

**U.S. Department of Labor**

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**Issue Date: 09 May 2012**

CASE NO.: 2010-BLA-5809

In the Matter of:

THEODORE M. LATUSEK, JR.,  
Claimant

v.

CONSOLIDATION COAL COMPANY,  
Employer

and

CONSOL ENERGY, INC.,  
Carrier

and

DIRECTOR, OFFICE OF WORKERS'  
COMPENSATION PROGRAMS,  
Party-in-Interest.

APPEARANCES:

Sue Anne Howard, Esq.  
For the Claimant

William S. Mattingly, Esq.  
For the Employer

Before: THOMAS M. BURKE  
Administrative Law Judge

**DECISION AND ORDER AWARDING BENEFITS**

This proceeding arises from a claim for lifetime benefits under the Black Lung Benefits Act, 30 U.S.C. §§ 901-945 and the regulations issued thereunder, found at Title 20 of the Code of Federal Regulations. The Act provides benefits to miners who are totally disabled due to pneumoconiosis and to eligible survivors of miners whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung disease, is a chronic dust disease of the lungs, including respiratory and pulmonary impairments arising out of coal mine employment. In this

claim Theodore M. Latusek, Jr. (“Claimant”) requests modification of a denial of his lifetime claim.

#### PROCEDURAL BACKGROUND

This claim for black lung disability benefits has a lengthy procedural history. This decision is the fourth by an administrative law judge, it has been reviewed on three occasions by the Benefits Review Board (“Board”) and on two occasions by the Fourth Circuit Court Appeals. It has been ongoing since Claimant filed his application for benefits on July 5, 1994. The district director, Office of Workers Compensation, reviewed the application and awarded benefits on May 12, 1995. Employer, Consolidation Coal Company, requested a hearing and the claim was referred to the Office of Administrative Law Judges (“OALJ”). Administrative Law Judge Daniel L. Leland issued a Decision and Order Awarding Benefits on June 26, 1997. Employer appealed to the Board on July 28, 1997. The Board affirmed the award of benefits by Decision and Order dated July 17, 1998. Employer appealed to the U.S. Fourth Circuit Court of Appeals. The Court vacated the decision awarding benefits and remanded the case for further consideration by unpublished opinion dated August 6, 1999. Judge Leland considered the case on remand, and after again weighing the medical evidence, adhered to his earlier decision that Claimant was totally disabled due to pneumoconiosis, and awarded benefits by Decision and Order On Remand-Awarding Benefits dated June 8, 2000. Employer again appealed to the Board. This time the Board in a September 17, 2001 Decision and Order vacated Judge Leland’s decision awarding benefits and remanded the case for a reevaluation of the evidence. Judge Leland reevaluated the evidence and, based on his review, again found Claimant to be totally disabled due to pneumoconiosis, and awarded benefits in a Decision and Order dated January 2, 2002. Employer appealed to the Board for review of Judge Leland’s decision awarding benefits, and the Board, after review, affirmed Judge Leland’s award of benefits in a Decision and Order dated November 26, 2002. Employer for a second time appealed the decision of the Board affirming Judge Leland’s award of benefits to the Fourth Circuit Court of Appeals. The Court of Appeals in an unpublished opinion dated January 23, 2004, reversed the award of benefits. (DX 102).<sup>1</sup>

The present case involves Claimant’s request for modification of the Fourth Circuit Court’s reversal of award of benefits by a letter to the district director dated January 19, 2005. The modification request is supported by a medical opinion from Dr. James Dauber, Claimant’s treating pulmonary specialist, stating that Claimant’s pulmonary condition is caused by exposure to coal mine dust. The district director granted Claimant’s request for modification in a Decision and Order dated May 24, 2005. (DX 107).

Employer requested a hearing and the matter was forwarded to OALJ on November 16, 2005. (DX 112). A hearing scheduled for August 2, 2006, in Morgantown, West Virginia was continued by Judge Michael Lesniak at the joint request of the parties as Claimant was hospitalized after having undergone lung transplant surgery. The claim was rescheduled for hearing on July 24, 2007, in Morgantown, West Virginia. The case was again continued by Order of Continuance dated June 6, 2007, in response to a joint request for continuance for the

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<sup>1</sup> Director’s Exhibits are marked as DX\_\_\_, Claimant’s Exhibits are marked as CX\_\_\_; Employer’s Exhibits are marked as EX\_\_\_; pages of the hearing transcripts are marked as Tr.\_\_\_\_.

purpose of allowing a West Virginia workers' compensation claim to proceed to resolution. By Order of Remand dated May 23, 2008, the claim was remanded to the district director until such time that the West Virginia workman's compensation claim was resolved. (DX 115).

Claimant's West Virginia workers' compensation claim was finally resolved by an Order of the West Virginia Supreme Court of Appeals on March 31, 2010. Consequently, the claim was referred back to OALJ on August 5, 2010. (DX 117, 118).

A hearing took place in Morgantown, West Virginia on April 27, 2011. Employer stipulated to 23 years of coal mine employment. Employer also agreed that the presence of pneumoconiosis, causal relationship of pneumoconiosis to coal mining, and total pulmonary disability had been established. (Tr. 7). Admitted into evidence were Director's Exhibits (DX) 1-121, and Employer's Exhibit (EX) 1-12. The record was held open for the submission by Claimant of the deposition of Dr. Cecile Stephanie Rose, and for the submission by Employer of depositions by Dr. Samuel V. Spagnolo and Dr. David M. Rosenberg. Dr. Rose's deposition was received on July 22, 2011, and admitted into evidence as CX 1. Dr. Spagnolo's deposition was received on May 2, 2011, and admitted into evidence as EX 13. Dr. Rosenberg's deposition was received on August 19, 2011, and admitted into evidence as EX 17. Post-hearing briefs were received from both parties on September 1, 2012.

