

No. 17-1982

**UNITED STATES COURT OF APPEALS
FOR THE FOURTH CIRCUIT**

CEDAR COAL COMPANY,

Petitioner

v.

PEARL M. BARTON, survivor of CLARENCE E. BARTON

and

**DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
UNITED STATES DEPARTMENT OF LABOR,**

Respondents

**On Petition for Review of an Order of the Benefits Review Board,
United States Department of Labor**

BRIEF FOR THE FEDERAL RESPONDENT

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BRIEF FOR THE FEDERAL RESPONDENT

**STATEMENT OF APPELLATE AND SUBJECT
MATTER JURISDICTION**

This case involves a 2013 claim for survivor benefits under the Black Lung Benefits Act (BLBA or the Act), 30 U.S.C. §§ 901-944, filed by Pearl M. Barton. Claimant is the widow of Clarence E. Barton, who worked as a coal miner for nineteen-plus years and died in 2008.

On June 14, 2016, United States Department of Labor (DOL) Administrative Law Judge Richard A. Morgan issued a decision awarding benefits and ordering

Cedar Coal Company (Cedar Coal or the coal company), the miner's former employer, to pay them. The coal company appealed the ALJ's decision to DOL's Benefits Review Board on July 8, 2016, within the thirty-day period prescribed by 33 U.S.C. § 921(a), as incorporated into the BLBA by 30 U.S.C. § 932(a). The Board had jurisdiction to review the decision pursuant to 33 U.S.C. § 921(b)(3), as incorporated by 30 U.S.C. § 932(a).

The Board affirmed the award on June 27, 2017, and Cedar Coal petitioned this Court for review on August 23, 2017. The Court has jurisdiction over this petition because 33 U.S.C. § 921(c), as incorporated by 30 U.S.C. § 932(a), allows an aggrieved party sixty days to seek review of a final Board decision in the court of appeals in which the injury occurred. Because the miner had exposure to coal-mine dust – the injury contemplated by 33 U.S.C. § 921(c) – in the state of West Virginia, within this Court's territorial jurisdiction, the Court has jurisdiction over the coal company's petition for review.

STATEMENT OF THE ISSUES

The issues in this case are:

1. Whether the ALJ used the correct standard to determine if Cedar Coal's medical evidence proved that the miner did not suffer from legal pneumoconiosis;
and

2. Whether substantial evidence supports the ALJ's conclusion that Cedar Coal failed to prove that the miner did not suffer from legal pneumoconiosis.

STATEMENT OF THE CASE

A. Legal Background

1. The Black Lung Benefits Act and the fifteen-year presumption

The BLBA provides disability compensation to survivors of former coal miners whose death was due at least in part to pneumoconiosis arising out of coal mine employment.¹ 30 U.S.C. §§ 901(a), 902(b); 20 C.F.R. § 718.1; *Milburn Colliery Co. v. Hicks*, 138 F.3d 524 (4th Cir. 1998). The Act contains several presumptions designed to aid survivors in establishing the causal connection between the miner's death and his pneumoconiosis. *See generally Usery v. Turner Elkhorn Mining Co.*, 428 U.S. 1 (1976). One such presumption, found at 30 U.S.C. § 921(c)(4), is known as the "fifteen-year presumption." It is invoked if the miner worked at least fifteen years in qualifying coal mine employment and had a totally disabling respiratory or pulmonary condition.² 30 U.S.C. § 921(c)(4); *Hobet Min., LLC v. Epling*, 783 F.3d 498, 501 (4th Cir. 2015).

¹ *Pneumoconiosis* is commonly referred to as "black lung disease."

² Here, there is no dispute that the ALJ properly invoked this presumption; the issue on appeal is rebuttal.

Once the fifteen-year presumption is invoked, the survivor is rebuttably presumed entitled to benefits, i.e., is presumed to have proved that the miner's death was due at least in part to pneumoconiosis. To defeat entitlement, the liable coal mine operator must satisfy one of two alternate methods of rebuttal: 1) that the miner had neither clinical pneumoconiosis arising out of coal mine employment nor legal pneumoconiosis," 20 C.F.R. § 718.305(d)(2)(i); or failing that, 2) that "no part of the miner's death was caused by [his] pneumoconiosis" ³ 20 C.F.R. § 718.305(d)(2)(ii); *Mingo Logan Coal Co. v. Owens*, 724 F.3d 550, 554 (4th Cir. 2013).

Because the two rebuttal methods refer to "pneumoconiosis," it is critical to understand what is meant by that term under the Act. Compensable pneumoconiosis under the BLBA includes two distinct forms, "clinical" and "legal." 20 C.F.R. § 718.201(a); see *Westmoreland Coal Co. v. Stallard*, No. 16-1416, --- F.3d ---- 2017 WL 5769516, at *2 (4th Cir. Nov. 29, 2017); *Harman Min. Co. v. Dir., Off. of Workers' Comp. Programs*, 678 F.3d 305, 308 (4th Cir. 2012). *Clinical (or "medical") pneumoconiosis* refers to a collection 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."

³ The second method is frequently called the "rule-out standard."

20 C.F.R. § 718.201(a)(1). It includes the disease medical professionals refer to as “coal workers’ pneumoconiosis” or “CWP,” and is typically diagnosed by chest x-ray, biopsy, or autopsy, 20 C.F.R. §§ 718.102, 718.106, 718.202(a)(1)-(2).

In contrast, *legal pneumoconiosis* is a broader category, including “*any chronic lung disease or impairment . . . arising out of coal mine employment.*” 20 C.F.R. § 718.201(a)(2) (emphasis added). A chronic lung disease or impairment (whether obstructive or restrictive) that is “significantly related to, or substantially aggravated by” exposure to coal-mine dust, is considered to have “arise[n] out of coal mine employment,” and is therefore considered to be legal pneumoconiosis. 20 C.F.R. § 718.201(b).

Turning back to the fifteen-year presumption – For a liable operator to rebut the fifteen-year presumption by the first method, the company must disprove both clinical and legal pneumoconiosis. Notably, the fact that the miner does not suffer from *clinical* pneumoconiosis – for example, does not have a positive x-ray – does not preclude a finding of *legal* pneumoconiosis. 20 C.F.R. § 718.202(b) (“A claim for benefits must not be denied solely on the basis of a negative chest X-ray.”); 20 C.F.R. § 718.202(a)(4) (“A physician may diagnose pneumoconiosis “notwithstanding a negative X-ray.”); *see also Westmoreland Coal Co, Inc. v. Dir., Off. of Workers’ Comp. Programs [Cochran]*, 718 F.3d 319, 324 (4th Cir. 2013) (“Both the preamble [to the regulatory definition of legal pneumoconiosis] and

regulations make clear that the absence of clinical pneumoconiosis cannot be used to rule out legal pneumoconiosis.”).

And if the responsible operator fails to rebut the presumption by the first method, i.e., fails to disprove both clinical and legal pneumoconiosis, the operator can rebut by the second method by proving that the miner’s death is completely unrelated to his pneumoconiosis. 20 C.F.R. § 718.305(d)(2)(ii).

2. The preamble to the regulatory definition of legal pneumoconiosis

The preamble to the 2000 BLBA regulations “sets forth the medical and scientific premises relied on by the Department in coming to . . . conclusions in its regulations.” *Harman Min. Co.*, 678 F.3d at 314. An ALJ may rely on the preamble in evaluating medical opinions. *See, e.g., Cochran*, 718 F.3d at 323 (discussing use of the preamble); *see also Stallard*, 2017 WL 5769516, at *6 (quoting *Cent. Ohio Coal Co. v. Dir., Off. of Workers’ Comp. Programs*, 762 F.3d 483, 491 (6th Cir. 2014) (“The sole issue presented here is whether the ALJ was entitled to discredit Dr. Rosenberg’s medical opinion because it was inconsistent with the [Labor Department] position set forth in the preamble, and the answer to that question is unequivocally yes.”)).

