# Mapping and Prediction of Coal Workers' Pneumoconiosis with Bioavailable Iron Content in the Bituminous Coals

### Xi Huang,<sup>1</sup> Weihong Li,<sup>1</sup> Michael D. Attfield,<sup>2</sup> Arthur Nádas,<sup>1</sup> Krystyna Frenkel,<sup>1</sup> and Robert B. Finkelman<sup>3</sup>

<sup>1</sup>Department of Environmental Medicine and NYU Cancer Institute, New York University School of Medicine, New York, New York, USA; <sup>2</sup>Division of Respiratory Diseases Studies, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Morgantown, West Virginia, USA; <sup>3</sup>U.S. Geological Survey, Reston, Virginia, USA

Based on the first National Study of Coal Workers' Pneumoconiosis (CWP) and the U.S. Geological Survey database of coal quality, we show that the prevalence of CWP in seven coal mine regions correlates with levels of bioavailable iron (BAI) in the coals from that particular region (correlation coefficient r = 0.94, p < 0.0015). CWP prevalence is also correlated with contents of pyritic sulfur (r = 0.91, p < 0.0048) or total iron (r = 0.85, p < 0.016) but not with coal rank (r = 0.59, p < 0.016)p < 0.16) or silica (r = 0.28, p < 0.54). BAI was calculated using our model, taking into account chemical interactions of pyrite, sulfuric acid, calcite, and total iron. That is, iron present in coals can become bioavailable by pyrite oxidation, which produces ferrous sulfate and sulfuric acid. Calcite is the major component in coals that neutralizes the available acid and inhibits iron's bioavailability. Therefore, levels of BAI in the coals are determined by the available amounts of acid after neutralization of calcite and the amount of total iron in the coals. Using the linear fit of CWP prevalence and the calculated BAI in the seven coal mine regions, we have derived and mapped the pneumoconiotic potencies of 7,000 coal samples. Our studies indicate that levels of BAI in the coals may be used to predict coal's toxicity, even before large-scale mining. Key words: bioavailable iron, calcite, coal, chronic obstructive pulmonary disease, COPD, pneumoconiosis. Environ Health Perspect 113:964-968 (2005). doi:10.1289/ehp.7679 available via http://dx.doi.org/ [Online 15 April 2005]

Coal remains a major energy resource worldwide. In the United States, > 50% of electricity is generated in coal-fired power plants. Recent debate in the United States has focused on increasing coal use. In fact, energy costs from a new coal power plant are low, between \$0.035 and \$0.04/kW-hr (Jacobson and Masters 2001). However, coal mining causes environmental problems such as acid mine drainage, whereas the inhaled coal particles at the work place may lead to the development of coal workers' pneumoconiosis (CWP; Castranova and Vallyathan 2000; Demchak et al. 2004). According to the National Institute of Occupational Safety and Health (NIOSH 2003), CWP deaths accounted for half of the pneumoconiosis deaths during the 10-year period from 1990 to 1999. Coal mining can also increase the risk of developing asthma and chronic obstructive pulmonary disease (COPD), such as emphysema and chronic bronchitis (Attfield and Kuempel 2003; Ruckley et al. 1984; Soutar et al. 2004). Among the occupations listed by the U.S. Census industry code, coal mining is the highest risk job associated with asthma and COPD death, with a proportionate mortality ratio of 1.98 [95% confidence interval (CI), 1.84-2.12, adjusted for age, sex, and race), compared with the second highest risk job of trucking service of 1.29 (95% CI, 1.22-1.37) (NIOSH 2003). Federal "black lung" program payments totaled > \$1.5 billion for nearly 190,000 beneficiaries in 1999. Health and environmental costs, such as occupational lung disease compensation, can bring the total cost from \$0.035–0.04/kW-hr to as high as \$0.0556–0.083/kW-hr (Jacobson and Masters 2001). If we can predict the toxicity of coal before mining, we may be able to develop screening and prevention programs that carefully monitor early adverse effects and, thus, reduce health care costs related to the coal use.

