

**IN THE UNITED STATES COURT OF APPEALS
FOR THE SIXTH CIRCUIT
No. 14-3375**

QUARTO MINING COMPANY,

Petitioner,

v.

CLIFFORD MARCUM, SR., and DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
UNITED STATES DEPARTMENT OF LABOR,

Respondents.

On Petition for Review of a Final Order of the Benefits
Review Board, United States Department of Labor

BRIEF FOR THE FEDERAL RESPONDENT

M. PATRICIA SMITH
Solicitor of Labor
RAE ELLEN JAMES
Associate Solicitor
SEAN BAJKOWSKI
Counsel for Appellate Litigation
JONATHAN ROLFE
Attorney
U.S. Department of Labor
Office of the Solicitor
Suite N-2117
200 Constitution Avenue, N.W.
Washington, D.C. 20210
(202) 693-5660

Attorneys for the Director,
Office of Workers'
Compensation Programs

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STATEMENT REGARDING ORAL ARGUMENT

Pursuant to Sixth Circuit Rule 34(a), the Director, Office of Workers' Compensation Programs, United States Department of Labor (the "Director"), respectfully submits that oral argument is unnecessary in this case.¹ To the extent that this appeal raises anything other than routine substantial-evidence issues, its

¹ The Director administers the Black Lung Benefits Act on the Secretary of Labor's behalf. 20 C.F.R. § 1.2(f); Secretary's Order 10-2009, 74 Fed. Reg. 58834 (Nov. 12, 2009). As the Secretary's delegate, the Director is a party to this action. 30 U.S.C. § 932(k).

outcome is controlled by this Court’s recently published decision in *Central Ohio Coal Co. v. Director, OWCP*, --- F.3d ---, No. 13-3712, 2014 WL 3858471 (6th Cir. August 7, 2014). Therefore, “the facts and legal arguments are adequately presented in the briefs and record[.]” Fed. R. App. P. 34(a)(2)(C).

JURISDICTIONAL STATEMENT

This case arises from Respondent Clifford Marcum, Sr.’s claim for benefits under the Black Lung Benefits Act (the “BLBA” or the “Act”), 30 U.S.C. §§ 901-944 (2006 & Supp. VI 2012), which was filed on June 15, 2010. Director’s Exhibit (“DX”) 5. On May 12, 2011, a Department of Labor claims examiner issued a Proposed Decision and Order awarding BLBA benefits to Marcum, payable by his former employer, petitioner Quarto Mining Company (“Quarto”). DX 27. Quarto requested a hearing before an administrative law judge on May 20, 2011, within the 30-day period established by 20 C.F.R. § 725.419(a). DX 28.²

On February 28, 2013, Administrative Law Judge Michael P. Lesniak (“the ALJ”) awarded BLBA benefits to Marcum. Joint Appendix (“JA”) 208. Quarto timely appealed to the Benefits Review Board on March 26, 2013. *Id.* at 225; *see* 33 U.S.C. § 921(a), as incorporated by 30 U.S.C. § 932(a) (providing a thirty-day

² Because Quarto refused to pay BLBA benefits to Marcum during the litigation of this case, those benefits were instead paid by the Black Lung Disability Trust Fund. *See* DX 29; 20 C.F.R. §§ 725.420(a), 725.522(a). Quarto is obligated to reimburse the Trust Fund for payments it made on the employer’s behalf. 30 U.S.C. § 934(b); 20 C.F.R. §§ 725.602-603.

period for appealing ALJ decisions). The Board had jurisdiction to review the ALJ's decision pursuant to 33 U.S.C. § 921(b)(3), as incorporated by 30 U.S.C. § 932(a).

On February 24, 2014, the Board issued a final order affirming the award of benefits. *Id.* at 208. Quarto timely petitioned this Court to review the Board's order on April 23, 2014. *Id.* at 218; *see* 33 U.S.C. § 921(c), as incorporated by 30 U.S.C. § 932(a) (providing a sixty-day period for appealing Board decisions). This Court has jurisdiction over Quarto's petition for review under 33 U.S.C. § 921(c), as incorporated by 30 U.S.C. § 932(a). The injury contemplated by 33 U.S.C. § 921(c)—Marcum's exposure to coal-mine dust—last occurred in Ohio, within the jurisdictional boundaries of this Court. *See Danko v. Director, OWCP*, 846 F.2d 366, 368 (6th Cir. 1988).³

STATEMENT OF THE ISSUES

30 U.S.C. § 921(c)(4) provides a rebuttable presumption that totally disabled former coal miners who worked at one or more an underground mines for at least fifteen years are entitled to federal black lung benefits. There is no dispute that

³ While the ALJ mistakenly stated that Marcum's last coal mine employment took place in Pennsylvania, JA 205 n.25, the parties do not dispute that his last coal mine employment took place at Quarto's Number 4 mine in Monroe County, Ohio. *See* Quarto's Brief in Support of Petition for Review to the Benefits Review Board at 1 ("It is a matter of public record that [Quarto's Number 4 Mine] is located in Monroe County, Ohio."); Pet. Br. 3.

Marcum established that he is totally disabled by a respiratory impairment. The ALJ decided, and the Benefits Review Board affirmed, that Marcum also established that he worked for more than fifteen years at an underground coal mine. The first question presented is whether substantial evidence supports the ALJ's decision on that length of employment, entitling Marcum to invoke the presumption.

To rebut the presumption, an employer must demonstrate either that the miner does not have pneumoconiosis, or that no part of the miner's respiratory disability was caused by pneumoconiosis. The ALJ determined, and the Benefits Review Board affirmed, that Quarto failed to rebut the presumption because the company's medical experts, who argued that Marcum's respiratory disease was caused solely by smoking and therefore was not pneumoconiosis, were not credible. The second question presented is whether substantial evidence supports that decision.

STATEMENT OF THE CASE

A. Legal and technical background.

1. The definition of pneumoconiosis.

The Black Lung Benefits Act, 30 U.S.C. §§ 901-944, provides disability compensation and certain medical benefits to coal miners who are totally disabled by pneumoconiosis, commonly referred to as "black lung disease." 30 U.S.C.

§ 901(a); 20 C.F.R. § 718.1(a). The Act defines “pneumoconiosis” as “a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment.” 30 U.S.C. § 902(b). Compensable pneumoconiosis takes two distinct forms, “clinical” and “legal.” 20 C.F.R. § 718.201(a).

“Clinical pneumoconiosis” refers to a cluster of diseases recognized by the medical community as fibrotic reactions of lung tissue to the “permanent deposition of substantial amounts of particulate matter in the lungs[.]” 20 C.F.R. § 718.201(a)(1), and is generally diagnosed by chest X-ray, biopsy or autopsy. 20 C.F.R. §§ 718.102, 718.106, 718.202(a)(1)-(2). Clinical pneumoconiosis is often referred to as “coal workers’ pneumoconiosis” or “CWP.” *See Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819, 821 (4th Cir. 1995) (explaining the difference between “the particular medical affliction ‘coal workers’ pneumoconiosis’ [and] the much broader legal definition of pneumoconiosis”).

“Legal pneumoconiosis” is a broader category that includes “any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.” 20 C.F.R. § 718.201(a)(2). A disease “aris[es] out of coal mine employment” if it is “significantly related to, or substantially aggravated by, dust exposure in coal mine employment.” 20 C.F.R. § 718.201(b). As a result, coal-mine dust need not

be the sole or even the primary cause of a chronic lung disease for that disease to be legal pneumoconiosis.

2. Elements of entitlement and the fifteen-year presumption.

The BLBA mandates the payment of benefits “in respect of total disability of any miner due to pneumoconiosis.” 30 U.S.C. § 921(a). “To establish entitlement to benefits,” a former miner must “prove by a preponderance of the evidence that (1) he has pneumoconiosis; (2) his pneumoconiosis arose at least in part out of his coal mine employment; (3) he is totally disabled; and (4) the total disability is due to pneumoconiosis.” *Morrison v. Tennessee Consol. Coal Co.*, 644 F.3d 473 (6th Cir. 2011) (citations omitted); *see* 30 U.S.C. §§ 901, 921; 20 C.F.R. § 725.202(d). The Act, however, contains various presumptions to assist miners in proving that they are totally disabled by pneumoconiosis, including 30 U.S.C. § 921(c)(4)’s fifteen-year presumption.⁴

The fifteen-year presumption provides a rebuttable presumption of entitlement to miners who (1) suffer from a totally disabling respiratory or

⁴ Section 921(c)(4), which Congress eliminated in 1981, was restored as part of the Affordable Care Act, Pub. L. No. 111-148, § 1556, 124 Stat. 119, 260 (2010), and applies to claims that were filed after January 1, 2005, and pending on or after March 23, 2010, the amendment’s enactment date. *Id.*; *see also Morrison*, 644 F.3d at 475. The regulation implementing the restored fifteen-year presumption applies to the same cohort of claims. 20 C.F.R. § 718.305(a). The amended statute and regulation therefore apply to this claim, which was filed on June 15, 2010. DX 5.

pulmonary condition and (2) worked for at least fifteen years “in one or more underground coal mines” or surface mines with substantially similar conditions. 20 C.F.R. § 718.305(b); see *Central Ohio Coal Co. v. Director, OWCP*, --- F.3d ---, No. 13-3712, 2014 WL 3858471 (6th Cir. August 7, 2014). The Department’s regulations define “underground coal mine” to include not only the underground mine shafts and tunnels, but “all land, structures, facilities, machinery, tools, equipment, . . . and other property, real or personal, appurtenant thereto.” 20 C.F.R. § 725.101(a)(30). As Quarto concedes, work in these surface areas of an underground coal mine therefore counts toward the required fifteen years needed to invoke the presumption. *Island Creek Kentucky Mining v. Ramage*, 737 F.3d 1050, 1058; Pet. Br. 32.

To rebut the presumption, the party opposing entitlement must establish either (A) the miner does not, or did not, have pneumoconiosis (in either clinical or legal form) arising out of coal mine employment; or (B) that no part of the miner’s respiratory or pulmonary disability was caused by pneumoconiosis. 20 C.F.R. § 718.305(d)(1)(i), (ii); see *Central Ohio Coal Co.*, 2014 WL 3858471, *5; *Big Branch Resources, Inc. v. Ogle*, 737 F.3d 1063, 1071 (6th Cir. 2013).

3. Pulmonary function tests.

The dispute over the ALJ's rebuttal analysis focuses largely on Dr. Rosenberg's analysis of pulmonary function test results. In BLBA claims, a pulmonary function test is typically used to establish total disability (which is not disputed in this case) but can be considered in a medical expert's analysis of other issues. *See* 20 C.F.R. § 718.202(a)(4) (A physician's diagnosis of pneumoconiosis "must be based on objective medical evidence such as . . . pulmonary function studies[.]").