The preamble makes clear a number of points relevant to the issues and medical evidence now before the Court. Notably, the preamble to the regulatory definition of pneumoconiosis at 20 C.F.R. § 718.201 states that the medical

literature has shown that “coal miners have increased risk of developing [chronic obstructive pulmonary disease],” 65 Fed. Reg. at 79943 (Dec. 20, 2000); “dust-induced emphysema and smoked-induced emphysema occur through similar mechanisms,” 65 Fed. Reg. at 79943; “[s]mokers who mine have additive risk for developing significant obstruction,” *id.*; and “[e]ven in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis,” 65 Fed. Reg. 79940. Further, in observing that different types of emphysema are due to coal mine employment, the preamble identified studies suggesting that whether a miner’s emphysema is due to coal mine employment may be related to the amount of dust in the lungs, as weighed pathologically. 65 Fed. Reg. at 79941-42.

Many observations and conclusions in the preamble come from the *Criteria for a Recommended Standard, Occupational Exposure to Respirable Coal Mine Dust (Criteria)*, <http://www/cdc.gov/niosh/docs/95-106/>, prepared by the National Institute for Occupational Safety and Health (NIOSH). Congress enlisted NIOSH to be the statutory scientific advisor to the black lung program. 30 U.S.C. § 902(f)(1)(D). And in revising the regulatory definition, the Department sought and received guidance from NIOSH, which supported the scientific analysis contained in the preamble. 65 Fed. Reg. 79937.

B. Procedural Facts

Claimant filed her claim for BLBA survivor's benefits in 2013. Joint Appendix (JA) 100. After reviewing evidence developed by Claimant and Cedar Coal, the district director of DOL's Office of Workers' Compensation Programs concluded that Claimant was entitled to benefits. JA 724. Dissatisfied with this decision, the coal company requested an administrative hearing, which was held before Administrative Law Judge Richard A. Morgan in February 2016. *Id.*

The ALJ issued a decision on June 14, 2016, awarding Claimant benefits. JA 723. First, he found no dispute that the fifteen-year presumption of entitlement was invoked: the miner had at least fifteen years of qualifying coal mine employment and he suffered from a totally disabling respiratory condition. JA 741-42. And second, the ALJ found the presumption un rebutted because, while Cedar Coal's medical evidence proved the absence of clinical pneumoconiosis, it did not disprove legal pneumoconiosis. JA 743-52. The failure to disprove legal pneumoconiosis precluded rebuttal not only by the first method – where disproving legal pneumoconiosis was a required element – it also precluded rebuttal by the second method because the coal company doctors' diagnoses of no causation between the miner's death and his pneumoconiosis were undermined by the fact that the doctors mistakenly assumed that the miner did not suffer from legal pneumoconiosis. JA 750-52.

Cedar Coal appealed this award to the Benefits Review Board, which affirmed the ALJ's decision on June 27, 2017. JA 756. The coal company's petition to this Court followed on August 23, 2017.⁴

C. Relevant Facts

1. Undisputed general facts

Claimant's husband was employed in coal mine work for almost twenty years, ending in 1984. JA 727. His work involved heavy manual labor. *Id.* The miner smoked 1-1/2 packs of cigarettes a day for approximately thirty-five years, ending in 1980. JA 728. He died in 2008 at the age of seventy-eight. JA 727. Prior to his death, the miner suffered from chronic obstructive disease (COPD) in the form of emphysema.⁵ JA 748. His COPD was totally disabling and resulted in his death. *Id.* The x-ray and ct-scan evidence did not prove the existence of clinical pneumoconiosis. JA 728-29, 747.

⁴ The decisions of the ALJ and Board are set forth in more detail following the statement of the relevant facts.

⁵ *COPD* "is persistent narrowing (obstruction) of the airways occurring with emphysema, chronic obstructive bronchitis, or both disorders." *The Merck Manual Consumer Version*, <http://www.merckmanuals.com/home/SearchResults?query=copd+chronic+obstr+uctive+pulmonary>. COPD is characterized by three disease processes: chronic bronchitis, emphysema, and asthma. *Emphysema* is "[t]he enlargement and partial amalgamation of the air sacs of the lungs, resulting in breathlessness and wheezing." *The New Shorter Oxford English Dictionary* (4th ed. 1993).

2. Results of pulmonary function study tests⁶

Date/ Page	<u>FEV₁</u>	<u>FVC</u>	<u>MVV</u>	<u>FEV₁ percent improve- ment w/bronchodilator</u>
10/28/85	1.45	3.26	73	
JA 251	1.40*	3.22*	79*	3%
1/8/04	.70	2.05	18	
JA 142	.82*	2.64*	28*	17%

*These figures were obtained after administration of a bronchodilator.⁷

The miner's treating physician, Dr. Lo' Ay Al-Asadi, reported that the 2004 study showed "minimal response to bronchodilators." JA 158.

3. Relevant medical opinions

It is undisputed that smoking contributed to the miner's COPD. JA 748.

The question is whether Cedar Coal has shown – as is its burden on rebuttal – that coal mine employment did not also significantly contribute to his COPD, thereby

⁶ *Pulmonary function tests*, also called spirometry, "measure the degree to which breathing is obstructed." See *Yauk v. Dir., Off. of Workers' Comp. Programs*, 912 F.2d 192, 196 n.2 (8th Cir. 1989). Tests resulting in certain values established in the regulations are evidence of total disability in BLBA claims. See 20 C.F.R. § 718.204(b)(2)(i); 20 C.F.R. Part 718 Appendix B.

While there is no dispute that the miner's respiratory condition is totally disabling, the results of the pulmonary function tests are set forth here because a number of doctors referenced the results in their conclusions concerning the cause of the miner's COPD.

⁷ A *bronchodilator* is a drug used to treat COPD. *The Merck Manual* 1894 (19th ed. 2011). It expands the "air passages of the lung." *Dorland's Illustrated Medical Dictionary* 253 (32nd ed. 2012).

disproving the presence of legal pneumoconiosis. Four doctors provided opinions on this question: Drs. Zaldivar and Sporn for the coal company, and Drs. Rasmussen and Forehand for Claimant. These doctors did not examine the miner but instead based their opinions on medical records developed prior to the miner's death. Only Drs. Zaldivar and Rasmussen are Board-certified internists and pulmonologists. JA 731, 733. Dr. Sporn is a Board-certified internist and pathologist, JA 731, and Dr. Forehand is Board-certified in pediatric medicine, allergy and immunology, JA 732. Their opinions are set forth below in chronological order.

Rasmussen #1, JA 244. At Claimant's request, Dr. Donald L. Rasmussen reviewed the medical reports and completed a February 2010 report addressing the cause of the miner's fatal COPD. JA 244. He explained that coal mine dust, like smoking, can independently cause COPD, and when combined they have an "additive" effect. JA 246. According to the doctor, coal mine dust and smoking both "destroy lung tissue and leave emphysema," with coal-related emphysema being indistinguishable from smoking-related emphysema: "[C]oal mine dust is capable of causing pulmonary emphysema indistinguishable from that caused by cigarette smoking." *Id.* In view of this, Dr. Rasmussen concluded that the miner's "20 years of coal mine employment [was] sufficient to be a major contributing cause" of the miner's COPD.