Coal is an aggregate of heterogeneous substances composed of organic and inorganic materials. The four major coal types ranked in order of increasing heat value are lignite, subbituminous, bituminous, and anthracite. The inorganic portion of coal can range from a few percent to > 50% (by weight) and is composed of phyllosilicates (kaolinite, illite, etc.), quartz, carbonates, sulfides, sulfates, and other minerals (Meyers 1982). In general, aluminum and iron are the main metals in the coals. Arsenic, nickel, zinc, cadmium, cobalt, and copper are trace metals that represent only a very small fraction of the mineral matter (Finkelman 1995).

Iron is the best-known transition metal capable of producing oxidants through Fenton, Haber-Weiss, or autoxidation reactions (Huang 2003). However, not all iron compounds in the coals are bioavailable for oxidant formation and subsequent adverse health effects. We have previously defined bioavailable iron (BAI) as the iron released in 10 mM phosphate solution, pH 4.5, which mimics the phagolysosomes of cells (Huang et al. 1998). Based on 30 coal samples from three coal mine regions, we have shown that levels of BAI in the coals correlated with the prevalence of CWP, and it was the BAI in the coals that transactivated the two important transcription factors of activator protein-1 and nuclear factor of activated T cells (Huang et al. 2002; Zhang et al. 2002; Zhang and Huang 2002).

The purpose of this study was to validate our hypothesis regarding BAI using a model based on various chemical interactions in the mixed coal dusts. Because it is impossible to obtain and measure BAI in all coal samples from the period when epidemiologic studies were performed during 1969 and 1971, we used the U.S. Geological Survey (USGS) database of coal quality (Bragg et al. 1998) for calculating BAI in each coal mine region. The USGS database is the largest publicly available database containing information on the chemistry and properties of U.S. coals. For the calculation of BAI for each individual coal, molar amounts of pyritic sulfur ( $S_{py}$ ) per gram of dry coal, as well as sulfate, calcium oxide (CaO), and total iron were taken into account. For correlation with CWP prevalence, other factors that were previously thought to contribute to CWP were also incorporated, such as coal rank or quartz. CWP prevalence in seven coal mine regions has been shown to be significantly correlated with the levels of BAI from the same region. Using the model that we developed, pneumoconiotic potencies of 7,000 coal samples were derived and mapped in the present study.

## **Materials and Methods**

*Coal data and samples.* Our hypothesis has been that BAI is the active component in the coals that induces CWP. If that proves to be the case, then the differences in the levels of BAI in the coals may be responsible for the observed regional differences in the prevalence

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Address correspondence to X. Huang, Department of Environmental Medicine and NYU Cancer Institute, New York University School of Medicine, 550 First Ave., PHL Room 802, New York, NY 10016 USA. Telephone: (212) 263-6650. Fax: (212) 263-6649. E-mail: xihuang@env.med.nyu.edu

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of CWP. To test our hypothesis, we used CWP prevalence data from the first National Study of Coal Workers' Pneumoconiosis (NSCWP) (Morgan et al. 1973) as well as physicochemical data from the USGS coal quality database (Bragg et al. 1998). In 1969, the first round of the NSCWP selected 31 coal mines, of which 29 were bituminous and two were anthracite mines (Morgan et al. 1973). Eight mines were located in Pennsylvania (PA; two anthracite, six bituminous); nine in West Virginia (WV); three in Kentucky (KY); two each in Virginia (VA), Alabama (AL), Illinois, and Utah (UT); and one each in Ohio (OH), Indiana (IN), and Colorado (CO). Participation in the first round was 90.5%. A total of 9,076 miners were fully examined, of which 8,553 were bituminous workers and 523 were anthracite workers. Because the properties of anthracite are different from bituminous coals and the number of anthracite miners was small, we excluded the two anthracite mines in the PA coal mine region from the present study. Only bituminous coals, including the six in PA, were used for BAI calculation and its correlation with CWP prevalence.

Based on the names of the coal mines, counties, and states, we searched the USGS coal quality database (Bragg et al. 1998) and matched 94 coal samples from 24 coal mines within seven states. These are bituminous coals obtained from mines within the same state, county, and coal seam as those samples used in the first NSCWP (Bragg et al. 1998). Most of the samples in the USGS database were collected in 1975–1985.

