

Coal Dust Exposure and Mortality From Ischemic Heart Disease Among a Cohort of U.S. Coal Miners

Deborah D. Landen, MD, PhD,^{1*} James T. Wassell, PhD,² Linda McWilliams, MS,¹
and Ami Patel, PhD, MPH³

Background Particulate exposure from air pollution increases the risk of ischemic heart disease (IHD) mortality. Although coal miners are highly exposed to coal dust particulate, studies of IHD mortality risk among coal miners have had inconsistent results. Previous studies may have been biased by the healthy worker effect.

Methods We examined the dose–response relationship between cumulative coal dust exposure, coal rank, and IHD mortality among a cohort of underground coal miners who participated in the National Study of Coal Workers' Pneumoconiosis.

Results After adjusting for age, smoking, and body mass index, risk of IHD mortality increased at higher levels of coal dust exposure. Mortality risk was also associated with coal rank region.

Conclusion There was an increased risk of mortality from IHD associated with cumulative exposure to coal dust, and with coal rank. The effect of coal rank may be due differences in the composition of coal mine dust particulate. The association of risk of IHD mortality with cumulative particulate exposure is consistent with air pollution studies. *Am. J. Ind. Med.* © 2011 Wiley-Liss, Inc.

KEY WORDS: coal dust; ischemic heart disease; mortality

INTRODUCTION

Particulate matter in air pollution is known to increase the risk of cardiovascular morbidity and mortality [Dockery et al., 1993; Schwartz, 1999, 2001; Laden et al., 2000; Pope et al., 2004; Vermylen et al., 2005; Ostro et al., 2006]. Although coal miners are exposed to high concentrations of particulate from coal mine dust, studies which

have examined the risk of ischemic heart disease (IHD) mortality among coal miners have had inconsistent results. Some have shown a risk no different, or lower than that of the general population [Rockette, 1977; Christie et al., 1995; Une et al., 1995], while others have shown increased risk, either overall, or among subgroups [Liddell, 1973; Costello et al., 1975; Atuhaire et al., 1986; Meijers et al., 1997; Attfield and Kuempel, 2008; Miller and MacCalman, 2010].

Most previously published studies of IHD among coal miners have used Standardized Mortality Ratios to compare their risk to that of general populations. These studies may have been biased by the healthy worker effect, since employed populations often experience lower mortality than the general population [Fox and Collier, 1976]. Bias from the healthy worker effect can be reduced by the use of study designs which use internal comparison groups [Gilbert, 1982]. A recent study of a large cohort of British coal miners [Miller and MacCalman, 2010] used Cox's proportional hazards model to examine the risk of coal

¹Office of Mine Safety and Health Research, National Institute for Occupational Safety and Health, Pittsburgh, Pennsylvania

²Division of Safety Research, National Institute for Occupational Safety and Health, Morgantown, West Virginia

³Office of Public Health Preparedness and Response, Centers for Disease Control and Prevention, Philadelphia, Pennsylvania

*Correspondence to: Deborah D. Landen, MD, PhD, Office of Mine Safety and Health Research, National Institute for Occupational Safety and Health, 626 Cochran Mill Road, Pittsburgh, PA 15236. E-mail: dlanden@cdc.gov

dust and quartz exposure on IHD mortality within the cohort. Coal dust exposure (lagged 15 years) was associated with IHD mortality when quartz exposure was included in the model.

We examined IHD mortality risk among a cohort of coal miners who had participated in the National Study of Coal Workers' Pneumoconiosis [Attfield and Morring, 1992] during 1969–1971, by comparing risk among miners with varying levels of dust exposure. We also examined the effect of coal rank, a factor associated with risk of pneumoconiosis. Coal rank is a measure of coalification, the bio- and geochemical process which transforms plant materials into coal [Coal Utilization, 2011]. Higher and lower ranked coals differ in their physical and chemical properties. Since the physical and chemical composition of particulates influence their risks for the development of chronic health conditions, including IHD [Franklin et al., 2008; Ostro et al., 2008] we considered coal rank to be a potential risk factor for IHD among this cohort.

