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Rapid Decline in Lung Function in Coal Miners: Evidence of Disease in Small Airways

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Background: *Coal mine dust exposure can cause both pneumoconiosis and chronic airflow limitation. The contributions of various pathophysiologic mechanisms to dust-related lung function decrements remain unclear.*
Methods: *Clinical and physiological findings were assessed for 15 underground coal miners who had demonstrated accelerated FEV_1 losses (decliners) over 6–18 years. Decliners' findings were evaluated in comparison to a group of 11 miners who had shown relatively stable lung function (referents) during the same period.*
Results: *At follow-up examination, the decliners showed significantly greater mean airway resistance (10.47 vs. 6.78 $cmH_2O/L/s$; $P = 0.05$) and more air trapping (RV/TLC = 37.5 vs. 29.1%; $P < 0.01$) compared to the referents. Decliners also demonstrated more evidence of small airways dysfunction and tended to have more bronchospasm than the referent group. Total lung capacity, lung compliance, diffusing capacity, and chest radiography did not differ significantly between the two groups. After cessation of mine dust exposures, the decliners' mean rate of FEV_1 loss normalized.*
Conclusion: *In a series of working coal miners, accelerated lung function declines were associated with air trapping and evidence of small airways dysfunction. A preventive benefit from controlling dust exposures was suggested.* Am. J. Ind. Med. 56:1107–1112, 2013. © 2013 Wiley Periodicals, Inc.

KEY WORDS: small airway disease; pneumoconiosis; spirometry; obstructive lung disease; coal mining; bronchial hyperreactivity

INTRODUCTION

Coal mine dust exposure is associated with progressive loss of lung function [Rogan et al., 1973; Marine et al., 1988]. Certain dust-exposed miners experience an accelerated decline in ventilatory lung function, compared to the expected age-related decline in the general population [Wang et al., 1999; Beeckman et al., 2001]. A review of the published evidence concluded that exposure to coal mine dust leads to the development of chronic obstructive pulmonary disease, although "the exact nature of the pathology underlying the loss of lung function in miners is still uncertain [Coggon and Newman-Taylor, 1998]."

Several dust-related disorders have been investigated in relation to airflow limitation among coal miners, including bronchitis, interstitial fibrotic processes, and emphysema [Rogan et al., 1973; Hankinson et al., 1977; Lyons et al., 1981; Cockcroft et al., 1982; Ruckley et al., 1984;

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Leigh et al., 1994; Coggon and Newman-Taylor, 1998; Wang et al., 2007; Wade et al., 2011]. Some previous data suggest that small airways disease may also play an important role in the decline of lung function in some coal miners [Seaton et al., 1972]. Improved understanding of the mechanism(s) of the rapid declines may facilitate medical interventions and a reduction in adverse health outcomes.

We previously described a group of coal miners who had experienced clinically important loss of lung function (decliners) during an average 12 years of mining [Wang et al., 1999; Beeckman et al., 2001]. Over time, this group of miners went on to develop more respiratory symptoms and chest illnesses, and experienced a higher risk of death from cardiovascular and non-malignant respiratory diseases compared to matched referent miners with more stable lung function. The current study describes clinical patterns and lung physiology from a series of rapid decliners and referent miners who returned for a follow-up examination that included extensive physiologic, radiologic, and allergy skin testing.

MATERIALS AND METHODS

Participants

Study participants were recruited from the original longitudinal cohort of male underground coal miners who had participated in the U.S. National Study of Coal Workers Pneumoconiosis (NSCWP; see Table I) [Beeckman et al., 2001; Wang et al., 2007]. Each decliner had shown an FEV_1 loss at least 60 ml/year greater than their matched (age, height, smoking status, initial FEV_1) referent miner over a 6- to 18-year interval between NSCWP surveys. An extensive follow-up examination was offered to 62 miners and completed by 26, including 16 of the miners who had originally been matched (eight pairs). The 15 rapid decliners and 11 referent miners who completed the clinical examination were found to be similar for all matched variables (age, height, smoking status, and initial FEV_1) to the entire study group of 634 miners from the NSCWP. Therefore, in assessing differences between the decliners and referent miners, we used group comparisons in the entire 26 miners, and matched pair statistics for the 16 miners in the matched pairs.

This report describes findings from a series of 26 miners who underwent the follow-up evaluation. All participants signed written informed consent and all methods used in the protocol were approved by the National Institute for Occupational Safety and Health (NIOSH) Institutional Review Board. The IRB committee title is: Centers for Disease Control and Prevention IRB #4-NIOSH. The IRB Registration # is 00000186,the Federal-Wide Assurance number (FWA#) is 00001413, and the study approval number is Protocol #HSRB 93-DRDS-01 "Airway Disease in Miners."