A pulmonary function test (also called a "ventilatory test" or "spirometry") is one measure of a miner's pulmonary capacity. The test measures several values, including the FEV₁ (forced expiratory volume), the FVC (forced vital capacity), and the FEV₁/FVC ratio. The FEV₁ value measures the amount of air exhaled in one second on maximum effort. It is expressed in terms of liters per second. The FVC value represents the total amount of air that can be exhaled. Obtaining a FVC value requires the miner to take a deep breath and then exhale as rapidly and forcibly as possible. The FEV₁ value is taken from the first second of the FVC exercise. The FEV₁/FVC ratio (also referred to as "FEV₁%") is derived by dividing the FEV₁ value by the FVC value. *See* 20 C.F.R. § 718.103; 20 C.F.R. Part 718 App. B; *see generally* Occupational Safety and Health

Administration, U.S. Dept. of Labor, Spirometry Testing in Occupational Health Programs: Best Practices for Healthcare Professionals, at 1-2 (2013).⁵

Pulmonary function study results meeting prescribed regulatory criteria establish presumptive total respiratory disability. *See* 20 C.F.R.

§ 718.204(b)(2)(i); 20 C.F.R. Part 718 App. B; *Slusher v. Director, OWCP*, 983 F.2d 1068 (6th Cir. 1992). For example, a test resulting in a FEV₁/FVC ratio of less than 55% establishes a miner's total disability, in the absence of contrary evidence. 20 C.F.R. § 718.204(b)(2)(i)(c).

B. Factual background.

The issues raised in this appeal are whether substantial evidence supports the ALJ's finding that Marcum's work for Quarto occurred at an underground coal mine site and his decision not to credit the opinions of Quarto's medical experts on the cause of Marcum's COPD. Only the facts relevant to those two issues are summarized below.

1. Evidence relevant to the ALJ's conclusion that Marcum worked at least fifteen years at an underground coal mine.

Marcum reported on his CM-911a claim form that he worked as an underground coal miner for Island Creek Coal Company and then Ogleby Norton Coal from May 1969 until November 1971. DX 6. He next reported working for

⁵ The OSHA best practices document is available at <https://www.osha.gov/Publications/OSHA3637.pdf> (last visited September 24, 2014).

Consolidation Coal Company, Quarto Mining's parent company, for the remainder of his coal mining career, which spanned from December 1971 until January 1994. *Id.*⁶ These dates are consistent with the Social Security Administration's Statement of Earnings for Marcum. DX 8 at 8-9. He identified the Quarto mine (like the Island Creek and Ogleby Norton mines) as "UNDERGROUND." DX 6. He described the type of industry at Quarto as being "coal mining extraction and preparation of coal" and he described his occupation there as a "bolter inside mine—also mechanic & welder outside mine." *Id.*

Quarto stipulated that Marcum worked as a coal miner for at least 23 years. JA 163; *see also* Pet. Br. at 5. During the hearing, Marcum testified about his employment history at some length. He stated that, after working underground for Island Creek and Ogelby Norton, he worked at Quarto's Number 4 mine from 1972 through 1994. JA 166. He stated that he spent his first three years at the Number 4 mine working underground before being allowed to bid for any outside work. JA 165. He further testified that, even after he successfully bid for outside work, he would still be periodically transferred back underground for sporadic assignments. JA 164-168, 171-172. When asked how long in total he spent

⁶ As the employer concedes, Consolidation Coal Company is Quarto's parent company. *See* Pet. Br. i. While the two entities are referred to somewhat interchangeably in the record below, the Director refers to both as "Quarto" in this brief for the sake of consistency.

underground at the Number 4 mine, he testified: “Well, it’s kind of hard to say because after I bided outside then they sent me back inside for a few months. Then I’s [sic] back outside again and they sent me back in again. So, it was kind of hard to keep up with what I was doing[.]” JA 167. He also described the amount of dust he was exposed to during his various surface jobs. JA 167-171, 173, 181-183.

Marcum never testified that he worked at any location other than Quarto’s Number 4 mine from 1982 through 1994. Quarto’s counsel did not question him on the matter. Quarto submitted no evidence suggesting that its Number 4 mine is not an underground mine. Nor has it submitted any evidence suggesting that that Marcum was transferred to another location during his years of employment with the company.

2. Evidence relevant to the ALJ’s conclusion that Quarto failed to prove that Marcum’s COPD was not legal pneumoconiosis.

All parties agree that Marcum is totally disabled by chronic obstructive pulmonary disease (“COPD”). Pet. Br. 8.⁷ The medical dispute in this case centers on the cause of Marcum’s totally disabling COPD. The DOL-sponsored physician, Dr. Paul Knight, and Marcum’s physician, Dr. John Schaaf, attribute the

⁷ COPD is an umbrella term that encompasses chronic bronchitis, emphysema, and certain forms of asthma. 65 Fed. Reg. 79939 (Dec. 20, 2000); *see also* The Merck Manual 1889 (19th ed. 2011).

disease to the combination of coal dust inhalation and smoking.⁸ Quarto's experts, Drs. David Rosenberg and Peter Tuteur, attribute Marcum's COPD solely to smoking.

a. Marcum's smoking history.

Marcum, who was born in 1931, testified that smoked cigarettes from "around [age] seventeen" until he quit in 1981. JA 1, 177. He stated that he did not keep track of the number of cigarettes he smoked, but that "I never smoked over a pack [per day.]" JA 178. He was certain that he stopped in 1981 because he "became a Christian" in that year and "didn't believe in smoking tobacco or drinking[.]" JA 178-179. While the various physicians reported slightly different smoking histories in their reports, none varied significantly from Marcum's testimony.

b. Dr. Knight's testimony.

Dr. Knight, who is board-certified in internal medicine, examined Marcum on October 21, 2010. JA 15. He noted that Marcum had been hospitalized for COPD in January, March, and September of that year. JA 16. He recorded a smoking history of a pack a day that started in 1948, but that stopped completely in

⁸ Each miner who files a claim must "be provided an opportunity to substantiate his or her claim by means of a complete pulmonary evaluation." 30 U.S.C. § 923(b). These exams are provided by the Department of Labor at no cost to the miner. 20 C.F.R. § 725.406(a).

1981. *Id.* Marcum reported sputum, wheezing, dyspnea, orthopnea, and ankle edema. *Id.* Dr. Knight further noted that Marcum’s chest x-ray showed “cardiomegaly, emphysema, S/P nodules, [and] perfusion 1/1.” *Id.* His pulmonary function study showed “[m]oderate obstruction” that showed “some” response to a bronchodilator with a “[s]ignificant decrease also in the forced vital capacity.” *Id.* Dr. Knight also recorded that Marcum “is on oxygen 24/7.” JA 17.

Dr. Knight diagnosed “COPD”, “pulmonary emphysema” and “coal worker’s pneumoconiosis” based on “a history of significant tobacco smoking,” chest x-rays, and a “history of extensive deep coal mining work.” JA 18. At his deposition, Dr. Knight clarified that Marcum “had both smoking reasons and coal workers’ dust exposure reasons to have chronic bronchitis.” JA 52. He attributed Marcum’s COPD, which he considered an umbrella term including both Marcum’s bronchitis and emphysema, to “a combination of smoking and his years of dust exposure in mining,” JA 61, which he thought were “co-equal” factors in his disability. JA 62.

c. Dr. Schaaf’s testimony.

Dr. Schaaf, who is board-certified in internal medicine, pulmonary disease, critical care medicine, and medical examining, examined Marcum on August 5, 2010. He noted that Marcum’s breathing difficulties had gotten progressively worse over the years, and that he had been hospitalized for breathing difficulties

several times in 2010. JA 5-6. He recorded a smoking history of “about 20 to 25 years” of a pack a day that ended in 1981. JA 6.

Dr. Schaaf wrote that Marcum suffers from “chronic bronchitis which began during his coal mine employment” and was “associated with severe obstructive airways disease.” JA 8. Dr. Schaaf noted that Marcum “quit smoking in 1981 and had smoked perhaps a pack of cigarettes a day for up to 30 years.” JA 9. But he “specifically note[d]” that Marcum’s “symptoms of chronic bronchitis [began] long after he stopped smoking.” JA 9. The doctor concluded that Marcum’s “coal dust exposure is a significant contributing factor to his chronic bronchitis [COPD].” JA 11.

In his deposition, Dr. Schaaf testified that the diagnosis of chronic bronchitis was based on Marcum’s history of a “chronic cough productive of sputum most days of the week for three consecutive months for two consecutive years.” JA 97. He attributed the airflow obstruction to two sources: Marcum’s coal dust exposure and his smoking. JA 100. While he could not definitively say which of those sources caused the bronchitis, he testified that because Marcum did not get bronchitis until several years after he quit smoking his continued coal dust exposure was “the more likely cause of [it].” JA 101.

Dr. Schaaf explained that smoker’s chronic bronchitis typically improves after smoking cessation “and if you smoke then quit and you don’t have chronic

bronchitis, you don't go on to develop [it]." JA 103. Still, he could not "attribute the entirety of the severe airways obstruction" solely to coal mine employment because of Marcum's smoking history. JA 122. Put another way, he attributed Marcum's chronic bronchitis to coal dust exposure, but could not "exclude that perhaps his cigarette smoking [also contributed] to [his] airflow obstruction." *Id.*

Dr. Schaaf further testified that he disagreed with Dr. Rosenberg's central position in this case: that "patients who have diseases of the lungs due to coal dust exposure have a normal FEV₁/FVC ratio." JA 144. Dr. Schaaf had trouble with this definition of COPD because he believes that "patients who have coal dust exposure can have obstructive airways disease" and "that is manifest when the FEV₁/FVC ratio is reduced." *Id.*

Dr. Schaaf therefore did not agree that COPD caused by coal dust exposure can be distinguished from COPD caused by smoking on this basis. He further did not believe that a study cited by Dr. Rosenberg supported the proposition that the COPD miners acquire is "uniquely different from the COPD found in the general population." *Id.* He also disagreed with Dr. Rosenberg's assessment of a study authored by Soutar and Hurley that Rosenberg claimed showed that "[s]moking was associated with reduction in the ratio of FEV₁/FVC, but dust exposure was not related to this ratio." JA 148. Instead, Dr. Schaaf testified that Dr. Rosenberg is

“almost, but not totally, unique” in his definition of obstruction, which is not “recognized widely.” JA 144.

d. Dr. Rosenberg’s testimony.

Dr. Rosenberg, who is board-certified in internal medicine, pulmonary disease, and occupational medicine, examined Marcum on February 28, 2011. JA 23. Dr. Rosenberg reported that Marcum had “been on oxygen 24/7 for the last several years” and that his breathing problems have worsened over the last five years. JA 28. Like Drs. Schaaf and Knight, Dr. Rosenberg concluded that Marcum was totally disabled by COPD. JA 29 (“Mr. Marcum has a severely disabling obstructive lung disease.”). Unlike those doctors, however, Dr. Rosenberg attributed that COPD solely to smoking. JA 32.

