment Of The Federal coal Mine Health And Safety Act Of 1969. *Am. J. Indus. Med.* 21:715-734 (1992).
13. Potts, J.W.; McCawley, M.A.; Jankowski, R.A.: Thoracic Dust Exposures On

Longwall And Continuous Mining Sections. Appl. Occup. Environ. Hyg. 5:440-447 (1990).

14. Burkhardt J.E.; McCawley, M.A.; Wheeler, R.W.: Particle Size Distributions in Underground Coal Mines. Am. Indus. Hyg. Assoc. J. 48:122-126 (1987).

15. Rubow, K.L.; Marple, V.A.; Olin, J.; McCawley, M.A.: A Personal Cascade Impactor: Design, Evaluation And Calibration. Am. Indus. Hyg. Assoc. J. 48:532-538 (1987).

16. Rader, D.J.; Mondy, L.A.; Brockmann, J.E.; et al.: Stage Response Calibration Of The Mark III And Marple Personal Cascade Impactors. Aerosol Sci Technol 14:365-379 (1991).

17. Hinds, W.C.: Data Analysis. in Lodge, J.P.; Chan, T.G.: Eds. Cascade Impactor Sampling and Data Analysis. pp. 45-78. American Industrial Hygiene Association, Akron, OH. (1986).

18. Caplan, K.J.; Doemeny, L.J.; Sorenson, S.D.: Performance Characteristics Of The 10 mm Cyclone Respirable Mass Sampler: Part 1 - Monodisperse Studies. Am. Indus. Hyg. Assoc. J. 38:83-95 (1977).

19. Seixas, N.S.; Moulton, L.H.; Robins, T.G.; et. al.: Estimation Of Cumulative Exposures For The National Study Of Coal Workers' Pneumoconiosis. Appl. Occup. Environ. Hyg. 6:1032-1041 (1991).

20. American Conference of Governmental Hygienists, Annual Reports of the Committees on Threshold Limit Values and Biological Exposure Indices. May, 1993. ACGIH, Akron Ohio, (1993)
21. Smith, T.J.: Exposure Assessment For Occupational Epidemiology. Am. J. Indus. Med. 12:249-268 1987.
22. Vincent, J.H.; Mark, D.: The Measurement Of Aerosols In Relation To Risk Assessment. In: Rappaport, S.M.; Smith, T.J.: Eds. Exposure Assessment For Epidemiology And Hazard Control. pp 41-52. Lewis, Chelsea (1991).
23. Rubow, K.L.; Marple, V.A.: Determining The Size Distribution Of Coal/Diesel Aerosol Mixtures With The Microorifice Uniform Deposit Impactor. In: Respirable Dust In The Mineral Industries. (1988).
24. Mark, D.; Cowie, H.; Vincent, J.H.; et. al.: The Variability Of Exposure Of Coalminers To Inspirable Dust. Institute of Occupational Medicine Report No. TM/88/02, Edinburgh, U.K. (1988).
25. Solderhom S.C.: Why Change ACGIH's Definition of Respirable Dust?. Appl. Occup. Environ. Hyg. 6:248-250 (1991).

Table I. Marple 298 Personal Impactor Characteristics With Particle Size Fraction Definitions

	Stage								
	1	2	3	4	5	6	7	8	Filter
Cut Point (microns)	21.1	15.0	9.8	6.0	3.5	1.54	0.91	0.53	<0.53
Correction Factor ^a	0.26	0.70	0.82	0.90	0.94	0.97	0.98	0.99	0.99
Mass Fractions^b									
Cyclone (Cyc)	0.000	0.000	0.000	0.002	0.180	0.921	1.000	1.000	1.000
ACGIH Respirable (RPM)	0.000	0.000	0.002	0.034	0.268	0.807	0.994	1.000	1.000
ACGIH Thoracic (TPM)	0.008	0.084	0.325	0.731	0.964	0.999	1.000	1.000	1.000
Alveolar Deposition (DE _a)	0.000	0.000	0.003	0.064	0.286	0.393	0.277	0.179	0.130
TB Deposition (DE _b)	0.006	0.031	0.110	0.285	0.314	0.138	0.040	0.020	0.022
Alv. + TB Deposition (DE _{ba})	0.006	0.031	0.113	0.349	0.600	0.531	0.317	0.199	0.152

^a Calculated using the inlet efficiency and internal loss functions developed by Rader et al (16).

^b Fraction definitions are: Cyc, mass fraction collected by the 10 mm nylon cyclone at 2.0 Lpm; RPM, respirable particulate mass by ACGIH definition; TPM, thoracic particulate mass by ACGIH definition; DE_a, alveolar deposition fraction; DE_b, tracheo-bronchial deposition fraction; DE_{ba}, alveolar plus tracheo-bronchial deposition fraction. See text for further explanation.