Zaldivar #1, JA 303. At Cedar Coal's s request, Dr. George L. Zaldivar in June 2014 reviewed the medical records, including Dr. Rasmussen's 2010 report. JA 303. He concluded that the miner's significant smoking history was itself enough to cause respiratory disability and death, regardless of the work the miner performed. JA 312. He then explained that the miner suffered from bronchospasm⁸ in the form of asthma, which developed in the miner's childhood but was undiagnosed until adulthood. JA 312-13. In support of his diagnosis of bronchospasm, Dr. Zaldivar directed attention to the pulmonary function study taken in January 2004, *see supra* at 10, which he stated showed improved results after administration of a bronchodilator.⁹ JA 311.

Based upon this diagnosis of bronchospasm/asthma, Dr. Zaldivar concluded that the miner's asthma combined with his COPD to produce "overlap syndrome." JA 312. That condition, according to Dr. Zaldivar, "has been described in COPD of smokers, but not COPD due to coal workers' pneumoconiosis." JA 312. He

⁸ *Bronchospasm* is the "spasmodic contraction of the smooth muscle of the bronchi as occurs in asthma." *Dorland's Illustrated Medical Dictionary* 254 (32nd ed. 2012). In contrast, chronic bronchitis is "a type of [COPD] in which there is bronchial irritation with increased secretions and a productive cough for at least three months, two years in succession; it is usually accompanied by pulmonary emphysema. The most common cause is long-term inhalation of irritants." *Dorland's* at 252.

⁹ Dr. Zaldivar's report does not indicate that he reviewed the 1985 pulmonary function study results that showed only three percent improvement after administration of a bronchodilator. *See supra* at 10.

explained that, without early treatment – which the miner did not have – and “continuation of smoking, the individual is bound to develop severe irreversible obstruction.” JA 313. He described the symptoms of this condition as wheezing and frequent hospitalizations due to frequent exacerbations.¹⁰ JA 312.

The doctor then addressed Dr. Rasmussen’s belief that smoking and coal mine dust use the same mechanisms to produce emphysema.¹¹ JA 313. Dr. Zaldivar admitted that “[t]he common pathway ultimately leading to the destruction of lung is similar for both coal mining or smoking or any other entities, because the final pathway to destruction will have to activate the same enzymes and the same oxygen radicals, etc.” JA 313, 315. But, he explained, the “seminal damage to the lung produced by smoking” has now been described in a 2014 study, whereas “coal workers’ pneumoconiosis has not been described as having the same mechanism of injury.” JA 313. He explained that this 2014 study was significant because it mapped out the changes in cell structure and DNA that resulted from smoking. JA 313-15. Because of this, the doctor stated that it was no longer true

¹⁰ In making this observation, the ALJ reviewed medical records revealing frequent hospitalizations in the 2000s, especially in 2003 to 2005. JA 304-06. These hospitalizations were more than twenty years after the miner ceased smoking in 1980.

¹¹ As explained *supra* at 7, the preamble to the regulatory definition of pneumoconiosis explained that “dust-related emphysema and smoked-induced emphysema occur through similar mechanisms. . . .”

that smoking and coal mine employment led to emphysema by the same mechanisms. JA 313.

Dr. Zaldivar concluded that the miner did not suffer from legal pneumoconiosis, asserting “there was absolutely no contribution by his coal mine work” to the miner’s respiratory disability and resultant death. JA 315-16. Dr. Zaldivar also concluded that the miner did not suffer from clinical pneumoconiosis because the miner’s x-rays and ct-scan result were negative for that condition. JA 312, 315.

Rasmussen #2, JA 225. After reading additional medical reports, including Dr. Zaldivar’s June 2014 opinion, Dr. Rasmussen completed a second report in April 2015. JA 225. He stated that the miner’s COPD, which was “progressively severe,” had two risk factors: smoking and coal mine dust exposure, and that both contributed to the miner’s COPD. JA 226. He also observed that most of the miner’s twenty years of coal mine employment (1964-84) occurred prior to implementation of dust suppression.¹² And he again stated that the miner’s coal mine dust exposure “was a significant contributing cause” of the miner’s COPD and resultant death. *Id.*

Dr. Rasmussen then addressed Dr. Zaldivar’s bases for finding that coal mine dust exposure did not cause or affect the miner’s respiratory condition. JA

¹² The federal guidelines for dust limits were instituted in 1971.

227. Concerning the new research showing that smoking causes certain changes in cell structure and DNA, Dr. Rasmussen observed that “[Dr. Zaldivar] ha[d] no basis in stating that coal mine dust does not cause the same abnormalities since it has not been studied in miners.” JA 228. Consequently, the doctor reasoned, there was no basis for Dr. Zaldivar’s conclusion that the new study made it easier to distinguish smoking from coal mine dust exposure. Dr. Rasmussen therefore maintained his position that it was not possible to distinguish between COPD related to smoking and that condition related to coal mine dust exposure.

Dr. Rasmussen’s response to Dr. Zaldivar’s diagnosis of “overlap syndrome” was in the same vein. He posited that even if the miner suffered from that condition, it did not prevent coal mine employment from contributing: “Certainly one cannot exclude overlap syndrome in this case, however, it does not exclude coal mine dust exposure as a significant co-contributor.” JA 228. Dr. Rasmussen also observed that the doctor’s “overlap” analysis was based in part on the doctor’s assumption that the miner’s pulmonary function study results showed partial “reversibility” (i.e., that the results improved upon administration of a bronchodilator). JA 228. Dr. Rasmussen, however, questioned this assumption because there was no reversibility in studies he reviewed that were performed from 1979 to 1987. *Id.*

Dr. Rasmussen reported it was “clearly recognized” that a miner may have COPD even without proof by x-ray, JA 227; that “x-ray [was] an imperfect tool for determining the presence or absence of pneumoconiosis”; that ct-scans had not been tested to show their accuracy when compared with pathological results, JA 229; that twenty years of coal mine employment, some before rules limiting dust, was enough to develop COPD, *id.*; and the fact that the miner continued to smoke after he left the mines did not argue against causation since the damage caused by coal mine dust may be progressive, *id.* Finally, Dr. Rasmussen questioned the theory of another doctor, whose report was apparently submitted in connection with Claimant’s state compensation claim. JA 227. The doctor theorized that, if a miner has improvement/reversibility in pulmonary function study results after administration of a bronchodilator, then the respiratory condition is not due to coal mine employment. Dr. Rasmussen rejected the doctor’s theory, explaining that coal mine employment can lead to conditions – like silicosis, emphysema, and chronic bronchitis – other than clinical pneumoconiosis. According to the doctor, those conditions, like smoking, can “show partial reversibility with bronchodilator medications.” *Id.*

Sporn, JA 326. At the coal company’s request, Dr. Thomas A. Sporn reviewed the medical records and at the outset observed that there was no positive evidence of clinical pneumoconiosis. JA 326. He stated “[i]t [was] possible that

Mr. Barton's employment resulted in the inhalation of coal dust which became deposited in his lungs," but concluded that it was only "to a minimal extent," and that it probably would show up only upon "microscopic/pathologic examination of lung tissue," which did not occur in this case. JA 327. Dr. Sporn concluded that smoking was the sole cause of the miner's COPD because the miner's exposure to smoke was greater than his exposure to coal mine dust: "To within a reasonable degree of medical certainty, and as determined or measured clinically and radiographically, the dosage of cigarette smoke sustained by Mr. Barton exceeded the lung burden of coal mine dust and therefore was the sole cause of his impairment and death from [COPD]." *Id.*

Forehand, JA 233. At the Claimant's request, Dr. J. Randolph Forehand in January 2016 reviewed the medical records, including Dr. Rasmussen's 2010 opinion and Dr. Zaldivar's 2014 opinion. JA 233. He set forth some general principles: "[a] 7% response to bronchodilator is neither a significant response nor an asthmatic response," JA 236; determining the cause of a miner's COPD is not "a choice between pneumoconiosis and emphysema" because both cause COPD, JA 239; "[f]requent exacerbations of shortness of breath with wheezing is a characteristic of [COPD] of which asthma is only one component – emphysema and chronic bronchitis are also associated with frequent exacerbations with wheezing," JA 240-41 (footnote omitted); many studies show that a negative x-ray

is not borne out by what is actually seen on autopsy, JA 241-42; and a coal-dust related disease cannot be ruled out based upon a negative x-ray, JA 242.

