

inversely proportional to dust exposure. Thus, Soutar and Hurley concluded that exposure to respirable coal mine dust can cause severe respiratory impairment, even without the presence of simple CWP or PMF. They speculated that the pathology of coal dust-induced lung disease differs from that induced by smoking.

Centriacinar emphysema in coal miners has been associated with the amount of dust retained in their lungs at the end of life and with their dust exposures during life and the years worked underground (Ruckley *et al.*, 1984; Leigh *et al.*, 1983, 1994). Emphysema in coal miners has also been associated with pathological measurements of pneumoconiosis (Cockcroft *et al.*, 1982a), and with lung function decrements and irregular opacities on chest x-rays (Cockcroft *et al.* 1982b,c; Cockcroft and Andersson, 1987).

Recent studies from China (Wang, *et al.*, 1997) and the European community (Bourgard, *et al.*, 1998; Carta, *et al.*, 1996; Lewis, S., *et al.*, 1996) have also supported the British and U.S. findings which demonstrated the correlation between occupational exposure to coal dust and respiratory symptoms and loss of lung function in miners.

Wang, *et al.* (1997) examined lung function in underground coal miners and other workers from several factories in Chongqing, China. For their study, information was obtained on exposure duration, results of radiographic tests, and smoking history. Pulmonary function tests were performed, providing the Forced Expiratory Volume in one second (FEV₁), the Forced Vital Capacity (FVC), and FEV₁/FVC data. Additionally, the diffusing capacity for carbon monoxide (DL_{CO}) was measured. This is an indicator of diffusion impairment at the "blood-gas barrier" which may occur, for example, when this barrier becomes thickened (West, 1990; 1992). Wang, *et al.* (1997) found that there was impairment of pulmonary function among the coal miners and they had evidence of obstructive disease. Like other studies, such effects were observed among coal miners even in the absence of simple CWP. Pulmonary function was further decreased when simple CWP was present. This study did not provide exposure measurements and there was no consideration of exposure-response relationships. Also, silica exposures and their potential effects were not examined in the underground coal miners.

As noted above, Bourgard, *et al.* (1998) was interested in the earlier stages of simple CWP (*i.e.*, Categories 0/

1 and 1/0) and the prognostic role of cumulative dust exposure, smoking patterns, respiratory symptoms, lung CT scans, and lung function indices for chest x-ray worsening and evolution to simple CWP category 1/1 or higher. Over a 4-year period, they studied French coal miners who were employed in underground and surface mines. Bourgard, *et al.* (1998) found that, at the first medical examination, the ratio of the Forced Expiratory Volume in one second (FEV₁) to the Forced Vital Capacity (FVC) (*i.e.*, FEV₁/FVC) and other airflows determined from a forced expiration (West, 1990; 1992) were lower among miners who later developed simple CWP category 1/1 or higher. These miners also experienced more wheezing at the first medical examination. Thus, the results of their study suggested that lung function changes may serve as an early indicator of miners who are at increased risk of developing simple CWP and PMF and who should be monitored more closely.

Carta, *et al.* (1996) have examined the role of dust exposure on the prevalence of respiratory symptoms and loss of lung function in a group of young Italian coal miners (*i.e.*, mean age at hire 28.9 years, mean age at first survey 31.2 years). These miners worked underground and were exposed to lignite (*i.e.*, low rank coal) which had a 5–7% sulfur content. They were followed for a period of 11 years, from 1983 and 1993. Carta, *et al.* (1996) found few abnormalities on miner chest x-rays taken throughout the 11-year study. However, there was an increased prevalence of respiratory symptoms and loss of lung function. This was particularly noteworthy since dust exposures were often below 1.0 mg/m³; the cumulative dust exposure for the whole cohort was 6.7 mg-yr/m³ after the first survey. Thus, Carta, *et al.* (1996) demonstrated that miners experience respiratory effects of exposure to dust generated from a lower rank coal and at lower concentrations. They have recommended yearly measurements of lung function for miners.

Lewis, *et al.* (1996) studied a group of British miners, many of whom entered the coal industry in the 1970s. Based upon chest x-rays, the miners had no evidence of simple CWP or PMF. The objective of this study was to determine whether coal mining (*i.e.*, exposure to respirable coal mine dust) is an independent risk factor for impairment of lung function. Lewis, *et al.* (1996) found that there was a loss of lung function in miners (smokers and nonsmokers), particularly among miners who were under approximately 55 years of age. For miners who smoked, there

was a greater loss of lung function than in nonsmoking miners with the same level of exposure to respirable coal mine dust. Above age 55, the loss of lung function was similar for miners and their controls, although all smokers continued to exhibit a greater loss of lung function than nonsmokers. Lewis, *et al.* (1996) concluded that the deficits in lung function may occur in the absence of simple CWP and PMF, and independent from the effects of smoking.

There have been two recent mortality studies that have demonstrated a relationship between exposure to respirable coal mine dust and development of COPD. This association was reported by Kuempel, *et al.* (1995) in the U.S., and by Meijers, *et al.* (1997) in the Netherlands. These two groups of investigators have reported that occupationally-induced COPD (*e.g.*, chronic bronchitis, emphysema) can occur in miners, with or without the presence of simple CWP or PMF. They also found that the risk of premature mortality from COPD was elevated among miners and could be separated from the effects of smoking and age.

Kuempel, *et al.* (1995) found an increase in relative risk (RR) of premature mortality from COPD among U.S. coal miners who participated in the NSCWP from 1969 through 1971. In their data analysis, the exposure-response relationship was evaluated using the Cox proportional hazards model. This model assumes that the hazard ratio between nonexposed and exposed groups does not significantly change with time. When fitting a curve to the data (*e.g.*, log-linear), cumulative exposure was expressed as a categorical or continuous variable. Due to model limitations (*e.g.*, less statistical power, influence of category scheme, use of lowest exposure group for comparisons vs. use of non-exposed group), Kuempel, *et al.*, (1995) believed that the exposure data should be expressed as a continuous variable. If, for example, the cumulative exposure was 90 mg-yr/m³ (*i.e.*, 2 mg/m³ for 45 years), then the relative risk of mortality from chronic bronchitis or emphysema was 7.67. Kuempel, *et al.* (1995) also showed that relative risk decreased with lower cumulative exposures (*i.e.*, below 90 mg-yr/m³) and increased with higher cumulative exposures (*i.e.*, above 90 mg-yr/m³). Thus, these investigators demonstrated a statistically significant exposure-response relationship for COPD.

Meijers, *et al.* (1997) have shown, among Dutch miners, reductions in lung volumes and capacities are good predictors of the increased risk of