Table II. Average (Standard Deviation) Cyclone Mass Fractions and Dust Fraction Ratios For All Samples and By Proximity To The Face And Mining Method

	n	Cyc^a	RPM/Cyc^b	TPM/Cyc^b	DE_a/Cyc^b	DE_b/Cyc^b	DE_{ba}/Cyc^b
All	180	0.07 ±0.04	1.07 ±0.06	3.84 ±1.46	0.63 ±0.17	1.18 ±0.58	1.81 ±0.73
<u>By Location</u>							
Face	145	0.07 ±0.04	1.06 ±0.05	3.84 ±1.33	0.63 ±0.15	1.18 ±0.53	1.81 ±0.67
Non-Face	35	0.07 ±0.04	1.07 ±0.08	3.84 ±1.93	0.62 ±0.22	1.18 ±0.75	1.80 ±0.96
<u>By Mining Method</u>							
Continuous	54	0.07 ±0.05	1.07 ±0.05	3.90 ±1.55	0.63 ±0.18	1.21 ±0.62	1.83 ±0.79
Longwall	48	0.06 ±0.03	1.06 ±0.04	3.86 ±1.19	0.64 ±0.13	1.19 ±0.47	1.84 ±0.58
Conventional	19	0.08 ±0.04	1.06 ±0.05	3.62 ±1.02	0.60 ±0.11	1.09 ±0.40	1.69 ±0.50

a Mass fraction collected by the 10 mm nylon cyclone at 2.0 lpm. See text for further definition.

b Ratio of the mass fraction according to the specified definition to the mass fraction collected by the cyclone. Fraction definitions are: RPM, respirable particulate mass by ACGIH definition; TPM, thoracic particulate mass by ACGIH definition; DE_a, alveolar deposition fraction; DE_b, tracheo-bronchial deposition fraction; DE_{ba}, alveolar plus tracheo-bronchial deposition fraction. (See text for further explanation).

c The number of samples by mining method do not sum to 180 because some occupations sampled are not unique to a particular method.

Table III. Average (Standard Deviation) Cyclone Mass Fractions and Dust Fraction Ratios by Mine and Location

	n	Cyc^a	RPM/Cyc^b	TPM/Cyc^b	DE_a/Cyc^b	DE_b/Cyc^b	DE_{ba}/Cyc^b
<u>Mine A</u>							
Face	41	0.06 ±0.03	1.07 ±0.05	4.04 ±1.05	0.68 ±0.12	1.26 ±0.41	1.94 ±0.52
Non-Face	9	0.05 ±0.03	1.14 ±0.12	5.61 ±2.31	0.83 ±0.20	1.86 ±0.87	2.66 ±1.03
<u>Mine B</u>							
Face	43	0.04 ±0.02	1.09 ±0.05	4.76 ±1.46	0.72 ±0.15	1.55 ±0.58	2.28 ±0.72
Non-Face	13	0.06 ±0.02	1.07 ±0.06	4.15 ±1.39	0.69 ±0.17	1.31 ±0.56	2.01 ±0.74
<u>Mine C</u>							
Face	42	0.09 ±0.05	1.03 ±0.03	2.81 ±0.64	0.50 ±0.10	0.77 ±0.26	1.27 ±0.36
Non-Face	13	0.10 ±0.03	1.02 ±0.01	2.32 ±0.40	0.41 ±0.07	0.57 ±0.17	0.99 ±0.23
<u>Mine D</u>							
Face	19	0.08 ±0.04	1.06 ±0.05	3.62 ±1.02	0.60 ±0.11	1.09 ±0.40	1.69 ±0.50

a Mass fraction collected by the 10 mm nylon cyclone at 2.0 lpm. See text for further definition.

b Ratio of the mass fraction according to the specified definition to the mass fraction collected by the cyclone. Fraction definitions are: RPM, respirable particulate mass by ACGIH definition; TPM, thoracic particulate mass by ACGIH definition; DE_a, alveolar deposition fraction; DE_b, tracheo-bronchial deposition fraction; DE_{ba}, alveolar plus tracheo-bronchial deposition fraction. (See text for further explanation).

