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An Evaluation of Suggestions of Rapidly Progressing Coal Mine Workers Pneumoconiosis (CWP) Supporting Major US Policy Changes

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Chapter 1 Introduction

Anato, et al., recently expressed concern that the continued incidence of Coal Mine Workers Pneumoconiosis (CWP) and "Rapidly Progressing Coal Mine Workers Pneumoconiosis (RPCWP)" in the United States indicate(s) the need for particular vigilance with regards to enforcement of the current dust limits in smaller mines and in specific regions, and suggests that additional measures to control exposures may be needed to enhance the protection of miners.[1] In particular, as recommended in the NIOSH Criteria document, reduction of the coal mine dust exposures, especially in high rank coal mining areas is desirable."[2] Without commenting on other potential policy or scientific reasons for these suggestions, an examination of the underlying Anato study discloses a lack of adequate support for the study conclusions and suggested response.

Using the computerized X-rays from participating in the voluntary coal miner X-ray program, Anato, et al. were able to identify 29,521 coal miners with two X-rays taken in 1996 and then again in 2002.[1] These X-rays were evaluated to determine the prevalence (number of existing cases of CWP, most recent radiograph showing an ILO category 1/1 or greater) of CWP and its progression during the 6 year period, 1996-2002. A total of 886 cases met this criterion, which was an initial X-ray defined as ILO category 1/0, resulting in an initial crude prevalence rate of 3% for CWP.

Of particular interest in the Anato, et al. study is what the authors defined as Rapidly Progressing Coal Mine Workers Pneumoconiosis (RPCWP). This disease entity was defined radiographically as an increase in the small opacity profusion by the equivalent of more than one ILO subcategory over five years after 1985, and/or the development or progression of PMF after 1985.

A problem with the definition of RPCWP is the interpretation of what is an increase in one ILO subcategory The intent of the standard is to prevent reaching major category

2 because the probability of progressing to PMF increases once category 2 is reached.[2] Thus if the authors consider progression from cactegories1/0 to 1/1, or 1/1 to 1/2, it is arguable that this should be considered a progression of meaningful significance or "rapidly progressing" CWP.

A subset of 783 coal miners with at least two X-rays was identified from the initial 886 coal miners meeting the qualifying criteria, having an X-ray showing CWP. Two hundred and seventy-seven X-rays of miners having at least two X-rays were categorized as having RPCWP. Additionally, 41 cases of PMF were identified. Further analysis indicated a strong geographic and demographic influence in the data set. Specifically, Southwestern Virginia, Southeastern West Virginia, and Eastern Kentucky showed highest relative amounts of RPCWP. Also, employment at small mines, less than 50 employees, was associated with increased risk of RPCWP. The authors concluded, based on assumed exposures at or below the current MSHA PEL of 2.0 mg/m^3, there is a risk of developing RPCWP, however, no exposure data was presented to support this conclusion. Because of the nature of the geographic distribution of the cases and their rarity, the authors have classified these observations as "Sentinel Events."

Chapter 2 Background

In most of the early studies of exposure to coal dust and its relationship to the development of coal mine worker's pneumoconiosis, some miner's were observed to develop the disease sooner than the average and progress to more advanced states more rapidly than other miners exposed to similar levels of coal dust. The reasoning for this pathological response has been speculated to arise from a variety of causes, some of which are elevated quartz exposures, differences in coal type, e.g. bituminous vs. anthracite, the presence of ferric iron and subsequent inflammatory response, or differences in the genetics of exposes workers.

In some of the earliest studies of the relationship between exposure to respirable coal dust and CWP, it was observed that in some miners the disease progressed more rapidly than others with the same exposures.[3-12] Studies by Hurley, Jacobsen, Attfield, and others, noted that miners developed CWP and progressed with continuing exposure to coal mine dust.[3-12] From these early studies, it was determined that reduced exposure to coal mine dust would slow or eliminate the progression of CWP. Jacobsen reported that with an average lifetime (35 years) of coal mine dust exposure of 2.0 mg/m^3 or less that the prevalence of CWP would be at or near 0. From these early studies it was also believed that the development of Pulmonary Massive Fibrosis (PMF) would be eliminated or reduced significantly with projected reduction of exposure to coal mine dust. (This is the basis for the MSHA Part 90 Miner program).