About two weeks after the hearing, Claimant filed a motion seeking authorization to serve post-hearing interrogatories on counsel for Employer for the purpose of obtaining 15 biopsy slides and 15 tissue blocks of Claimant's native left lung from UPMC lung pathology department which had been in Employer's counsel's possession since 2007, as well as any reports generated from examination of the lung pathology specimens, for review and comment by Dr. Rose. Counsel for Claimant averred that prior to her deposition, Dr. Rose requested that she be provided with native lung pathology specimens from Claimant's lung transplant, which had taken place on July 3, 2006, at UPMC. When Claimant contacted his transplant physician to have the biopsy specimens forwarded to Dr. Rose, Claimant was informed by UPMC that the law firm of Jackson Kelly had signed out all available specimens consisting of 15 biopsy slides and 15 tissue blocks on June 18, 2007, and had never returned the specimens. Claimant's counsel offered that none of Employer's consulting physicians, including Drs. Renn, Spagnolo, Tuteur, or Rosenberg, refer to any pathology evidence other than the July 3, 2006 UPMC report. Claimant asserted that, "in order to provide a full and fair hearing, satisfy the interests of due process, and promote the integrity of the adjudicative process," production of the 15 biopsy slides and 15 tissue blocks of Claimant's native left lung from UPMC lung pathology department, as well as any reports generated from examination of the lung pathology specimens, was necessary.

Employer opposed Claimant's motion on the grounds that the request was untimely and improper as it sought production of materials protected as attorney work product. Specifically, Employer asserted that Claimant waited at his own peril until after the hearing to seek to have the biopsy slides reviewed for the first time and asserted that it has no duty to produce evidence from non-testifying experts, and that such reports are protected from discovery as attorney work product under Rule 26 of the Federal Rules of Civil Procedure.

Claimant's motion was granted and Employer was ordered to produce the biopsy slides as well as any reports generated from examination of the lung pathology. Employer's argument that the pathology reports requested by Claimant were protected from compelled production as attorney work product was rejected under 29 C.F.R. § 18.14(c), and the reasoning of the Benefits Review Board's decision in *Fox v. Elk Run Coal Company, Inc.*, No. 09-0438 BLA (BRB Apr. 16, 2010). (work product not protected as it is product of physicians, not attorneys). Employer's argument that Claimant's request was untimely was also rejected as the evidentiary record remained open.

In compliance with the order Employer submitted the July 19, 2007 biopsy report by Dr. Richard L. Naeye, marked as EX 14, August 21, 2009 biopsy report by Dr. Erika C. Crouch, marked as EX 15, and the curriculum vitae of Dr. Crouch, marked as EX 16.

#### FINDINGS OF FACT

Claimant worked as an underground coal miner for 23.5 years for Employer, from September 23, 1970 to April 3, 1994. He was the low wall operation coordinator. His job in the early 1970s was long wall section foreman "when long wall mining was really on the move in the United States." (Tr. 13). He testified that his work with the new technology exposed him to large amounts of dust:

We had a lot of cave-ins and things like that. We'd have to mine the rock up. Long wall mining is kind of unique because once you get into a position, the face is all set up, there's no way to go backwards. You only have to go forward. So, if you get --- not matter what you encounter, you have to cut through that. You may have a fall on 200 feet high and under that cave-in is all sandstone, limestone, various materials and you have to cut that up and it has to be discharged out of the mine because you can't put it back.

(Tr. 13, 14).

At Claimant's prior hearing before Judge Leland on February 25, 1997, he testified with more specificity about his job and resulting coal dust and sandstone exposure:

Today that panel, with the technology of the roof supports, you could probably extract that coal in maybe two months of operations, would be considered a reasonable time to extract that amount of coal. But then at that time it took us 14 months. And primarily we were - - due to the fact that the roof supports could not hold the roof up, we were constantly mining pure rock and sandstone and a lot of that. And when we got in those kind of conditions, I literally lived at the Ross Run Mine. I would work 12 to 16 hours a day underground because it was such dangerous conditions that I felt that at the time that I didn't want anybody doing anything that I wouldn't do as far as safety goes. And I tried to stay with the face as much as I could when we got in the adverse conditions. A lot of times I would spend 24, 36 hours

underground when we had those kinds of conditions because of the fact that they were in such a dangerous situation with the equipment, the equipment plus injury, too, to the miners. And since I was the responsible person in charge of that long wall, I felt that it was my duty and responsibility to stay in there and actually see if we could make the conditions any better.

(Feb. 25, 1997 hearing, Tr. 17, 18).

The follow up question to Claimant was: What was the dust actually like?

The dust was pretty severe when we got into the rock depending on the ventilation. And a lot of times we would have cave-ins at the tailgate, and that's when the ventilation would become much of a standstill because the flow - - the restriction of the airway was reduced significantly, and the dust would be a tremendous amount at that time. It wouldn't be coal dust. I would mostly be rock dust.

(*Id.* at 18).

Claimant retired because of pulmonary difficulties. (Tr. 10, 11). He presented a letter to Employer from his treating physician stating that he had a pulmonary condition that was being caused by his coal dust exposure. Consequently, Employer agreed to remove him from coal dust exposure, but the alternate job he was given paid forty percent less salary. He testified that he considered his best option was to go on short term disability and subsequently long term disability. (Tr. 12).

Claimant's pulmonary problems were first recognized in 1990 when he went for a company physical. The examining physician observed breathing problems and suggested he see a pulmonologist. Claimant initially saw Dr. Abraham in Morgantown, West Virginia, and later sought treatment by Dr. Joseph Renn. He treated with Dr. Renn for about a year and a half. Claimant testified that Dr. Renn's treatment was not helping, his condition was not improving, and Dr. Renn thought his "outlook was sort of bleak." (Tr. 28). He asked Dr. Renn if there was any place he could find help. Dr. Renn recommended National Jewish Hospital in Denver Colorado. (*Id.*).

Claimant treated with Dr. Cecile Stephanie Rose and Dr. Constance A. Jennings at National Jewish Hospital from September of 1993 to August of 1996. (Tr. 25, 26). In September of 1996 he moved the treatment for his pulmonary condition to the University of Pittsburgh Medical Center ("UPMC"), where he saw Dr. James Dauber who placed him on the lung transplant list. (Tr. 15, 16) Claimant saw Dr. Dauber every four to six months depending on his condition and test results. (Tr. 16). Dr. Dauber examined Claimant at each visit. The examination included pulmonary function tests and chest x-rays. Its focus was judging lung capacity to follow the progression of the disease. (Tr. 17). Claimant's condition leveled off and he was taken off the transplant list in 1998. (Tr. 18). His condition remained stable until 2006, when his condition worsened. He underwent a single left lung transplant on July 3, 2006. (Tr. 21). Claimant continues to treat at UPMC every three to six months. (Tr. 27).

