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Exposure-Response Analysis of Mortality Among Coal Miners in the United States

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The quantitative relationship between exposure to respirable coal mine dust and mortality from nonmalignant respiratory diseases was investigated in a study of 8,878 working male coal miners who were medically examined from 1969 to 1971 and followed to 1979. Exposure-related mortality was evaluated using Cox proportional hazards modeling for underlying or contributing causes of death and modified lifetable methods for underlying causes. For pneumoconiosis mortality, the lifetable analyses showed increasing standardized mortality ratios (SMRs) with increasing cumulative exposure category. Significant exposure-response relationships for mortality from pneumoconiosis ($p < 0.001$) and from chronic bronchitis or emphysema ($p < 0.05$) were observed in the proportional hazards models after controlling for age and smoking. No exposure-related increases in lung cancer or stomach cancer were observed. Pneumoconiosis mortality was found to vary significantly by the rank of coal dust to which miners were exposed. Miners exposed at or below the current U.S. coal dust standard of 2 mg/m^3 over a working lifetime, based on these analyses, have an elevated risk of dying from pneumoconiosis or from chronic bronchitis or emphysema.

Key words: pneumoconiosis, COPD, dust, proportional hazards model, survival analysis, risk, coal dust exposures, coal rank, lung cancer, stomach cancer

INTRODUCTION

Elevated standardized mortality ratios (SMRs) among miners with radiographic evidence of complicated coal workers' pneumoconiosis (CWP) have been reported previously in the United States and the United Kingdom, compared to miners with simple CWP or without CWP [Atuhaire et al., 1985; Cochrane et al., 1979; Ortmeyer et al., 1974]. Regional differences in SMRs among coal miners have also been observed, and miners in the anthracite coal regions of the United States have had

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higher SMRs for all-cause mortality [Ortmeyer et al., 1973]. Elevated SMRs have been reported for nonmalignant respiratory diseases (including pneumoconiosis and bronchitis), stomach cancer, and accidents [Cochrane et al., 1979], while SMRs for ischemic heart disease [Costello et al., 1975] and lung cancer [Costello et al., 1974] have been reported to be lower among coal miners than in the general population.

These previous mortality studies have been limited for several reasons. First, all of these studies have been based on comparisons with the general population mortality data from a country, state, or region; this approach may result in negatively biased estimates of occupation-related mortality risks because of the healthy worker effect [Fox and Collier, 1976]. Second, none of these studies examined the exposure-response relationship using quantitative exposure estimates. Third, previous studies were restricted to the recorded underlying causes of death, without examination of contributing causes mentioned on the death certificates.

Exposure data were included in a study of British coal miners, in which the 22-year survival rates were determined for 19,500 miners who were medically examined between 1953 and 1958 [Miller and Jacobsen, 1985]. In age-specific comparisons, significant associations were found between increasing exposure categories and increasing mortality from all nonviolent causes, pneumoconiosis, and chronic bronchitis and emphysema, as the underlying causes of death. Also observed was a marginally significant association between coal dust exposure and mortality from cancer of the digestive system, but no association was found between coal dust exposure and lung cancer [Miller and Jacobsen, 1985].

The primary objective of the present analysis was to investigate the relationship between exposure to respirable coal mine dust and mortality, particularly from nonmalignant respiratory diseases, in U.S. coal miners. Exposure-response relationships for mortality from nonmalignant respiratory diseases, including pneumoconiosis, chronic bronchitis, and emphysema, as well as from lung cancer, stomach cancer, and all causes were investigated using lifetable analyses and proportional hazards modeling. The effects of intensity and duration of exposure and the effect of coal rank on mortality risk were also investigated. Coal rank is a classification based on the geologic age, percent carbon, hardness, and other characteristics of the coal [Given, 1984]. The rank of coal tends to increase from the western to the eastern United States, with high-rank anthracite coal occurring in eastern Pennsylvania.

In an earlier analysis of the white miners in this cohort, SMRs were computed for underlying (but not contributing) causes of death, but exposure data were not considered [Attfield et al., 1985]. The present study greatly enhances previous information on mortality among U.S. coal miners by utilizing quantitative exposure estimates, by modeling mortality rates within the cohort (in addition to comparisons with the U.S. population), and by considering contributing causes of death.

MATERIALS AND METHODS

Background

The cohort for this analysis consists of the 9,078 male coal miners from 31 mines across the United States who participated in the medical examinations of the National Study of Coal Workers' Pneumoconiosis (NSCWP) from 1969 through 1971 [Attfield and Morring, 1992b]. Mortality follow-up was 9 years later, to November

30, 1979, the end of study date. The follow-up procedures to determine vital status included searches by Social Security number of files from the Social Security Administration, United Mine Workers Welfare and Retirement Fund, and State Vital Statistics [Attfield et al., 1985]. Of the 9,078 miners who were medically examined during 1969–1971, a total of 8,878 miners were included in the present study. Omitted were 139 miners with incomplete data for variables that were to be used in the analyses. Also omitted before beginning analyses were 61 miners of age 65 or older who, given their continued work in coal mining, were thought to represent a selected group.

Participants were administered questionnaires on work history and smoking history and were given medical examinations including chest radiographs. The 8th revision of the International Classification of Diseases (ICD-8) was used to classify the causes of deaths recorded on the death certificates.

Cumulative exposure estimates. Individual cumulative exposure estimates were computed by the method described in Attfield and Morring [1992a]. Briefly, for each miner, the intensity of exposure associated with a given job was multiplied by the duration of employment in that job, and these products were summed over all jobs. The work histories for each miner were collected as part of the NSCWP medical survey in 1969–1971. The job-specific exposure estimates were based on approximately 4,300 gravimetric samples of airborne respirable dust concentrations collected by the U.S. Bureau of Mines (BOM) during 1968 and 1969 in 29 underground coal mines across the United States, 17 of which were later included in the NSCWP. The BOM data included sampling of at least 10 shifts for certain jobs, including jobs at the coal face, but there were few or no samples for other underground or surface jobs. Exposure data for these jobs were based on samples collected from 1970 to 1972 by mine operators, with an adjustment for the reduction in exposures that occurred after 1969, when the Federal Coal Mine Safety and Health Act was enacted [Attfield and Morring, 1992a; 30 USC 801–962]. Cumulative exposure estimates were computed for the period from starting work in mining until the date of medical examination in 1969–1971. Exposures that occurred during the approximately 9-year follow-up period were not determined because work history information was not available after 1969–1971 for most of the miners who participated in the 1969–1971 survey.