Recent studies by Goodwin and Attfield, Attfield and Sexias and others, on CWP in the American coal mining industry have demonstrated that the prevalence has continued to fall with the continuing control of respirable coal mine dust. These studies have demonstrated that CWP has been declining for more than 20 years.[13-19]

Studies that have focused on the rate of progression in CWP have indicated progression is associated with and dependent on exposure to respirable coal mine dust. Continued exposure at the current MSHA permissible coal mine dust exposure, 2.0 mg/m^3 can result in a fraction of the exposed miner population developing category 2/0 pneumoconiosis (radiologically present, however, without pulmonary impairment). The development of category 2/0 with continuing exposure result in a small fraction, typically less than 1%, of exposed miners developing PMF.[17] The mechanism of PMF development is unknown and does not appear to be predictable.[20]

Recent studies of the pulmonary inflammation process have suggested that ferric iron can play a significant role in the development of reactive oxygen species and inflammatory disease.[21] Additionally, there appears to be a genetic component that makes certain miners more susceptible to pneumoconiosis and its progression that remains unidentified. [22]

Chapter 3 Issues

While the studies of Anato, et al., provide provocative results, there are a number of issues associated with their methods and data that bring into question their results and conclusions. The issues include problems with the representativeness of the study population, B-reader variability in the diagnosis of CWP and RPCWP, case placement within a specific ILO category, the geographic distribution of CWP and RPCWP cases and the suggestion that current coal dust exposure levels could be responsible for their observations.

To understand the conclusions of the Anato, et al. study and their implications, the relationship of the coal miner population studied and the total population of coal miners needs to be reviewed. A critical question focuses on how well does the study population represent the population of coal miners? Is the population biased in a way that would inflate or reduce the observed risk of disease? The source of X-ray data in the Anato study is a **semi-voluntary** health surveillance program administered by NIOSH. Participation can be considered semi-voluntary because miners entering the profession are required by law to have an initial radiograph followed by an additional radiograph after three years. After this time frame participation in the NIOSH surveillance program is voluntary. The participation rate of coal miners in this program has declined since its inception.[23] The reason(s) for this decline have not been investigated or explained. However, the low participation of coal miners in this surveillance program can have a significant influence in the interpretation of study results.

A **Nonresponse bias** can occur because individuals who do not respond to a call to participate in research studies are generally different from those who do respond.[24] Typically, this type of bias has been thought to be associated with masking of the true health risk as it is assumed that those responding are in better health than those who did respond.[24] This has not been demonstrated for the population of miners participating in

NIOSH and MSHA X-ray programs. It is just as likely that miners who chose not to participate are miners who have had an X-ray that did not show pneumoconiosis during earlier screenings. This type of non-response could lead to an increase in the apparent prevalence by decreasing the total population at risk.

The NIOSH X-ray miner surveillance programs (Coal Worker's X-ray Program, CWXP) has been plagued with problems from the start with low miner participation. (See Table 1) Historically, other than the first two rounds conducted in the 1970's, the participation rates averaged less than 35%. If the above scenario is true then the observed prevalence for CWP would be inflated and, in reality, be less than the 3-4% as reported by Anato, Attfield and others.

Case Definition and ILO Characterization Issues

In the study by Anato, each radiograph was classified when read by two separate certified B-readers at NIOSH. Disease presence and progression was assessed for miners meeting one of three criteria:

- who had two radiographs in 1996 and 2002; or,
- had at least two examinations;
- or, if the most recent radiograph showed at least a category 1/1 pneumoconiosis.[1]

The definition of CWP follows the ILO placement of individuals within pneumoconiosis category 1/0 based on the profusion and the number and shape of opacities. This is a categorical scale with 4 major and 12 sub-categories. When a radiograph is judged to fall into a certain major and sub-category it is assumed to fall into the middle of the category, however, the case could be borderline and in a position to transition into the next major category or sub-category or be read as appearing in the next highest category. The diagnosis of CWP and RPCWP is highly subjective and is subject to large potential categorization errors (disease misclassification) solely on the basis of diagnostic category. According to Lynch, et al. it is probably impossible to score profusion within a sub-category.[25]

The categorical placement of subjects suspected of having early stage CWP, 1/0, provides additional concern with respect to the number of cases and the change of CWP category across the study time defined by Anato. Inter-observer variation in reading x-rays for pneumoconiosis also can play a significant role in the observations made by Anato. Anato, et al. suggest that disease misclassification was reduced by only classifying those individuals whose diagnosis demonstrated a two category change and that 97% of the alleged cases of RPCWP moved through at least two categories (sub-categories). [1] Figure 1 demonstrates graphically how a two sub-category change in CWP can appear to happen when only a small change in radiographic appearance occurs.