Table IV. Average (Standard Deviation) Cyclone Mass Fractions and Dust Fraction Ratios For Selected Occupations

	<u>n</u>	<u>Cyc</u> ^a	<u>RPM/Cyc</u> ^b	<u>TPM/Cyc</u> ^b	<u>DE_a/Cyc</u> ^b	<u>DE_b/Cyc</u> ^b	<u>DE_{ba}/Cyc</u> ^b
Continuous Miner Operator	22	0.07 ±0.05	1.07 ±0.05	3.86 ±1.16	0.64 ±0.15	1.19 ±0.46	1.84 ±0.61
Roof Bolter	22	0.06 ±0.04	1.07 ±0.06	3.97 ±1.73	0.63 ±0.19	1.23 ±0.69	1.86 ±0.87
Long Wall Jacksetter	16	0.06 ±0.03	1.06 ±0.04	3.82 ±1.23	0.63 ±0.13	1.17 ±0.48	1.80 ±0.61
Long Wall Operator	28	0.06 ±0.02	1.06 ±0.04	3.77 ±0.85	0.65 ±0.11	1.16 ±0.34	1.81 ±0.43
Beltman	10	0.06 ±0.02	1.07 ±0.04	3.99 ±1.09	0.67 ±0.15	1.24 ±0.43	1.90 ±0.56

a Mass fraction collected by the 10 mm nylon cyclone at 2.0 lpm. See text for further definition.

b Ratio of the mass fraction according to the specified definition to the mass fraction collected by the cyclone. Fraction definitions are: RPM, respirable particulate mass by ACGIH definition; TPM, thoracic particulate mass by ACGIH definition; DE_a, alveolar deposition fraction; DE_b, tracheo-bronchial deposition fraction; DE_{ba}, alveolar plus tracheo-bronchial deposition fraction. (See text for further explanation).

Figure Captions

Figure 1. Histograms representing the average particle-size distribution for face (n=145) and non-face (n=35) personal cascade samples.

Figure 2. Histograms representing the average particle-size distribution for face occupations specific to Continuous (n=52), Longwall (n=48) and Conventional (n=19) mining technologies.

Figure 3. Histograms representing the average particle-size distribution for face occupations only from Mine A (n=41), Mine B (n=43), Mine C (n=42) and Mine D (n=19).