The lung pathology report from UPMC dated July 3, 2006, diagnosed usual interstitial pneumonia; hamartoma (3.0 cm in diameter) in left lower lobe; and multiple hilar lymph nodes with focally calcified anthracosilicotic nodules. (EX 1). The pathologist's comment was: histologic sections demonstrate usual interstitial pneumonia pattern of the lung injury of uncertain etiology. Scattered rare poorly formed granulomas are identified, and these may represent a nonspecific finding. However, a possibility of chronic hypersensitivity reaction or aspiration cannot be excluded. The Gross Description provided in part: pleural surface is tan to red and anthracotic. The surrounding lymph nodes are anthracotic and of normal size. Palpable at the lower left lobe is a firm mass with a cut surface consisting of cartilaginous gray-white, rubbery to firm tissue. The adjacent parenchyma is red to anthracotic and soft. Serial sectioning through the remainder of the lung exposes similar fibrotic tan-red to anthracotic soft to rubbery parenchyma. No other suspicious lesions are seen. (EX 1).

Employer had the pathology of the explanted left lung reviewed by two pathologists: Dr. Richard Naeye and Dr. Erica Crouch.

Dr. Naeye reported that the tissues show that collagen has massively replaced normal or near normal tissues in the lungs. He found no black pigment and no very tiny birefringent crystals of toxic silica associated with the fibrosis. He offered the opinion that the near absence of fibrosis in nearby lymph nodes is categorical confirmation that the fibrosis is not occupational-silicotic in origin. He explained that when silica or other environmental fibrogenic agents damage lung tissue they eventually drain into nearby lymph nodes where they produce fibrosis. He observed that with one small exception the lymph nodes that are available for microscopic review are free of the lesions that are present in his lung tissues. He knew of no available information to determine the origin of the fibrosis so it must be labeled idiopathic. He concluded that there is no evidence that it would be occupational in origin. (July 19, 2007 report; EX 14).

Dr. Erika Crouch reviewed the tissue slides from the explanted left lung and slides from a biopsy of the right middle and upper lobes of lung in an August 21, 2009 report. Her diagnosis of the lung, left and right was

:chronic organizing interstitial pneumonia  
:coal dust deposition with small numbers of coal dust macules consistent with mild simple coal workers' pneumoconiosis.  
:pulmonary hamartoma in explanted lung

Dr. Crouch reported that the lungs show evidence of a severe diffuse fibrosing lung disorder, first recognized in 1992 and eventually progressing to end-stage pulmonary fibrosis as is evident from the explants in 2006. Histologic findings in the 1992 biopsy are generally consistent with usual interstitial pneumonia (UIP) and the clinical diagnosis of idiopathic pulmonary fibrosis (IPF). The changes appear more severe in the upper lobe, but this could reflect limited sampling. There are a few coal dust macules, but no larger dust related lesions, and there is also no concordance between the distribution of dust and the distribution or severity of the observed fibrosis. The explanted lung shows end-stage remodeling and pulmonary fibrosis, which is most severe in the lower lobes.

Dr. Crouch reported it notable that Claimant had an unusually long survival for one with IPF, raising the possibility of other less aggressive disorders, but not suggesting a contributing role of coal dust. She offered the opinion that Claimant was undoubtedly disabled from lung disease, but found no evidence that occupational dust exposure contributed to any significant degree to clinical impairment or disability.

#### CONCLUSIONS OF LAW

There is agreement that Claimant is totally disabled from a pulmonary condition. It is also agreed that Claimant worked over twenty years as an underground coal miner and has simple pneumoconiosis resulting from his coal mine employment. The sole issue is whether his total and permanent pulmonary disability results from his coal dust exposure. As shown by the recitation of the history of the litigation of this claim, this issue of causation has been wrestled with by administrative agencies and the Fourth Circuit Court of Appeals since Claimant filed his claim for benefits in 1994, some 18 years ago. The Office of Workers' Compensation Programs found entitlement in 1995 and Administrative Law Judge Daniel Leland awarded benefits based on the evidence before him in decisions issued in 1997, 2000 and November 26, 2002. The Board affirmed Judge Leland's initial award of benefits but on appeal the Fourth Circuit Court of Appeals vacated the award and remanded the claim to him for further consideration. After again considering the evidence, Judge Leland issued his second award of benefits. This decision awarding benefits was vacated by the Board and again returned for evaluation of the evidence. After awarding benefits for a third time, his award was affirmed by the Board. However, his decision was reversed by a decision of the Fourth Circuit Court of Appeals dated January 23, 2004. The Circuit Court held that it was error for the Administrative Law Judge to discredit the opinions of Drs. Renn, Fino, Morgan, and Kleinerman. Judge Leland's decision - Decision and Order on Remand - found these physicians to have offered "irrational" and "not well reasoned" medical opinions because they were able to draw a conclusion as to what is not the cause of Claimant's idiopathic pulmonary fibrosis when they could not give an opinion as to its cause.

The Court of Appeals reversal did not end the matter on January 23, 2004. Claimant had an ongoing claim for black lung benefits before the State of West Virginia. In light of the state claim and his conviction that the medical community's understanding of the cause of idiopathic pulmonary fibrosis has substantially evolved over the past 15 years, Claimant filed a request for modification on January 19, 2005, before the district director. Claimant asserted that the modification was necessary because: "in order to render justice, the contemporary state of medical literature should be considered in determining whether [Claimant] suffers from IPF associated with his coal dust exposure." The district director granted Claimant's request for modification in a Decision and Order dated May 24, 2005. (DX 107).

The modification provisions at Section 22 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 922, incorporated into the Black Lung Benefits Act at 30 U.S.C. § 932(a), provide the statutory authority to modify orders. A decision awarding or denying benefits in a black lung claim may be modified (increased, decreased, or terminated) at the behest of the claimant, employer, or district director upon demonstrating either: (1) a change in condition or (2) a mistake in a determination of fact. 20 C.F.R. § 725.310 (2010). Modification may be sought by the claimant at any time before one year after the denial of a claim. 20 C.F.R.