**BAI calculation.** BAI consists mainly of water-soluble iron, such as ferrous and ferric sulfate, which can be originally present in the coals or can be obtained by the oxidation of pyrite (FeS<sub>2</sub>). Another possible source of BAI is acid solubilization of siderite (FeCO<sub>3</sub>) or ferrous silicate (FeSiO<sub>3</sub>). Using the USGS coal database (Bragg et al. 1998), we have calculated levels of BAI in each coal. Considering that most BAI originates from FeS<sub>2</sub> oxidation (Huggins et al. 1983), we used the following formula for the calculations:

$2\text{FeS}_2 + 7\text{O}_2 + 2\text{H}_2\text{O}$	
$\rightarrow 2FeSO_4 + 2H_2SO_4$	

One mole  $FeS_2$  will produce 1 mol BAI as ferrous sulfate (FeSO<sub>4</sub>) and 1 mol sulfuric acid (H<sub>2</sub>SO<sub>4</sub>). However, levels of FeS<sub>2</sub> in the USGS coal database (Bragg et al. 1998) were not measured directly. Because only S<sub>py</sub> content is available in the database, reaction 1 shows that 1 mol S<sub>py</sub> will produce 0.5 mol BAI and 0.5 mol H<sub>2</sub>SO<sub>4</sub>.

Previous studies have shown that BAI is stable only in an acidic environment (Huang et al. 1994). If calcite ( $CaCO_3$ ) is present in the coal,  $CaCO_3$  will consume the acid and neutralize the pH as follows:

$$CaCO_3 + H_2SO_4$$
  

$$\rightarrow CaSO_4 + H_2O + CO_2$$
[2]

Increasing the pH would facilitate ferrous and ferric ion oxidation to goethite (FeOOH), which is water insoluble and thus not bioavailable for redox reactions (Lowson 1982; Singer and Stumm 1969). Therefore, no BAI will accumulate when CaCO<sub>3</sub> is present. If CaCO<sub>3</sub> is absent in the coals,  $H_2SO_4$  produced from reaction 1 would solubilize other iron compounds (e.g., FeCO<sub>3</sub>) and release more BAI as follows:

$$FeCO_3 + H_2SO_4$$
  
 $\rightarrow FeSO_4 + H_2O + CO_2$  [3]

Based on the above chemical reactions, we concluded that the total available molar amounts of  $H_2SO_4$  in a given amount of coal would be as follows:  $[H_2SO_4] = [\text{one-half } S_{py} + \text{sulfate} - \text{CaCO}_3]$ . If  $[H_2SO_4] \leq 0$ , the acid is completely consumed by  $\text{CaCO}_3$  and, concomitantly, iron will be oxidized. Therefore, there will be no BAI in that coal. If  $[H_2SO_4] > 0$ , the excess acid would stabilize BAI and, possibly, also leach out other iron compounds, such as FeCO<sub>3</sub>, thus releasing additional BAI.

Based on the USGS coal database,  $S_{py}$  and sulfates as percentages of coal are available for calculations. CaCO<sub>3</sub> and FeCO<sub>3</sub> in the coals were not measured. However, levels of CaO and the total amount of iron (shown as Fe<sub>2</sub>O<sub>3</sub>) were measured in high-temperature ashes of the coals; the ash yield in the coal is available from the USGS database. We assumed that the CaO was all derived from CaCO<sub>3</sub> and the iron was derived from FeS<sub>2</sub>, two of the most common minerals in coal, thus maximizing the CaCO<sub>3</sub> and FeS<sub>2</sub> estimates. Because 1 mol CaO is formed by the decomposition of 1 mol CaCO<sub>3</sub> at high temperature, this allowed us to use the same molar amounts of CaO as measures of CaCO<sub>3</sub>. We then calculated the millimolar amounts of S<sub>py</sub> and sulfates per 100 g dry coal in each individual coal. The millimolar amounts of CaO and total iron (Fe<sub>2</sub>O<sub>3</sub>) per 100 g dry coal were also calculated after taking into consideration the ash yield in each individual coal.