Radiographic category. The chest radiographs were read by a panel of three readers [Morgan et al., 1973], and classifications were based on the UICC/Cincinnati criteria [Bohlig et al., 1970]. A consensus opinion (in which at least two of the three readers agreed) was obtained for about 90% of the radiographs. For approximately 10%, a fourth reader was used, and for less than 0.2%, a fifth reader was used to arrive at consensus [Morgan et al., 1973].

Modified Lifetable Analyses

The National Institute for Occupational Safety and Health (NIOSH) Life Table Analysis System was used to compare the observed deaths in this study with the expected deaths from the general U.S. population rates [Steenland et al., 1990]. Lifetable analyses were based on underlying causes of death. Person-days were calculated from the specific date of medical examination during 1969–1971 until death or end of follow-up (November 30, 1979). Expected deaths for each cause-of-death category were computed by applying the age-, race-, and calendar year-specific mortality rates for the general U.S. population to the corresponding person-years in

each stratum of the study data. SMRs and standardized rate ratios (SRRs) were computed for six cumulative exposure strata (within the range of 0.1–234 milligram × years per cubic meter, mg-yr/m^3). These exposure groups were chosen before analyses began and were based on inclusion of approximately equal number of deaths (about 132) in each group. The number of miners in exposure groups I through VI (lowest to highest exposure) were 3,536; 1,316; 1,292; 1,076; 802; and 856, respectively, for a total of 8,878.

Proportional Hazards Models

The exposure-response relationship was further examined using the Cox proportional hazards model [Cox, 1972, 1975], which was fitted with the SAS PHREG procedure [SAS, 1991]. In the Cox model, the hazard rates were compared within the coal miner cohort, thus minimizing the potential bias from using general U.S. population mortality rates. Risk sets were created for each cause of death in the analysis, which consisted of all miners who survived to at least the same length of follow-up as a miner who died of a given cause. Deaths from a given cause were assumed to have occurred if there was any mention of that cause on the death certificate. Thus, the proportional hazards analyses were performed using underlying causes alone or using both underlying and contributing causes. Chronic bronchitis and emphysema were combined in these analyses because they both represent obstructive ventilatory defects, which may be difficult to distinguish clinically, and because there were insufficient deaths from either cause to evaluate separately. Miller and Jacobsen [1985] also combined chronic bronchitis and emphysema in their mortality analysis of U.K. coal miners.

An exponential (log-linear) form of the Cox proportional hazards model was the principal model fitted to the data. Cumulative exposure was modeled as each of the following: 1) categorical variables (described in the preceding section); 2) a continuous variable; and 3) duration of exposure (as a continuous variable) defined within average intensity categories. Covariates for age at the start of follow-up and pack-years of smoking were treated as continuous predictor variables, and smoking at the start of follow-up and coal rank were incorporated as one or more indicator variables (value equals 0 or 1). Coal rank was assigned by the geographical region in which the miner worked at the start of follow-up [Attfield and Morring, 1992b].

Variables of a priori interest as predictors of mortality from causes of interest were included in the initial models (age, cumulative exposure, and coal rank). Additional variables (pack-years of smoking, smoking status at the start of follow-up, and race) and all two-way interactions between cumulative exposure and covariates were tested for inclusion in the model using a stepwise (forward) selection procedure [SAS, 1991]. Higher order terms (quadratic and cubic) for the cumulative exposure and age variables were also tested for inclusion in the model. The statistical significance of the variables retained in the model was determined by likelihood ratio tests [Kleinbaum et al., 1988].

The basic time dimension used in the proportional hazards models was time from start of follow-up until death or censor (time on study). Age at death or censor was not selected as the time dimension because it is highly correlated with cumulative exposure (Pearson correlation coefficient of 0.77 for age at death or censor, compared to 0.055 for time on study). If a variable that is highly correlated with cumulative exposure had been used as the time dimension, then some of the effects of cumulative

TABLE I. Vital Status of Coal Miners on November 30, 1979, Including Causes of Death Used in Exposure-Response Analyses*

	UCOD only		UCOD or CCOD[a]	
Vital status and cause of death	Number	Percent	Number	Percent
Alive[b]	8,085	91.1		
Deceased	793	8.9		
Nonmalignant respiratory diseases (ICD-8 codes 460–519)	85	1.0	303	3.4
Pneumoconiosis (ICD-8 code 515)	54	0.6	207	2.3
Chronic bronchitis or emphysema (ICD codes 491–492)	9	0.1	76	0.9
Other nonmalignant respiratory diseases	22	0.2	70	0.8
Malignancy of trachea, bronchus, or lung (ICD-8 code 162)	65	0.8	69	0.8
Malignancy of stomach (ICD-8 code 151)	8	0.09	12	0.1
Other causes	635	7.1	442	4.9
Study total[c]	8,878	100.0		

*Abbreviations: UCOD, underlying cause of death; CCOD, contributing cause of death; ICD-8, International Classification of Diseases, 8th Revision.

[a]Overlap of CCODs: 50 death certificates mentioned both pneumoconiosis and either chronic bronchitis or emphysema; 33 death certificates mentioned both nonmalignant respiratory diseases and either malignancy of the trachea, bronchus, or lung (28), or malignancy of the stomach (5).

[b]Miners lost to follow-up were assumed alive for the analyses.

[c]From the original cohort of 9,078, 61 miners who were age 65 or older at the start of follow-up and 139 individuals with incomplete data were omitted prior to analysis.

exposure would have been included in the estimated baseline risk function [Breslow and Day, 1987], resulting in bias toward the null.