Turning directly to the miner's situation, Dr. Forehand stated that, "[b]ecause Mr. Barton had severe, end stage lung disease, no criteria exist[ed] for differentiating the effects of cigarette smoke from the effects of inhaling coal mine dust," JA 242; and the miner's longer smoking history "obscured the effects of coal mine dust on Mr. Barton's lungs, but that fact [did] not eliminate or prevent the effects of coal mine dust endured by Mr. Barton – coal mine dust-related lung diseases are latent and progressive and continue to get worse over time, even in the absence of further coal mine dust exposure," JA 241.

Dr. Forehand disagreed with Dr. Zaldivar that coal mine employment played no role in the miner's condition. JA 239. He expressed doubt that the doctor "took into serious consideration the nature of [the miner's] coal mine dust exposure or the effects on Mr. Barton's lungs of the prolonged exposure of coal mine dust." *Id.* He also criticized the doctor for not seriously considering the combined effects of cigarette smoke and coal mine dust on the lungs," or "that the contribution made by coal mine dust to Mr. Barton's lung disease was not insignificant." *Id.* To Dr. Forehand, it was not "a choice between pneumoconiosis and emphysema"; he explained that both smoking and coal mine employment were "both scientific proven causes of Mr. Barton's [COPD]." *Id.*

Dr. Forehand also observed that, while Dr. Zaldivar stated that bronchodilators helped the miner's asthma, he ignored their other uses, as with occupational exposures:

No space was devoted to discussing the facts [sic] that other forms of obstructive lung disease are treated with bronchodilators, that Mr. Barton's obstructive lung disease most likely arose from more than a single cause (asthma, cigarette smoking, occupational exposure) and that bronchodilators had no effect on more than 70% of Mr. Barton's airway obstruction – the portion of Mr. Barton's obstructive lung disease contributed to by coal mine dust exposure.

JA 240.

Dr. Forehand acknowledged that there was some dispute concerning the length of the miner's smoking history. JA 243. But given the miner's lengthy coal-mine dust exposure, the doctor concluded it could not be denied that the dust exposure was a significant contributor to the miner's COPD and death: "Whether [the miner] smoked 20 years or 80 years does not diminish the fact that Mr. Barton's coal mine employment for 20 years [] substantially contributed (on an ongoing basis) to his obstructive lung disease, which eventually caused his death."

Id.

Zaldivar, #2, JA 365. In February 2016, Dr. Zaldivar reviewed Dr. Rasmussen's 2014 report and Dr. Forehand's 2016 report. At the outset, Dr. Zaldivar reported again that smoking was the cause of the miner's asthma and COPD. JA 365. And he explained that the miner's asthma could not be due to

coal mine employment because neither coal nor silica is allergenic. He acknowledged there was a condition called occupational asthma, but explained that condition occurred when “chemicals are used in the process of creating a product,” and ended when the exposure ceased. JA 365-66. He stated that the asthma-overlap syndrome has recently received more notice in in the scientific community, and stressed that the condition was serious, where “patients with features or diagnos[e]s of both asthma and COPD have worse clinical outcomes than patients with either condition alone.” JA 367.

Turning to Dr. Rasmussen’s recent opinion, Dr. Zaldivar suggested that that doctor did not read certain studies accurately. JA 367-69. He then responded to Dr. Rasmussen’s statement that the “similar mechanisms” understanding still held true. Dr. Zaldivar admitted, as Dr. Rasmussen had pointed out, that there had been no study done with coal mine employment as there had been with smoking. But Dr. Zaldivar then used that fact to criticize the general understanding [as set forth in the preamble] that coal mine employment and smoking occur through similar mechanisms. He asserted “it [was] not logical to state that two diseases, that look the same, must have occurred through . . . the same mechanism of damage without doing empirical studies.” JA 369. Dr. Zaldivar also questioned Dr. Rasmussen’s conclusion that coal mine employment may cause respiratory impairment even without a positive x-ray, pointing out that one of the studies the doctor relied upon

involved Sicilian miners who – presumably unlike the miner here – were exposed to dust with a high silicosis content. JA 370.

Dr. Zaldivar also explained that, according to a study reported in NIOSH's *Criteria* at section 4.1.2.3, there was a correlation between the amount of dust seen on x-ray and a miner's impairment. JA 371. While admitting that there may be dust that the x-ray cannot see, the doctor suggested it could be seen by ct-scan since that type of testing, according to the doctor, is better than x-rays. Ultimately, Dr. Zaldivar admitted the superiority of pathology: "Of course, the pathologist, under the microscope, may still find micronodules of pneumoconiosis where the CT-scan has not shown it." *Id.*

Finally, Dr. Zaldivar reiterated that there was "a clear diagnosis of a smoker's asthma-COPD overlap syndrome with a very extensive history of smoking, which [] in itself is sufficient to cause such problem [as the miner had], and [there was] no evidence of radiographic pneumoconiosis by any means." JA 371. As a last point, Dr. Zaldivar cited a *Criteria* statement that nonsmoking miners with forty years of coal mine employment were only 1.6 to 6.3% likely to suffer from COPD, whereas smoking miners with the same work history were 8 to 17.3% more like to suffer that condition. JA 373.

Zaldivar, #3, JA 538. Dr. Zaldivar was deposed in March 2016. JA 538. His deposition, over 180 pages in length, reiterated his belief that smoking and

asthma, which combined to form asthma-overlap syndrome, were the sole causes of the miner's condition. JA 544. He also stated that the miner had chronic bronchitis which was not due to coal mine employment. JA 551. When asked why there was no connection with the miner's employment, he indicated it was because the miner smoked and had asthma, as well as chronic allergic rhinitis and gastroesophageal reflex. JA 552. With those conditions, which he identified as "powerful inducers," he suggested there was no reason to look further:

[I]t's very hard to implicate the working, the working environment as the cause of your bronchitis, because all those other four factors are so much more powerful inducers. So it is a diagnosis of exclusion. . . . He was a smoker and he had asthma, so yes, he had plenty of reasons to develop chronic bronchitis. So that is the explanation as to why this man doesn't have bronchitis due to his work.

JA 552.

D. Decisions Below

1. ALJ decision awarding benefits (JA 723)

At the outset, the ALJ found it undisputed that the miner worked in coal mine employment for 19-1/2 years, JA 726; that this employment involved underground coal mining or surface coal mining in conditions substantially similar to underground coal mining, JA 727; that the miner had a totally disabled respiratory impairment in the form of COPD prior to his death, JA 748; and that the miner's respiratory impairment caused his death, *id.* He also found that the

miner had a smoking history of 52.5 pack years (thirty-five years of smoking 1-1/2 packs of cigarettes a day, ending in 1980). JA 728.