While this claim is unsubstantiated, it is clear that classifying early stages of CWP is difficult at best.[20] Without an understanding of the concordance for the miners in the study group, which is the agreement between readers, it is impossible to rely on the results of this study. Even under very controlled study conditions where concordance of x-ray readings has been evaluated the results are not terribly encouraging especially when small changes in radiography are being interpreted as disease progression.[26]

Moreover, the true average time course of CWP progression is not well defined and Anato, et al. does not relate their observations to what might be considered "normal progression".

To reach the conclusion of a rapid progress of CWP, Anato, et al:

- employed a novel analysis of two successive lung X-Rays, on each of 783 miners, with CWP, separated by 6 years;
- read by different X-Ray readers; and
- The creation of a unique, non-peer reviewed, definition of RPCWP that relies on a one step shift in variable X-Ray ILO diagnosis categories.[1]

Unfortunately, Anato, et al. used a small, self selecting subset of almost 30,000 miners with at least one X Ray, who returned for a second, and did not consider the actual exposures at the mines where the subject miners were employed, data readily available from the US

Mine Safety and Health Administration (MSHA). Moreover; the authors did not adequately account for widely recognized X-Ray reader variability or intensity and time of exposure. All of these factors have been scientifically acknowledged in prior research to substantially impact lung disease risk analysis in miners, and their individual error impacts must be compounded for accurate analysis, thus rendering the Anato, et al analysis and conclusions unreliable.

Geographic clustering

Anato, et al., state that the rates of PMF and CWP have been increasing and, specifically have suggested these rates are increasing primarily in younger coal miners, and, in specific areas, e.g. southern West Virginia, Kentucky, and Virginia.[1] While the author's acknowledge that coal rank may play a role in the appearance of RPCWP, they state that this is not the full explanation and the mining methods, approaches to dust control and enforcement may also play a role. No support for these speculations is provided. It should be noted that a similar geographic distribution of pneumoconiosis was noted in the study of Coggon in the United Kingdom.[27] In this study, Coggon reported that mortality from CWP varied from a proportionate mortality ratio of 135 to over 3500 depending on the county under study.

Exposure to coal dust -- the critical variable

It is clear from the numerous studies of Attfield and others that exposure to respirable coal mine dust has decreased considerably across time. Since the 1969 Coal Mine Act was passed, respirable coal dust exposures have been reduced from averages above 3 mg/m^3 to less than 1 mg/m^3 in most mines. A recent survey of mines in one of the areas where RPCWP is suspected to occur demonstrates the reduction in respirable coal mine dust, Figure 2. In Figure 2, it can also be seen the respirable quartz concentrations have declined significantly across time, at least in the two mines studied, by a factor of approximately 5 or 6.

A recent study by Pollock, et al. of coal mine dust exposures in the southern Appalachian region of the United States (area including Southwestern Virginia, southern West Virginia and western Kentucky) indicated that small mines dominated (comprising more than

70% of active mines) the area under study.[28] Additionally, in these small mines, exposures to respirable coal mine dust were at or near the MSHA standard, 2.0 mg/m^3. However, an analysis of continuous miner operators (a face job) samples showed that many of the measured exposures were above the current MSHA allowable exposure for respirable coal mine dust.