#### Results

Table 1 shows that marked regional differences in the prevalence of CWP existed, with the disease being most common in bituminous miners of PA [cumulated prevalence of 45.4%, including diseases of categories 1, 2, 3 (Henry 2002; Jacobsen 1991), and progressive massive fibrosis] and least common in miners of CO (4.6%), after adjusting for age and years spent underground (Morgan et al. 1973). The follow-up studies at the same mines (in 1972-1975, 1977-1981, 1985-1988, and 1996-2002) have shown that the overall prevalence of CWP decreased in the United States because of the lowered dust levels, but the regional difference persisted with a greater risk in eastern coal miners (PA and WV) than in western coal miners (UT and CO) (Attfield and Morring 1992a; Attfield and Seixas 1995; Centers for Disease Control and Prevention 2003; Goodwin and Attfield 1998). After taking into consideration the slight differences present in exposure concentration or mining techniques, as well as the X-ray reader variation or changes in X-ray standard, these epidemiologic results indicate that physicochemical characteristics of the coals responsible for toxicity are different in the eastern and western states (Attfield and Morring 1992a). Table 1 also summarizes the molar ratio of C:H as one of the indicators of coal rank, Spy, sulfate, silicon dioxide content, CaO, iron oxide, arsenic, and nickel (mmol/100 g dry coal) from the USGS coal quality database (Bragg et al.). The sample sizes varied from one state to another because of the availability of coal samples in the USGS database. There were also wide variations on physicochemical characteristics,

Table 1. CWP prevalence and mean levels (± SD; mmol/100 g dry coal) of physicochemical properties of coal samples from each coal mine region.

State	No. of mines	No. of samples	CWP (%)	MR (C:H)	SiO <sub>2</sub>	CaO	S <sub>py</sub>	SO <sub>4</sub>	Fe <sub>2</sub> O <sub>3</sub>	As	Ni
PA	3	9	45.35	1.28 ± 0.12	71.05 ± 46.22	3.97 ± 2.13	34.76 ± 21.88	1.23 ± 1.06	12.48 ± 7.4	$0.33 \pm 0.4$	0.26 ± 0.17
OH	1	6	31.80	$1.09 \pm 0.07$	84.66 ± 26.96	5.23 ± 7.17	37.08 ± 20.03	1.37 ± 1.20	12.86 ± 8.8	0.17 ± 0.13	$0.14 \pm 0.19$
KY	4	13	29.00	$1.20 \pm 0.07$	69.82 ± 24.06	$5.69 \pm 9.70$	25.48 ± 30.53	$0.43 \pm 0.6$	9.78 ± 9.11	$0.15 \pm 0.22$	$0.22 \pm 0.11$
WV	4	8	28.25	$1.32 \pm 0.13$	49.53 ± 24.00	4.58 ± 3.17	17.89 ± 29.57	0.21 ± 0.21	7.27 ± 8.22	0.22 ± 0.25	$0.13 \pm 0.05$
AL	1	13	16.70	$1.26 \pm 0.07$	71.84 ± 49.59	2.88 ± 2.96	17.07 ± 19.74	0.11 ± 0.99	8.85 ± 5.86	$0.40 \pm 0.29$	0.17 ± 0.13
UT	2	4	13.10	$1.08 \pm 0.07$	40.82 ± 8.51	7.33 ± 5.27	7.97 ± 4.35	$0.16 \pm 0.06$	2.69 ± 2.61	$0.01 \pm 0.00$	$0.03 \pm 0.00$
CO	9	41	4.60	$1.04 \pm 0.06$	117.3 ± 82.53	4.61 ± 2.3	3.43 ± 3.08	0.21 ± 0.14	3.68 ± 1.86	$0.03 \pm 0.05$	0.07 ± 0.06

MR, molar ratio.

[1]

as reflected by large SDs, which were probably due to the heterogeneity of coal samples. Levels of coal rank and silica content, two parameters that were previously thought important in contributing to CWP development, do not vary as much as CWP prevalence does from east to west (Table 1). CaO, a product from the decomposition of CaCO<sub>3</sub> in the high-temperature ashes of the coals, does not differ much from one state to another. In general, levels of S<sub>py</sub>, total iron, arsenic, and nickel are higher in the eastern coal mine regions (PA, OH, KY, WV) than in western coal mine regions (UT and CO).