Dr. Rosenberg’s diagnosis was based on Marcum’s pulmonary function test results, which revealed a “a marked reduction of [Marcum’s] FEV₁/FVC ratio” in addition to a “severe reduction in his FEV₁ value.” JA 32; *see supra* at 7-9 (explaining pulmonary function tests). Dr. Rosenberg acknowledged that the “Global Initiative for Chronic Obstructive Pulmonary Disease defines COPD broadly as a reduction in the FEV₁/FVC ratio[.]” JA 30. But he claimed that recent medical literature establishes that COPD can exist even in the absence of a reduced FEV₁/FVC ratio. *Id.*

According to Dr. Rosenberg, this ratio-preserving form of COPD “is the ‘norm’ in patients with coal mine induced obstructive disease.” JA 30. “[T]he opposite is true” in patients with COPD caused by cigarette smoking, “where the ratio is decreased.” *Id.* Because Marcum’s pulmonary function test results revealed the “classic” ratio-reducing form of COPD that Dr. Rosenberg associates with smoking rather than exposure to coal-mine dust, the doctor concluded, “with a reasonable degree of medical certainty,” that Marcum does not suffer from legal pneumoconiosis and that, while the miner “is disabled from a pulmonary perspective, this relates to his long smoking history with resultant smoking-related COPD.” JA 32.

e. Dr. Tuteur’s testimony.

Dr. Tuteur, who is board-certified in internal medicine and pulmonary disease, submitted a consultative report on Quarto’s behalf dated August 22, 2011. JA 72. After reviewing the medical record, Dr. Tuteur found that Marcum suffered from COPD, but gleaned that there was “no convincing data to indicate the presence of a coal-mine induced pulmonary problem.” JA 80. According to Dr. Tuteur, since the severity of Marcum’s airflow obstruction varied over time, it suggested that his COPD was instead related to tobacco smoke exposure. *Id.* This variability indicated “the absence of fixed abnormalities so characteristic of coal mine dust-induced pulmonary disease.” *Id.*

Dr. Tuteur admitted that “coal mine dust may be the etiological agent, in general, for the development of this COPD phenotype as depicted by Mr. Marcum.” JA. 80. But the doctor identified four “lines of reasoning” that allowed him “to conclude with reasonable medical certainty that coal dust was not etiological [sic] responsible here.” *Id.* First, according to Dr. Tuteur, “it is extremely well established that non-mining cigarette smokers with a history similar to Marcum (thirty to sixty pack years) develop a COPD phenotype approximately 20% of the time” and that North American coal miners develop legal pneumoconiosis “less than 2%” of the time. JA 80-81. Second, he stated that “the average rate of [FEV₁] fall of never mining non-smokers compared to miners who never smoked is about the same” which indicates that coal-mine dust causes COPD “so infrequently that it does not affect average values.” JA 81. Third, he cited a 1994 study that “asks the question of whether or not coal mine dust ever produces clinically meaningful airflow obstruction” and that found a 3% prevalence of COPD in a population of 37 non-smoking miners. *Id.* According to Dr. Tuteur, the study “indicates the infrequent occurrence of coal mine dust-induced COPD.” *Id.*

The fourth reason Dr. Tuteur gave for attributing Marcum’s COPD to smoking was Dr. Rosenberg’s opinion. Dr. Tuteur found his colleague’s analysis persuasive, stating “as elegantly presented by Dr. David M. Rosenberg in his report

of March 14, 2011, . . . the typical distribution of pulmonary changes of COPD caused by cigarette smoking and coal mine dust differ.” JA 81. Thus, the “argument of Dr. Rosenberg further reduces the likelihood” of legal pneumoconiosis and “increases the robustness of the conclusion that with reasonable medical certainty, in this case the etiology of Mr. Marcum’s chronic obstructive pulmonary disease is the inhalation of cigarette smoking superimposed on the substantial risk imposed by childhood pulmonary illnesses.” *Id.*

3. The decisions below.

a. The ALJ’s decision awarding benefits.

As a threshold matter, the ALJ found that Marcum had enough qualifying employment at an underground mine to invoke the fifteen year presumption. JA 200. The ALJ agreed with Quarto that Marcum had not established that he worked underground for more than fifteen years. *Id.* But he found that Quarto’s Number 4 mine “was clearly an underground mine as miner testified that he initially worked underground and was later switched from above to belowground and back up again.” JA 200-201 (citing JA 171-172). He recognized Quarto’s argument that Marcum had not established that the Number 4 mine was an underground mine, but rejected it on the basis of (1) Marcum’s indication that the mine was underground on his CM-911a form and (2) the logical observation that “if this

were not an underground mine site, [Marcum] could not have been ‘sent underground.’” JA 172 n.18.

As a result, all of Marcum’s time working at the Number 4 mine qualified toward the fifteen-year presumption. JA 201 (citing Board authorities). The ALJ accordingly found that Marcum had established “at least 25 years of coal mine employment” (23 years at Quarto and 30 months total with Island Creek and Ogelby Norton). JA 201 and n.19. Because there was no dispute that Marcum suffered from a totally disabling respiratory impairment, the ALJ ruled that the fifteen-year presumption had been invoked and turned to the question of rebuttal. JA 201.

The ALJ correctly observed that Quarto could rebut the presumption by proving (1) that Marcum did not suffer from either clinical or legal pneumoconiosis, or (2) that no part of Marcum’s disability was due to pneumoconiosis. JA 201, 204. Based primarily on the x-ray evidence, the ALJ determined that Quarto had established that Marcum did not suffer from clinical pneumoconiosis. JA 202. He found, however, that the employer had failed to prove that Marcum’s COPD was not legal pneumoconiosis. JA 204. He recognized that Drs. Rosenberg and Tuteur attributed Marcum’s COPD entirely to smoking, but he did not credit their testimony.

The ALJ found Dr. Rosenberg’s theory—that Marcum’s decreased FEV₁/FVC ratio indicated that smoking rather than dust caused his COPD—to be plainly “inconsistent with the preamble to the regulations, which recognizes ‘that coal dust can cause clinically significant obstructive disease in the absence of clinical pneumoconiosis, as shown by a reduced FEV₁/FVC ratio.’” *Id.* (citing *Taylor v. Manalapan Mining Co.*, BRB No. 10-0403 BLA (Mar. 11, 2011) (unpub.) (rejecting Dr. Rosenberg’s opinion that a reduction in the FEV₁/FVC ratio cannot be caused by coal dust exposure as contrary to the preamble)). The ALJ therefore decided that Dr. Rosenberg’s opinion was entitled to “little weight.” *Id.* (citing, *inter alia*, *Jericol Mining Inc. v. Napier*, 301 F.3d 703 (6th Cir. 2002) (an ALJ must determine if a medical opinion is supported by the cited medical literature and whether it is consistent with the DOL’s comments to the regulations)).

The ALJ rejected Dr. Tuteur’s opinion for several reasons. First, he found the portion of Dr. Tuteur’s opinion that cited medical literature for the proposition that legal pneumoconiosis occurs “less than 2 percent of the time” in coal miners to rely on “gross generalities” rather “than on the specifics of the miner’s condition.” JA 204, citation omitted. Second, he found that Dr. Tuteur’s reliance on Dr. Rosenberg’s discredited views also affected his credibility: “to the extent Dr.

Tuteur relied on Dr. Rosenberg’s analysis to ‘increase[] the robustness’ of his conclusion, this reliance is misplaced, for the reasons outlined above.” *Id.*

Having found that Quarto had failed to rebut the presumption by proving that Marcum’s COPD was not legal pneumoconiosis, the ALJ turned to the question of whether the employer had successfully ruled out any connection between pneumoconiosis and Marcum’s disability. JA 204-205. He found that Drs. Rosenberg and Tuteur’s opinions on disability-causation were unpersuasive because they “erroneously dismissed the possibility of pneumoconiosis.” JA 205 (citing *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109 (4th Cir. 1995) and *Grigg v. Director, OWCP*, 28 F.3d 416 (4th Cir. 1994)). Finding that the fifteen-year presumption had been invoked and not rebutted, the ALJ awarded BLBA benefits. JA 205-206.

b. The Board decision affirming the award.

Marcum timely appealed to the Benefits Review Board, which affirmed. The Board flatly rejected Quarto’s arguments regarding Marcum’s length of qualifying coal mine employment. According to the Board, the ALJ’s “determination that [Marcum] worked for [Quarto] as an aboveground worker at an underground coal mine from 1971-1994, is rational and supported by substantial evidence in the form of claimant’s testimony at the hearing, his employment records, and the absence of evidence to the contrary.” JA 211, citations omitted.

As a result, the ALJ properly found that Marcum “was not required to show comparability of environmental conditions in order to qualify for the [fifteen year] presumption.” *Id.*, citations omitted.

The Board also found that Quarto’s arguments regarding the rejection of its doctors’ opinions were “without merit.” JA 213. Instead, the ALJ rationally determined that:

Dr. Rosenberg’s opinion that [Marcum’s] reduced FEV₁/FVC ratio indicated that [Marcum’s] impairment was not due to coal dust exposure is in conflict with the preamble to 2001 regulations, ‘which recognizes that coal dust can cause clinically significant obstructive disease in the absence of clinical pneumoconiosis, as shown by a reduced FEV₁/FVC ratio.’

Id., citations omitted. Moreover, the Board found that “the fact that Dr. Rosenberg cited more recent medical literature did not require the administrative law judge to conclude that advancements in science have negated the medical literature addressing the effects of coal mine dust exposure on the lungs that was endorsed by DOL in the preamble.” *Id.*, citations omitted.

The Board also found that the ALJ was correct in discrediting Dr. Tuteur’s opinion because, in addition to adopting Dr. Rosenberg’s analysis, “he relied, at least in part, on generalities and statistics, rather than the miner’s specific condition, and believed that pneumoconiosis is rare.” *Id.*, citations omitted. The Board thus concluded that the opinions of Rosenberg and Tuteur were insufficient to rule out a possible causal connection between Marcum’s disability and his coal

mine employment. In light of that determination, the Board affirmed the award of benefits. JA 215.⁹ This appeal followed.

SUMMARY OF THE ARGUMENT

The ALJ correctly ruled that Marcum was entitled to the fifteen year presumption by virtue of working for more than fifteen years at underground coal mines, even though Marcum may not have actually worked underground for fifteen years. It is undisputed that Marcum worked for at least 23 years for Quarto at the end of his career. It is equally undisputed that if that period of employment occurred at a site including an underground mine, regardless of where Marcum worked at the site, it counts toward the fifteen years of employment needed to invoke the fifteen-year presumption.