Anato, et al, did not analyze the extensive US MSHA coal dust and quartz sampling data base, available for every mine, to estimate the exposures at the mines where the 783 study miners were employed. Yet, concurrent exposure to respirable coal mine dust and high levels of respirable quartz, in addition to excessive coal dust alone have shown to result in an increased risk of CWP development. In some of the earliest studies of CWP by the British National Coal Board's Pneumoconiosis Field Research (PFR) the presence of high quartz exposure levels, 2-4 mg/m^3, were observed to significantly influence both the appearance and progression of CWP. [5-6] Low levels of quartz, however, do not appear to have similar pathological effect. Moorfield, et al. described the relationship between quartz and CWP as having an inverse effect.[29-30]

Anato, et al did not address the long term excessive coal dust or silica exposures needed for the development of CWP and even RPCWP, as they defined this new term. The literature documents that CWP takes years of exposure to develop. Therefore, the intensity of exposures over time for the miners in the Anato study group was a critical factor that required analysis to understand if miners were at risk at or below the 2 mg/m^3 current exposure limit or at levels far above or suffered excessive silica exposures as well. In fact, the exposures that resulted in the development of the Anato defined RPCWP would have occurred many years ago, when dust levels were more variable than today, and, in many cases, significantly higher than miner's today routinely experience. Since exposure is the single most important factor creating risk of CWP, the Anato study, without an extensive exposure analysis on readily available MSHA data, does not justify its conclusions and suggestions.

Chapter 4 Conclusions

The following issues make conclusions from the Anato, et al. study difficult, if not impossible to relate to the cohort of miners currently working in the mining industry.

1. The study population is a small subset of the entire population.

2. The study population is a self-selected sub-set of the entire respirable coal dust exposure miner population. How this effects the conclusions drawn by Anato, et al. is not explained or discussed.

3. Natural progression of CWP is not defined, therefore, defining an entity of RPCWP" is not possible.

4. The changes in respirable coal dust exposure and the impact of these changes has not been evaluated.

5. Given that CWP is a slowly developing disease, occurring over many years, it is not possible to relate exposures occurring today to disease appearing in the near past.

6. Exposure to respirable coal mine dust and respirable quartz, while changing over time, have not been evaluated with respect to the miners reported to have RPCWP.

7. Geographic clustering of CWP has not been explained by either exposure to respirable coal mine dust or respirable quartz.

8. The role of employment in small coal mines is not explained.

Table 1. Coal Mine worker's participation rates in NIOSH Coal Worker's X-Ray Program*

Participation and Disease Rates for the Coal Workers' X-Ray Surveillance Program

Time Period	Number of X-Rays Processed by NIOSH[1]	Average Number of Underground Coal Miners for the Period[2]	Participation Rate	Number of X-Rays showing evidence of CWP[1]	CWP Disease Rate
1970-1974	122,425	152,066	81	13,259	11
1975-1979	116,014	150,474	77	3,156	3
1980-1984	45,289	131,112	38	1,080	2
1985-1989	17,830	91,122	20	546	3
1990-1994	15,523	69,424	22	453	3
1995-1999	14,629	50,319	29	283	2
2000-2004	15,236	31,826	48	604	4

1 Data provided by the NIOSH Underground Coal Mine System

2 Average calculated using the MSHA Address & Employment Data Sets

*NIOSH/DRDS/CWHSP Celebrates the 35th Anniversary of the Federal Coal Mine Health and Safety Act, May 21, 2009.

Table 2

Prevalence of coal workers' pneumoconiosis (CWP) and progressive massive fibrosis (PMF) among examined noncontract miners*, estimated number of employees, and participation rates, by state — U.S. National Coal Workers' X-ray Surveillance Program and Miners' Choice Program, fiscal years 1996–2002