METHODS

Study Population

The cohort included 9,078 working underground coal miners who had participated in medical examinations during the first round of the National Study of Coal Workers' Pneumoconiosis (NSCWP) from 1969 through 1971. The first round of the NSCWP was designed to estimate the prevalence of pneumoconiosis among coal miners in the United States [Morgan et al., 1973]. Thirty-one underground coal mines in ten states were chosen to represent all major U.S. coal fields and mining methods. Miner participation was reported as 90.5%; all participating miners signed consent forms for their participation in the study. Data on demographics, smoking, and work history were collected by questionnaire. Participants were given medical examinations, which included collection of data on height and weight, as well as chest radiographs and pulmonary function tests. Of the 9,078 miners, 107 were excluded because of missing or invalid data on one or more of vital status, smoking, or coal dust exposure.

Vital Status and Cause of Death

Follow-up of vital status through 1993 was determined through the Social Security Administration, the files of the United Mine Workers of America Welfare and Retirement Fund, state Vital Statistics offices and the National Death Index database. The underlying cause of death on the death certificate was classified according to the International Classification of Diseases, Revision 8 (ICD-8) for deaths through 1979, and Revision 9 (ICD-9) for deaths

from 1980 to 1993. Deaths categorized as IHD were codes 410–414 in both ICD-8 and ICD-9. For pneumoconiosis deaths, codes were 515, 515.0, 515.1, and 515.9 in ICD-8, and 500, 502, and 505 in ICD-9.

Measurement of Exposure

Estimates of cumulative individual exposure to respirable coal mine dust were derived from each miner's work history up until the time of study enrollment. Using a job-exposure matrix, the time worked in each job title was multiplied by a job title-specific exposure estimate, and the result summed across all jobs. The job title-specific exposure estimates came from two sources: surveys undertaken by the U.S. Bureau of Mines between 1968 and 1969, which obtained dust samples from 17 of the mines which later participated in the NSCWP; and dust samples collected in the mines between 1969 and 1971 by coal mine operators and processed by the Mine Safety and Health Administration (MSHA) under their compliance program [Schlick, 1971]. The methodology for estimation of dust exposure is described by Attfield and Morring [1992] and by Seixas et al. [1990].

Coal rank region was assigned based on the location of the mine in which the miner worked at the start of follow-up. We grouped mines into the coal rank regions described in previous studies of this cohort [Attfield and Morring, 1992; Kuempel et al., 1995; Attfield and Kuempel, 2008]. Two mines in eastern Pennsylvania mined anthracite coal. Mines in other areas all mined bituminous coals, but the coals differed by region in their volatile content and percentage carbon, with higher ranked coals in the east and lower ranked in the west. The bituminous coals are identified simply by their geographic region: Eastern Appalachian, Western Appalachian, Midwest, and West.

Statistical Analysis

Death from IHD and death from pneumoconiosis are both known to be associated with particulate exposure. Deaths from these two causes represent non-independent competing risks. A competing risks situation arises when an individual can experience more than one type of event which could result in his removal from a cohort [David and Moeschberger, 1978]. In this case, although our cause of death of interest was IHD, miners may have been removed from the cohort by death from pneumoconiosis, for which coal miners have a substantial increased mortality risk [Morgan et al. 1973; Kuempel et al. 1995; Attfield and Kuempel 2008], or from other non-IHD causes.

In order to accommodate the effect of competing causes of mortality, we used a regression method for competing risks developed by Fine and Gray, which models

the effect of covariates on the cumulative incidence function (CIF) by a proportional hazards regression model [Fine and Gray, 1999]. Unlike Cox proportional hazards regression, which assumes non-informative censoring of causes of death other than the cause of interest, the Fine–Gray regression model accounts for the distribution of a competing risk from other causes of death. Models using the CIF provide a better estimator in a competing risks situation [Pepe and Mori, 1993; Lau et al., 2009; Grambauer et al., 2010].

Analyses were conducted using the `cmprisk` package [Gray, 2010] in R software [R Development Core Team, 2011]. Time at risk was calculated from the date of the baseline examination to either date of death or study end. To examine the relationship between dust exposure and IHD mortality, as well as the effect of competing mortality risks, we first generated CIF plots for deaths from IHD by exposure level within each coal region. We tested for differences between exposure groups, comparing the results obtained from the log rank test, which does not include the effects of competing risks, with those obtained from the Gray test, which does include the effects of competing risks. Comparison of the results of these two tests has been recommended by Pintilie [2006] as a way of assessing the importance of a competing risk.