Marcum's reported employment history and detailed testimony establish that the mine was underground. Nothing suggests that he was ever transferred away from the site. Quarto never challenged Marcum's testimony or the documentation below. Nor did it introduce any of its own evidence that the mine it owned (Number 4) was solely a surface mine or that it transferred a miner it exclusively employed (Marcum) to any other work site. The *only* reasonable conclusion under the circumstances is that Marcum worked those 23 years at an underground mine

⁹ The Board also rejected Quarto's challenge to the legal standards governing rebuttal, which are not challenged in this appeal. JA 212-213; *see* Pet. Br. 8-9.

site. The ALJ's decision is therefore supported by much more than substantial evidence.

Quarto's challenges to the ALJ's evaluation of its medical experts should also be rejected. The ALJ permissibly discredited Dr. Rosenberg's diagnosis because it conflicts with the scientific underpinnings of the BLBA regulations as expressed in their preamble. This Court recently upheld an ALJ's rejection of an essentially identical report submitted by Dr. Rosenberg for the same reasons in *Central Ohio Coal*, and the same result should obtain here. The ALJ also correctly recognized that Dr. Tuteur's opinion was based on statistical generalities rather than an analysis of Marcum's specific condition. The ALJ permissibly concluded that neither doctor provided evidence sufficient to meet Quarto's burden of proving that coal-mine dust did not cause or contribute to Marcum's COPD. The ALJ's weighing of the evidence and resulting award of benefits should be affirmed.

ARGUMENT

A. Standard of Review

Quarto challenges the ALJ's credibility determinations, which must be affirmed if they are supported by substantial evidence, *Peabody Coal Co. v. Hill*, 123 F.3d 412, 415 (6th Cir. 1997), "even if the facts permit an alternative conclusion[.]" *Youghiogheny & Ohio Coal Co. v. Webb*, 49 F.3d 244, 246 (6th Cir. 1995). To satisfy the substantial evidence standard, the ALJ must adequately

explain why he weighed the evidence as he did. *Morrison*, 644 F.3d at 478. “A remand or reversal is only appropriate when the ALJ fails to consider all of the evidence under the proper legal standard or there is insufficient evidence to support the ALJ’s finding.” *McCain v. Director, OWCP*, 58 F. Appx. 184, 201 (6th Cir. 2003).

B. The ALJ’s finding that Marcum’s work for Quarto occurred at an underground coal mine is supported by substantial evidence.

The fifteen-year presumption is available to miners employed for fifteen years or more in one or more underground mines who suffer from a totally disabling respiratory or pulmonary impairment. 30 U.S.C. § 921(4)(c). For purposes of the presumption, time spent working aboveground at an underground mine counts towards the threshold based on the DOL’s definition of “underground mine.” After notice-and-comment rulemaking, the Department of Labor defined an “underground coal mine” as “a coal mine in which the earth and other materials which lie above and around the natural deposit of coal (i.e., overburden) are not removed in mining; including all land, structures, facilities, machinery, tools, equipment, shafts, slopes, tunnels, excavations and other property, real or personal, appurtenant thereto.” 20 C.F.R. § 725.101(a)(30).¹⁰

¹⁰ Time a miner spends working in a coal mine other than an underground mine also counts toward the fifteen-year requirement, but only if conditions in that mine “were substantially similar to conditions in an underground mine.” 30 U.S.C. § 921(c)(4); see *Central Ohio Coal Co.*, 2014 WL 3858471, *6; 20 C.F.R.

This Court recently upheld the validity of the definition in applying the fifteen-year presumption. *Island Creek Kentucky Mining v. Ramage*, 737 F.3d 1050, 1058 (6th Cir. 2013) (accepting the DOL definition of underground mine and concluding “that no showing of comparability of conditions is necessary for an aboveground employee at an underground coal mine.”). Quarto does not challenge that finding in *Ramage*. See Pet. Br. 32 (“The issue is not whether Mr. Marcum’s years of aboveground mining, assuming they occurred at an underground mine site, constitute ‘qualifying coal mine employment’ for the purposes of invoking the 15-year presumption—in the Sixth Circuit they do.”).

The sole question here is thus whether substantial evidence supports the ALJ’s factual finding that the Number 4 mine where Marcum worked during his 23 years with Quarto was an underground coal mine. Quarto faces a heavy burden in making this substantial-evidence challenge, because the ALJ’s finding must be upheld so long as it is supported by more than a

§ 718.305(b)(1)(i), (c). Because the ALJ found that Marcum worked at underground coal mines for more than fifteen years, it was unnecessary for him to rule on the issue of substantial similarity. See *Ramage*, 737 F.3d at 1058-59.

“mere scintilla” of evidence in the record. *Peabody Coal Co. v. Odom*, 342 F.3d 486 (6th Cir. 2003).

Using this deferential standard, the ALJ’s finding easily passes muster. Marcum reported on his employment history form that the mine was “underground,” that he was a “bolter inside the mine” as well as a welder and mechanic outside of the mine. DX 6. He testified that he worked underground at the Quarto mine for three years before being allowed to bid on aboveground work, and then was periodically transferred back underground for sporadic assignments even after those first three years. JA 164-168, 171-172. It is difficult to quibble with the ALJ’s logic: “if this were not an underground mine site, [Marcum] could not have been ‘sent underground.’” JA 172 n.18.

Quarto—which, as the owner of the mine was in a unique position to discredit Marcum’s statements—did nothing to cast any doubt whatsoever on the miner’s testimony. It did not submit any evidence of its own. It did not cross-examine Marcum on the issue at the hearing. Indeed, Quarto’s argument on appeal is not that its Number 4 mine is actually a surface mine or that it transferred Marcum to other mines during those periods when he worked underground, but only that the record lacks direct evidence establishing that all of Marcum’s work for Quarto took place at one mine site that contained an underground mine. Pet. Br. 33. But direct evidence of that sort is not required. Marcum’s testimony that

his work for Quarto occurred at the Number 4 mine and his credible description of working both aboveground and underground during his tenure with Quarto is a sufficient basis—particularly in the absence of any contrary evidence—for the ALJ’s finding that Marcum’s work for Quarto occurred at an underground coal mine. The Court should decline Quarto’s invitation to usurp the ALJ’s fact-finding function.

C. The ALJ permissibly discredited Dr. Rosenberg’s opinion as contrary to the regulatory preamble.

Quarto also challenges the ALJ’s conclusion that it failed to rebut the fifteen-year presumption. Pet. Br. 13-16. Primarily, this attack focuses on the ALJ’s conclusion that Dr. Rosenberg’s analysis conflicted with the Department of Labor’s evaluation of the scientific literature in the preamble to the BLBA’s implementing regulations. Quarto concedes that “[a]n agency fact-finder may as a valid exercise of discretion consult the regulatory preamble as a means of determining the credibility of expert medical opinions.” Pet. Br. 13-14 (citing *A&E Coal Co. v. Adams*, 694 F.3d 798, 802 (6th Cir. 2012); *Harman Mining Co. v. Director, OWCP*, 678 F.3d 305, 312 (4th Cir. 2012)). But it argues that the ALJ incorrectly discredited Dr. Rosenberg’s opinion in this case. The argument should be rejected. As this Court recently held in *Central Ohio Coal Co. v. Director, OWCP*, --- F.3d ---, No. 13-3712, 2014 WL 3858471 (6th Cir. August 7, 2014), Dr.

Rosenberg's theory of COPD conflicts with the preamble, and ALJs can discredit his analysis on that ground.

1. Dr. Rosenberg's theory of COPD.

Dr. Rosenberg agrees with the Department's view that occupational exposure to coal-mine dust can cause COPD. Pet. Br. 14. He also agrees that "COPD may be detected by a decrease in the FEV₁ and FEV₁[/FVC] ratio." *Id.* (citing JA 30). Indeed, he admits that the Global Initiative for Chronic Obstructive Pulmonary Disease "defines COPD broadly as a reduction in the FEV₁/FVC ratio." JA 30.¹¹ But Dr. Rosenberg believes that this definition is too narrow. In addition to "classic COPD" that decreases both FEV₁ and FEV₁/FVC ratio, he argues that there is another form of COPD that decreases FEV₁ but has no impact on the ratio. *Id.* His key claim is that, while cigarette smoking causes classic COPD, coal-mine dust typically causes only this newly recognized, ratio-preserving form of COPD. *Id.* This is the linchpin of his diagnosis: he attributes Marcum's COPD to smoking primarily because the miner's FEV₁/FVC ratio decreased, which is "not characteristic of obstruction related to past coal mine dust exposure" but "is classic for obstruction related to one's past smoking history." JA 32.

¹¹ *Accord*, The Merck Manual 1853 (19th ed. 2011) ("Obstructive disorders are characterized by a reduction in airflow, *particularly the FEV₁ and the FEV₁ expressed as a percentage of the FVC (FEV₁/FVC).*" (emphasis added).

2. Dr. Rosenberg’s theory is inconsistent with the preamble.

Quarto’s lead argument is that Rosenberg’s theory is not actually contrary to the preamble. Pet. Br. 13-16. It is difficult to square this claim with the plain language of the preamble itself, which states that “epidemiological studies have shown that coal miners have an increased risk of developing COPD. COPD may be detected from decrements in certain measures of lung function, especially FEV1 and the ratio of FEV1/FVC.” 65 Fed. Reg. 79943 (quoting the National Institute for Occupational Safety and Health’s *Criteria for a Recommended Standard, Occupational Exposure to Respirable Coal Mine Dust*, § 4.2.3.2 (1995)). In any event, the argument is barred by *Central Ohio Coal Co.*, which was issued shortly after Quarto’s brief was filed.

In *Central Ohio Coal Co.*, as in this case, Dr. Rosenberg concluded that the miner’s “COPD was not attributable to coal-dust exposure “because it was ‘characterized by a severe reduction of his FEV1 and FEV1/FVC ratio, while the FEV1/FVC ratio is generally preserved’ when an individual’s COPD is caused by coal-dust exposure.” 2014 WL 3858471, at * 6. Indeed, the key section of Dr. Rosenberg’s medical opinion in *Central Ohio Coal Co.*—five paragraphs explaining the basis for his theory that coal-mine-dust exposure does not cause classic, ratio-reducing COPD—is identical to the opinion he submitted in this case. Compare JA 30-31 with Joint Appendix at 296-297, *Central Ohio Coal Co.*, No.

13-3712.¹² The ALJ in *Central Ohio Coal Co.* discredited Dr. Rosenberg’s theory as contrary to the preamble, and the employer appealed.

This Court affirmed the award, holding that “[t]he ALJ appropriately declined to credit Dr. Rosenberg’s medical opinion because it was inconsistent with the DOL’s position that coal mine dust exposure may cause COPD, with associated decrements in FEV₁/FVC.” *Central Ohio Coal Co.*, 2014 WL 3858471, *6. *Central Ohio Coal Co.* controls this case. Dr. Rosenberg’s opinion “plainly contradicts” the DOL’s evaluation of the medical literature in the preamble. *Id.* The ALJ was therefore permitted to discredit Dr. Rosenberg’s diagnosis on that ground.¹³

3. Dr. Rosenberg’s citation of post-2000 medical studies does not rehabilitate his opinion.

Quarto next argues that, “even if Dr. Rosenberg’s discussion is deemed inconsistent with the science relied upon in the preamble,” the ALJ erred in discrediting the doctor because his conclusions were based “on medical research

¹² Dr. Rosenberg’s full medical report in *Central Ohio Coal Co.* is appended to this brief for the Court’s convenience.