	Underground miners					Surface miners					Average employment and estimated participation†			
	No. miners examined	CWP		PMF		No. miners examined	CWP		PMF		Underground miners		Surface miners	
State		No.	(%)	No.	(%)		No.	(%)	No.	(%)	No.	(%)	No.	(%)
Alabama	2,308	25	(1.1)	3	(0.1)	524	5	(1.0)	1	(0.2)	3,904	(59.1)	2,200	(23.8)
Arizona	0	—	—	—	—	520	5	(1.0)	0	(0)	0	—	737	(70.6)
Arkansas	9	0	(0)	0	(0)	0	—	—	—	—	9	(100.0)	13	(0)
Colorado	1,655	24	(1.5)	3	(0.2)	180	3	(1.7)	0	(0)	1,655	(100.0)	712	(25.3)
Illinois	2,863	31	(1.1)	1	(0.0)	175	1	(0.6)	0	(0)	4,300	(66.6)	1,212	(14.4)
Indiana	816	5	(0.6)	0	(0)	397	2	(0.5)	0	(0)	816	(100.0)	2,836	(14.0)
Kentucky	3,073	106	(3.5)	9	(0.3)	1,253	34	(2.7)	3	(0.2)	19,220	(16.0)	13,910	(9.0)
Louisiana	0	—	—	—	—	112	0	(0)	0	(0)	0	—	168	(66.7)
Maryland	249	24	(9.6)	0	(0)	52	2	(3.9)	0	(0)	273	(91.2)	247	(21.1)
Montana	0	—	—	—	—	183	0	(0)	0	(0)	13	(0)	902	(20.3)
New Mexico	123	1	(0.8)	0	(0)	919	7	(0.8)	0	(0)	123	(100.0)	1,654	(55.6)
North Dakota	0	—	—	—	—	278	2	(0.7)	0	(0)	0	—	966	(28.8)
Ohio	530	9	(1.7)	0	(0)	406	10	(2.5)	0	(0)	1,952	(27.2)	2,241	(18.1)
Oklahoma	21	0	(0)	0	(0)	0	—	—	—	—	43	(48.8)	259	(0)
Pennsylvania	2,468	44	(1.8)	3	(0.1)	778	22	(2.8)	3	(0.4)	6,204	(39.8)	5,468	(14.2)
Tennessee	102	5	(4.9)	0	(0)	52	2	(3.9)	0	(0)	681	(15.0)	712	(7.3)
Texas	0	—	—	—	—	1,292	11	(0.9)	0	(0)	0	—	2,598	(49.7)
Utah	1,586	8	(0.5)	1	(0.1)	48	1	(2.1)	0	(0)	2,184	(72.6)	96	(50.0)
Virginia	1,749	150	(8.6)	11	(0.6)	743	28	(3.8)	1	(0.1)	6,771	(25.8)	3,718	(20.0)
Washington	0	—	—			81	0	0	0	(0)	0	—	580	(14.0)
West Virginia	3,069	232	(7.6)	17	(0.6)	1,221	58	(4.8)	5	(0.4)	16,289	(16.8)	8,939	(13.7)
Wyoming	26	0	(0)	0	(0)	1,252	3	(0.2)	0	(0)	95	(27.4)	4,771	(26.2)
Others§	0	—	—	—	—	0	—	—	—	—	8	—	550	(0)
Total	**20,647**	**664**	**(3.2)**	**48**	**(0.2)**	**10,466**	**196**	**(1.9)**	**13**	**(0.1)**	**66,540**	**(31.0)**	**55,489**	**(18.9)**

*Among 66 examined contract miners from nine states (Alabama, Indiana, Kentucky, Michigan, Pennsylvania, Tennessee, Utah, Virginia, and West Virginia), two were determined to have CWP and one was determined to have PMF.

†Participation by contract miners was low; of an estimated 47,662 contract miners working at surface or underground coal mines, only 66 (0.1%) were examined in the federal programs.

§Alaska, California, Kansas, Mississippi, and Missouri.

From Pneumoconiosis Prevalence Among Working Coal Miners Examined in Federal Chest Radiograph Surveillance Programs — United States, 1996—2002, CDC Monthly Morbidity and Mortality Report,

Advancement of 1 ILO Category

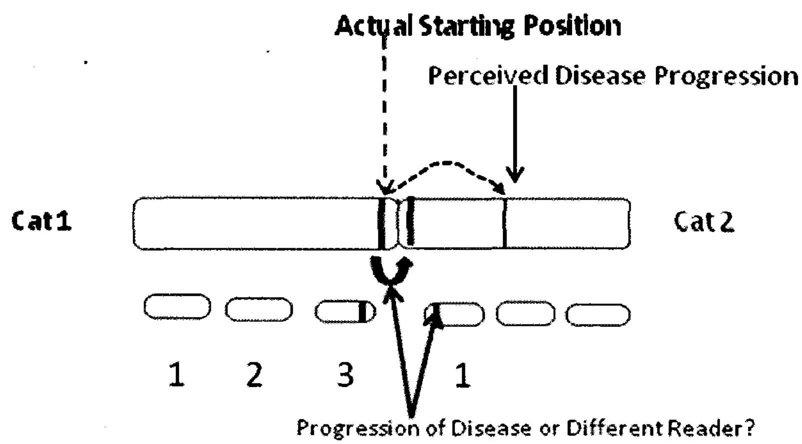

Figure 1

Mean concentrations of respirable coal mine dust and crystalline silica in coal mine dust* for underground workers at the coal face† — Lee and Wise counties, Virginia, 1970–2005