We then fitted competing risks regression models to estimate the effect of coal dust exposure and coal rank region on IHD mortality, adjusted for body mass index, pack-years of smoking, and age. For this analysis, the Midwest and West coal regions, which had similar low risk, were combined. Cumulative exposure was modeled as each of the following: (1) a continuous linear variable, (2) a continuous linear variable with the addition of a quadratic term, and (3) a categorical variable defined as quartiles of cumulative exposure. Competing risks were all other causes of mortality, including pneumoconiosis. We compared model fit using likelihood ratio tests.

We tested for interactions between confounders (age, smoking, and body mass index) and exposure, and between coal region and exposure, using the likelihood ratio

test. We tested the validity of the proportional hazards model assumption (that the hazard ratio (HR) does not change over time) using a time-dependent variable representing the interaction between time since start of follow-up and cumulative exposure.

We then fit a separate model using Cox regression, after excluding the Anthracite region, and compared the results to the Fine–Gray competing risks regression model.

RESULTS

Table I shows characteristics of the cohort at study entry during 1969–1971. Forty-six percent were current or ex-smokers, and 56.5% had a body mass index (BMI) in the overweight or obese range (>25). Age, BMI, smoking prevalence, and cumulative dust exposure were highest among miners in the Anthracite coal region. During the follow-up period through 1993, 36% of the cohort (3,224) died; 957 deaths (29.5%) were due to IHD; and 245 (7.6%) due to pneumoconiosis.

Figure 1 shows CIF plots for mortality from IHD in the Anthracite region, and in the four bituminous regions. The bituminous regions were combined for presentation, since the CIFs for all four regions were quite similar. In the bituminous regions, IHD mortality risk increased with quartile of dust exposure. In the Anthracite region, however, risk was highest in the second quartile of exposure, while risk in the highest quartile of exposure was similar to that for the lowest quartile. This pattern is probably due to the effect of a competing risk from pneumoconiosis mortality, which was much higher in the Anthracite region than in the bituminous regions (Fig. 2). Although the log rank test showed a significant difference in IHD mortality between exposure groups in the Anthracite region ($P = 0.04$) Gray’s test, which includes the effect of the competing risk, showed no difference ($P = 0.10$). The disparate results of these two tests indicated that a competing risk was affecting the data [Pintilie, 2006].

TABLE I. Characteristics of the National Study of Pneumoconiosis Cohort at Enrollment, 1969–1970

Characteristic	All coal rank regions	Anthracite	Eastern Appalachian	Western Appalachian	Midwest	West
No. of miners	8,971	519	1,354	4,923	1,219	956
Mean (SD) age, years	44.5 (11.9)	51.6 (8.8)	43.4 (11.4)	44.1 (11.8)	44.1 (12.5)	44.7 (12.9)
Race, % White	95.1	99.4	92.9	93.6	98.0	99.6
Body mass index, % >25 kg/m ³	56.5	62.6	51.3	57.2	61.5	50.8
Smoking status, % ever smoked	46.0	51.3	46.8	45.3	41.3	51.3
Mean (SD) pack-years of smoking	17.5 (17.4)	23.4 (20.3)	15.7 (16.0)	17.6 (17.4)	19.0 (18.0)	14.5 (16.1)
Mean cumulative coal dust exposure, mg-yr/m ³	64.4 (46.4)	91.3 (40.3)	62.7 (42.8)	65.1 (46.6)	57.0 (46.5)	58.6 (47.7)

SD, standard deviation.

