

approximately 5,800 miners who worked underground from the late 1970s to mid-1980s. Morfeld, *et al.* observed increases in relative risks (RRs) of developing early x-ray changes, category 0/1, that were exposure-dependent. Relative risks (RRs) increased with higher dust concentrations.

Starzynski, *et al.* (1996) conducted a mortality study on a group of 11,224 Polish males diagnosed with silicosis, simple CWP, or PMF between 1970 and 1985. This cohort was subdivided by occupation into four subcohorts: Coal miners (63%); employees of underground work enterprises (8%) (*i.e.*, drift cutting and shaft construction jobs); metallurgical industry and iron, and nonferrous foundry workers (16%); and refractory materials, china, ceramics and quarry workers (13%). The investigators found that coal miners had a slight, statistically significant excess overall mortality (*i.e.*, all causes) as indicated by a standardized mortality ratio (SMR) of 105 (with a 95% confidence interval (C.I.) of 100–110). Also, excess of deaths from diseases of the respiratory system among coal miners was nearly four times that of the referent population (SMR of 383 with a 95% C.I. of 345–424). The study of Starzynski, *et al.* (1996) agrees with others that there is premature mortality among coal miners from simple CWP and PMF. Unfortunately, there is little or no information presented on miner work history, exposure assessment (*e.g.*, respirable coal mine dust, silica), and mine environment (*e.g.*, coal rank(s), underground vs. surface mining).

Yi and Zhang (1996) conducted a study to measure the progression from simple CWP to PMF or death among a cohort of 2,738 miners with simple CWP who were employed at the Huai-Bei coal mine in China. Relative risks (*i.e.*, RRs) were calculated for progression from simple CWP category 1 to simple CWP category 3 and for progression from simple CWP category 3 to death. Their results demonstrated that miners with simple CWP category 1 are at risk of developing simple CWP category 2 and simple CWP category 3 (*e.g.*, RRs of 1.101 and 2.360, respectively). They also found that miners with PMF had a decreased life expectancy. Other risk factors for development of PMF included long-term work underground, and drilling. This study was limited by a lack of exposure assessment, estimation of miner smoking histories, and use of a radiological classification system that differs from that of the ILO.

Hurley and Maclaren (1987) studied British coal miners who were examined between 1953 and 1978, over 5-year

intervals. They have shown that exposure to respirable coal dust increases the risks of developing simple CWP and of progressing to PMF. As seen in their data analysis, these responses were dependent upon dust concentration and coal rank. That is, greater responses were seen at higher dust concentrations and with higher rank coal (*i.e.*, increasing percent carbon). The investigators also noted that estimated risks were unaffected by changes in the proportion of miners with simple CWP who transferred jobs. The authors concluded that “limiting exposure to respirable coal dust is the only reliable way of limiting the risks of radiological changes to miners.”

#### b. Other Health Effects

As noted in Table V–2, there were 21 studies in which the loss of lung function (LLF) was examined in coal miners. Fourteen of these studies also included an evaluation of respiratory symptoms (RS) in the miners. There were nine studies describing chronic obstructive pulmonary disease (COPD) in miners.

Henneberger and Attfield (1997; 1996), Kuempel, *et al.* (1997), Seixas, *et al.* (1993), Attfield and Hodous (1992), and Seixas, *et al.* (1992) evaluated data from pulmonary function tests and standardized questionnaires to miners in the NSCWP. A common finding in their studies was an increase in respiratory symptoms such as cough, shortness of breath, and wheezing. The symptoms were dependent upon the dust concentration to which the miners had been exposed, with more pronounced symptoms occurring after long-term exposures to higher exposure levels. These studies also demonstrated that a loss of lung function occurred among miners.

Attfield and Hodous (1992) studied U.S. miners who had spent 18 years underground (on average) and who participated in Round 1 (1969–1971) of the NSCWP. They observed that greater reductions in pulmonary function were associated with exposure to higher ranks of coal (*i.e.*, anthracite vs. bituminous vs. lignite). Using linear regression models, Kuempel *et al.*, (1997) predicted the excess (exposure attributable) prevalence of lung function decrements among miners with cumulative exposures to respirable coal mine dust of 2 mg/m<sup>3</sup> for 45 years (*i.e.*, 90 mg-yr/m<sup>3</sup>). The excess prevalence estimated were 315 and 139 cases per thousand for forced expiratory volume in one second (FEV<sub>1</sub>) of <80% and <65% of predicted normal values, respectively, among never-smoking miners (a sub-group of 977 NSCWP

participants studied in Seixas *et al.*, 1993). Such reductions in FEV<sub>1</sub> are clinically significant; FEV<sub>1</sub> <80% (of predicted normal values) is a measure that is used to determine ventilatory defects (American Thoracic Society, 1991). Three recent studies found impaired FEV<sub>1</sub> to be a predictor of increased pre-mature mortality (Weiss, *et al.*, 1995; Meijers, *et al.*, 1997; Hansen *et al.*, 1999).

Seixas, *et al.* (1993) conducted an analysis of 977 underground coal miners who began working in or after 1970 and were participants of both NSCWP Round 2 (1972–1975) and Round 4 (1985–1988). They found a rapid loss of lung function in miners and further declines in lung function with continuing exposure to coal mine dust. Collectively these studies have shown that the prevalence of decreased lung function was proportional to cumulative exposure. That is, with exposure to higher coal dust levels over a working lifetime, there were more miners who experienced a loss of lung function. Also, the types of respiratory symptoms and patterns of pulmonary function decrements observed by both Attfield and Hodous (1992) Seixas, *et al.* (1992;1993) are characteristic of COPD.

The U.S. findings on respiratory symptoms and loss of lung function in miners have agreed with those of previous British studies by Marine, *et al.* (1988) and Soutar and Hurley (1986). Marine, *et al.* (1988) analyzed data from British coal miners and focused their attention on respiratory conditions other than simple CWP and PMF. In particular, they examined the Forced Expiratory Volume in one second (FEV<sub>1</sub>) among smoking and nonsmoking miners and, on the basis of reported respiratory symptoms, identified those miners with bronchitis. Using these data, logistic regression models were used to estimate the prevalence of chronic bronchitis and loss of lung function. Marine, *et al.* concluded that both exposure to respirable coal mine dust and smoking independently cause decrements in lung function; their contributions to COPD appeared to be additive in coal miners.

Soutar and Hurley (1986) examined the relationship between dust exposure and lung function in British coal miners and ex-miners. The men who were studied were employed in coal mines in the 1950s and were followed up and examined 22 years later. These miners and ex-miners were categorized as smokers, ex-smokers, or nonsmokers. The Forced Expiratory Volume in one second (FEV<sub>1</sub>), the Forced Vital Capacity (FVC), and the FEV<sub>1</sub>/FVC ratios decreased in all study groups and these reductions in lung function were