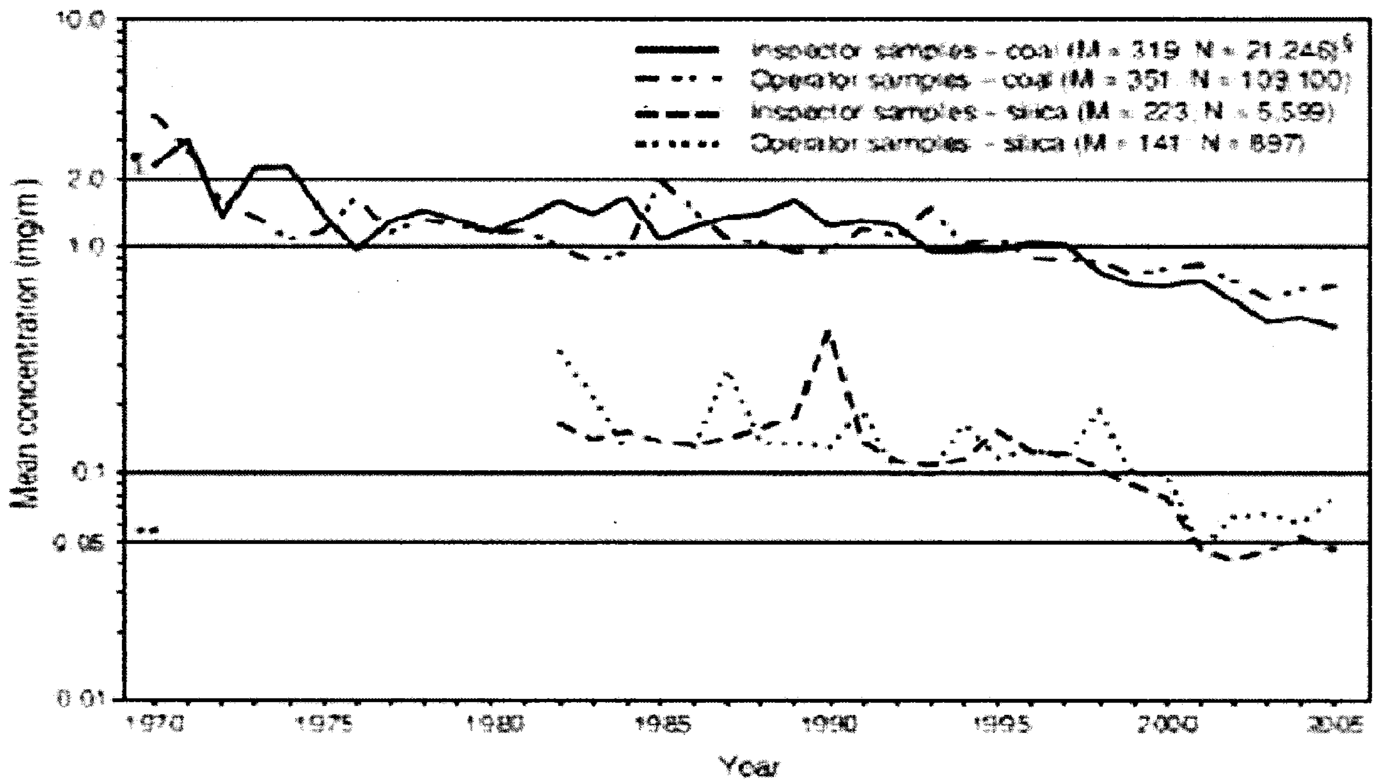

Figure 2

Table 3. Small
Mines in the Southern Appalachian Region of the U.S.[23]

MSHA District	Small Underground Mines	Total Active Underground Mines	Percent Small Mines
4	85	148	57%
5	39	53	74%
6	88	105	84%
7	50	70	71%
Total	**262**	**376**	**70%**

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