The validity of the proportional hazards assumption (i.e., that the hazard ratio does not change with time for the groups being compared, exposed, and unexposed) was tested using a time-dependent explanatory variable representing the interaction between time since start of follow-up and cumulative exposure. The hazard rates (or rate ratios) were computed from the final models for various values of the independent variables.

Additional analyses were also performed to evaluate the sensitivity of the regression results by restricting the analysis to underlying causes of death or to cases of pneumoconiosis or chronic bronchitis or emphysema that did not have the other cause listed as well. Finally, an analysis was performed in which the effect of radiographic category at the beginning of the follow-up was examined.

RESULTS

Descriptive Statistics

Table I lists the vital status and causes of death for miners included in the study. The overall percentage of follow-up was at least 95.2%. Miners with unknown status were assumed alive for the analyses. By the end of follow-up in 1979, there were 207 reported deaths from pneumoconiosis and 76 reported deaths from chronic bronchitis or emphysema as an underlying or contributing cause of death. Of these deaths, 50 included mention of both pneumoconiosis and chronic bronchitis or emphysema on the death certificate. Thus, of the 207 miners who died of pneumoconiosis as a listed cause of death, 157 had no mention of chronic bronchitis or emphysema; and of the

TABLE II. Characteristics of the Study Population: National Study Coal Workers' Pneumoconiosis

Variable (units)	Deaths (n = 793)	Whole cohort (n = 8,878)
Mean (standard deviation)		
Age at start of follow-up (years)	51.7 (8.9)	44.3 (11.8)
Age at start of mining (years)	23.4 (6.4)	23.9 (6.2)
Cumulative exposure (mg-yr/m^3)	87.6 (41.2)	63.9 (45.9)
Dust concentration (mg/m^3)	3.1 (0.9)	3.0 (1.0)
Duration of exposure (years)	28.3 (11.2)	20.4 (13.1)
Cumulative smoking (pack-years)	23.4 (6.4)	23.9 (6.2)
Percentage (count)		
Smoking status at start of follow-up		
Current	60.2 (477)	54.2 (4,812)
Ex-smoker	23.8 (189)	25.5 (2,262)
Never smoker	16.0 (127)	20.3 (1,804)
Race		
White	92.6 (734)	95.1 (8,443)
Nonwhite	7.4 (59)	4.9 (435)
Coal rank		
Anthracite	8.8 (70)	5.7 (506)
Medium/low volume bituminous	14.6 (116)	15.1 (1,345)
High volume bituminous A	56.4 (447)	55.0 (4,881)
High volume bituminous MW	11.6 (92)	13.5 (1,200)
High volume bituminous W	8.6 (68)	10.7 (946)

76 miners who died of chronic bronchitis or emphysema as a listed cause of death, 26 had no mention of pneumoconiosis on the death certificate.

Characteristics of the study population are provided in Table II. The average age at the start of follow-up was 44 years, and the average length of employment in coal mining was 20 years. For more than two thirds of the miners in the study, cumulative exposures were below 90 mg-yr/m^3, which is the cumulative exposure equivalent to working for 45 years at the current U.S. standard of 2 mg/m^3 for respirable coal mine dust. About one third of the deaths either from pneumoconiosis or from chronic bronchitis or emphysema (as underlying or contributing causes) occurred among miners with cumulative exposures below 90 mg-yr/m^3.

Lifetable Analyses

SMRs for underlying causes of death are shown in Table III, by cumulative exposure groups, for the causes of death that were of a priori interest and for all causes combined. The SMR for all-cause mortality was 0.85 ($p < 0.01$), which indicates lower mortality from all causes among working miners than among individuals in the general population. SMRs for pneumoconiosis are significantly elevated for all exposure groups (except the lowest), and SMRs increase with increasing exposure group (except the highest). SRRs are also provided for mortality from pneumoconiosis, using miners in the lowest exposure group as the reference group (Table III). A statistically significant trend in the SRRs for pneumoconiosis was observed with increasing cumulative exposure group ($p < 0.05$, trend test for directly standardized rates [Rothman, 1986]). There was no indication of a trend of increasing mortality with cumulative exposure for any of the other underlying causes examined. For either chronic bronchitis mortality or emphysema mortality (underlying causes only), SMRs were not elevated in the lifetable analyses (Table III). However, there were just eight

TABLE III. SMRs by Exposure Group for Causes of Death Investigated in Study: National Study of Coal Workers' Pneumoconiosis

Underlying cause	Cumulative exposure group (mg-yr/m^3) I (0.1–48.9)	II (49.0–69.3)	III (69.4–88.3)	IV (88.4–107.0)	V (107.1–127.0)	VI (127.1–234)	Total
Pneumoconioses and other respiratory diseases (ICD-8 codes 500–519[a])							
Observed	4	8	12	13	15	16	68
Expected	1.99	2.56	3.35	3.28	2.97	4.14	18.30
SMR	2.01	3.13[b]	3.58[c]	3.96[c]	5.04[c]	3.86[c]	3.72[c]
95% CI	0.55–5.15	1.35–6.15	1.85–6.26	2.11–6.78	2.82–8.33	2.21–6.28	2.89–4.71
SRR[d]	1.00	1.26	1.46	1.62	1.96	1.96	1.57
Chronic bronchitis and unspecified bronchitis (ICD-8 codes 490, 491)							
Observed	0	0	0	1	0	0	1
Expected	0.27	0.37	0.49	0.49	0.45	0.62	2.70
SMR	0.00	0.00	0.00	2.04	0.00	0.00	0.37
Emphysema (ICD-8 code 492)							
Observed	0	0	2	3	2	1	8
Expected	1.19	1.74	2.35	2.35	2.17	3.05	12.85
SMR	0.00	0.00	0.85	1.28	0.92	0.33	0.62
Malignancy of trachea, bronchus, and lung (ICD-8 code 162)							
Observed	12	12	8	9	15	9	65
Expected	10.21	12.95	16.00	15.13	13.02	16.79	84.10
SMR	1.18	0.93	0.50[b]	0.59	1.15	0.54	0.77[b]
Malignancy of stomach (ICD-8 code 151)							
Observed	1	0	1	3	0	3	8
Expected	1.06	1.30	1.66	1.55	1.36	1.83	8.76
SMR	0.94	0.00	0.60	1.94	0.00	1.64	0.91
All causes							
Observed	133	132	131	132	134	131	793
Expected	149.16	141.04	171.78	159.84	137.34	178.30	937.47
SMR	0.89	0.94	0.76[c]	0.83[b]	0.98	0.73[c]	0.85[c]
Distribution of person-years							
	31,165	11,392	11,323	9,302	6,941	7,287	77,409