¹³ Dr. Rosenberg’s theory conflicts not only with the preamble, but with the BLBA regulations themselves, which allow miners to establish total disability by, *inter alia*, a pulmonary function test demonstrating a decreased FEV₁/FVC ratio (equal to or less than 55%). 20 C.F.R. § 718.204(b)(2)(i)(C). It would not have made sense to use a reduced ratio as a measure of disability in BLBA claims if, as Dr. Rosenberg believes, a reduced ratio indicates that the disability is not caused by coal-mine dust.

published years after the preamble was issued.” Pet. Br. 16-21. It is true that Dr. Rosenberg cites five articles—Baldi (2001), Bernstein (2004), Fabbri (2007), Rabe (2007), and Tuder (2006)—that were published after the 2000 preamble. JA 33-34. But Quarto’s suggestion that these citations effectively immunize Rosenberg’s theory from critique as contrary to the preamble is inconsistent with both precedent and logic.

The precedential problem is, again, *Central Ohio Coal Co.* Dr. Rosenberg cited the exact same studies in his opinion in that case. *Central Ohio Coal Co.* recognized that an employer could “challenge the substance of the DOL’s position as articulated in the regulation’s preamble” by arguing that “COPD resulting from coal-dust exposure is not correlated with a reduced FEV₁/FVC ratio.” *Central Ohio Coal Co.*, 2014 WL 3858471, *6. And it explained that, to substantiate such a challenge, an employer would need to “submit[] ‘the type and quality of medical evidence that would invalidate’ the DOL’s position in that scientific dispute.” *Id.* (quoting *Midland Coal Co. v. Director, OWCP*, 358 F.3d 486, 490 (7th Cir. 2004)). But the court found it unnecessary to “engage the substance of the scientific dispute” because the employer “*presented no such evidence* and [asked] the court to make no such determination.” *Id.* (emphasis added). If Dr. Rosenberg’s citations were insufficient to challenge the substance of DOL’s

position in *Central Ohio Coal Co.*, those same citations are necessarily insufficient to sustain such a challenge in this case.

Second, Dr. Rosenberg does not rely on any post-2000 articles to support his key theory: that exposure to coal-mine dust does not cause classic, ratio-reducing COPD. Here is how Dr. Rosenberg uses the post-2000 articles he cites:

- Rabe (2007): cited for the proposition that “The Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) defines COPD broadly as a reduction in the FEV_1/FVC ratio[.]” JA 30. Dr. Rosenberg, of course, disagrees with this definition. He believes that COPD can be diagnosed even in the absence of a reduced FEV_1/FVC ratio, and that this ratio-preserving form of COPD is the only form of the disease caused by coal-mine dust. *Id.*
- Fabbri (2007): cited for the proposition that “recent literature (including literature published after D.O.L.’s revisions to the black lung regulations) establishes the limitation of defining COPD as simply a reduction in FEV_1 or $FEV_1\%$ [*i.e.*, FEV_1/FVC ratio].” JA 30. This might support Dr. Rosenberg’s claim that there are forms of COPD that do not cause reductions in the FEV_1/FVC ratio, but says nothing at all about whether coal miners are susceptible to classic, ratio-reducing COPD.
- Tudor (2006) and Bernstein (2004): cited to substantiate claims about the content and size of particles contained in cigarette smoke. JA 31. Dr. Rosenberg does not claim that these articles address coal miners at all.

- Baldi (2001): cited to substantiate the claim that emphysema in cigarette smokers “characteristically” results in a reduced diffusing capacity. JA 32. This claim is unrelated to Dr. Rosenberg’s theory that coal-mine dust does not cause classic ratio-reducing COPD. Moreover, the article does not appear to address miners; Dr. Rosenberg instead cites a pair of pre-preamble articles addressing diffusing capacity in coal miners.

Thus, while Dr. Rosenberg cites five post-preamble studies, he cites them for points that are at best only tangentially related to his central thesis that exposure to coal-mine dust does not cause classic, ratio-reducing COPD. When Dr. Rosenberg articulates that thesis, those post-preamble studies are nowhere to be found:

Thus, while I agree with the D.O.L. that COPD may be detected by a decrease in the FEV₁ and FEV₁/FVC ratio, this does not generally apply to patients with legal CWP. Among the latter, *based on epidemiologic studies accepted by the D.O.L.*, the obstruction is characterized by a preservation of the FEV₁/FVC ratio. In fact, what has been outlined is that the preservation of the FEV₁/FVC ratio is the “norm” in patients with coal mine induced obstructive lung disease. The opposite is true with respect to smoking-related COPD where the ratio is decreased. Thus, patterns of airflow obstruction help determine the etiology of a given miner’s airway obstruction.

JA 30 (emphasis added).

Dr. Rosenberg’s failure to cite the articles he relies on in this paragraph makes it difficult to be certain, but he appears to believe that studies evaluated by

DOL in the preamble itself support his view. What is clear, however, is that Dr. Rosenberg has not identified any post-preamble medical literature supporting his theory that exposure to coal-mine dust does not cause classic, ratio-reducing COPD. The ALJ therefore correctly treated the Department of Labor's evaluation of the relevant medical literature in the preamble as unrebutted, and permissibly discredited Dr. Rosenberg's opinion because it conflicted with that evaluation. *See Westmoreland Coal Co. v. Cochran*, 718 F.3d 319 (4th Cir 2013) (ALJ appropriately discredited medical opinion as contrary to preamble despite doctor's citation of post-preamble studies, "none of which appears to even discuss the effects of coal mine dust exposure on the lungs."). The ALJ committed no error in evaluating Dr. Rosenberg's opinion.¹⁴

D. The ALJ permissibly discredited Dr. Tuteur's medical opinion because it was based on statistical generalities rather than the specifics of Marcum's condition.

The ALJ gave little weight to Dr. Tuteur's diagnosis because it was based on "gross generalities, rather than the specifics of the miner's condition." JA 204. As the ALJ read it, Dr. Tuteur's analysis boiled down to this: coal-mine dust rarely causes COPD, ergo Marcum's COPD was not caused by coal-mine dust. Closer

¹⁴ Quarto objects that the Board erred by finding yet another reason to discredit Dr. Rosenberg: that it was contrary to the DOL's finding in the preamble that coal-mine dust and cigarette smoke cause obstructive impairments through similar mechanisms. Pet. Br. 21-24. At most, this was harmless error by the Board, which also affirmed the ALJ's reasoning. JA 213.

inspection reveals that the ALJ's contrary interpretation was not only reasonable, but correct.

Dr. Tuteur's report purports to give four reasons supporting his conclusion that exposure to coal-mine dust did not cause Marcum's COPD. JA 80. The fourth is simply an incorporation of Dr. Rosenberg's analysis, which the ALJ properly discounted as contrary to the preamble. The first three are, as the ALJ correctly observed, statistical studies purporting to show: (1) that only 2% of miners develop COPD while 20% of smokers with a history similar to Mr. Marcum's develop COPD; (2) that coal-mine-dust induced COPD "occurs so infrequently that it does not affect average values" (comparing FEV₁ decreases in non-smoking miners and non-smoking non-miners); and (3) the "infrequent occurrence of coal mine dust-induced COPD." JA 80-81.

Even assuming that Dr. Tuteur's statistics are correct, they are insufficient to satisfy Quarto's burden of proving that Marcum's COPD was not "significantly related to, or substantially aggravated by, dust exposure in coal mine employment." 20 C.F.R. § 718.201(b). It is well-established that ALJs can discredit medical opinions that "rel[y] heavily on general statistics rather than particularized facts about" the miner. *Harman Mining Co. v. Director, OWCP*, 678 F.3d 305, 312 (4th Cir. 2012); *see also Antelope Coal Co./Rio Tinto Energy Amer. v. Goodin*, 743 F.3d 1331, 1345-46 (10th Cir. 2014) (affirming ALJ's

finding that employer’s “experts’ reliance on statistical probabilities undermined their ultimate conclusion that Mr. Goodin did not have pneumoconiosis because they did not show why Mr. Goodin is not among the cohort of those who suffer COPD from surface coal mining.”); *Consolidation Coal Co. v. Director, OWCP*, 732 F.3d 723, 734 (7th Cir. 2013) (affirming award where ALJ rejected employer’s expert “because the doctor relied on general statistics without relating them to [the miner] in particular.”). Similarly, Dr. Tuteur has failed to explain why *Marcum’s* COPD was not caused or aggravated by coal-mine dust. Because Dr. Tuteur relied on nothing but blunt generalizations (and Dr. Rosenberg’s discredited theory) to exclude coal-mine dust as a potential cause of Marcum’s COPD, the ALJ permissibly gave his opinion little weight.¹⁵

Quarto argues that Dr. Tuteur’s statistical analysis was “but one part of a multi-faceted and objective scientific analysis that concluded coal mine dust exposure was not the cause of Mr. Marcum’s impairment.” Pet. Br. 25. But it

¹⁵ While the ALJ did not discredit Dr. Tuteur on this ground, others have discounted similar opinions as contrary to the preamble, and the courts of appeals have upheld those determinations. *See, e.g., Peabody Coal Co. v. Director, OWCP*, 746 F.3d 1119, 1127 (9th Cir. 2014) (“The ALJ rationally discounted the testimony of Peabody’s medical experts, who based their opinions on the premise that coal dust exposure never, or very rarely, causes COPD.”). *See also* 65 Fed. Reg. 79939 (“there is a clear relationship between coal mine dust and COPD and lung dysfunction[.]”); *Adams*, 694 F.3d at 801 (summarizing the preamble’s explanation for including COPD within the definition of legal pneumoconiosis).

searches only briefly for a non-statistical justification for Dr. Tuteur’s diagnosis. Quarto seizes on Dr. Tuteur’s observation that some examinations revealed “variable findings of airflow obstruction, typical of COPD, but not of simple coal workers’ pneumoconiosis.” Pet. Br. 25 (citing JA 76). Quarto goes on to point out that Dr. Tuteur noted the absence of various symptoms that would be expected in patients suffering from simple coal workers’ pneumoconiosis. Pet. Br. 25-26 (citing JA 76, 78).