§ 725.310(a).

On a request for modification the entire record must be reviewed *de novo* to determine whether a "mistake in a determination of fact" has been made. *See e.g., Kovac*, 14 B.L.R. 1-156; *Dingess v. Director, OWCP*, 12 B.L.R. 1-141 (1989); *Cooper v. Director, OWCP*, 11 B.L.R. 1-95 (1988). In considering a request for modification, the fact-finder is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." *O'Keeffe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 257 (1971). As the initiator of modification proceedings in this case, Claimant bears the burden of persuasion in establishing a basis for modification of the decision denying benefits. *Branham v. BethEnergy Mines, Inc.*, 20 BLR 1-27, 1-34 (1996).

The State of West Virginia Workers' Compensation Office decided that Claimant's pulmonary impairment was caused by his coal mine dust exposure. The decision was based principally on a report by Dr. Jack Parker. Dr. Edward Doyle, the Clinic Director, Institute of Occupational & Environmental Health, West Virginia University Department of Occupational Medicine, provided a report dated December 7, 2001, to the West Virginia compensation office finding Claimant to have a work related physical impairment. His report referenced a letter to him from Dr. Parker wherein Dr. Parker stated, "[b]oth [Claimant's] coal worker's pneumoconiosis and his pulmonary fibrosis are more likely than not the result of coal mine dust exposure." (DX 103) Dr. Doyle's testimony in the state case described Dr. Parker as not only a pulmonary specialist, but a world class public health epidemiologist with "hundreds of publications and things like that." (DX 103, Doyle dep. at 23).

Dr. Parker testified in the state case on December 6, 2004. (DX 103, Parker dep.) He testified to his qualifications in pulmonary medicine. He testified that he was on staff of NIOSH until 1998, then took a full-time position at West Virginia University, becoming chief of pulmonary and critical care in 2000. At NIOSH he was the chief or acting chief of the clinical investigation branch and the coal workers' x-ray surveillance program, including B-reader certification program which he ran from 1991 to 1998. He is a NIOSH B-reader, has taught the ILO classification system for dust related lung diseases, has served as a consultant to both the World Health Organization and the International Labor Office in the teaching of the recognition of pneumoconiosis, and was a consultant for the revision of the ILO classification system in 2000 revision. (DX 103, Parker dep. at 26) Dr. Parker also has a significant clinical practice including patients with occupational lung diseases ranging from mineral dust, asbestos, coal and silica, to occupational asthma, and hypersensitivity pneumonitis, and other lung diseases, toxic lung inhalation from work.

Dr. Parker evaluated Claimant's pulmonary condition in May of 2001 for his state claim. He examined Claimant and reviewed records provided by Dr. Doyle or Claimant, including pathology reports and reports from Drs. Renn, Rose, Honma, Klinerman, Spagnolo and Morgan. He characterized the physicians who reported on the claim as a "variety of who's who of pulmonary physician's and pathologists." Dr. Parker was asked for the basis of his opinion that the Claimant's coal worker's pneumoconiosis and his pulmonary fibrosis are both more likely than not the result of his coal mine dust exposure. He answered:

Because he's a lifetime nonsmoker, and because his occupational exposure to coal mine dust did result in pathologic changes consistent with the typical response to coal mine dust, and that his pulmonary fibrosis was recognized, appeared and did not follow the typical time course of pulmonary fibrosis, that is by the time I saw him he was nine years out from a biopsy and not very impaired. Most people with pulmonary fibrosis, it's quite typical to die within five years rather than to survive, so I think he's an atypical case that isn't a typical case of pulmonary fibrosis and that based upon the extensive pathology read by Dr. Honma, I feel quite comfortable that his lung function abnormality was a result of his coal mine dust exposure and not the idiopathic variety of pulmonary fibrosis.

*(Id. at 21).*

Dr. Parker was asked about the opinions of other physicians who believe Claimant's pulmonary fibrosis has no known ideology, and whether he could rule out the fact that Claimant could have an IPF with no known ideology. He answered:

Nor can they rule out that his pulmonary fibrosis was related to his coal mine dust exposure. I think that they're not willing to think outside the box in their understanding of these diseases.

*(Id. at 22).*

Earlier in his testimony, Dr. Parker was asked whether the opinion espoused by Dr. Koichi Honma, the Japanese pathologist who offered an opinion on the cause of Claimant's pulmonary fibrosis, that interstitial pulmonary fibrosis is caused by occupational dust exposure, is generally accepted in the scientific community that Dr. Parker practices. Dr. Parker responded:

I think it's slowly becoming more broadly accepted. I think the traditional dogma in occupational lung disease related to coal and silica dust exposed workers is that they develop coal macules and silicotic nodules, but it's clear that the lung has a limited number of ways to respond to insults from dust, whether it's an interstitial response or whether it's an obstructive airways response. And some people can respond with airflow obstruction, even without significant pathologic change on the radiograph or on the lung biopsy, and similarly the lung is capable of responding at the interstitial level with minimal evidence of traditional macules or silicotic nodules.

*(Id. at 14, 15).*

Dr. Parker testified that he had authored papers on coal worker's pneumoconiosis. He was asked to summarize his articles. He answered that their subject involved:

Both coal worker's pneumoconiosis and silica related lung changes tend to be upper zone rounded lung nodules, but other patterns have been seen and described which include lower zone irregular shadows on the chest radiograph.

(*Id.* at 15).

Based primarily on the reports of Drs. Doyle and Parker the West Virginia Workers' Compensation Commission awarded benefits to Claimant for partial permanent disability caused by occupational pneumoconiosis. Employer's appeal to the West Virginia Supreme Court of Appeals was denied.

In addition to Drs. Doyle and Parker, the physicians who treated Claimant after he left Dr. Renn's care in 1993 found his pulmonary disability to be caused by his coal dust exposure. Those physicians are Dr. Constance A. Jennings and Dr. Cecile Stephanie Rose, who treated Claimant at National Jewish Hospital in Denver, Colorado from 1993 to 1996, as well as Dr. James H. Dauber who treated Claimant at UPMC.