Based upon the miner's coal mine employment history and the fact that his respiratory condition was totally disabling, the ALJ concluded the fifteen-presumption of entitlement was invoked. JA 742. The ALJ next considered whether this presumption was rebutted, i.e., whether Cedar Coal's evidence proved that 1) the miner suffered from neither clinical pneumoconiosis nor legal pneumoconiosis; or 2) the miner's pneumoconiosis did not contribute in any part to the miner's death. *Id.*

The ALJ concluded that Cedar Coal rebutted the presumption of clinical pneumoconiosis because, while there were two x-rays positive for that condition, the x-ray readings and CT-scan report found in the treatment records did not diagnose clinical pneumoconiosis, nor did the medical opinion evidence. JA 745-48. The ALJ then considered whether the company's evidence rebutted the presumption that the miner suffered from legal pneumoconiosis: "The only issue is whether the miner's disabling COPD is significantly related to his coal mine dust exposure." JA 748.

The ALJ first addressed the reasons Drs. Rasmussen and Forehand provided for diagnosing the requisite contribution: the miner's COPD involved emphysema, and coal mine dust exposure can cause emphysema; smoking and coal mine

employment independently cause damage but the resulting damage— the destruction of lung tissue and the creation of emphysema – is the same; it was impossible to separate the effect of smoking from that of coal mine dust exposure; the effects of smoking and coal mine employment are additive; and even if the miner did suffer from asthma-COPD overlap syndrome, as Dr. Zaldivar espoused, that did not prevent coal mine employment from contributing to the miner’s respiratory condition. *Id.* The ALJ concluded that the opinions of the two doctors were consistent with the preamble and the scientific studies supporting the preamble.¹³ *Id.*

Next, the ALJ considered the contrary opinions of Drs. Zaldivar and Sporn. JA 748. The ALJ was not persuaded by Dr. Zaldivar’s overlap diagnosis because it ignored the underlying question of whether coal mine employment contributed to the miner’s COPD: “Even assuming Dr. Zaldivar is correct in diagnosing both asthma and cigarette induced COPD, and the negative implications of the overlap of these two conditions, he never adequately provides a documented or well-reasoned basis for excluding the contribution of the miner’s nearly 20 years of coal mine dust exposure to his disabling lung disease.” JA 748.

The ALJ was also not persuaded by Dr. Zaldivar’s reliance on the negative x-ray and ct-scan evidence to preclude contribution to the miner’s COPD by the

¹³ The ALJ referred to the preamble to the regulatory definition of pneumoconiosis at 65 Fed. Reg. 79937-45.

miner's coal mine work. JA 748-49. The ALJ observed that the regulation at 20 C.F.R. § 718.202, which explained how pneumoconiosis could be established, specifically provided that legal pneumoconiosis could be found “notwithstanding a negative X-ray. . . .” 20 C.F.R. § 718.202(a)(4). JA 748. The ALJ also cited *Cumberland River Coal Co. v. Banks*, 690 F.3d 477 (6th Cir. 2012), where the Sixth Circuit affirmed an ALJ's discrediting a doctor's opinion finding no connection between the miner's respiratory impairment and coal mine employment based upon negative x-ray evidence:

[T]he Department of Labor and the Board have made clear that a miner can be found to have legal pneumoconiosis, even in the absence of clinical pneumoconiosis. . . . Further, the Board has found it proper to discredit a physician's opinion based on the notion that emphysema caused by coal dust does not occur absent clinical pneumoconiosis.

690 F.3d at 487.

And the ALJ questioned Dr. Zaldivar's use of x-rays and ct-scans to determine the amount of dust in the lungs. JA 748-49. According to the doctor, there must be dust in the lungs for coal mine employment to cause COPD; and if the x-ray or ct-scan is negative, that means there is no dust in the lungs. JA 748. While the ALJ did not reject the dust/damage theory, he questioned its application here because no autopsy or biopsy had been done to measure the amount of dust.¹⁴

¹⁴ As explained *supra* at 7, the preamble at 65 Fed. Reg. 79941-42 reported studies showing a correlation between emphysema and the amount of dust in the lungs as measured pathologically.

JA 749. The ALJ rejected the doctor's suggestion that dust seen by x-ray was the equivalent of dust measured by autopsy or biopsy: "Dr. Zaldivar's opinion in this case does not rely on the amount of dust seen on pathology as there is no available autopsy or biopsy evidence in this case. Instead he *assumes* that the dust burden is minimal because of the absence of radiographic pneumoconiosis." *Id.* (citations omitted).

Next, the ALJ considered Dr. Sporn's opinion. JA 749. As he did with Dr. Zaldivar's opinion, the ALJ discredited the doctor's opinion because it failed to explain why coal mine employment did not at least contribute to the miner's condition: "Although none of the medical experts excludes cigarette smoking as a major contributor to the miner's severe COPD, Dr. Sporn does not adequately explain how a significant smoking history excludes contribution from the miner's nearly 20 years of coal mine dust exposure." *Id.*

Concluding that "Dr. Zaldivar and Dr. Sporn [did] not adequately explain the basis for ruling out significant contribution from coal mine dust exposure to the miner's severe COPD," the ALJ found their opinions both unreasoned and undocumented. JA 750. In contrast, the ALJ found credible the opinions of Drs. Rasmussen and Forehand, who concluded that both smoking and coal mine employment were "significant co-contributors" to the miner's disability and death from COPD. *Id.*

Because rebuttal by the first method requires evidence disproving both clinical and legal pneumoconiosis, and Cedar Coal failed in this latter burden, the ALJ turned to the second method of rebuttal: proving that pneumoconiosis contributed in no part to the miner's death from COPD. JA 750. Quoting the Court's decision in *West Virginia CWP Fund v. Bender*, 782 F.3d 129 (4th Cir. 2015), the ALJ stated Cedar Coal was required to affirmatively prove "that the miner's disability is attributable exclusively to a cause or causes other than pneumoconiosis." JA 751. Citing *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109 (4th Cir. 1995), the ALJ found this burden not met because "both doctors failed to diagnose legal pneumoconiosis, contrary to [the ALJ's] finding on this issue." JA 752. Accordingly, the ALJ found the fifteen-year presumption unrebutted and awarded benefits to the miner's widow. *Id.*

2. Board decision affirming the ALJ's award (JA 756)

Cedar Coal's argument to the Board was three-fold: 1) the ALJ used the wrong standard in determining whether the coal company disproved legal pneumoconiosis; 2) Drs. Zaldivar and Sporn did, in fact, explain why coal mine employment did not significantly contribute to the miner's COPD; and 3) the ALJ mistakenly assumed the two doctors believed a miner needed a positive x-ray before legal pneumoconiosis would be diagnosed. JA 757, 760 n.4. The Board rejected the first two assertions as being without merit, JA 759-60, and declined to

address the third assertion because the Board had “affirm[ed] the administrative law judge’s decision to discredit employer’s physicians on other valid grounds,” JA 760, n.8.

Addressing the coal company’s first assertion, the Board turned to the definition of legal pneumoconiosis at 20 C.F.R. § 718.201(a)(2), (b). Those provisions define legal pneumoconiosis as any respiratory condition arising out of coal mine employment, with “‘arising out of coal mine employment’ includ[ing] any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.” Consequently, to disprove legal pneumoconiosis, the coal company had to prove that the miner’s COPD was “not significantly related to, or substantially aggravated by, dust exposure in coal mine employment.”