[a]International Classification of Diseases, 8th Revision (ICD-8): pneumoconioses (ICD-8 code 515) listed as the underlying cause of death for 54 miners; NIOSH modified lifetable analysis included "other respiratory diseases" (ICD-8 code 510–519), which added 14 miners.
[b]$p < 0.05$.
[c]$p < 0.01$.
[d]Trend test [Rothman, 1986]: Chi-square = 4.87 (1 d.f.): $p < 0.05$.

deaths from emphysema and one death from chronic bronchitis (as the underlying cause).

Proportional Hazards Models

Tests for the validity of the proportional hazards assumption demonstrated that the proportional hazards assumption was satisfied (i.e., the hazard ratio between the comparison groups of exposed and unexposed did not change significantly over time). These tests showed no evidence of significant interaction between the variables for time since the start of follow-up and cumulative exposure (p values > 0.7) for each cause of death evaluated.

In the models for either lung cancer or stomach cancer mortality, there was no evidence of an exposure-response relationship in models that included covariates for

TABLE IV. National Study Coal Workers' Pneumoconiosis: RRs for Mortality From Pneumoconiosis and From Chronic Bronchitis or Emphysema as Either an Underlying or Contributing Cause of Death, by Cumulative Exposure Categories*

Cumulative exposure (mg-yr/m^3)	RR	95% CI	p value
Pneumoconiosis (207 deaths)			
II (49.0–69.3)	2.55	1.20–5.41	0.015
III (69.4–88.3)	2.84	1.36–5.94	0.0055
IV (88.4–107.0)	3.48	1.66–7.23	0.0009
V (107.1–127.0)	5.22	2.50–10.84	0.0001
VI (127.1–234.0)	3.83	1.80–8.14	0.0005
Chronic bronchitis and emphysema (76 deaths)			
II (49.0–69.3)	3.07	0.83–11.50	0.096
III (69.4–88.3)	2.89	0.80–10.85	0.11
IV (88.4–107.0)	4.20	1.17–15.22	0.0029
V (107.1–127.0)	4.97	1.38–18.55	0.016
VI (127.1–234.0)	3.58	0.97–13.73	0.060

*RRs based on proportional hazards model with covariates for age, pack-years of smoking, and current smoking status; RRs relative to the lowest cumulative exposure group (0.1–48.9 mg-yr/m^3).

age and pack-years of smoking (p values > 0.2). The exposure-response relationship was negative for lung cancer mortality, and the rate ratio (RR) was 0.68, 95% confidence interval (CI) = 0.36–1.25, for cumulative exposure to respirable coal mine dust at 90 mg-yr/m^3. There was a positive, but not statistically significant, exposure-response relationship for stomach cancer (RR = 1.19 and 95% CI = 0.30–4.78 for 90 mg-yr/m^3). Thus, the remainder of this section focuses on results of models for pneumoconiosis mortality or chronic bronchitis or emphysema mortality.

Cumulative exposure (categorical variables). A model with cumulative exposure represented as categorical variables was fitted using the same cumulative exposure groups as those used in the lifetable analyses. The lowest exposure group was treated as the background (or unexposed) group in this analysis. Age, pack-years of smoking, and smoking status at the start of follow-up were included as covariates in the model. Table IV shows that, except for the highest group, RRs increase with increasing cumulative exposure for mortality from pneumoconiosis as an underlying or contributing cause of death (207 cases). For chronic bronchitis and emphysema as underlying or contributing causes of death (76 cases), the RRs were elevated, but the exposure-response relationship was not as consistent.

Cumulative exposure (continuous variable). The final proportional hazards models for pneumoconiosis mortality and chronic bronchitis or emphysema mortality, using cumulative exposure as a continuous variable, are provided in Table V. Race was not included in the final models because inclusion of this variable did not significantly improve the fit of the model for either pneumoconiosis ($p > 0.6$) or chronic bronchitis or emphysema ($p > 0.8$), nor did its inclusion appreciably modify the coefficient for cumulative exposure. For pneumoconiosis mortality, parameters for age, smoking, and coal rank, in addition to cumulative exposure, significantly improved the fit of the model (Table V). For chronic bronchitis and emphysema mortality, the parameters for age, smoking, and cumulative exposure were statistically significant; however, of the coal rank terms, only coal rank 1 (anthracite) was a significant additional predictor of mortality. None of the two-way interactions

TABLE V. National Study of Coal Workers' Pneumoconiosis: Cox Proportional Hazards Models for Mortality From Pneumoconiosis and From Chronic Bronchitis or Emphysema (as either an Underlying or Contributing Cause of Death), Using Cumulative Exposure as a Continuous Variable

Parameter	Estimated coefficient	Standard error	χ^2	p value
Pneumoconiosis				
Model −2 log likelihood = 3,374.810; 8 d.f. (without covariates: 3,307.366)				
Age	0.0902	0.0132	47.74	<0.00001
Pack-years smoking	0.00822	0.00347	5.30	0.02
Smoking status	0.304	0.148	4.24	0.04
Cumulative exposure	0.0301	0.00935	12.33	0.0004
(Cumulative exposure)^2	−0.000115	0.0000428	8.49	0.004
Coal rank 1[a]	2.207	0.292	75.82	<0.00001
Coal rank 2[b]	1.245	0.317	16.74	0.00004
Coal rank 3[c]	0.987	0.278	15.81	0.00007
Chronic bronchitis or emphysema				
Model −2 log likelihood: 1,218.292; 6 d.f. (without covariates: 1,359.401)				
Age	0.102	0.0223	21.94	<0.00001
Pack-years smoking	0.00579	0.00588	0.93	0.3
Smoking status	0.584	0.252	5.57	0.02
Cumulative exposure	0.0371	0.0167	6.08	0.01
(Cumulative exposure)^2	−0.000161	0.0000783	5.20	0.02
Coal rank 1[a]	1.635	0.244	36.91	<0.00001

[a]Anthracite coal (Eastern Pennsylvania).
[b]Medium/low volatile bituminous coal (Eastern Appalachia).
[c]High-volatile bituminous "A" coal (Western Appalachia). Reference group for categorical coal rank variables included coal rank 4 (high-volatile bituminous, Midwest) and coal rank 5 (high-volatile bituminous, West).

between cumulative exposure and the other covariates were statistically significant ($p > 0.2$).