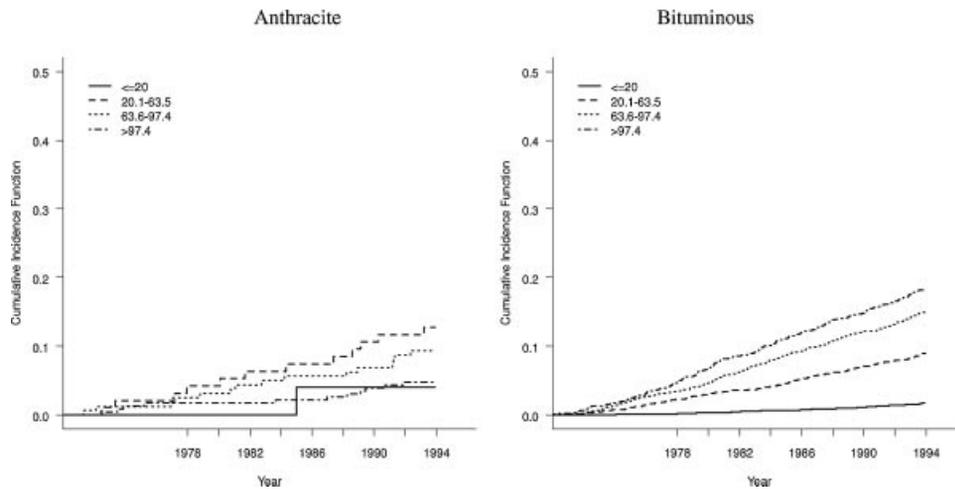


FIGURE 1. Cumulative incidence function for mortality from (ischemic heart disease) by quartile of dust exposure in the anthracite and bituminous regions.

We next modeled the relationship between cumulative dust exposure and IHD mortality using the Fine–Gray competing risks method. The best fit was provided by a model with exposure categorized in quartiles (likelihood ratio test with 4 df $P = 0.03$ compared to the model with continuous linear exposure and a quadratic term). This final model is shown in Table II. There was a significant interaction between coal dust exposure and coal region ($P = 0.007$). The HRs are presented by quartile of exposure within region, compared to a reference group consisting of the lowest quartile of dust exposure in the Western Appalachian region. Estimates have been adjusted for age, smoking, and BMI. In the Midwest–West region, point estimates for the two highest quartiles were above the reference level, but only quartile 3 was significantly different

(HR 1.92, 95% CI 1.12, 3.28). In the Eastern and Western Appalachian regions, risk was significantly above reference for quartiles 2–4, and increased with increasing levels of coal dust exposure in Western Appalachia. In the Anthracite region, none of the HRs were statistically significant. There was no interaction between time since study enrollment and cumulative exposure, indicating that the proportional hazards assumption was met.

As expected, risk increased with increasing years of age, BMI, and pack-years of smoking. The HR for age was 1.07 (95% CI 1.06, 1.08) and the HR for BMI was 1.04 (95% CI 1.02, 1.06). The HR for smoking increased for each level of exposure, with the highest HR, 1.56 (95% CI 1.28, 1.90), in the group with over 30 pack years of smoking.

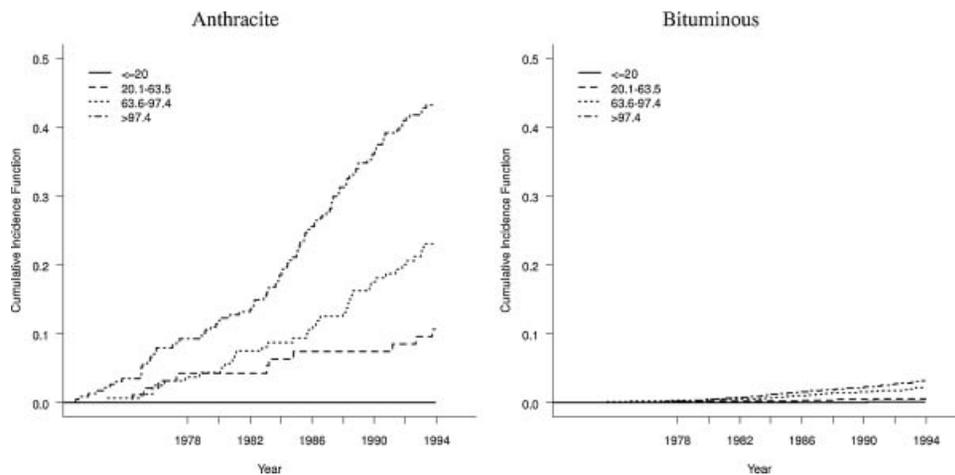


FIGURE 2. Cumulative incidence function for mortality from (pneumoconiosis) by quartile of dust exposure in the anthracite and bituminous regions.