TABLE I. Characteristics of Cases and Referents at the Time of Follow-Up Clinical Examination*

	Cases (n = 15)	Referents (n = 11)	*P* value
Age, year	56.3 (9.3)	57.9 (9.4)	0.61
Height, in	69.1 (3.3)	68.7 (2.7)	0.75
Weight, lb	199.6 (32.8)	186.4 (28.3)	0.29
Smoking, pack years	21.3 (16.0)	19.1 (15.7)	0.73
Smoking status			
Current, n (%)	4 (26.7)	3 (27.2)	0.66
Former, n (%)	8 (53.3)	7 (63.6)	
Never, n (%)	3 (20)	1 (9.1)	
Mining tenure			
At initial NSCWP, year	10.7 (9.3)	9.7 (8.9	0.78
At final NSCWP, year	21.9 (9.8)	19.0 (8.3)	0.43
At follow-up examination, year	26.7 (11.2)	24.5 (9.7)	0.61
After final NSCWP, year	4.9 (4.1)	5.6 (4.9)	0.69
Silica exposure, year	11.5	13.6	0.59

*Values are mean (SD) except for smoking status. Pack years = 0 for never smokers.

Clinical Categorization Criteria

Methods and interpretation for physiologic testing, chest radiographs, HRCT scans, and allergy skin testing are described in the online supplement. Each participant was assigned up to four clinical categories: bronchospasm, interstitial lung disease, emphysema, or coal workers' pneumoconiosis (CWP), based upon radiographic and physiologic criteria specified a priori. This categorization was undertaken to help delineate contributions of various mechanisms to the loss of function observed in decliners.

Statistics

SAS® software (SAS Institute, Inc., Cary, NC) was used for data analyses [SAS Institute, Inc., 1999]. Group comparison and matched pair statistics were used to analyze differences between miners with rapid declines and referents. Student's *t*-test was used for continuous variables; Fisher's exact test was used for dichotomous variables. Probability levels of $P \leq 0.05$ were considered significant.

RESULTS

Participants

The 26 miners who participated in the follow-up examination included 16 from the original matched

decliner–referent pairs, and 10 other miners (7 decliners and 3 referents) for whom the corresponding match did not participate in follow-up testing. Table I shows the characteristics of the miners at the time of the follow-up examination. Mean tenure in mining and in jobs exposed to silica was similar for decliners and referents at all evaluations The two groups were also similar with respect to mean age, height, weight, resting heart rate, resting breathing frequency, resting blood pressure, pack years, and proportions of current, former, and never smokers.

Compared to referents, decliners were not categorized as having more emphysema or CWP, and allergy skin test results were similar. Although not statistically significant, decliners did have a higher prevalence of bronchospasm (33%) than the referents (9%, $P = 0.20$). However, none the six miners categorized with bronchospasm reported a history of childhood asthma.

Physiologic Tests

Spirometry indices from the initial and final NSCWP mine surveys and from the follow-up clinical examination are shown in Table II. At baseline, spirometry results were very similar for decliners and referents. As expected from the selection criteria, mean FEV_1 losses from the initial to final mine surveys were greater for the decliners than for the referents. In contrast, mean decline in FEV_1 from the final NSCWP survey to the clinical follow-up examination did not differ between the groups. The decliners had lower mean forced vital capacity (FVC) and FEV_1/FVC ratios compared to the referents, at both the final NSCWP and at the follow-up examination.

Forced Expiratory Flow Between 25% and 75% of FVC ($FEF_{25–75}$) at the initial spirometry did not differ significantly; however, at both the final NSCWP and follow-up examination, the decliners had significantly lower mean $FEF_{25–75}$ (Table II).

After bronchodilator, FVC increased in decliners ($P = 0.07$), suggesting the presence of air trapping, but declined slightly in referents. The FEV_1 response to bronchodilator did not differ between groups.

Results of spirometry while breathing a helium–oxygen mixture are displayed in Table III. The increase in maximal flows at 50% of vital capacity (%$\Delta Vmax_{50}$) when breathing a gas less dense than air (i.e., helium) reflects the resistance of turbulent flow regimes in the large central airways. A low %$\Delta Vmax_{50}$ (<20%) suggests that the predominant site of airflow resistance is in the small ($\leq$2 mm) airways [Pedersen and Ingram, 1987]. Among the 15 miners who performed a valid test, the mean $\Delta Vmax_{50}$ breathing was 18% for decliners and 40% for the referents ($P = 0.12$). The helium–

TABLE II. Comparison of Spirometry Indices From the Initial and Final National Study of Coal Workers' Pneumoconiosis (NSCWP) Surveys and the Follow-Up Examination for Case and Referent Miners