Departure from linearity of the effect of cumulative exposure on mortality risk was assessed by inclusion of higher order terms. Addition of a quadratic term for cumulative exposure significantly improved the fit of the model for either pneumoconiosis mortality or chronic bronchitis or emphysema mortality. The coefficients were negative, indicating a downward trend in the mortality risks at higher exposures (above about 125 mg-yr/m^3). This finding is consistent with the findings of analyses using cumulative exposure categories, including both proportional hazards analyses (Table IV) and lifetable analyses (Table III), in which an apparent downturn in risk at high exposures is observed (evaluated in Discussion section). The addition of a cubic term for cumulative exposure did not significantly improve the model fit. A power function form of the proportional hazards model was also evaluated (using log of cumulative exposure), but the likelihood was not improved compared to the exponential form of the model.

Mortality RRs and 95% CIs for pneumoconiosis and for chronic bronchitis or emphysema are presented in Figures 1 and 2 (based on the final models, Table V). Exposure for a 45-year working lifetime at 2 mg/m^3 is equivalent to cumulative exposure of 90 mg-yr/m^3 (assuming no exposure-rate effects, at least for the intensity and duration of exposures experienced within the cohort). The rate ratio at 90 mg-yr/m^3 is 5.92 for pneumoconiosis (95% CI = 2.18–16.10) and 7.67 for chronic bronchitis and emphysema (95% CI = 1.31–44.98).

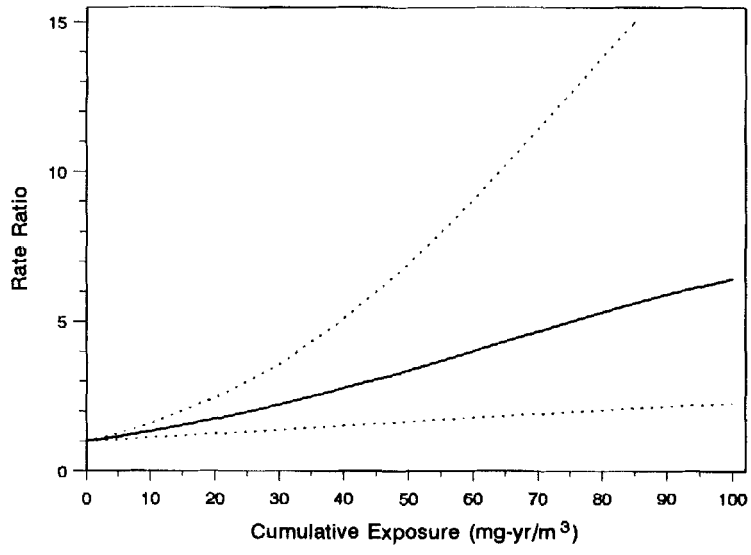

Fig. 1. Mortality rates for pneumoconiosis as an underlying or contributing cause of death by cumulative exposure to respirable coal mine dust (based on Cox proportional hazards model in Table V; dotted lines represent 95% CI estimates).

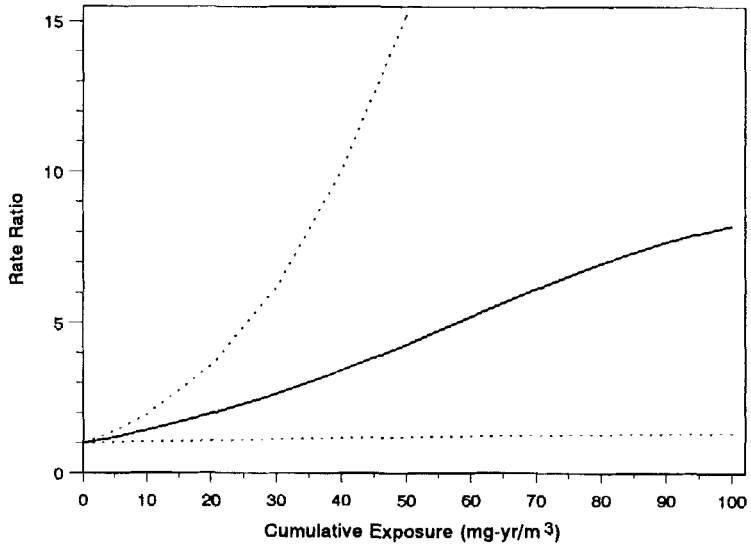

Fig. 2. Mortality rates for chronic bronchitis or emphysema as underlying or contributing causes of death by cumulative exposure to respirable coal mine dust (based on Cox proportional hazards model in Table V; dotted lines represent 95% CI estimates).