The average levels of total  $H_2SO_4$  (1/2  $S_{py}$ + sulfate), the amount of acid available for solubilization of other iron compounds (1/2 S<sub>py</sub> + sulfate - CaO), total iron, and BAI in each coal mine region are summarized in Table 2. In calculating BAI, we discovered that the amount of BAI in the coal should be equal to the lesser value between the amount of available acid (1/2  $S_{py}$  +  $SO_4^{2-}$  – CaO) and Fe<sub>2</sub>O<sub>3</sub>: *a*) if the coal has an excessive amount of acid and a limited amount of iron, BAI will be limited by the amount of iron present; and b) if the coal has less acid but more iron present, BAI will then be limited by the amount of acid because excess iron cannot be solubilized and therefore cannot become bioavailable. Table 2 shows the average levels of BAI (mmol/100 g dry coal) from seven states with corresponding CWP prevalence reported in the first NSCWP (Morgan et al. 1973).

Table 3 shows a very good correlation between CWP prevalence and BAI (correlation coefficient r = 0.94; 95% CI, 0.66–0.999; p < 0.0015), as well as with S<sub>py</sub> (r = 0.91; 95% CI, 0.35–0.99; p < 0.0048) and total iron (r =0.85; 95% CI, 0.20–0.97; p < 0.016), but not significantly with coal rank (r = 0.59; 95% CI, -0.26 to 0.91; p < 0.16) or silica (r = 0.28; 95% CI, -0.55 to 0.82; p < 0.54). No association of CWP with CaO itself was observed (r = -0.18; 95% CI, -0.78 to 0.60; p < 0.69).

The relationship between CWP and BAI is well described by a linear model. Figure 1 displays the fitted line and a scatterplot of the data tagged by the coal mine region of its origin. Based on the levels of BAI in each coal that we calculated, we derived the pneumoconiotic potency in 7,000 coal samples collected by the USGS. Figure 2 shows that there is a geographic distribution of coals with different levels of BAI and therefore possibly different pneumoconiotic potencies. For example, in the western states, most coals do not have BAI, which may pose less risk to coal miners (shown in green, Figure 2). In the eastern states, there is a trend for possibly high risk coals (black and gray), ranging from PA to OH to WV and KY. There is also an apparent trend of low-risk coal (blue and green) from WV to TN to AL. Because CWP prevalence was much higher in the first round of the NSCWP (Morgan et al. 1973) than in the current epidemiologic data, the prevalence of CWP in the map is probably overestimated, in part due to reduced dust exposure. However, the indication of the relative risk of CWP in coal mining in various coal mine regions may still be valid and useful for CWP prediction. For example, today most of U.S. coals come from Wyoming, a state that was not studied in the first round of NSCWP but has a low CWP prevalence predicted, as shown in Figure 2.

# Discussion

CWP is one of the occupational diseases that has been most studied by epidemiologists. In

**Table 2.** Average levels (mmol/100 g dry coal) of total  $H_2SO_4$  (1/2  $S_{py} + SO_4$ ), available amount of acid (1/2  $S_{py} + SO_4 - CaO$ ), total iron, and predicted BAI.<sup>*a*</sup>

PI						
State	No. of mines	CWP (%)	1/2 S <sub>py</sub> + SO <sub>4</sub>	$1/2 S_{py} + SO_4 - CaO^{b,c}$	Fe <sub>2</sub> O <sub>3</sub>	BAI <sup>d</sup>
PA	9	45.35	18.61	14.63	12.48	11.82
OH	6	31.80	19.91	14.69	12.86	9.07
KY	13	29.00	13.17	7.49	9.78	6.25
WV	8	28.25	9.15	4.57	7.27	4.77
AL	13	16.70	9.65	6.77	8.85	5.29
UT	4	13.10	4.14	-3.19	2.69	1.09
CO	41	4.60	1.92	-2.69	3.68	0.15

<sup>a</sup>Levels of S<sub>py</sub>, SO<sub>4</sub>, CaO, and Fe<sub>2</sub>O<sub>3</sub> were obtained from the USGS database for each coal mine (Bragg et al. 1998). <sup>b</sup>CaO was presented as percentage of high-temperature ashes in the USGS database and was converted to percentage of dry coal based on the ash yield. Because 1 mol CaCO<sub>3</sub> produces 1 mol CaO in the ashes, the molar amount of CaO per 100 g coal was used as a measure of CaCO<sub>3</sub> levels in the coals. Values in the individual coal samples were calculated first and then averaged for the coal mine region for each of physicochemical parameters listed. <sup>d</sup>BAI was calculated as follows: if the difference in [1/2 S<sub>py</sub> + SO<sub>4</sub> - CaO] is  $\geq$  0 for the individual coal mine, the BAI is given as 0. If the difference in [1/2 S<sub>py</sub> + SO<sub>4</sub> - CaO] is > 0, a lesser value between [1/2 S<sub>py</sub> + SO<sub>4</sub> - CaO] or total iron (Fe<sub>2</sub>O<sub>3</sub>) is given for BAI (see text for details).