	Cases (n = 15)				Referents (n = 11)			
	FEV_1 (L)	FVC (L)	$FEF_{25–75}$ (L/S)	FEV_1/FVC (%)	FEV_1 (L)	FVC (L)	$FEF_{25–75}$ (L/S)	FEV_1/FVC (%)
Initial NSCWP	4.16 (0.87)	5.52 (1.17)	4.59 (1.61)	75.9 (8.27)	4.06 (0.43)	5.36 (0.62)	5.04 (1.19)	76.0 (6.16)
Final NSCWP	2.96 (0.83)**	4.18 (1.20)*	2.87 (1.17)**	71.9 (10.8)	3.96 (0.54)	5.16 (0.70)	4.35 (0.93)	76.9 (4.7)
Follow-up	2.74 (0.84)*	4.14 (0.95)*	1.81 (0.94)**	65.9 (13.7)	3.59 (0.67)	4.84 (0.68)	2.88 (1.15)	74.1 (7.3)
Follow-up, bronchodilator	2.87 (0.81)**	4.28 (0.95)			3.71 (0.66)	4.82 (0.71)		
Percent predicted[a]								
Initial NSCWP	96.8 (14.5)	104.1 (14.4)			96.6 (12.8)	102.8 (12.2)		
Final NSCWP	74.6 (16.8)**	83.0 (19.6)**			102.2 (14.0)	104.0 (11.7)		
Follow-up	77.2 (22.6)**	89.0 (17.1)**			102.1 (15.3)	104.5 (9.2)		
Percent below LLN[a]								
Initial NSCWP	20	13.3			9.1	0		
Final NSCWP	66.7**	40.0*			0	0		
Follow-up	46.7	33.3			9.1	0		
Slope								
Initial to final NSCWP, ml/year	−93 (30.3)***	−102 (49.5)***			−7 (24.8)	−17 (16.1)		
Final NSCWP to follow-up, ml/year	−20 (70.8)	−2 (83.0)			−34 (25.1)	−31 (31.2)		

Values are mean (SD). *P* values compare cases and referents.
*$P < 0.05$, **$P < 0.01$, ***$P < 0.0001$ *values are mean (SD).
[a]LLNPredicted and LLN (lower limit of normal) values from Hankinson [1999].

TABLE III. Lung Volumes, Diffusing Capacity, Lung Mechanics, and Helium–Oxygen Response for Cases and Referents*

	Cases (n = 15)	Referents (n = 11)	*P* value
TLC, L	6.99 (1.75)	6.79 (0.99)	0.73
RV, L	2.70 (1.13)[a]	2.01 (0.63)	0.06
RV/TLC, %	37.5 (7.8)	29.1 (5.8)	<0.01
FRC, L	3.64 (1.36)	3.04 (0.75)	0.21
D_LCO_{uncorr}, ml/min/mmHg	24.45 (6.48)[b]	27.28 (5.18)[c]	0.14
D_LCO_{corr}, ml/min/mmHg	23.71 (6.86)[b]	27.53 (4.63)[c]	0.24
DL/VA, L/min/mmHg	4.13 (1.15)[b]	4.44 (0.85)[c]	0.49
sRaw, cmH_2O/L/s	10.47 (4.97)[d]	6.78 (3.47)	0.05
C_{stat}, L/cmH_2O	0.245 (0.059)[e]	0.271 (0.071)[f]	0.39
C_{dyn}, L/cmH_2O	0.229 (0.100)[e]	0.256 (0.075)[f]	0.53
ΔVmax50 (%)			
Mean (SD)	18 (21.0)[g]	40 (30.5)[h]	0.12
Responders[a]	33.30%[g]	66.70%[h]	0.31

*Values are mean (SD). *P*-values compare cases and referents.
[a]Responders were those whose ΔVmax50 increased by at least 20% when breathing the helium–oxygen mixture.
[b]n = 14.
[c]n = 10.
[d]n = 13.
[e]n = 11.
[f]n = 8.
[g]n = 9.
[h]n = 6.

oxygen response was lower among miners with bronchospasm (10.1%) than among those without (35.1%; $P = 0.07$).

Table III displays the results of lung volumes, diffusing capacity, and lung mechanical indices at the follow-up examination. The decliners demonstrated a higher mean ratio of residual volume to total lung capacity (RV/TLC) than the referents ($P < 0.01$), evidence of air trapping. Decliners also had higher ($P = 0.05$) mean airways resistance (sRaw). RVs were high in the decliners but not the referent miners (118% vs. 88% predicted), while TLC was normal in both groups (108% and 106% predicted, respectively). Lung compliance and diffusing capacity did not differ among decliners and referents. Matched pair *T*-tests in the 16 miners in decliner–referent pairs confirmed the results of grouped analysis among all 26 miners: RV/TLC% was greater among decliners ($P < 0.001$), but there were no significant differences for either TLC or DLCO.