Intensity and duration of exposure. A model was evaluated with separate slopes for duration of exposure at different levels of intensity of exposure (Table VI). The mean concentration categories chosen a priori were <2 mg/m^3, 2–3 mg/m^3, and >3 mg/m^3. The models with duration by intensity category provided a poorer fit to

TABLE VI. National Study of Coal Workers' Pneumoconiosis: Cox Proportional Hazards Model for Mortality From Pneumoconiosis (as Either an Underlying or Contributing Cause of Death), Using Duration of Exposure as a Continuous Variable Within Intensity Categories*

Parameter	Estimated coefficient	Standard error	χ^2	RR[a]	95% CI
Age	0.0792	0.0171	20.49		
Pack-years smoking	0.00814	0.00344	5.28		
Smoking status	0.322	0.148	4.78		
Duration of exposure					
Mean concentration <2.0 mg/m^3	0.0230	0.0146	2.47	2.82	0.78–10.16
Mean concentration 2.0–<3.0 mg/m^3	0.0328	0.0120	7.98	4.37	1.51–12.63
Mean concentration ≥3.0 mg/m^3	0.0366	0.0121	9.15	9.92	1.78–15.09
Coal rank 1[b]	2.202	0.294	73.99		
Coal rank 2[c]	1.255	0.318	16.90		
Coal rank 3[d]	0.984	0.279	15.56		

*Model −2 log likelihood = 3,378.460; 9 d.f. (without covariates: 3,707.366)
[a]RRs computed for a 45-year duration of exposure within each intensity category, relative to zero exposure.
[b]Anthracite coal (Eastern Pennsylvania).
[c]Medium/low volatile bituminous coal (Eastern Appalachia).
[d]High-volatile bituminous "A" coal (Western Appalachia). Reference group for categorical coal rank variables includes coal rank 4 (high-volatile bituminous, Midwest) and coal rank 5 (high-volatile bituminous, West).

the data than did the models using cumulative exposure (Table V). The model of duration by intensity category shows that, for a given duration of exposure the RR for mortality from pneumoconiosis increases with increasing intensity category (Table VI). For mortality from chronic bronchitis or emphysema, duration of exposure, within concentration category, was not statistically significant in any of the a priori categories (results not shown).

Effects of overlapping causes of death. For the proportional hazards models presented above, the response variables were defined as any mention of that cause on the death certificate, without regard to other causes listed. To test the possible effect of overlapping causes for mortality from pneumoconiosis or from chronic bronchitis or emphysema (i.e., to determine if overlapping causes could be driving the exposure-response relationship), alternative response variables were defined by excluding the overlapping cases (50 cases). The final model for pneumoconiosis (Table V) was refitted for the 157 miners with any mention of pneumoconiosis on the death certificate but without any mention of chronic bronchitis or emphysema. The results were similar to the original analysis. Based on this subset of deaths, the RR for mortality from pneumoconiosis was 5.47 (95% CI = 1.78–16.79) for exposure to 90 mg-yr/m^3, compared to 5.92 (95% CI = 2.17–16.14) from the full analysis. Both pack-years of smoking and smoking status at the start of follow-up remained statistically significant predictors of mortality from pneumoconiosis.

The full model for chronic bronchitis and emphysema (Table V) was refitted by defining the response variable as the 26 deaths involving chronic bronchitis or emphysema without any mention of pneumoconiosis on the death certificate. In the analysis of this subset of deaths, the coefficient for cumulative exposure was mar-

ginally significant based on the likelihood ratio test ($p = 0.053$), and the RR for mortality from chronic bronchitis or emphysema increased from 7.76 to 22.4 for exposure to 90 mg-yr/m^3. Coal rank 1 (anthracite) was not a significant predictor ($p = 0.99$) of mortality from chronic bronchitis or emphysema when the response was limited to miners without mention of pneumoconiosis on the death certificate.

Effect of limiting analyses to underlying causes of death. Proportional hazards analyses were performed to determine the effect of limiting the response to underlying cause only for pneumoconiosis mortality (54 cases). For chronic bronchitis or emphysema, data were insufficient to perform analyses using only underlying causes (9 cases). For pneumoconiosis as the underlying cause of death, the RRs increased with increasing exposure category (i.e., 4.09, 4.74, 9.00, 13.56, 14.76) in a model using cumulative exposures as categorical variables (described in the Materials and Methods section). However, when age was added to the model, the coefficients for the exposure categories were no longer significant ($p > 0.2$), and the RRs were decreased (i.e., 1.18, 1.03, 1.66, 2.04, 1.63). The standard errors were large for the parameter estimates associated with these RRs, and the 95% CIs were wide and included one.

In a model for pneumoconiosis as the underlying cause of death, using cumulative exposure as a continuous variable, a significant exposure-response relationship was observed for cumulative exposure ($p < 0.0001$) when age was not in the model. As in the categorical exposure model, the effect of adding age was to decrease the effect of exposure on mortality risk. Pack-years of smoking and coal rank 1 (anthracite) remained statistically significant predictors of pneumoconiosis mortality using underlying causes only, and including these parameters to the model did not appreciably reduce the effect of exposure to respirable coal mine dust on mortality risk. RRs for pneumoconiosis as the underlying cause of death, at a cumulative exposure of 90 mg-yr/m^3, were 5.02 (95% CI = 3.02–8.34) in the model with cumulative exposure only and 1.43 (95% CI = 0.73–2.78) when age was added.

Relationship between radiographic category and mortality from pneumoconiosis. The relationship between radiographic category of the chest X-ray at the start of follow-up and mortality from pneumoconiosis during the subsequent 9-year period was investigated in a proportional hazards model using categorical variables for radiographic category. Category 0, or no evidence of pneumoconiosis, was treated as the background radiographic category. In all models, the effects of age, pack-years of smoking, and smoking status at the start of follow-up were included as covariates. Radiographic evidence of pneumoconiosis at the start of follow-up was found to be significantly associated with mortality from pneumoconiosis, either as any cause of death or as the underlying cause listed on the death certificate (Table VII). Further, the magnitude of the RRs tended to increase with increasing radiographic category (Table VI).