Those observations, however, are simply irrelevant to the question at hand because “simple coal workers’ pneumoconiosis” is a species of *clinical* pneumoconiosis—typically, as Dr. Tuteur admits, a *restrictive* lung condition. JA 78. See *Mitchell v. OWCP*, 25 F.3d 500, 501 (7th Cir. 1994) (“Clinical pneumoconiosis exists in two forms, simple and complicated.”); 20 C.F.R. § 718.202(a)(1) (defining clinical pneumoconiosis). The ALJ ruled in Quarto’s favor on the question of clinical pneumoconiosis, so his failure to explicitly consider this aspect of Dr. Tuteur’s opinion caused Quarto no prejudice. Instead, the ALJ found that Quarto had failed to establish that Marcum’s chronic *obstructive* pulmonary disease was not *legal* pneumoconiosis. JA 203-205. The presence or absence of restrictive clinical pneumoconiosis is simply not relevant to that inquiry. The ALJ therefore committed no error in not considering these observations by Dr. Tuteur in his analysis of legal pneumoconiosis.

The remainder of Quarto's defense of Dr. Tuteur is devoted to proving that the doctor's statistical assumptions were "derived from medical studies." Pet. Br. 26-28. But those statistics, even if true, cannot prove that Marcum's COPD was unrelated to his occupational exposure to coal-mine dust. *See supra* at 36-38. They are therefore insufficient to rebut the presumption that Marcum's COPD is legal pneumoconiosis.

In sum, the ALJ's factual findings and credibility determinations are reasonable and supported by substantial evidence. The ALJ permissibly credited Marcum's uncontradicted testimony about his own work history to find that the miner worked for more than fifteen years at underground coal mines as defined by the BLBA's implementing regulations. That finding, paired with the uncontested fact that Marcum suffers from totally disabling COPD, invoked the fifteen-year presumption. The ALJ's finding that Quarto failed to rebut the presumption by proving that coal-mine dust did not cause or aggravate Marcum's COPD rests on equally solid footing. He reasonably discredited Dr. Rosenberg's opinion because it was based on premises contrary to the Department of Labor's evaluation of the relevant scientific literature in the regulatory preamble, and Dr. Tuteur's because it was based on statistical generalities rather than Marcum's specific condition. Quarto's challenges to these findings, and to the award of BLBA benefits that flowed from them, should be rejected.

CONCLUSION

The ALJ's award of black lung benefits to Marcum should be affirmed.

Respectfully submitted,

M. PATRICIA SMITH
Solicitor of Labor

RAE ELLEN JAMES
Associate Solicitor

SEAN BAJKOWSKI
Counsel for Appellate Litigation

s/Jonathan Rolfe
JONATHAN ROLFE
Attorney, U.S. Department of Labor
Office of the Solicitor
Frances Perkins Building
Suite N-2119
200 Constitution Ave, N.W.
Washington, D.C. 20210
(202) 693-5660
rolfe.jonathan@dol.gov

Attorneys for the Director, Office
of Workers' Compensation Programs

CERTIFICATE OF COMPLIANCE

I hereby certify that this brief complies with the type-volume limitation of Fed. R. App. P. 32(a)(7)(B). This brief contains 9,375 words, excluding the parts of the brief exempted by Fed. R. App. P. 32(a)(7)(B)(iii). I also certify that this brief complies with the typeface requirements of Fed. R. App. P. 32(a)(5) and the type style requirements of Fed. R. App. P. 32(a)(6) because it has been prepared in a proportionally-spaced typeface using Microsoft Word 2010 in fourteen-point Times New Roman font.

s/Jonathan Rolfe
Jonathan Rolfe
Attorney
U.S. Department of Labor

CERTIFICATE OF SERVICE

I hereby certify that on September 24, 2014, an electronic copy of this brief was served through the CM/ECF system on the following:

Jeffrey R. Soukup
Jackson Kelly PLLC
175 E. Main Street, Suite 500
Lexington, KY 40507

Heath M. Long
Pawlowski, Bilonick & Long
603 North Julian Street
P.O. Box 658
Ebensburg, PA 15932

s/Jonathan Rolfe
Jonathan Rolfe
Attorney
U.S. Department of Labor

ADDENDUM

DR. ROSENBERG'S REPORT FROM *Central Ohio Coal Co. v. Director, OWCP*, ---F.3d---, 2014 WL 3858471 (6th Cir. August 7, 2014)



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William S. Mattingly
Jackson Kelly, P.L.L.C.
150 Clay St. – Suite 500
P.O. Box 619
Morgantown, WV 26507

Corporate Health

Occupational & Environmental Health Services

3909 Orange Place, Suite 2300
Orange Village, OH 44122

216 895 1855 Phone
216 896 1851 Fax

**RE: LARRY T. STERLING
OWCP NO. XXX-XX-4000
J&k REFERENCE NO. 17272/305**

Dear Mr. Mattingly:

The following correspondence is in reference to Larry T. Sterling who is a 66 year old gentleman formerly employed in the coal mine industry who evaluated by myself on May 11, 2011. Consequent to this employment, the issues to be addressed are whether or not he has coal workers' pneumoconiosis of the medical or legal variety. In addition, it should be opined whether or not he has any respiratory impairment and/or disability, and if so, was it caused in whole or part by past coal mine dust exposure and the presence of CWP. In preparing this report, in addition to personally examining Mr. Sterling, I have reviewed the following with respect to him:

- 1.) Claim Application;
- 2.) Good Samaritan Medical Center records;
- 3.) Genesis Healthcare System records;
- 4.) Correspondence of Dr. Schowengerdt from August 8, 2000;
- 5.) Records of Dr. Knell;
- 6.) Records of Dr. Forrestal;
- 7.) Evaluation of Dr. Knight from November 9, 2006;
- 8.) Quality evaluation of the pulmonary function tests from November 9, 2006 by Dr. Gerblich;
- 9.) Quality B reading of the film from November 9, 2006 by Dr. Gaziano;
- 10.) Breathing of the film from November 9, 2006 by Drs. Muchnock, Meyer and Miller;
- 11.) Report of Dr. Grodner from April 6, 2007 regarding his evaluation from March 29, 2007;
- 12.) B reading of the chest X-ray from March 29, 2007 by Drs. Fox and Ahmed;
- 13.) Deposition of Dr. Knight;
- 14.) Report of Dr. Diaz from December 2, 2009; and
- 15.) Current pulmonary function tests, arterial blood gas, EKG and chest X-ray and nicotine.

First the Claim Application was reviewed. It indicated that Mr. Sterling had 24½ years of coal mine employment up until 1999. Also, it reported that he had been on supplemental oxygen since 1997, with oxygen at 24/7 dating back to 2003. It was noted that his last job in 1999 was as a strip mine worker, and he also did that dating back to 1969.

**Employer's
Exhibit No. 3**

000290

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Next, pulmonary function tests from Good Samaritan Medical Center from October 2, 1998 were said to reveal severe bronchial obstruction with moderate improvement after bronchodilators. It was reported that the FVC was 1.87 liters (24% predicted). Also, there was increased residual volume of 172% predicted, and the diffusing capacity was 69% predicted corrected for lung volumes. Pulmonary function tests from July 11, 2000 revealed a FVC of 1.79 liters (36% predicted) with an FEV₁ of 0.61 liters (15% predicted) and an FEV₁% of 34%. The flow-volume curves were consistent with severe airflow obstruction.

A chest X-ray from Genesis Healthcare System Bethesda's Hospital from July 11, 2000 was said to reveal mild emphysema.

Next, Dr. Schowengerdt authored a correspondence dated August 8, 2000. Shortness of breath was outlined, and smoking cessation was attempted with Mr. Sterling. He was currently smoking a pack of cigarettes per day down from three packs per day. His medications included Theo 24 600mg/day, Serevent, Atrovent, Proventil, Flovent, Azmacort, as well as Flonase nasal spray, Lamisil and Motrin. It was noted that his room air percent saturation was 87%, and he had low diaphragms with clear lung fields. He was felt to have advanced emphysema, and it was reported that he would be expected to die within several years if he did not stop smoking. Various medications were prescribed to help with smoking cessation.

Dr. Knell evaluated Mr. Sterling on August 6, 2002. He was said to have undergone a colorectal screen and was on Social Security Disability for COPD. Decreased breath sounds were heard, and he had guaiac positive stools, and a colonoscopy was to be performed. Multiple polyps were found.

Dr. Forrestal's records from October 24, 2002 outlined chest congestion, and he was smoking a pack of cigarettes per day and had done so for 36 years. No rales, rhonchi or wheezes were heard, and he was felt to have hypertension with unspecified asthma. On February 3, 2003, it was noted that he was on albuterol 2 puffs every 4 hours as needed, Allegra D, Atrovent inhaler 2 puffs b.i.d., Flonase, Flovent inhaler 110 2 puffs b.i.d., Serevent 2 puffs b.i.d., Theo 24 300mg t.i.d. along with Zocor, Vicodin and Viagra. Chest congestion was outlined, and he was felt to have acute bronchitis. A blood gas from April 7, 2003 revealed a pH of 7.40, a PCO₂ of 45mmHg and a PO₂ of 57.9mmHg. Dr. Forrestal outlined congestion on May 12, 2003, and he was treated with antibiotics. Continued congestion was outlined by Dr. Forrestal in different notes, and he was felt to have asthma and chronic obstruction without status asthmaticus. On December 2, 2003, continued cough and congestion with shortness of breath were outlined. Next, it was noted that he was admitted between December 17, 2003 and December 23, 2003 for a COPD exacerbation. He was treated with aerosols, corticosteroids, bronchodilators, etc. Also, he was treated with nasal BiPAP. A COPD exacerbation was noted on December 18, 2003, and he was treated during this admission. It was also noted that he had CO₂ retention with a pH of 7.24. He was to be treated with a BiPAP mask. Dr. Branditz on January 29, 2004 stated that Mr. Sterling had COPD, and he had presented with respiratory insufficiency and CO₂ retention. He was treated with positive pressure. It was felt that he likely had COPD with sleep apnea, and he was to be scheduled for a sleep study. His breath sounds were reduced but quiet. Dr. Branditz considered Mr. Sterling to have sleep apnea and COPD secondary to his smoking history.

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Dr. Forrestal on April 20, 2004 outlined continued airways disease with asthma and chronic obstruction, and he was to be followed. Similar diagnoses were outlined in the file by Dr. Forrestal, and acute bronchitis was treated on November 24, 2004. He also received some corticosteroids on February 13, 2005. He was said to have end-stage COPD, on home oxygen with nebulization treatment. He had been found at home unresponsive, and he was brought to the emergency room and intubated. The initial blood gas revealed a pH of 7.18 and a PCO₂ of 91mmHg, and he was placed on a FIO₂ of 50% with improved oxygenation. Overall, he was felt to have end-stage COPD with end-stage emphysema and acute bronchitis versus pneumonia. He was admitted to the ICU and treated with ventilatory support, IV steroids and antibiotics, etc. per Dr. Alahakoon. The echocardiogram from February 15, 2005 revealed a left ventricular ejection fraction of 45% with a moderately dilated right ventricle with moderate hypokinesia. A dilated inferior vena cava was also seen. The chest X-ray from February 28, 2005 revealed bibasilar pulmonary infiltrates, and underlying COPD was suggested.