Table 3. Correlation among average levels of various parameters with CWP prevalence.

						1/2 S <sub>py</sub>	1/2 S <sub>py</sub>		
	C/H	SiO <sub>2</sub>	CaO	S <sub>py</sub>	SO4 <sup>2-</sup>	$+ SO_4^{2-}$	$+ SO_4^{2-} - CaO$	$Fe_2O_3$	BAI
r-Value	0.59	0.28	-0.18	0.91	0.58	0.90	0.87	0.85	0.94
Lower 95% Cl	-0.26	-0.55	-0.78	0.35	-0.26	0.40	0.25	0.20	0.66
Upper 95% CI	0.91	0.82	0.60	0.99	0.90	0.99	0.98	0.97	0.999
<i>p</i> -Value	0.16	0.54	0.69	0.0048	0.17	0.006	0.01	0.016	0.0015

the United States, Great Britain, France, and Germany, the prevalence and severity of CWP have been shown to differ markedly among coal mines despite exposures comparable with respirable dust. In the United States, there has been a decline in the prevalence from east to west, the disease being most common in PA coal miners and least common in coal miners from UT. In France, coal miners of Provence never had reported CWP (0%), and the prevalence of CWP in coal miners of Nord Pas de Calais was 24% (Amoudru 1987). In Great Britain, the proportional mortality ratios for CWP varied from 135 in Leicestershire county to 3,825 in South Glamorgan county (Coggon et al. 1995). These epidemiologic results indicate that physicochemical characteristics of the coals responsible for toxicity may be different from one coal mine region to another. This fact allowed us to correlate certain physicochemical characteristics of coals with the epidemiologic results.

In the present study we found a significant correlation between CWP prevalence and levels of BAI in the coals. FeS<sub>2</sub>, a typical contaminant in coals, readily undergoes oxidation and forms BAI and acid. The formed acid in the coal mines causes acid mine drainage in the environment, and CaCO3 is used for its treatment (Aziz et al. 2004; Cravotta 2003; Demchak et al. 2004). Burning of Spy-containing coal produces sulfuric dioxide (SO<sub>2</sub>), a major component of acid rain (Carmichael et al. 2002; Srivastava and Jozewicz 2001). BAI, a fraction of total iron, can catalyze oxidant formation and lead to oxidative lung damage. CaCO<sub>3</sub>, a mineral existing only in certain coals, such as those in the western coal mines, can oxidize BAI and make iron less bioavailable for adverse health effects (Huang et al. 1998, 2002; Zhang et al. 2002; Zhang and Huang 2002). Therefore, our results indicate that certain minerals in the coals can interact and thus contribute to different levels of BAI. This might provide an explanation for the observed regional differences in the

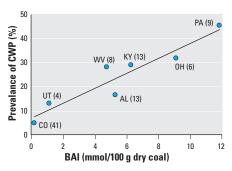


Figure 1. Correlation between prevalence of CWP and BAI in seven U.S. states' coal mine regions. Numbers in parentheses indicate the number of coal samples per state for which analytical data were available. The expected prevalence of CWP (%) = 3.11 BAI + 7.04.

prevalence of CWP and the associated COPD. Increasing evidence demonstrates that iron present in the coal fly ash, asbestos, or urban particles can lead to increased oxidant, ferritin, and cytokine formation (Chao et al. 1994; Fang and Aust 1997; Smith and Aust 1997; Smith et al. 2000). These studies support our hypothesis that BAI may be responsible for coaldust–induced lung injury.