The Board concluded that the ALJ used that standard based upon his statement that legal pneumoconiosis includes “any chronic pulmonary disease or respiratory or pulmonary impairment that is significantly related to, or substantially aggravated by, dust exposure in coal mine employment.” JA 759. While acknowledging that the ALJ had used the term “rule out” in his discussion of the evidence, the Board explained its use derived from the fact that Drs. Zaldivar and

Sporn had themselves “ruled out” coal mine dust exposure as contributing to the miner’s COPD:

In rejecting the opinions of Drs. Zaldivar and Sporn, the administrative law judge did not, as employer asserts, require these physicians to “rule out” all contributions from coal dust exposure to the miner’s COPD in order to disprove legal pneumoconiosis. Rather, the administrative law judge simply concluded that neither Dr. Zaldivar nor Dr. Sporn adequately explained why *they* completely excluded coal dust exposure and as significant contributor to the miner’s COPD.

JA 759.

The Board also concluded that the ALJ properly rejected the opinions of Drs. Zaldivar and Sporn because, as the ALJ found, the doctors emphasized how toxic the miner’s smoking and alleged asthma were at the expense of explaining why coal mine employment was not a significant contributor. JA 759-60. In support, the Board pointed to the ALJ’s observation that, “even assuming” Dr. Zaldivar properly diagnosed overlap syndrome, he “failed to give a ‘documented or well-reasoned basis for excluding the contribution of the miner’s nearly [twenty] years of coal mine dust exposure to his disabling lung disease.” JA 760 (quoting the ALJ at JA 749). And the Board pointed to Dr. Sporn’s conclusion that smoking was the sole cause merely because it “exceeded the lung burden of coal mine dust.” JA 760 (quoting Dr. Sporn at JA 327).

Because Cedar Coal failed to establish error in the ALJ’s award, the Board affirmed the ALJ’s award of benefits to Claimant.

SUMMARY OF THE ARGUMENT

Claimant, widow of the miner, is rebuttably presumed entitled to survivor's benefits based upon her husband's nineteen-plus years of coal mine employment and the fact that his COPD was totally disabling. Cedar Coal can defeat her claim if it can prove that the miner did not suffer from either clinical or legal pneumoconiosis, or that the miner's pneumoconiosis played no part in his death from end-stage COPD.

Cedar Coal proved that the miner did not suffer from clinical pneumoconiosis, but was unable to prove that he did not have legal pneumoconiosis (i.e., a respiratory condition significantly related to coal mine employment). The ALJ properly found that the coal company's doctors – Drs. Zaldivar and Sporn – while credible in proving that smoking and asthma were perhaps primary causes, were unable to convincingly support their conclusion that *none* of the miner's COPD was due to coal mine employment.

The doctors tried to prove smoking and asthma to be the sole culprits simply by showing how much more toxic those factors were. But this reasoning, as the ALJ found, failed to explain why coal mine employment did not at least contribute to or aggravate the miner's COPD. As the Court has frequently held, this is a proper basis to discredit a doctor's opinion on the issue of legal pneumoconiosis.

The ALJ used the correct standard – significantly related to or substantially aggravated by – to determine whether Cedar Coal’s evidence disproved the presumed existence of legal pneumoconiosis. It was Drs. Zaldivar and Sporn who muddied the waters by stating that the miner’s coal mine employment provided absolutely no contribution to the miner’s COPD. The ALJ simply addressed these opinions on their own terms.

The ALJ also properly found that Cedar Coal failed in its burden to prove that the miner did not suffer from legal pneumoconiosis. That finding precluded rebuttal by the first method. It also doomed chances of rebuttal by the second method because doctors who mistakenly assume the miner does not have legal pneumoconiosis, have little credibility when the issue is whether the miner’s pneumoconiosis contributed in any way to his death. Here, because Drs. Zaldivar and Sporn wrongly believed that the miner’s COPD was unrelated to coal mine employment, they could not credibly opine that the miner’s death, which was due to COPD, was unrelated to legal pneumoconiosis or coal dust exposure. In short, their opinions on the role of legal pneumoconiosis in causing the miner’s death are per se unreliable.

This Court should affirm the ALJ’s award of survivor benefits.

ARGUMENT

A. Standard of Review

This Court's review of decisions under the BLBA is "limited." *Harman Min. Co. v. Dir., Off. of Workers' Comp. Programs*, 678 F.3d 305, 310 (4th Cir. 2012). The Court evaluates "whether substantial evidence supports the factual findings of the ALJ and whether the legal conclusions of the Board and ALJ are rational and consistent with applicable law." *Id.* The Court must "defer to the ALJ's determination regarding the proper weight to be accorded competing medical evidence, and [the Court] must be careful not to substitute [its] judgment for that of the ALJ." *West Virginia CWP Fund v. Bender*, 782 F.3d 129, 144 (4th Cir. 2015) (internal quotation marks omitted).

B. Introduction

After finding invocation of the fifteen-year presumption, the ALJ considered whether Cedar Coal rebutted the presumption by proving that 1) the miner suffered from neither clinical nor legal pneumoconiosis; or 2) the miner's pneumoconiosis did not contribute in any way to the miner's death. The ALJ found neither criterion established: the first method was not satisfied because the coal company failed to prove the absence of legal pneumoconiosis; and the second method was not satisfied because the opinions of the Cedar Coal's doctors on the question of whether pneumoconiosis caused the miner's fatal COPD were undermined by their

belief – contrary to the ALJ’s determination – that the miner suffered from legal pneumoconiosis.

In its opening brief, Cedar Coal asserts that the ALJ erred in finding that the coal company failed to prove that the miner did not suffer from legal pneumoconiosis. Specifically, Cedar Coal argues that the ALJ used the wrong standard for disproving legal pneumoconiosis, and improperly weighed the evidence relative to disproving that condition. Both arguments are without merit.

C. The ALJ used the right standard to determine if Cedar Coal proved the absence of legal pneumoconiosis.

There is no dispute that, based upon the regulation at 20 C.F.R. § 718.201, legal pneumoconiosis is any respiratory condition arising out of coal mine employment, 20 C.F.R. § 718.201(a)(2), with the “arising out of” provision “includ[ing] any chronic pulmonary disease or respiratory or pulmonary impairment *significantly related to, or substantially aggravated by*, dust exposure in coal mine employment,” 20 C.F.R. § 718.201(b) (emphasis added). And there is no dispute that for Cedar Coal to prove the absence of legal pneumoconiosis, the coal company was required to prove the negative of the definition of legal pneumoconiosis, i.e., the coal company was required to prove that the miner had no respiratory condition “*significantly related to, or substantially aggravated by*, dust exposure in coal mine employment.”

The ALJ used this standard. He stated that the regulatory definition of legal pneumoconiosis included the “significantly related to, or substantially aggravated by” requirement, JA. 743; and that the issue in the case was whether the miner’s “disabling COPD [was] significantly related to his coal mine employment,” JA 748. And when discussing the opinions of Drs. Zaldivar and Sporn, the ALJ used the regulation’s “significant” contribution language: “Dr. Zaldivar and Sporn [did] not adequately explain the basis for ruling out *significant* contribution from coal mine dust exposure to the miner’s severe COPD,” JA 750 (emphasis added).

The coal company, however, resists. Opening Brief at (OB) 21-22. While acknowledging that the ALJ “identified” the correct standard, OB 21, Cedar Coal argues that the ALJ effectively used a more difficult standard that required the company to rule out any and all contributions from the miner’s employment. In support, Cedar Coal points to where the ALJ used “contribute” or “rule out” without the “significant” or “substantial aggravation,” qualification. OB 21-22. A typical example, according to Cedar Coal, was where the ALJ discredited the opinions of Drs. Zaldivar and Sporn for not explaining why they “ruled out legal pneumoconiosis.” OB 21.

Cedar Coal, however, fails to see the forest from the trees. When discussing the defects in the doctors’ opinions, the ALJ used terms like “rule out” because that is what the doctors reported: they “ruled out” coal mine employment and found

that the work in no way or manner contributed to the miner's COPD.