Dr. Jennings is Board-certified in internal medicine and pulmonary medicine. She is presently the Clinical Director of the Interstitial Lung Disease Laboratory at the National Jewish Center for Immunology and Respiratory Medicine in Denver, Colorado. She is also an Assistant Professor of Medicine, Pulmonary Division, University of Colorado Health Sciences Center, Denver, Colorado. (DX 45) She was a staff member in the pulmonary branch of NIH for two years working exclusively in interstitial lung disease. She has worked at National Jewish Center for Immunology and Respiratory Medicine since 1993, exclusively in interstitial lung disease. (DX 45; dep. of Jennings at 5)<sup>2</sup>. She testified that National Jewish is a research center and one of the most well-known centers for the studies of interstitial lung disease. (*Id.* at 7). She sees patients from all over the country that suffer from interstitial lung disease including some who have worked in and about coal mines. (*Id.* at 8). Dr. Jennings was Claimant's primary pulmonologist for the years that he treated at National Jewish. (*Id.* at 9). She developed and implemented a treatment regiment for Claimant. She also monitored the side effects of Claimant's medication. She diagnosed diffuse interstitial lung disease caused related to coal workers' pneumoconiosis. (DX 10). She recommended that Claimant avoid all exposure to coal dust and silica. (*Id.*). Dr. Jennings based her finding that the interstitial fibrosis was caused by coal dust in part on the histology report from the biopsy as Dr. Waldren, the consulting pathologist, observed silicate deposits, dust macules and small airways disease. (*Id.* at 20). She opined that silicates were probably the cause, as she noted that the histologic evidence showed evidence of silicotic deposition. (*Id.* at 22). Specifically, she reported that the histologic findings included abundant polarized silicates within the alveolar space out of proportion, silicate crystals implicating the presence of the silicates in the pathogenesis of the large number of inflammatory cells within the lung, and silicate deposits observed within the areas of honeycomb, strongly indicating silicates in the pathogenesis of the fibrotic process. (*Id.* at 28, 29). A second reason was Claimant's age. Dr. Jennings considered Claimant's age of 39 years to be way out of range of the age of a person treated for idiopathic pulmonary fibrosis, as it strikes people as they age, typically at about 66 years of age. She recalled that about 90 percent of her idiopathic pulmonary fibrosis patients are over 50 years of age, and although they

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<sup>2</sup> Dr. Jennings' deposition is EX 15 in the record before Judge Leland.

occasionally see someone in their 40s, they almost never see someone in their 30s. (*Id.* at 27). A third ground is the Claimant's history of extensive exposure to sandstone and coal dust in his drilling and long wall operation. (*Id.* at 28). Dr. Jennings testified that she sent the histologic slides to Dr. Honma in Japan because he has extensive experience in silicosis-induced lung disease. (*Id.* at 29). She reported that Dr. Honma was of the opinion that the biopsy was consistent with silica-diffuse interstitial lung disease. (*Id.*). Dr. Honma has authored a study looking at histologic abnormalities within the lungs of patients with nonasbestos pneumoconiosis and found patterns consistent with interstitial fibrosis in 25 per cent of those patients. (*Id.* at 24). Dr. Jennings also referenced studies published in the American Review of Respiratory Diseases about 1994 and in Archives of Environmental Health in 1990 of epidemiologic research suggesting that individuals with exposure to silicates and other dusts have a much higher frequency of developing interstitial pulmonary fibrosis, and cases of idiopathic etiology within a group of dust-exposed individuals implicating silicates in etiology of idiopathic pulmonary fibrosis. (*Id.* at 23, 24). She also noted that Dr. Churg and Dr. Vancouver show diffuse interstitial fibrosis with silicate exposure in slate miners. (*Id.* at 43). Dr. Jennings was asked whether it is generally accepted among physicians knowledgeable in this area that silicates can cause this type of reaction. She answered that, "Well, it's not the classical description but people are increasingly writing about the observance of usual interstitial pneumonia in patients with silicate exposure that is not compatible with chance." (*Id.* at 26). Dr. Jennings listed histologic findings on Claimant's lung tissue that are not typical of interstitial fibrosis: changes in upper lobe were more severe than in lower lobe whereas in IPF disease begins in lower lobes; the number of cells within the lung were four times that seen in patient with IPF who are non-smokers implicating irritants to the lung; and one never see silicates within areas of honeycombing in IPF.

Dr. Jennings also ruled out other diseases that can cause interstitial lung disease as she eliminated manifestation of collagen and vascular disease and rheumatoid arthritis. (*Id.* at 38). Dr. Jennings' testimony concluded that the diffuse interstitial fibrosis should be presumed to be secondary to silicate exposure because of the silicate deposition within areas of honeycombing together with the atypical features. (*Id.* at 50).

Dr. Jennings referred Claimant to Dr. Rose to evaluate his pulmonary condition considering his coal mine dust exposure, but Dr. Jennings continued as his primary pulmonologist. (*Id.* at 9). Dr. Rose's emphasis as a physician has been on interstitial lung disease arising from occupational origins whereas Dr. Jennings' studies have been with individuals who have interstitial lung disease both from occupational and non-occupational origins. (*Id.* at 10)

Dr. Rose saw Claimant for an initial clinical evaluation on October 28, 1993. Her deposition testimony was taken on July 6, 2011. (CX 1). Dr. Rose is most qualified to offer an opinion on the cause of Claimant's pulmonary condition. She is Board-certified in internal medicine, pulmonary medicine and occupational medicine. (CX 1 at 7). She is Professor of Medicine in the Division of Pulmonary and Critical Care at the University of Colorado, Professor of Medicine at National Jewish Health in the Department of Medicine's Division of Environmental and Occupational Health. She has a secondary academic appointment as Professor of Environmental and Occupational Health in the Colorado School of Public Health.