Mr. Sterling was seen in follow-up as an outpatient on April 1, 2005 and was given Spiriva. He was felt to have classic angina per Dr. Van Gilder as reported on April 13, 2005. A catheterization was to be performed, and he also was considered to have right-sided heart failure due to cor pulmonale with advanced COPD. A subsequent catheterization as outlined on April 15, 2005 revealed only minor coronary artery disease with up to 25% blockages or so in the left main coronary artery.

He was seen by Dr. Van Gilder on May 18, 2005 with mild coronary artery disease and COPD. It was noted that he was using oxygen, and some wheezes were heard. Dr. Forrestal's records contained routine follow-up with stable coughing with shortness of breath and wheezing as was noted on October 12, 2005. He was said to have acute bronchitis on January 17, 2006, and was treated accordingly. It was noted that he was on chronic oxygen on April 13, 2006, and complained of weakness and shortness of breath. The diagnoses continued unchanged, and on September 27, 2006, cough, shortness of breath and wheezing were noted with chest congestion.

Next, Dr. Knight performed an evaluation on November 9, 2006. It was reported that he had smoked starting at age 21 up until February, 2005, smoking one pack of cigarettes per day for ten years, and then two packs per day until he quit. He had had breathing problems dating back to 2002 or so, and had been on a ventilator in February, 2005. Attacks of wheezing were outlined, along with sputum production and dyspnea, hemoptysis, chest pains, orthopnea and ankle edema. Trouble sleeping was outlined, and his chest was hyperresonant with decreased breath sounds. An arterial blood gas revealed a pH of 7.40, a PCO₂ of 49.3mmHg and a PO₂ of 64.4mmHg. The pulmonary function tests were felt to be acceptable by Dr. Gerblisch. They revealed a FVC of 2.58 liters (58% predicted) with an FEV₁ of 0.69 liters (21% predicted) and an FEV₁% of 27%. The MVV was 31 liters/minute (23% predicted). After bronchodilators, the FVC was 2.56 liters (58% predicted) with an FEV₁ of 0.65 liters (19% predicted) and an FEV₁% of 25%. The MVV was 33 liters/minute (25% predicted). The flow-volume and volume-time curves were consistent with severe obstructive lung disease. Dr. Muchnock felt that the chest X-ray from November 9, 2006 revealed predominantly t opacities with some q opacities with a profusion of 1/1. The lung fields were emphysematous. Dr. Gaziano felt the film was quality 2. Dr. Meyer stated the film was 0/0 without changes of a pneumoconiosis. Emphysema was present. Dr. Miller felt the film revealed

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t/q opacities in all lung zones with a profusion of 1/1 with left-sided pleural thickening. Overall, Dr. Knight felt that Mr. Sterling had pulmonary emphysema and CWP. He was considered to have severe impairment and was disabled.

Further notes of Dr. Forrestal from December 14, 2006 outlined acute bronchitis with asthma and chronic obstruction. He was treated with corticosteroids and antibiotics.

Next, Dr. Grodner evaluated Mr. Sterling on March 29, 2007 as reported on April 6, 2007. Shortness of breath was outlined dating back to 1995, and he had been on home oxygen dating back to that time in 1997. In 2003, he began using oxygen 24/7. Dyspnea on exertion was outlined, and he had been on a ventilator in 2005. He complained of shortness of breath with wheezing and sputum production, and his medications included Nexium, Lipitor, Allegra, Theo-Dur, Flovent, Atrovent and albuterol. It was reported that he quit smoking two years before, having smoked for 38 years, 1½ packs of cigarettes per day. He also had worked in the coal mines from 1975 to 1999. He worked in the strip mines, and thereafter, he operated a dozer, tearing down power plants. He also worked for a foundry, and on examination, he was 69½ inches in height and weighed 289 pounds, on supplemental oxygen. Diminished breath sounds were heard, but were clear, and the X-ray was read by Dr. Fox as being 0/0. His spirometry was said to reveal severe airflow obstruction, and his percent saturation dropped with minimal exertion. Overall, he was felt to have severe COPD without CWP. In addition, he was considered disabled, but this did not relate to past coal mine dust exposure. The recorded FVC was 1.13 liters (25% predicted) with an FEV₁ of 0.45 liters (13% predicted) and an FEV₁% of 39%. Another set of pulmonary function tests revealed a FVC of 1.45 liters (32% predicted) with an FEV₁ of 0.54 liters (15% predicted) and an FEV₁% of 37%. The FVC was variable, with the best value being 1.45 liters and the second best being 1.25 liters. The flow-volume curves revealed severe airflow obstruction. It was noted that Dr. Fox stated the film was 0/0 with streaking at the left base. An arterial blood gas revealed a pH of 7.42, a PCO₂ of 49.9mmHg and a PO₂ of 66.2mmHg with a carboxyhemoglobin level of 2.1%. Another blood gas revealed a pH of 7.42, a PCO₂ of 50.6mmHg and a PO₂ of 38.6mmHg. Dr. Ahmed felt that the chest X-ray revealed s/t opacities in the mid and lower lung zones with a profusion of 1/1 with emphysematous changes.

Next, the deposition of Dr. Knight from December 13, 2007 was reviewed. His evaluation of Mr. Sterling was outlined, and it was noted that he last worked in the coal mines in 1999, having begun in 1969, working on the surface. It was noted that he operated heavy equipment. His smoking history was a pack per day for ten years, and two packs per day up until 2005, which was around 86 pack-years. Thereafter, it was corrected to 68 pack-years. It was felt that his lung disease related to both cigarette smoking and coal dust exposure. He also was felt to have chronic bronchitis. Wheezing was reported, and he stopped smoking in 2005 at the time he was placed on a ventilator. It was noted that he had been on oral corticosteroids and inhaled corticosteroids, as well as various bronchodilators, and he had a very hyperresonant chest on examination consistent with significant obstruction and emphysema. It was also felt that obesity was playing an adverse role on his lung function. It was noted that his pulmonary function tests revealed the most severe form of obstructive lung disease. It was noted that he had hypoventilation and it was contraindicated for him to exercise. It was noted that Dr. Muchnock outlined p and q opacities with emphysema. He was felt to have CWP based on the presence of nodules and the history of dust exposure. Also, it was felt that his impairments were more related to smoking than dust

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exposure. It was felt that the obstructive pattern observed very uncommonly was seen with coal dust exposure, but a component of his impairment was coal mine dust related.

Next reviewed was a correspondence of Dr. Diaz from the Ohio State Medical Center dated December 2, 2009. He had evaluated Mr. Sterling who reported severe dyspnea. It was reported that he had severe COPD with an FEV₁ of 21% predicted which was in the range to consider lung transplantation. It was also his opinion that occupational dust exposure had contributed significantly to his disease, which was rendering him unable to perform his coal mine employment. Mr. Sterling was said to have worked in the coal mines for 31 years up until 1999. Also, he was said to have a 57 pack-year history of smoking. Furthermore, it was stated that coal dust exposure was evident on his X-ray documented by the presence of linear and nodular opacities.

At the time of **MY EVALUATION** Mr. Sterling reported that he had had shortness of breath dating back to around 1993 which was getting worse. Recently, he began pulmonary rehabilitation which was helping. He normally was on around 3 liters/minute of oxygen, and he would increase his oxygen flow rate up to 4 to 6 liters/minute with exercise. He went on to report that he stopped smoking on February 14, 2005 when he was intubated with respiratory failure. Currently, because of his respiratory problems, he had difficulty performing activities of daily living. In the past, he had cough and sputum production, but bronchitis was not a major issue since he stopped smoking. He slept in a recliner and had some swelling which was treated with a water pill. He also had atrial fibrillation last year that was treated with ablation. Also, he had been on Coumadin for a period of time.

His **PAST MEDICAL HISTORY** was notable for no medicine allergies, and currently, he was on Theo 24 300mg t.i.d., Proventil HFA as needed, one baby aspirin per day, alprazolam .25mg PRN, Actos plus metformin 15/500 b.i.d., prednisone PRN, metoprolol 50mg b.i.d., Lasix 40mg/day, Lipitor 40mg/day, promethazine-codeine cough syrup, Flovent 220 2 puffs b.i.d., Spiriva q.d. and fluticasone nasal spray 2 puffs q.d. He had had the cardiac ablation for atrial fibrillation last year, with right knee surgery. He had had four to five admissions for respiratory failure, most recently in 2008. He was intubated in 2005, being on a ventilator for an extended timeframe. He reported having pneumonia at least on one occasion, with the usual childhood illnesses, without a history of whooping cough, TB or asthma. He had no history of hiatal hernia, eczema, hayfever, nasal polyps, fractured ribs or congestive heart failure.

His **FAMILY HISTORY** was notable in that his mother died with diabetes and his father had Black Lung Disease, having worked in the deep mines.

His **SOCIAL HISTORY** was notable in that he was married, and he smoked from 1966 to 2005. Overall, he was said to have smoked a pack of cigarettes per day for the first 12 to 13 years, then he smoked 1½ packs of cigarettes per day for another 13 years, and thereafter, 2 packs per day. Overall, he reported averaging 1½ packs per day for 38 years.

His **WORK HISTORY** was notable for 31 years of coal mine employment up until the year 2000. He became disabled at that point in time. He had been laid off and could not pass a physical examination to return to the mines. He was a mechanic and equipment operator during

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his timeframe of employment, about half of the time doing each. As such, he would drive trucks loaders, dozers, etc. He would load trucks with coal, and he also had cleaned the dozer with a shovel which he reported to be quite hard work. The cabs were closed at the end, but during the first portion of his employment were open, and respiratory protection was not utilized. He also worked as a mechanic, and a lot of road dust was created, since he would be working in areas where other vehicles drove by. Finally, for a period of time, he also worked at the tipple, and for two years, he shot coal.

His **REVIEW OF SYSTEMS** was pretty much negative except as noted.

On **PHYSICAL EXAMINATION** he appeared in no distress on oxygen, with a respiratory rate of around 16 to 20 breaths/minute and a pulse rate of around 68 beats/minute and regular. His head, ears, eyes, nose and throat revealed no use of accessory muscles. He had hyperresonance with markedly diminished breath sounds. His cardiac sounds were distant, without murmurs, gallops or rubs, and his abdomen was protuberant, without masses or areas of tenderness. Spirometry was attempted. He was taken off of his oxygen, and his **percent saturation** quickly dropped to 79%. The **spirometry** that was obtained revealed a FVC of 1.68 liters (41% predicted) with an FEV₁ of 0.48 liters (14% predicted) and an FEV₁% of 28%. The flow-volume and volume-time curves were consistent with severe airflow obstruction. He was immediately placed back on the oxygen. A **blood gas** while on oxygen at 3 liters/minute revealed pH of 7.36, a PCO₂ of 67mmHg and a PO₂ of 72mmHg. Next, his **chest X-ray** was reviewed which demonstrated cardiomegaly with diffuse emphysematous changes. There were increased markings in the mid and lower lung zones compatible with his chronic obstructive pulmonary disease without micronodularity (0/0) related to past coal mine exposure.