TABLE II. Fine–Gray Subdistribution Hazard Ratios (HRs) and 95% Confidence Intervals for Ischemic Heart Disease Mortality by Quartiles of Coal Dust Exposure* and Coal Rank, Adjusted for Age, BMI, and Smoking

Coal region and dust quartile	Person-years follow-up	No. of cases	HR and 95% CI
Western Appalachia			
Q1 (reference group) ^a	40,061	20	1.00
Q2	37,262	98	1.75 (1.06, 2.89)
Q3	37,039	184	2.08 (1.26, 3.43)
Q4	35,282	252	2.26 (1.37, 3.75)
Eastern Appalachia			
Q1	10,442	7	1.35 (0.58, 3.15)
Q2	11,347	41	2.63 (1.52, 4.53)
Q3	11,578	32	2.38 (1.40, 4.07)
Q4	7,905	98	2.29 (1.31, 4.00)
Midwest/West			
Q1	21,702	11	0.92 (0.45, 1.89)
Q2	18,637	53	1.46 (0.86, 2.50)
Q3	13,415	66	1.92 (1.12, 3.28)
Q4	13,543	71	1.51 (0.87, 2.61)
Anthracite			
Q1	886	1	1.94 (0.27, 13.81)
Q2	2,815	11	2.04 (0.96, 4.33)
Q3	4,860	15	1.18 (0.59, 2.34)
Q4	6,043	11	0.43 (0.20, 0.92)

*Cumulative coal dust exposure (mg-yr/m³) at study entry: 1st quartile: ≤20, 2nd quartile: 20.1–63.5, 3rd quartile: 63.6–97.4, and 4th quartile: >97.4.

^aQ1–Q4 = quartiles 1–4.

Table III shows the results of the Cox proportional hazards regression, with the Anthracite region (the region affected by competing mortality risk) excluded. In this analysis, there was no interaction between coal region and dust exposure. There was a significant increase in the HRs for IHD mortality risk associated with higher quartiles of dust exposure. HRs for the coal rank regions were not statistically significant, although point estimates were in the same direction as in the competing risks regression; increased in the Eastern Appalachian region, and decreased in the Midwest/West.

DISCUSSION

In this analysis of data collected for the National Study of Coal Workers' Pneumoconiosis, we have shown that the risk of IHD increased with cumulative exposure to respirable coal mine dust. The strongest effect was seen in the two Appalachian regions. Results were uninformative in the Anthracite region, which had the smallest sample size. In the other regions, the exposure–response effect for coal dust did not increase linearly, but flattened out at

TABLE III. Cox Proportional Hazards Regression Hazard Ratios (HRs) and 95% Confidence Intervals for Ischemic Heart Disease Mortality by Quartiles of Dust Exposure* and Coal Rank Region, Adjusted for Age, BMI, and Smoking

	Person-years follow-up	No. of cases	HR, 95% CI
Dust exposure quartile			
Q1 (reference) ^a	72,205	38	1.00
Q2	67,246	192	1.58 (1.09, 2.30)
Q3	62,032	282	1.81 (1.23, 2.66)
Q4	56,730	421	1.92 (1.29, 2.86)
Coal rank			
Western Appalachia (reference)	149,644	554	1.00
Eastern Appalachia	41,272	178	1.18 (0.99, 1.41)
Midwest/West	67,297	201	0.75 (0.64, 0.88)

Anthracite region excluded.

*Cumulative coal dust exposure (mg-yr/m³) at study entry: 1st quartile: ≤20, 2nd quartile: 20.1–63.5, 3rd quartile: 63.6–97.4, and 4th quartile: >97.4.

^aQ1–Q4 = quartiles 1–4.

higher levels of exposure. This pattern has also been found in studies of particulate air pollution, which show steep dose–response effects on cardiovascular mortality at low levels, with attenuation at higher levels [Pope et al., 2009]. In addition, attenuation of effect at high dose in occupational studies may occur due to biases, including healthy worker survivor effects, and depletion of the susceptible population in long-term studies [Stayner et al., 2003].