S<sub>py</sub> and total iron also significantly correlate with CWP prevalence in the present study (Table 3), suggesting that  $S_{py}$  and total iron levels, which are available in the USGS coal database (Bragg et al. 1998), may be used as simple indexes for predicting coal's toxicity. However, we have previously noticed that samples from the coal mines of Provence, France, had high levels of FeS<sub>2</sub> (Huang et al. 1994), but coal workers in the Provence coal mine region do not have reported CWP (Amoudru 1987). It is this observation that leads us to search for other factor(s) that may contribute to or inhibit CWP development. In fact, the coals of Provence contain large amounts of  $CaCO_3 (\geq 10\% \text{ wt/wt})$ . By suspending these coals in acid (100 mM H<sub>2</sub>SO<sub>4</sub>), no BAI was released. Therefore, it may not be coincidental that the coals having no BAI did not report CWP in miners working in the coal mine regions. Similarly, coals from UT did not release iron at pH 4.5 mimicking phagalososomes but released iron in 50 mM HCl. These results suggest that not all iron compounds are bioavailable. In fact, the presence of CaCO<sub>3</sub> in

certain coals makes iron less bioavailable. Our results indicate that oxidation of FeS2 and subsequent neutralization of acid by CaCO3 most likely determine the levels of BAI in the coal dusts. This is further supported by the improved correlation between CWP prevalence and BAI over the correlation with available acid  $(1/2 S_{py} + SO_4^{2-} - CaO)$  or total iron. From a chemical point of view, CaCO3 is more basic than FeCO<sub>3</sub>, consuming acid first before FeCO<sub>3</sub>. Therefore, CaCO<sub>3</sub> limits iron's bioavailability. This should not be surprising because, in nutrition, calcium carbonate supplements depress iron's bioavailability (Cook et al. 1991; Prather and Miller 1992; Wienk et al. 1996).

CWP, which was originally thought to be a variant of silicosis, results from the inhalation of coal mine dust that usually contains relatively small amounts of free crystalline silica (quartz) (Borm and Tran 2002; Castranova and Vallyathan 2000). Coal rank was found to play a role, because CWP risk increases with coal rank (Attfield and Morring 1992b; Maclaren et al. 1989). Laboratory coal breakage studies have shown a positive correlation with the amount of respirable-size particles found in the product increasing with coal rank (Moore and Bise 1984). It has been suggested that higher rank coals with a higher electrostatic charge on breakage may contribute to the increased incidence of CWP in the high-rank coal regions (Page and Organiscak 2000). However, a correlation

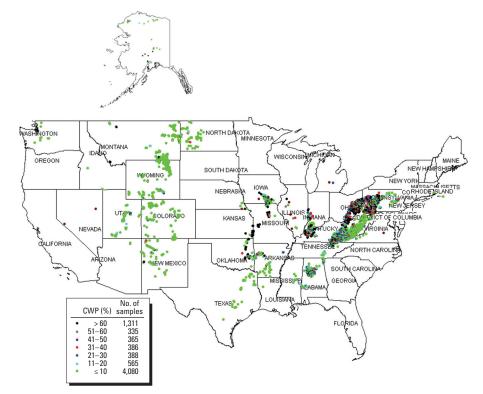


Figure 2. Mapping and prediction of the pneumoconiotic potency of coal by the level of BAI.

between coal rank and cell cytotoxicity has not yet been established in biologic studies (Christian and Nelson 1978; Christian et al. 1979). In the present study, no significant correlation between CWP prevalence and coal rank or silica was observed (r = 0.59, p < 0.16for coal rank and r = 0.28, p < 0.54 for silica).

Based on the present study using the calculated model of BAI, we believe that it may be possible to predict which coal is likely to be toxic, even before large-scale mining. However, this study is far from concluding the causeand-effect relationship between BAI and CWP and the associated COPD development. Further studies on the role of BAI in cell and lung injury, as well as the protective role of CaCO<sub>3</sub> in inhibiting BAI and the associated injuries, are still needed. Other limits of the present study are a) noninclusion of anthracite coals for the correlation study but use of only bituminous coals, which restricts the usefulness of comparing BAI with coal rank or extrapolation to anthracite coals; b) incomplete coal samples from all states as in the first NSCWP (Morgan et al. 1973), and no recognition of many surface coal mines in the western states (e.g., Wyoming and Texas) where CWP may be less likely to occur than in underground coal mines; and c) lack of consideration of coal particle sizes, effects of other transition metals that may become bioavailable, and effects of phagolysosomes of cells in contributing to acid solubilization of iron in the coals.

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