(CX 1 at 7). She is active and involved in the Environmental and Occupational Health Section at American Thoracic Society. She is involved in American College of Occupational and Environmental Medicine. (CX 1 at 8). She has published chapters in a number of textbooks on hypersensitivity pneumonitis and on occupational lung disease. (CX 1 at 9). She has presented a number of case reports on occupational causes of interstitial lung disease in a review article in Seminars in Respiratory and Critical Care Medicine. (CX 1 at 9). She has a clinical practice at National Jewish Hospital in Denver, Colorado that focuses on occupational and environmental lung disease. She treats patients with interstitial lung disease, including those with diffuse interstitial fibrosis. She also treats coal miners with coal workers' pneumoconiosis. (CX 1 at 5, 6). Her "practice has intensified its focus on the medical evaluation and management of miners, including coal miners." She sees on average ten miners a month at the clinic in Denver and sees between 35 and 120 miners at outreach clinics in distant rural areas of Colorado. (CX 16). She sees miners for evaluation of coal workers' pneumoconiosis on referral from the Department of Labor. (CX 17, 12).

Dr. Rose testified that studies since 1995 have increasingly linked UIP, usual interstitial pneumonia, pattern of IPF, idiopathic pulmonary fibrosis, to occupational and environmental exposures. (CX 1 at 15). The studies "seem to increasingly support the finding that in a minority of coal miners who develop lung disease that this form of diffuse interstitial fibrosis can be one of the manifestations of exposure to coal mine dust and subsequent lung disease." (CX 1 at 16). Dr. Rose disagreed with Dr. Renn's testimony that there is no data to support a link between coal mine dust exposures and diffuse interstitial fibrosis. (CX 1 at 17). She testified about the study, *Interstitial Fibrosis In Coal Workers – Experience in Wales and West Virginia*, by K. McConnochie, F.H.Y. Green, and V. Vallyathan et al. (1988). (McConnochie et al. study) which looked at Welsh coal miners and found that the prevalence of diffuse pulmonary fibrosis at autopsy was substantially higher than would be expected. (CX 1 at 17) The study's Welsh coal miner results were verified when the study reviewed findings of diffuse interstitial fibrosis in coal miners in Southern West Virginia, where the prevalence of diffuse interstitial fibrosis was 17% higher than would be expected in the general population who get interstitial fibrosis or what's termed clinically idiopathic fibrosis. (CX 1 at 18, 19). Dr. Rose testified that the McConnochie et al. study had particular relevance to Claimant's claim as it discovered that coal miners who developed interstitial fibrosis develop it at a statistically significantly younger age than patients with non-occupational related idiopathic pulmonary fibrosis, and that miners with severe interstitial fibrosis had a fairly benign clinical course, in that about two thirds of them survived for ten years, a much longer duration than for patients who have idiopathic pulmonary fibrosis. Thus, the age of onset and relatively benign course was different in coal miners compared to patients with IPF. (CX 1 at 22). Dr. Rose explained her finding that Claimant's interstitial fibrosis had a slow progression by pointing to the lung transplant not occurring until 13 years after exercise testing revealed significant decline in arterial oxygen tension. (CX 1 at 44, 45). Dr. Rose testified to other studies that are pertinent here as they show that although the classical form of coal workers' pneumoconiosis with upper lung zone predominant rounded opacities is the more common radiologic presentation, a substantial minority of coal miners may present with lower lobe irregular or linear interstitial opacities as occurs in patients such as Claimant who presents lower lobe predominant interstitial fibrosis on imaging. The studies were by Anne Cockcroft, published in the early the 90s, and another in British Journal of Industrial Medicine. (CX 1 at 22, 23). Dr. Rose also referenced *Idiopathic Pulmonary Fibrosis:*

*Epidemiologic Approaches to Occupational Exposure*, a study by Dr. Kazuro Iwai et al., in Japanese medical literature showing diffuse interstitial fibrosis in miners with mixed dust exposures. (CX 1 at 24). Dr. Rose testified that NIOSH as well as NIH, National Institute of Health, are increasingly recognizing diffuse interstitial fibrosis with irregular linear opacities and without classical findings of nodular opacities on the imaging is occurring in this country and elsewhere. She noted that the NIH's website states that coal dust exposure is associated with risk for IPF. (CX 1 at 25)

Dr. Rose was asked about Dr. W.K.C. Morgan's testimony before Judge Leland that a textbook by Dr. W. Raymond Parkes states that there is absolutely no relationship between idiopathic pulmonary fibrosis and coal dust exposure. Dr. Rose answered by pointing to a third edition of Dr. Parkes' textbook where there is a discussion of the McConnochie et al. study finding interstitial fibrosis at autopsy in around 16 to 18 % of Welsh and West Virginia coal miners. (CX 1 at 26, 27). She recalled that Dr. Parkes' text also discussed the clinical differences between miners and other patients with diffuse interstitial fibrosis, in that the miners have a much more benign clinical course and less intensively progressive and rapid downhill decline compared to the general population of people who suffer from IPF. (CX 1 at 28). Dr. Rose testified to being familiar with the Morgan and Seaton textbook on occupational lung diseases, 3d. edition, which describes the difference in clinical course of diffuse interstitial fibrosis in coal miners. (CX 1 at 29).

Dr. Rose identified records she reviewed prior to her deposition, including medical records of Claimant at National Jewish back to the early 90s, and pathology reports of Drs. Naeye and Crouch. (CX 1 at 29, 30). She concluded, based on those records and her treatment, that Claimant suffered from interstitial fibrosis that led to the lung transplant. (CX 1 at 30). She found the course of Claimant's disease to be consistent with the interstitial disease of coal miners described in the McConnochie et al. study. (CX 1 at 31). She interpreted the lung transplant pathology to show end-stage lung disease in a UIP pattern with findings of air-way-centered injury consistent with an inhalation exposure, and she opined, with a reasonable degree of probability, that the cause was Claimant's coal dust exposure. (CX 1 at 33). She also testified that the findings from the transplant pathology were consistent with findings from the 1992 biopsy pathology in that the 1992 biopsy showed interstitial lung disease in a UIP pattern, progressing to end-stage fibrotic lung disease with areas of pigmentation and some coal macules. (CX 1 at 33). Dr. Rose noted that the 1992 pathology report showed in addition to anthracotic dusts, macules containing abundant polarizable silicates. (CX 1 at 38).