DISCUSSION

This study reports an extensive evaluation of respiratory physiology in a group of coal miners who had prospectively demonstrated clinically important FEV_1 losses during an average 12 years of mining. The physiologic findings in the decliners were compared to referent miners who were similar to the decliners in age, height, smoking status, and initial FEV_1, but whose lung function was relatively stable over the 12 years.

At the follow-up examination, the decliners demonstrated significantly higher airways resistance and more air trapping than the referents. The decliners were similar to the referent miners for mean TLC, lung compliance, and diffusing capacity, findings which argue against a substantial contribution from either emphysema or fibrotic lung disease to their FEV_1 losses. In contrast, three findings from the study suggest the importance of disease in small peripheral airways: On average miners with rapid declines (1) showed little change in flow rates after breathing helium–oxygen; (2) showed improved air trapping after bronchodilator inhalation (increased FVC, without a change in FEV_1); and (3) had significantly lower mid-flows ($FEF_{25–75}$). Taken together, these results suggest an important contribution of small airways disease to the lung function declines observed in the study coal miners.

Findings from the current study are consistent with a number of previous American and British studies which found that coal miners experience exposure-related declines in FVC that are often nearly parallel to the FEV_1 losses, suggesting a restrictive defect [Pern et al., 1984; Soutar and Hurley, 1986; Attfield and Hodous, 1992]. TLC has not generally been reported in previous studies. However, when lung volumes have been measured in groups of working coal miners, increases in RVs have been documented [Morgan et al., 1971, 1972; Lapp, 1972]. In the current study, the miners who demonstrated excessive declines in FVC, FEV_1, and the ratio of FEV_1/FVC also demonstrated significant elevations in RV/TLC ratios, but no difference in TLC. Thus, the declines of lung function in the study miners appears to have been principally due to obstruction and associated air trapping, rather than underlying restrictive mechanisms that are typically associated with fibrotic interstitial lung diseases.

The pathologic lesion of pigmentation and fibrosis specific to walls of the respiratory bronchioles of workers exposed to mineral dusts has been labelled "mineral dust-related airway disease" (MDAD) [Heppleston, 1953; Churg and Wright, 1983; Churg et al., 1985; Gevenois et al., 1998]. This pigmented lesion is considered a non-specific reaction of the small airways to inorganic dusts [Churg and Wright, 1983]. In an important study correlating physiology with dust-related small airways pathology, Churg et al. reported on 174 patients who had undergone pulmonary resection for carcinoma of the lung [Churg et al., 1985]. A detailed occupational questionnaire identified 53 individuals who were exposed to mineral dusts. Of these 53 patients, 13 (24.5%) had pathologic changes of MDAD in their respiratory bronchioles. Compared to matched (age, gender, and smoking history) controls, the patients with MDAD had significantly reduced FVC, FEV_1, and $FEF_{25–75}$, suggesting that the pathologic lesions in the small airway had important functional consequences. The patients with MDAD also had

an increased RV/TLC ratio and normal TLC, findings quite similar to the miners with rapid declines in the current study. The similarity in physiologic findings between the current study and that of Churg et al. may reflect a comparable small airways pathology induced by mineral dusts. The findings from the current study, taken together with prior studies do suggest that, in addition to the previously documented mechanisms of dust-induced bronchitis, emphysema, and interstitial inflammatory and fibrotic responses, in some miners, small airways disease may lead to clinically important declines in lung function.

Of note, many of the rapid decliners in the current study demonstrated normalization in their rate of FEV_1 decline after they ceased mining. This finding suggests a preventive benefit for longitudinal monitoring of lung function among dust exposed workers, and more strict control of exposures if excessive loss of lung function is detected.

The principal limitation of this study is the small number of miners who performed the clinical evaluations. The participants may not be representative of the 264 decliner and referent miners from the larger study or of affected individuals in the general mining population. However, the physiologic abnormalities documented in association with rapidly declining lung function are quite consistent with findings from a number of previous published studies of coal miners and offer a unique picture of the respiratory physiology for at least a sub-group of miners who develop work-related respiratory impairments.

Additionally, decliners and referents were similar for smoking status**;** however, only three of the decliners and one referent miner reported never smoking and it was not possible to fully separate dust and smoking effects.

Summary

We performed extensive clinical follow-up examinations among a series of 26 underground coal miners whose lung function had been followed longitudinally for an average of 12 years. The findings indicated that rapid declines in FVC and FEV_1 observed in this group of miners were associated primarily with airflow obstruction and air trapping, rather than an underlying restrictive process. Additionally, the testing results suggest that for some individuals, important lung functional losses observed during coal mining work may be related at least in part to disease in the small airways, corroborating an important physiologic effect of the mineral dust airway lesions described by Churg et al.

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Supporting Information

Additional supporting information may be found in the online version of this article at the publisher's web-site.