Figure 1

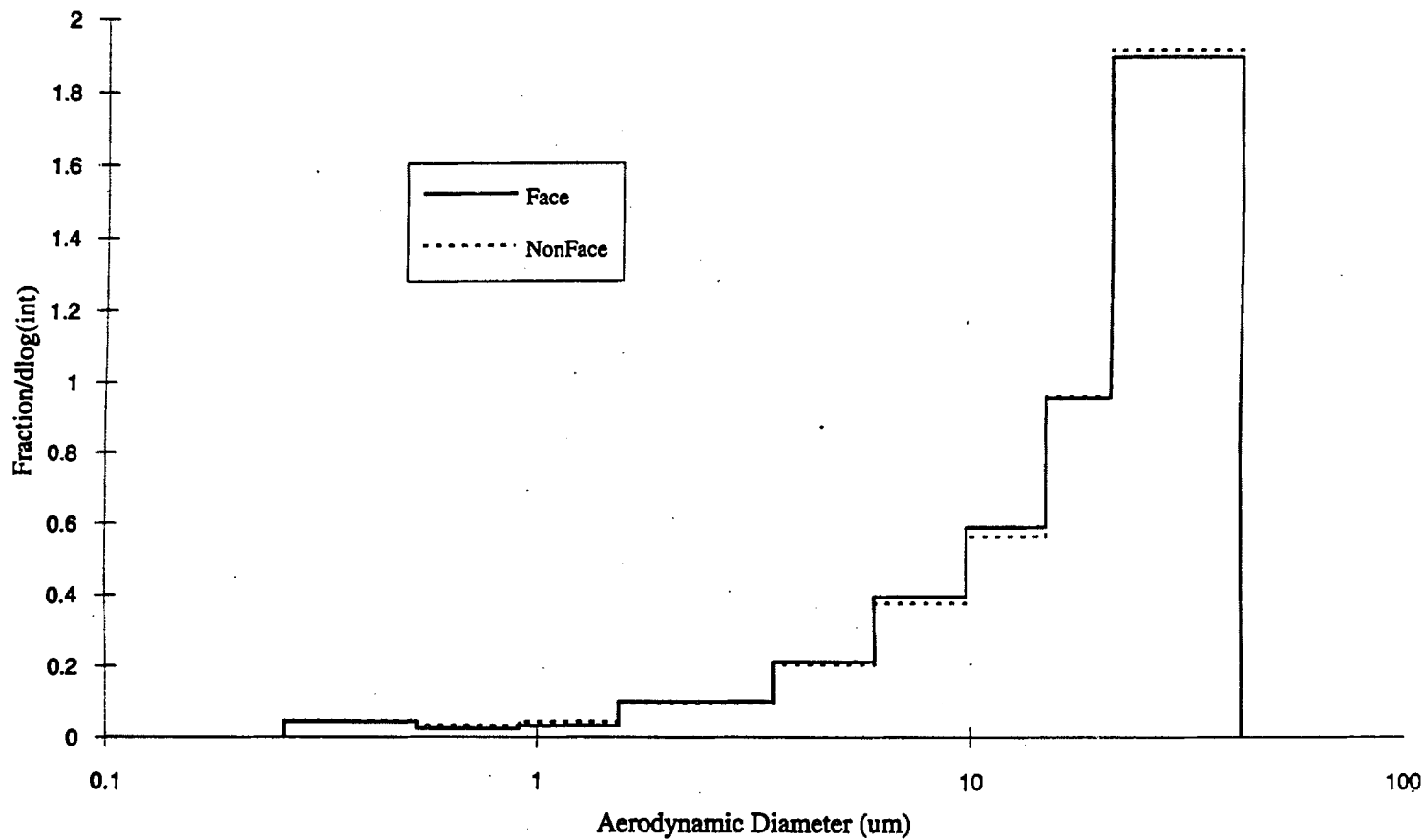


Figure 2

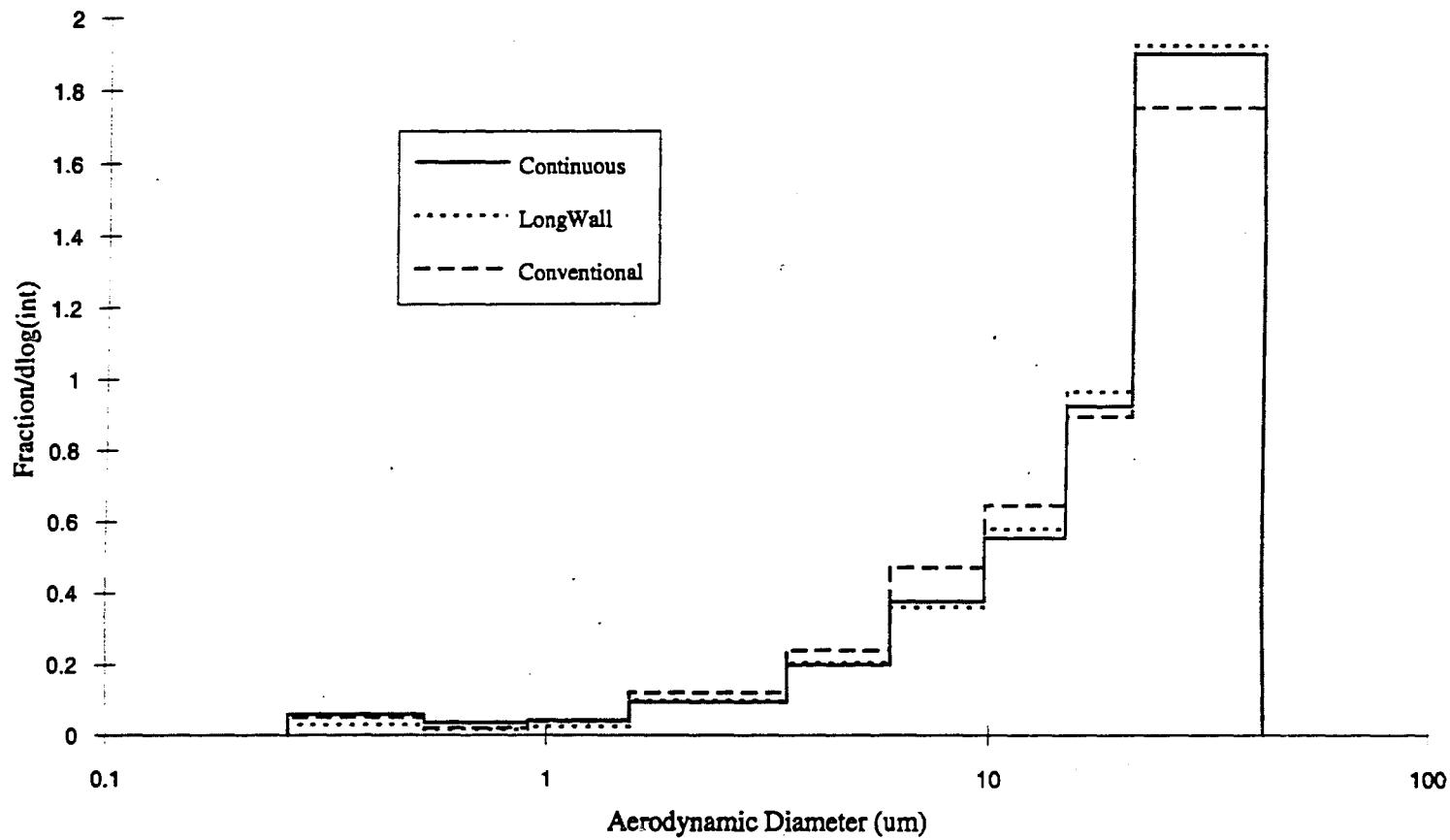


Figure 3