Dr. Rose concluded with reasonable medical certainty that Claimant had a small airways injury due to his coal mine dust exposure that resulted in epithelium damage, and the loss of the integrity of the airway epithelium precipitated a fibroproliferative response that led to his diffuse interstitial fibrosis. "...our thinking based on recent studies of patients with pulmonary fibrosis, is that it's not due simply to inflammation but to epithelial damage that triggers this cascade of fibroproliferation and end-stage lung disease." (CX 1 at 35, 36).

In September of 1996, Claimant transferred his treatment to UPMC for evaluation of lung transplantation and to have his care closer to home. He saw Dr. James H. Dauber, who placed him on a lung transplant recipient list. Dr. Dauber saw him about every six months for treatment

until Dr. Dauber's retirement in 2006. Claimant was listed as a lung transplant recipient in 1996 but his condition leveled off and he was taken off the transplant list in 1998. His condition worsened in 2006. He underwent a single left lung transplant on July 3, 2006.

Dr. James H. Dauber testified by deposition on on April 21, 2011. (EX 12). Dr. Dauber is Board-certified in internal medicine, pulmonary disease and critical care medicine. He was an Assistant Professor of Medicine at the University of Pennsylvania from 1977 until 1982 when he was recruited to UPMC as Professor of Medicine, Director of the Pulmonary Transplant Program and the Medical Director of the Center for Interstitial Lung Disease. (EX 12, ex.1). He retired from active practice in 2006. His position with UPMC involved direct patient care, administrative and academic responsibilities. (EX 12 at 6). About forty per cent of his time was spent with patient care. In 2002 Dr. Dauber started an interstitial lung disease program at UPMC. He spent the last four years of his career building the program, involving diagnosis and management of interstitial lung disease. The largest emphasis of his publications was on lung transplantation. (EX 12 at 8).

Prior to his deposition testimony, Dr. Dauber reviewed the reports of Drs. Renn, Repsher, Spagnolo, Rosenberg and Tuteur. (EX 12 at 9).

Dr. Dauber issued a report on Claimant's condition in 2004. At that time he was treating Claimant, as he had been seeing him every six months since 1996. His review of the transplant records as well as of the pathology of the lung tissue showed usual interstitial pneumonia, dust deposition and anthracosilicotic nodules. It confirmed his clinical impressions. (EX 12 at 12, 13). Dr. Dauber diagnosed pulmonary fibrosis, and considered the presence of idiopathic pulmonary fibrosis to be very unlikely. (EX 12 at 13, 14). He rejected the diagnosis of idiopathic pulmonary fibrosis because it is a disease that strikes older people, rarely before the age of 50, whereas the onset of Claimant disease was when he was in his mid-30s. (EX 12 at 14, 26). Also, he considered Claimant's duration of survival to be exceedingly unusual for idiopathic pulmonary fibrosis, as the median survival is only three years, whereas he was followed for 16 to 18 years before the lung transplant. (EX 12 at 15). Further, the course of Claimant's disease has not shown the typical acute exacerbations of idiopathic pulmonary fibrosis, as it is not unusual for the idiopathic pulmonary fibrosis patients to experience a sudden worsening in their symptoms, which is frequently associated with changes on x-ray or CT scan showing a lot more infiltrates, suggesting an ongoing very active process. Claimant did not show such exacerbations before his transplant. (EX 12 at 15, 26). Lastly, at times there is a family history of the disease, but no history in Claimant's family. (EX 12 at 16).

Dr. Dauber testified that the pathology of the lung was also convincing that coal dust was the genesis of the process that led to the usual interstitial pneumonia, particularly the presence anthracosilicotic nodules. (EX 12 at 18). He considered that the coal dust was not benign or Claimant would not have had nodules forming, as nodules, particularly when forming, have an inflammatory response with release of mediators that can affect other processes in lung. (EX 12 at 19). He testified that the pathology from the transplant showed multiple hilar lymph nodes with focally calcified anthracosilicotic nodules, meaning that the regional lymph nodes were anthracotic due to the fact that the coal particles will transport through the lymphatics, through the local lymph nodes, and stay there, and that typically, in idiopathic interstitial pneumonia,

there is no enlarged lymph nodes. (EX 12 at 28). Dr. Dauber reasoned that “[Claimant] does have usual interstitial pneumonia, and since he was exposed to coal dust sufficiently to produce silicotic nodules and coal nodules in his lung, its clear that he got a lot of dust in the lung at the time that this other disease was developing.” (EX 12 at 27).

Dr. Dauber testified that the American Thoracic Society teaches that idiopathic interstitial fibrosis is the correct diagnosis only when no other cause for the UIP can be determined after an extensive and thorough evaluation. (EX 12 at 31). Dr. Dauber found the McConnochie et al. study convincing in helping to make a causal relationship between dust exposure and UIP. (EX 12 at 41).

These physicians who evaluated Claimant’s condition for his state claim or treated Claimant all opined that his pulmonary condition was caused by his coal dust exposure. In contrast, Employer offered consulting expert opinions from physicians to show that Claimant’s pulmonary disability is not caused by coal dust. Those physicians are Dr. David Rosenberg, Dr. Samuel Spagnolo, Dr. Peter Tuteur and Dr. Lawrence Repsher, as well as a report from Dr. Joseph Renn, who treated Claimant for about a year and a half and recommended that Claimant contact National Jewish for treatment. Also, when the claim was before Judge Leland, Employer offered the consulting reports of Drs. Jerome Kleinerman, W.K.C. Morgan, Dr. Gregory Fino and Dr. Spagnolo.