Consequently, it was reasonable for the ALJ to inquire concerning the reasons the doctors "ruled out" any contribution. The Court, therefore, must reject the coal company's assertion that the ALJ used an improper standard when considering legal pneumoconiosis.

D. The ALJ properly determined that Cedar Coal failed in its burden to disprove the existence of legal pneumoconiosis.

The ALJ found that Cedar Coal did not prove the absence of legal pneumoconiosis because neither Dr. Zaldivar nor Dr. Sporn explained why – even if the miner's COPD was mostly due to smoking – his nineteen-plus years of coal mine dust exposure did not at least contribute to his disabling and fatal COPD. JA 747-50. The ALJ found the opinions of these two doctors wanting.

This Court addressed the same situation in its recent decision in *Westmoreland Coal Co. v. Stallard*, No. 16-1416, --- F.3d ---- 2017 WL 5769516, at *7 (4th Cir. Nov. 29, 2017). There, the employer's doctors concluded that the miner's smoking was the sole cause of his respiratory condition, and explained that the miner "would have been equally disabled had he never worked in the coal mining industry." *Stallard*, 2017 WL 5769516, at *7. The Court affirmed the ALJ's discrediting of these doctors because they focused on smoking as the cause of the miner's respiratory condition at the expense of discussing coal-mine dust

exposure as a co-contributor. The Court stated:

[The] doctors ruled out coal dust exposure as a potential cause simply because they viewed smoking to be the sole cause; however, because they solely focused on smoking, they nowhere addressed why coal dust could not have been an *additional* cause—a fundamental aspect of the legal inquiry [concerning the existence of legal pneumoconiosis].”

Id. at n.4. The Court affirmed an ALJ’s rejection of a similar analysis in *Westmoreland Coal Co. v. Stidham*, 561 Fed.Appx 280, 284 (4th Cir. 2014)

(emphasis added):

[A]lthough both physicians asserted that Stidham’s symptoms were “related to” or “classic” for cigarette smoking disease, *neither explained why*, assuming that cigarette smoking played the main role in causing the Claimant’s acknowledged pulmonary and respiratory disability, *coal mine dust exposure could not have played some less, but nevertheless significant, role*, consistent with the discussion of the epidemiology in the Preamble to the regulations.

See also Consolidation Coal Co. v. Galusky, 648 Fed.Appx 316, 2016 WL 2642784, at *5 (4th Cir. May 10, 2016) (“The problem, as both the ALJ and Board noted, is that neither expert explained why the asthma component of Galusky’s condition was not aggravated by exposure to coal dust.”).

That Drs. Sporn and Zaldivar were guilty of shortsightedness towards coal-mine dust exposure can easily be seen. Dr. Sporn concluded his report by stating that smoking was the sole cause of the miner’s fatal COPD simply because it was the most dangerous: “[T]he dosage of cigarette smoke sustained by [the miner] exceeded the lung burden of coal mine dust and *therefore* was the sole cause of his

impairment and death from [COPD].” JA 327 (emphasis added). The doctor failed to consider that, even with smoking as the primary causes, coal mine dust exposure could have contributed to or aggravated the miner’s condition.

Similarly, Dr. Zaldivar considered the search for the causation-culprit to be a competition, to see which exposure was the more likely. To this end he enthusiastically discussed his overlap syndrome theory and explained why it “fit” with the miner’s medical history and symptoms. He then concluded that the match precluded any contribution from coal mine employment: “In this case, we have a clear diagnosis of a smoker’s asthma-COPD overlap syndrome with a very extensive history of smoking, which is in itself [] sufficient to cause such problem” JA 371. And Dr. Zaldivar admitted in deposition that he based causation on which conditions likely did the most damage to the miner’s lungs. *See supra* at 22.

Both doctors seemed oblivious to the fact that a lung disease may have more than one cause; that coal dust exposure need not be the primary cause; and that as long as coal dust exposure significantly contributes, the lung disease is, by definition, legal pneumoconiosis.¹⁵ *Consolidation Coal Co. v. Dir., Office of*

¹⁵ Notably, 20 C.F.R. § 718.204(c)(1)(ii) provides that, even if a miner is already totally disabled by a respiratory condition unrelated to coal mine employment, pneumoconiosis may still be considered a “substantially contributing cause of the miner’s disability” if the pneumoconiosis “[m]aterially worsens [the] totally disabling respiratory or pulmonary impairment. . . .” *See also* 20 C.F.R. § 718.204(a) (providing that a miner may prove the required total respiratory disability even if a non-respiratory condition disabled the miner first).

Workers' Comp. Programs, 721 F.3d 789, 791 (7th Cir. 2013) (“Any chronic lung disease that is ‘significantly related to, or substantially aggravated by’ exposure to coal mine dust is legal pneumoconiosis; dust need not be the sole or even primary cause of the disease.”)

While the ALJ correctly observed that Drs. Sporn and Zaldivar did not sufficiently explain why they viewed coal-mine dust exposure as a non-contributor, that does not mean no attempt was made. Dr. Zaldivar tried, but was not persuasive. For instance, he attempted to prove that the miner’s coal mine employment resulted in no contribution to the miner’s COPD based upon the negative x-rays and ct-scan. He explained that coal mine employment contributed to the miner’s COPD to the extent the miner’s x-rays and ct-scan showed coal dust. JA 731. In support, he stated that studies reviewed by NIOSH in the *Criteria* showed that there is a correlation between the amount of dust in lungs and a miner’s emphysema, and that the amount of dust may be determined by looking at x-rays. Because the x-rays were negative in this case, Dr. Zaldivar concluded that the miner’s coal mine employment was not a contributing cause of his COPD. *Id.*

The ALJ, however, was quick to debunk the doctor’s assertion because there was no biopsy or autopsy in this case that reliably measured the amount of dust:

Although an opining physician may be credited for distinguishing between coal dust and smoking-induced emphysema based on the absence of dust retention seen on pathology, Dr. Zaldivar’s opinion in this case does not rely on the amount of dust seen on pathology as

there is no available autopsy or biopsy evidence in this case. Instead, he *assumes* that the dust burden is minimal because of the absence of radiographic pneumoconiosis.

JA 149. The ALJ's expectation that there be autopsy or biopsy evidence to determine the amount of dust in the lungs is consistent with the preamble at 65 Fed. Reg. at 79941-42, where the Department discussed a study suggesting a correlation between causation due to coal mine employment and the amount of dust measured pathologically.

While Cedar Coal admits the significance of the lack of biopsy or autopsy evidence, *see* OB 16, it nonetheless presses Dr. Zaldivar's belief, *see supra* at 21, that x-rays and ct-scans can, in fact, be used to determine the weight of dust in the lungs. OB 36. But the doctor's reliance on the NIOSH's *Criteria* at 4.1.2.3 is misplaced. NIOSH did not state, and the cited medical literature simply did not establish, that a negative x-ray indicates the absence of coal dust in the lungs. Indeed, NIOSH cited studies showing that the *smallest* opacities correlated to the greatest lung weights, while some opacities did not correlate to dust content at all. And most important, NIOSH reiterated the significant possibility of a false negative x-ray (up to 33%). This is the underlying reason for permitting physicians under 20 CFR § 718.202(a)(4) to diagnose legal pneumoconiosis without a positive x-ray. *See Usery v. Turner Elkhorn Min. Co.*, 428 U.S. 1, 31-34 (discussing the unreliability of x-ray evidence for disclosing the presence of the

pneumoconiosis). In any event, the Department did not include the studies Dr. Zaldivar cites in the preamble, *see* 65 Fed. Reg. at 79941-42; NIOSH supported the scientific analysis in the preamble, 65 Fed. Reg. 79937; and Dr. Zaldivar himself admitted the superiority of pathology in determining the amount of dust. JA 371.