Particulate exposures to coal miners are far greater than particulate exposures to the general population from ambient air. The current Environmental Protection Agency (EPA) 24-hr standard for particulate matter with a mass median aerodynamic diameter of ≤2.5 μm (PM_{2.5}) is 35 μg/m³, and the 24-hr standard for particulate matter with a mass median aerodynamic diameter of ≤10 μm (PM₁₀) is 150 μg/m³ [EPA, 2006]. The current standard for respirable coal mine dust, which has a particle size distribution in between that of PM_{2.5} and PM₁₀, is 2,000 μg/m³ per work shift [U.S. Congress, 1969].

In view of the high exposure levels, the risk for IHD mortality among coal miners is smaller than would be expected on the basis of studies of atmospheric particulate [Pope and Dockery, 2006], even given that respirable coal mine dust exposures are more limited, since they occur only at work. While both the healthy worker effect and attenuation of risk at higher exposures may play a role in the lower effect size for coal dust exposure, the most important factor is probably a difference in the particulate, both in terms of particle size distribution and particle composition. The particulate fraction under 2.5 μm (PM_{2.5}, fine particles) is most closely linked with IHD in

atmospheric pollution studies [Schwartz et al., 1996; Kan et al., 2007; Puett et al., 2008]. Most particles of this size in ambient air come from combustion sources. A small portion of fine particulate in ambient air comes from crustal material; this portion, however, has not been associated with an increased risk of either IHD [Laden et al., 2000] or of all-cause mortality [Ozkaynak and Thurston, 1987] in air pollution studies. Coal particulate is mechanically generated, so the particle size distribution within PM10 would tend toward larger particles. Since coal comes from earth's crust, fine particulate coal dust might be expected to be less hazardous than particulate of similar size derived from combustion, perhaps on the basis of a differing particle composition.

The influence of particle composition on IHD mortality has been studied less than the influence of particle size. We found the IHD mortality risk varied with coal rank, an indicator of differences in the composition of coal dust. In studies of pneumoconiosis among coal miners, higher coal rank is associated with a significantly increased risk of development of pneumoconiosis [Morgan et al., 1973; Attfield and Moring, 1992; Attfield and Seixas, 1995] and of pneumoconiosis mortality [Kuempel et al., 1995]. Differences in risk related to coal rank have not been studied extensively, but may involve differences in particle charge, which affects the degree to which dust penetrates the lung [Page and Organiscak, 2000], as well as the iron content of the coal [Huang et al., 2002].

Limitations in our study include lack of information on individual risk factors for IHD other than BMI and smoking, and the use of a job-exposure matrix to estimate miners' exposures, rather than direct individual measurements. Another limitation is lack of information on employment or exposure following study enrollment, which resulted in some degree of exposure misclassification. However, the largest part of the miners' exposure occurred prior to the study start, before implementation of the dust control regulations under the Federal Coal Mine Health and Safety Act of 1969 [U.S. Congress, 1969], which led to lower dust levels in coal mines.

We were unable to assess the effect of silica exposure, which has been found to be associated with an increased risk of IHD in a surveillance study of silica exposed workers in other industries [Weiner et al., 2007]. Exposure to silica from quartz dust generated during the mining process may contribute to miners' IHD risk, particularly in the Appalachian region, where a considerable amount of rock may be cut in order to access coal deposits [Laney et al., 2009].

Exposure to diesel emissions could also affect risk of IHD among underground coal miners; however, diesel equipment was not used extensively during the years of the study. The use of diesel engines was prohibited in Pennsylvania and West Virginia underground mines until

after the study's end. Diesel equipment was used to a limited extent in the Midwest and South, and more frequently in the West, a region which had a lower risk of IHD in this study. Overall, any effect of diesel particulate exposure on study estimates of the effect of coal dust exposure on IHD mortality should be small.

In conclusion, we found an increased risk of mortality from IHD associated with cumulative exposure to coal dust in this cohort. Mortality risk varied by coal region, which may be due to regional variations in the composition of the coal mine dust particulate. The association of increased risk of IHD with cumulative particulate exposure is consistent with air pollution studies, which have shown that long-term cumulative exposures to particulate are strongly related to IHD mortality [Schwartz et al., 2008; Pope et al., 2009].

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