In **SUMMARY**, Mr. Sterling is a 66 year old gentleman who complains of shortness of breath with minimal exertion. He has been on oxygen for an extended timeframe and has a long smoking history, having worked on the surface mines for around 30 years. He has had a problem with atrial fibrillation, undergoing ablation, and he has been intubated on one occasion for a prolonged timeframe because of respiratory failure. On examination, currently, he had marked hyperresonance with decreased breath sounds. He desaturated quickly, being taken off of his oxygen at 3 liters/minute. Spirometry revealed severe airflow obstruction, and it was reported in the past that he had had a low diffusing capacity. His pulmonary function tests were characterized by a severe reduction in his FEV₁ with a marked reduction of the FEV₁ / FVC ratio. Also, he has marked hypoxemia.

DISCUSSION: Based on a review of the above information, it can be appreciated that Mr. Sterling does not have micronodularity related to past coal mine dust exposure. Rather, any linear markings in the mid and lower lung zones related to his chronic obstructive pulmonary disease (COPD). Linear markings of this nature would not relate to the past inhalation of coal mine dust exposure. One should also appreciate that while his FVC was reduced, this undoubtedly relates to the presence of severe air trapping consequent to his severe chronic obstructive pulmonary disease (COPD). With respect to air trapping, his residual volume (RV) when measured in 1998 was 172% predicted. Mr. Sterling's severe COPD correlates with his marked hypoxemia and his examination revealing marked hyperresonance and decreased breath

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sounds. When all of the above information is looked at in total, Mr. Sterling does not have the condition of clinical coal workers' pneumoconiosis (CWP).

From an impairment perspective, Mr. Sterling has severe disabling obstructive lung disease characterized by marked decrement of his FEV_1 and FEV_1 / FVC ratio with marked hypoxemia. He clearly can not perform his previous coal mine job or other similarly arduous types of labor. The next issue pertains to what is the etiology for his disabling obstruction.

While there is no question coal mine dust exposure can cause significant airflow obstruction which is disabling, just because a given miner has airflow obstruction does not automatically mean that legal CWP is present. The reason for this is that miners are also susceptible for developing disorders which affect the general public. In order to ascertain whether a given miner's airways disease represents legal CWP versus obstruction caused by other factors, the specific characteristics of the miner's airways disease need to be assessed. With this understanding, one should appreciate that various epidemiologic studies have been performed in miners to determine the characteristic pattern of obstruction that develops in relationship to past coal mine dust exposure (Morgan; Soutar and Hurley; Attfield and Houdos). In regards to this, these authors have determined that while the FEV_1 decreases in relationship to coal mine dust exposure, the measurement of the FEV_1 / FVC ratio generally is preserved. In contrast, with smoking-related forms of COPD, the FEV_1 / FVC ratio is generally reduced (Huhti; Ashley; Balchum; Coates). Furthermore, one should appreciate that while the FEV_1 / FVC ratio characteristically is preserved in relationship to restrictive lung disorders, the presence of a normal ratio does not exclude the presence of obstructive lung disease. The reason for this is that as is illustrated in Figure 1, air trapping (the presence of which is based on an increased residual volume or RV, which increases the RV/TLC ratio) forces a reduction of the FVC. This reduction of the FVC in the setting of obstruction normalizes the FEV_1 / FVC ratio, resulting in a pattern of "pseudo-restriction". In this setting, obstruction is really present and its related air trapping has normalized the ratio. Furthermore, restriction is not really present despite the reduced FVC. The FVC is reduced because of the air trapping, and if the total lung capacity (TLC) was measured, under such circumstances, it would be either normal or increased. It should be remembered, as defined by the American Thoracic Society (1991), the presence of restriction or small lung size is defined by having a reduced TLC measurement. The relationships between FEV_1 , FVC and the FEV_1 / FVC ratio or $FEV_1\%$, in the settings of smoking-related obstruction, restriction and legal CWP or "pseudo-restriction", are illustrated in Figure 2.

Thus, the above information indicates there is no basis for finding that COPD is only defined by decrements in FEV_1 and $FEV_1\%$. The Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) defines COPD broadly as a reduction in the FEV_1 / FVC ratio (Rabe), but this definition does not purport to comprehensively define the problem. The definition applies to the population generally, but this is heavily influenced by the fact that cigarette smoking makes up the large portion of COPD, which typically is characterized by a decreased $FEV_1\%$. In fact, recent literature (including literature published after D.O.L.'s revisions to the black lung regulations) establishes the limitation of defining COPD as simply a reduction in FEV_1 or $FEV_1\%$ values (Fabbri).

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Thus, while I agree with the D.O.L. that COPD may be detected by a decrease in the FEV₁ and FEV₁ / FVC ratio, this does not generally apply to patients with legal CWP. Among the latter, based on epidemiologic studies accepted by the D.O.L., the obstruction is characterized by a preservation of the FEV₁ / FVC ratio. In fact, what has been outlined is that the preservation of the FEV₁ / FVC ratio is the "norm" in patients with coal mine induced obstructive lung disease. The opposite is true with respect to smoking-related COPD where the ratio is decreased. Thus, patterns of airflow obstruction help determine the etiology of a given miner's airway obstruction.

Furthermore, while both coal dust and cigarette smoke can cause emphysema based on similar pathogenic mechanisms, the differences exist between the types of emphysema these agents induce. This is logical, since the characteristics of the inhaled agents are disparate. In order to appreciate this fact, as outlined by the Surgeon General (1989), over 4,000 different components are contained within cigarette smoke with an estimated 10¹⁰ particles/ml. In addition, it is estimated that 10¹⁵ free radicals exist in the gas phase of each puff of tobacco smoke with 10¹⁸ free radicals being present per each gram of tar (Tuder). Furthermore, the various particles contained within cigarette smoke are dispersed in a vapor phase, with the particles being a median diameter of between 0.18 to 0.34 microns (Bernstein). Also, the Surgeon General (1984) outlined that 30 to 40% of these predominantly submicron particles end up reaching alveolar structures. The contrasting differences between tobacco smoke and coal mine dust exposures are first illustrated by the characterization of particle size distribution within coal mine dust. In regards to this, Seixas determined that there was a bimodal distribution of particle diameter size within coal dust, centering around 17 microns and 5 microns. Additionally, in the Burkart investigation, the distribution of the particle diameters was assessed in relationship to different mining operations. Overall, it was found that the particle sizes were comparable to that observed by Seixas, with only an extremely small fraction being below 1 micron in diameter. Additionally, coal dust is composed of a heterogeneous inorganic (nonliving) materials composed largely of carbon, hydrogen oxygen and nitrogen with rank being defined by the carbon content in relationship to other components (Parkes). These other components include various minerals, the amount of which decreases as the rank increases. The most common minerals include clays, quartz, pyrite and calcite, with trace metals also being present. In contrast, cigarette smoke is a combustion product of tobacco (an organic or living substance), which contains, as noted, 1000's of different components in both solid and gaseous phases, coupled with an abundance of free radicals. It should be noted the gases include toluene, benzene and phenol.

Based on the above information, the character of the components contained within coal dust and cigarette smoke are vastly different. As such, it follows that the disruption and alteration of tissue structures by these two agents within the lungs would be quite disparate. The larger coal dust particles would not have the same distribution pattern within the lungs as the submicron particles contained within cigarette smoke. Also, the abundance of free radicals contained within cigarette smoke (10¹⁵ / puff), along with the abundance of associated submicron particles, readily explains why cigarette smoke penetrates more deeply into alveolar structures than coal dust. This deeper penetration causes an inflammatory response in a different location than coal dust, resulting in a diffuse pattern of emphysema formation, in contrast to a more localized form of emphysema induced by coal mine dust. Furthermore, the diffuse emphysematous process that characterizes that related to cigarette smoking is supported by an associated diffusing capacity (DLCO) reduction, which develops as the emphysema becomes more advanced. The presence of a DLCO

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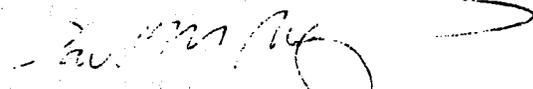
reduction indicates there has been a diffuse destruction of the alveolar capillary bed. A decreased DLCO is characteristically seen in relationship to emphysema caused by cigarette smoking (Berend, Baldi, Klein). In contrast, with coal mine dust exposure, generally the diffusing capacity is preserved (Nemery; Morgan, 1972). This supports the fact that a diffuse emphysematous destruction of lung tissue is not characteristic of coal mine dust exposure. Furthermore, the diffuse emphysematous process related to cigarette smoking often is associated with large lung volumes (increased total lung capacity), coupled with air trapping (increased RV/TLC).

Finally, it should be appreciated that mineral dust causes airway scarring which encroaches on luminal diameter (Churg and Wright). As such, this fibrosis within the airways would not be expected to allow airflow improvement in association with the administration of broncho-dilators.

Specific to Mr. Sterling, one could appreciate that he has a marked reduction of his FEV₁ coupled with a severe reduction of his FEV₁ / FVC ratio. This pattern of obstruction is uncharacteristic of the pattern of obstruction observed in relationship to past coal mine dust exposure. Rather, it is classic for a smoking-related form of COPD, as is the appearance of his flow-volume curve. Furthermore, his physical examination findings (decreased breath sounds with marked hyperresonance) and X-ray appearance is indicative of a diffuse emphysematous pattern. This is also consistent with his previously measured reduced diffusing capacity measurement. The latter indicates diffuse destruction of the alveolar capillary bed related to emphysema. This is also consistent with his marked oxygenation abnormality. Mr. Sterling's emphysematous pattern is classic for a smoking-related form of COPD and not that developing in relationship to past coal mine dust exposure. Also, smoking related COPD has caused his right ventricular dysfunction. Finally, it should be emphasized that hypoventilation (increased PCO₂) is characteristic of smoking related COPD and not airflow obstruction developing in relationship to past coal mine dust exposure. When all of the above information is looked at in total, Mr. Sterling's severe COPD does not represent the presence of legal CWP.

In **CONCLUSION**, it can be stated with a reasonable degree of medical certainty that Mr. Sterling does not have clinical or legal CWP. While he is disabled from a pulmonary perspective, this relates to the presence of smoking-related COPD and not a coal mine related disorder. If you have any questions, please feel free to contact me.

Sincerely,



David M. Rosenberg, M.D., M.P.H.
Medical Director Corporate Health
Occupational Health Services at Chagrin Highlands
University Hospitals

DMR/mmw

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