Dr. Zaldivar also attempted to undermine Dr. Rasmussen's and the preamble's observation that "tobacco smoking and mine work both produce emphysema through the same mechanism." JA 313. He touted a new study that showed how to identify the harm caused by smoking, and suggested that was a way to distinguish smoking from coal-mine dust exposure. *Id.* But Dr. Rasmussen astutely responded by stating that there has been no similar study of the effects of coal mining, so the dilemma continued. JA 228. Dr. Zaldivar's only answer was that there needed to be studies looking into the mechanisms: "[I]t is not logical to state that two diseases[] that look the same[], must have occurred through . . . the same mechanism of damage without doing empirical studies." JA 369. But there were studies done, and that is how the "similar mechanisms" observation ended up in the preamble, 65 Fed. Reg. at 79942-43.

In sum, Drs. Zaldivar and Sporn both tried to exclude coal-mine dust exposure as a cause of the miner's COPD because smoking – and asthma in Dr. Zaldivar's view – amounted to a sufficient explanation. But just because the medical data "fits" a particular cause does not mean that another cause could not

also have contributed. The medical literature cited in the preamble, by recognizing the additive effects of coal dust exposure and smoking, demonstrated that very possibility. Consequently, though the coal company's doctors tried, their opinions are just not persuasive in disproving legal pneumoconiosis. The ALJ reasonably and properly ruled against Cedar Coal on this issue.¹⁶

E. The Court need not address Cedar Coal's remaining two arguments because one was not considered by the Board, and the other is undermined by the coal company's failure to disprove the existence of legal pneumoconiosis.

In addition to the just-discussed reasons why the ALJ discredited the opinions of Drs. Zaldivar and Sporn, the ALJ added another: the doctors based their opinions on the assumption that a miner's COPD is never due to coal mine employment unless there is a positive x-ray or ct-scan. JA 748-49. The ALJ correctly determined that such a belief would be contrary to the BLBA, the regulations, and the preamble. *Id. See Westmoreland Coal Co, Inc. v. Dir., Off. of*

¹⁶ Cedar Coal last asserts (OB 44-46) that the ALJ erred by not giving weight to state compensation decisions when considering whether the coal company disproved the existence of legal pneumoconiosis. JA 738. Not true. The ALJ accorded them little weight because they relied primarily on evidence that was not in the administrative record, i.e., Dr. Fino's opinion and the testimony of Dr. Kinder, *see e.g.* JA 385. Moreover, the ALJ correctly observed that the State standards for entitlement differed from the federal ones. *See Freeman United Coal Min. Co. v. Off. of Workers' Comp. Programs*, 20 F.3d 289, 294-65 (7th Cir. 1994) (finding no collateral estoppel because the claimant had the burden of proof in state proceedings but was entitled to a presumption in the federal proceedings). Notably, Claimant here has the benefit of the fifteen-year presumption. The state decisions do not indicate a similar provision.

Workers' Comp. Programs [Cochran], 718 F.3d 319, 324 (4th Cir. 2013) (“Both the preamble [to the regulatory definition of legal pneumoconiosis] and regulations make clear that the absence of clinical pneumoconiosis cannot be used to rule out legal pneumoconiosis.”); *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173, 174 n.* (4th Cir. 1995) (holding doctor’s opinion that “a diagnosis of pneumoconiosis. . . . cannot be made without evidence by x-ray” is “in direct conflict with the regulations promulgated under the Act,” and cannot be “afforded much, if any, weight”).

The Board, however, properly concluded that it was not necessary to address the correctness of the ALJ’s interpretation because the ALJ’s other reasons for discrediting the doctors were valid. JA 760 n.8. *See Cochran*, 718 F.3d at 324 (explaining that an ALJ’s decision may be affirmed if “the ALJ provided independent reasons . . . for dismissing [the doctor’s] opinion”) (internal quotation marks omitted).

Despite this, Cedar Coal argues the issue to the Court. OB 23-27. In doing so, the coal company ignores clear precedent from this Court. Relying on *SEC v. Chenery*, 318 U.S. 80 (1943), this Court determined that “[its] review [of Board decisions] is *confined exclusively to the grounds actually invoked by the Board.*” *Island Creek Coal Co. v. Henline*, 456 F.3d 421, 426 (4th Cir. 2006) (emphasis added) (citations omitted). Consequently, the Court cannot consider the issue and

must remand the case to the Board only if it does not affirm the ALJ's other bases for finding that Cedar Coal failed to rebut the presumption.

Cedar Coal's remaining argument is that the ALJ erred in not finding the fifteen-year presumption rebutted by the second method of rebuttal – by proof that the miner's death by end-stage COPD was unrelated to his pneumoconiosis. OB 41-43. But when the coal company failed to disprove legal pneumoconiosis, it effectively lost its ability to prove that the miner's death was unrelated to his pneumoconiosis: the presumption that the miner's COPD was related to his coal mine employment went unrebutted, and it was the COPD that caused his death. Thus, because the company's doctors were wrong about the cause of the miner's COPD, they could not fairly answer the question of whether the miner's pneumoconiosis contributed to his death.

That was the ALJ's conclusion, and he was correct. In so deciding, the ALJ looked to this Court's decision in *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109, 116 (4th Cir. 1995). There, the Court held that little credit should be given to a doctor's opinion concerning the cause of a miner's impairment or death where the doctor, contrary to the ALJ's determination, mistakenly assumed the miner did not suffer from pneumoconiosis. See *Soubik v. Dir., Office of Workers' Comp. Programs*, 366 F.3d 226, 234 (3d Cir. 2004) (“Common sense suggests that it is usually exceedingly difficult for a doctor to properly assess the contribution, if any,

of pneumoconiosis to a miner's death if he/she does not believe it was present.”), quoted in *Hobet Min., LLC v. Soubik*, 783 F.3d 498, 504 (4th Cir. 2015); *see also* *Island Creek Ky Min. v. Ramage*, 737 F.3d 1050, 1062 (6th Cir. 2013) (explaining that the existence of legal pneumoconiosis and disability causation “are closely related” issues, and the ALJ’s discrediting of certain experts as to the former applied to the latter); *cf. Collins v. Pond Creek Min. Co.*, 751 F.3d 180, 185 (4th Cir. 2014) (holding that doctors’ opinions which incorrectly diagnosed smoking-related COPD rather than legal pneumoconiosis, nonetheless supported entitlement where COPD caused the miner’s death).

Cedar Coal does not address or cite *Toler*. With blindfolds, the coal company continues to argue that its doctors correctly reported that the miner’s COPD – and thus his death – was due solely to smoking and asthma, and that the ALJ erred in finding to the contrary. OB 41-45. But the ALJ and the Board correctly found that Cedar Coal lost that argument. The Director respectfully requests that the Court agree.

CONCLUSION

The ALJ correctly determined that Cedar Coal did not rebut the fifteen-year presumption that Claimant is entitled to survivor benefits. The Court, therefore, should affirm the award.

Respectfully submitted,

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CERTIFICATE OF COMPLIANCE

Pursuant to Federal Rule of Appellate Procedure 32(a)(7)(C), I certify that this brief is proportionally spaced, using Times New Roman 14-point typeface, and contains 10,408 words, as counted by Microsoft Office Word 2010.

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CERTIFICATE OF SERVICE

I hereby certify that on December 15, 2017, the Director's brief was served electronically using the Court's CM/ECF system on the Court and the following:

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