DISCUSSION

Respirable Coal Mine Dust Exposure as a Predictor of Mortality

An exposure-response relationship was evident in both lifetable analyses (using underlying causes) and in proportional hazards models (using underlying and contributing causes) between increasing cumulative exposure to respirable coal mine dust and mortality from pneumoconiosis, whether dust exposure was modeled as a con-

TABLE VII. National Study of Coal Workers' Pneumoconiosis: RRs for Mortality From Pneumoconiosis Either as an Underlying or Contributing Cause of Death, by Radiographic Category at the Start of the Study

Radiographic category[a]	RR[b]	95% CI	p value
Pneumoconiosis: underlying or contributing causes (207 cases)			
Model −2 log likelihood = 3,445.480; 8 d.f. (without covariates: 3,707.366)			
CWP 1	1.83	1.33–2.52	0.0002
CWP 2	2.06	1.32–3.22	0.002
CWP 3	3.00	1.21–4.75	0.02
PMF A	2.78	1.47–5.26	0.002
PMF BC	4.42	2.40–8.15	0.0001
Pneumoconiosis: underlying causes (54 cases)			
Model −2 log likelihood = 851.507; 8 d.f. (without covariates: 965.423)			
CWP 1	2.48	1.22–5.06	0.01
CWP 2	3.11	1.25–7.75	0.02
CWP 3	13.26	4.30–40.9	0.0001
PMF A	7.00	2.48–19.7	0.0002
PMF BC	14.07	5.61–35.3	0.0001

[a]Abbreviations for radiographic category [Bohlig et al., 1970]: CWP 1, CWP 2, CWP 3: simple coal workers' pneumoconiosis category 1, 2, or 3; PMF A, PMF BC: progressive massive fibrosis category A or categories B and C.

[b]Based on Cox proportional hazards model with covariates for age, pack-years of smoking, and smoking status at the start of follow-up; rate ratios for radiographic categories are relative to CWP 0 (no evidence of pneumoconiosis).

tinuous variable or as categorical variables. These results are consistent with previous mortality studies of coal miners in finding elevated SMRs for pneumoconiosis as the underlying cause of death among coal miners, compared to the general population. For chronic bronchitis or emphysema as an underlying or contributing cause of death, a significant exposure-response relationship was observed in the proportional hazards model with cumulative exposure as a continuous variable, although the exposure-response was not as clear in the model using categorical variables.

Other Predictors of Mortality

The coal rank of respirable dust to which miners were exposed influenced the rate of mortality from pneumoconiosis. Previous U.S. studies have reported a coal rank effect on the development of simple and complicated CWP [Attfield and Morring, 1992b; Lainhart, 1969] and on mortality from pneumoconiosis [Ortmeyer et al., 1973]. This additional effect of coal rank on mortality rate (i.e., in addition to the effect of cumulative exposure) is thought to be due to the greater toxicity of dust from coal of increasing rank.

Cigarette smoking was a statistically significant predictor of mortality either from pneumoconiosis or from chronic bronchitis or emphysema. In both cases, the magnitude of the effect for smoking was less than that for cumulative dust exposure. For example, among current smokers with 30 pack-years of smoking (which is about the average among miners aged 55 and older in this study), the RR for pneumoconiosis mortality was 1.73, and that for chronic bronchitis or emphysema mortality was 1.79, relative to never smokers. Although the relationship between smoking and chronic bronchitis or emphysema mortality was expected, that for pneumoconiosis mortality was not because smoking has not been associated with the development of

pneumoconiosis [Jacobsen et al., 1977; Muir et al., 1977]. The relationship between pneumoconiosis mortality and smoking remained even when miners with any mention of chronic bronchitis or emphysema on the death certificate were excluded from the analysis. A possible explanation is that among miners who have already developed pneumoconiosis, cigarette smoking may exacerbate their condition, resulting in elevated mortality from pneumoconiosis among miners who smoke. Yet, interactions between either pack-years of smoking or smoking status and mortality either from pneumoconiosis or from chronic bronchitis or emphysema were not statistically significant ($p > 0.2$). An alternative explanation is that there may have been a bias toward over-reporting of pneumoconiosis as a cause of death for miners, particularly among smokers. Such bias could result in an apparent smoking effect and could also obscure the actual relationship between dust exposure and mortality from pneumoconiosis.

Age at start of follow-up was a significant predictor of mortality, which was expected for all causes, but the continued significance of age in the model for pneumoconiosis mortality was initially surprising. One explanation is that because pneumoconiosis can continue to progress, even after dust exposure ceases, mortality from pneumoconiosis increases with age. That is, for a given cumulative dust exposure, the risk of death from pneumoconiosis is greater for an older rather than a younger miner. Another explanation is the possible underestimation of dust exposures for older miners, which may have been manifested as an age effect on mortality. Lastly, physicians may have been more likely to list pneumoconiosis as a cause of death for older miners, regardless of the level of exposure. This may have led to the observed age effect and also may have weakened the relationship with dust exposure.

Radiographic Category and Pneumoconiosis Mortality

In a study of mortality among British coal miners, Miller and Jacobsen [1985] reported that mortality from all nonviolent causes was significantly elevated among miners with progressive massive fibrosis (PMF) (compared to miners without radiographic evidence of pneumoconiosis, or category 0). Although a trend was not observed for increasing mortality with increasing categories of simple CWP, survival was slightly decreased (2–3%) among miners with simple CWP category 1 compared to those without pneumoconiosis (category 0). It is of interest, in the present study, that a relationship was observed between increasing radiographic category of simple pneumoconiosis and increasing mortality from pneumoconiosis (either as the underlying cause or as the underlying or contributing cause), based on proportional hazards models with covariates for age and smoking.

Cause of Death Information and Possible Misclassification Bias

The primary objective of this study was to investigate the exposure-response relationship using both underlying and contributing causes of death. It was thought that analyses using all of the cause of death information would be more sensitive and would be less affected by bias due to disagreement among physicians in determinations of the underlying cause of death [Gau and Diehl, 1982]. In this study of coal miners, there may have been a tendency toward ascribing pneumoconiosis as the underlying cause of death (rather than alternative causes such as chronic bronchitis or emphysema), given the known association between coal mining and pneumoconiosis.

Consistent with this possible bias is the relatively low number of deaths from chronic bronchitis or emphysema as the observed underlying cause (nine deaths, Table III). By using both underlying and contributing causes, fewer deaths from chronic bronchitis or emphysema would be missed. In addition, without the inclusion of contributing causes, it would not be possible to do exposure-response analyses of mortality from chronic bronchitis and emphysema (due to the small number of deaths attributed to either of those causes as the underlying cause).

For pneumoconiosis mortality, clear exposure-response relationships were evident in all analyses using underlying and contributing causes, but equivocal results were obtained in analyses restricted to underlying causes of death. Given the relationship between exposure to respirable coal mine dust and the development of pneumoconiosis [Attfield and Morring, 1992b; Hurley et al., 1987; Jacobsen et al., 1971], it was unexpected to find an equivocal exposure-response relationship for mortality from pneumoconiosis as the underlying cause and yet to find a significant relationship between increasing radiographic category and mortality from pneumoconiosis as the underlying cause. A possible explanation is that knowledge of the radiographic category of pneumoconiosis might have influenced a physician's decision to list pneumoconiosis as the underlying cause of death. Thus, given the range of cumulative exposures experienced among miners with radiographic evidence of pneumoconiosis (which reflects, in part, a range of sensitivities to the effects of dust exposure), it is not surprising that the response variable (pneumoconiosis listed as the underlying cause on the death certificate) would correlate better with radiographic category of pneumoconiosis than with dust exposure.

A potential limitation of exposure-response studies of mortality is that it is not always clear to what extent inferences can be made about the pathologic causes of death based upon death certificate information, or the extent to which determinations of underlying vs. contributing causes of death agree with pathologic findings. The discordance between causes of death listed on the death certificate and clinical or pathology data has been observed [Selikoff, 1992]. In this study, the effects of misclassification bias due to comparison with an external group would be diminished because, in the proportional hazards analyses, the mortality rates are compared within the cohort.

Cumulative Exposure Estimates

An effect of estimating cumulative exposures for the period of beginning work until the start of follow-up in 1969–1971 may have been to underestimate cumulative exposures. However, the magnitude of this underestimation is expected to be small, as is the associated bias in the risk estimates, for the following reasons. For most miners, the largest portion of their cumulative exposures was received prior to the start of follow-up because most of their work experience occurred before follow-up began and because the respirable coal mine dust concentrations were reduced after 1969, when the U.S. coal dust standard was enacted [30 USC 841–845]. Further, an important factor in disease development may be the residence time of dust in the lungs [Maclaren et al., 1989; Hurley et al., 1987], such that the earlier exposures might have a greater influence on disease development and progression. For these reasons, it is unlikely that miners' exposures were underestimated appreciably, particularly among the older miners, who were also those most likely to have developed disease.

Possible Selection Bias

In both the lifetable analyses and the proportional hazards models using either categorical or continuous variables for cumulative exposure, an apparent downward trend in the mortality at high exposures was observed. A possible explanation for this apparent decline in mortality with high exposures is that estimates of exposure may have been biased toward overestimation among miners with high cumulative exposures, resulting in the underestimation of risk. Miners with the highest cumulative exposures are also more likely to be older, and exposure estimation may have been inaccurate both because of recall bias in the occupational histories and because the mean concentrations measured for coal mining occupations in the late 1960s may have been poor reflections of the mean concentrations experienced in jobs worked several decades earlier.

However, since it is more reasonable to suspect that exposures were underestimated rather than overestimated in older miners, a more likely explanation is that miners with high exposures (who were more likely to have developed disease) self-selected out of the workforce before the start of the study. A basis for this explanation is the enactment of black lung benefits in the Federal Coal Mine Health and Safety Act in 1969 (30 USC 901–945), under which miners disabled by pneumoconiosis could receive compensation. A similar decline was seen in exposure-response data involving the *development* of pneumoconiosis, in which the apparent disease prevalence was lower among miners with high exposures [Attfield and Morring, 1992b].

Mortality Risk Among Miners Exposed at the Current U.S. Coal Dust Standard

Based on the proportional hazards model with cumulative exposure as a continuous variable, the RR for pneumoconiosis mortality is 5.92 (95% CI = 2.18–16.10) for miners exposed for a 45-year working lifetime at 2 mg/m^3, the current standard for respirable coal mine dust in the United States (i.e., cumulative exposure of 90 mg-yr/m^3). Because of the significant exposure-response relationship for pneumoconiosis mortality and the no-threshold form of the proportional hazards model, any nonzero exposure would result in predicted RRs greater than 1.0 (Fig. 1). However, the findings based on the no-threshold model are supported by the results of the analysis using cumulative exposure categories, in which the possible effect of exposure on mortality is evaluated directly within each exposure category relative to the reference category. In this categorical analysis, the RRs for pneumoconiosis mortality as an underlying or contributing cause of death were significantly elevated at cumulative exposures *below* 90 mg-yr/m^3 (Table IV).

Given that miners in the reference group had also been exposed to respirable coal mine dust and that there was a loss of statistical power from categorizing the cumulative exposure variable, it is not unexpected that the rate ratios are lower in the categorical model (Table IV) compared to the model using cumulative exposure as a continuous variable (Table V). The proportional hazards model of duration within concentration categories (Table VI) is consistent with the analyses using cumulative exposure, as either a continuous variable or as categorical variables.

For chronic bronchitis and emphysema as an underlying or contributing cause of death, a significant exposure-response relationship was observed in a proportional hazards model using cumulative exposure as a continuous variable. A marginally

significant exposure-response relationship remained when the response was limited to the 26 deaths with any mention of chronic bronchitis or emphysema mortality but *without* any mention of pneumoconiosis, and the magnitude of that exposure effect was doubled. In a proportional hazards model using cumulative exposure categories and controlling for age and smoking, an elevated and statistically significant RR was observed in exposure group IV (88.4–107.0 mg-yr/m^3) relative to the lowest exposure group (0.1–48.9 mg-yr/m^3; Table IV). This analysis provides evidence that miners with cumulative exposures near 90 mg-yr/m^3 are at a significantly elevated risk of mortality from chronic bronchitis and emphysema, apart from the effects of age and smoking.

CONCLUSIONS

Significant exposure-response relationships were found for cumulative exposure to respirable coal mine dust and mortality either from pneumoconiosis or from chronic bronchitis or emphysema as underlying and contributing causes of death. Miners exposed for a working lifetime at or below the current U.S. standard of 2 mg/m^3 for respirable coal mine dust are at increased risk of dying from these causes. Both the coal rank of the dust to which miners are exposed and the radiographic category of pneumoconiosis at the start of follow-up, including simple CWP category 1, are significant predictors of mortality from pneumoconiosis.

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