Dr. Renn reported on the cause of Claimant’s pulmonary condition in a February 17, 2006 medical report. (EX 2). The report includes a physical examination, review of medical and occupational histories, and treatment records supplied by Employer. Dr. Renn diagnosed UIP, simple pneumoconiosis, and moderately severe restrictive ventilatory defect owing to the UIP. He reported that the coal workers’ pneumoconiosis was very mild by pathology standards. Dr. Renn testified by deposition on April 11, 2011. (EX 11). Dr. Renn is Board-certified in internal medicine and pulmonary medicine. (EX 11 at 5). He is a full clinical professor at West Virginia University Medical Center. (*Id.*) He ceased the active treatment of patients in January of 2003, at which time he was seeing individuals referred to him by law firms and agencies, including the Office of Workers’ Compensation Programs. (EX 11 at 4). He keeps current with pulmonary medicine issues by regularly reading the American Journal of Respiratory and Critical Care Medicine. (EX 11 at 5). He examined Claimant on occasions as his treating physician and examined him on two other occasions at request of Employer. (EX 11 at 7). He last saw Claimant in 2006 for a disability evaluation at request of Employer. (EX 11 at 8). He diagnosed Claimant with usual interstitial pneumonitis in the 1990s after being suspicious of interstitial pulmonary fibrosis. (EX 11 at 13). The pathology report of the tissue from Claimant’s lung transplant showed usual interstitial pneumonia and multiple hilar lymph nodes with focally calcified anthracosilicotic nodules. (EX 11 at 18). He testified that the diagnosis of the pathology report is not one of coal workers’ pneumoconiosis, and in fact, the pathology report did not mention coal workers’ pneumoconiosis. (EX 11 at 19, 21). Dr. Renn found Claimant to be totally disabled from a restrictive impairment caused by usual interstitial pneumonitis pattern of interstitial pulmonary fibrosis. (EX 11 at 25). Dr. Renn testified that his reading of the medical literature, including the statement on idiopathic pulmonary fibrosis by the American Thoracic Society in 2000, shows no suggestion that idiopathic pulmonary fibrosis is caused by coal dust exposure. (EX 11 at 26, 29).

Dr. Rosenberg issued a report on June 13, 2006, and testified by deposition on July 15, 2011. (EX. 5; EX 17). Dr. Rosenberg concluded with reasonable degree of medical certainty, after reviewing Complainant's extensive file that Complainant has a minimal degree of pathologic involvement with simple pneumoconiosis, but his overwhelming disease entity is UIP, the etiology of which is unrelated to and has not been aggravated by coal mine dust exposure. Dr. Rosenberg testified that he is Board-certified in internal medicine, pulmonary disease and occupational medicine. (EX 17 at 3). He had a fellowship at National Institute of Health where his research was directly in area of idiopathic pulmonary fibrosis, his main interest during the three years he was there. (EX 17 at 4). He is an Assistant Clinical Professor at Case Western Reserve University School of Medicine in Cleveland. (EX 17 at 6). He sees about 75 to 100 patients a week, predominantly in the area of pulmonary disease and occupational medicine. (EX 17 at 6). He has performed disability evaluations of several thousand coal miners. (EX 17 at 7).

Dr. Rosenberg considers Claimant's condition to be consistent with IPF even though the median survival time for IPF is three to five years because it is consistent with the overall general pattern of IPF. (EX 17 at 18). Dr. Rosenberg's review of the pathology from the explanted lung showed pattern of UIP, consisting of end-stage fibrosis, along with some silica depositions a few macules. (*Id.*).

Dr. Rosenberg offered the opinion that coal mine dust exposure does not cause the condition of idiopathic pulmonary fibrosis suffered by Claimant. (EX 17 at 20). He explained that coal workers pneumoconiosis begins as coal macules, evolves into micronodules, macronodules and then progressive massive fibrosis, whereas idiopathic pulmonary fibrosis is diffuse fibrosis that spreads out and forms honeycombing and destruction of capillary bed. (*Id.*). Dr. Rosenberg testified that research done over the years revealed no evidence that coal dust causes the pathologic findings and clinical correlation of IPF. (EX 17 at 20, 21). He testified that the American Thoracic Society issued a statement in 2000 about the diagnosis and treatment of idiopathic pulmonary diagnosis which listed potential causative factors but not coal dust exposure. (EX 17 at 21). Dr. Rosenberg reported that the ATS statement provides that there is usually a three to five year survival period with IPF, but that the disease course is variable, and some patients do live longer. (EX 17 at 22, 23).

Dr. Rosenberg testified about the McConnochie et al. study. He explained that it shows through autopsies of coal miners that miners suffered from IPF at a greater rate than the general population, as around 15% of miners had changes consistent with IPF as seen by pathologic tissue. (EX 17 at 24, 25). Dr. Rosenberg testified that generalized statements on coal miners and IPF cannot be made as these samples were "selected pathology specimens," not randomly selected. (*Id.*). Dr. Rosenberg quoted the McConnochie et al. study as stating, "[t]here is no doubt that interstitial pulmonary fibrosis occurs in coal miners but it is unknown whether this is related to coal dust inhalation." (EX 17 at 25). Dr. Rosenberg considered the value of the McConnochie et al. study to be raising an issue, but that more information must be gathered over time to prove the hypothesis raised. (EX 17 at 26). Dr. Rosenberg was asked about an article by Eduard Monzo, *Mineralogical Microanalysis of Idiopathic Pulmonary Fibrosis*. He explained that the article found that certain patients with IPF had increased silica content in lungs, but he considered it to have nothing to do with coal miners. (EX 17 at 27). He also testified that

studies, *Diffuse Fibrosis in Nonasbestos Pneumoconiosis-a Pathological Study*, by Koichi Honma, and *Idiopathic Pulmonary Fibrosis: Epidemiologic Approaches to Occupational Exposure*, by Dr. Kazuro Iwai et al. looked at patients with IPF and their exposures to see if there was a correlation, and found possibly an association, but that the studies had no applicability to coal mining. (EX 17 at 28). Dr. Rosenberg was questioned about the article *Lung Disease Caused by Exposure to Coal Mine and Silica Dust* by Drs. Cohen, Patel and Green. He disagreed with the article's findings that diffuse interstitial fibrosis is a recognized type of pneumoconiosis following exposure to coal dust, silica, and dusts containing a mixture of minerals. He reasons that the articles conclusions are based on the McConnochie et al. study, which he does not consider as evidence that IPF is coal mine dust related. (EX 17 at 49-54).

Dr. Rosenberg testified to a 2011 update by ATS on idiopathic pulmonary fibrosis, dated March 15, 2011. (EX 17 at 55). He testified that the update changed some of the recommendations for diagnosis including diagnosis of patients younger than age 50 at time of presentation. He quoted it as stating that patients with IPF under 50 years of age are rare. (EX 17 at 57). He noted that the diagnostic criteria was changed to stress that accuracy depends on a multidisciplinary approach and that domestic and occupational, environmental exposures, connective tissue diseases and drug toxicity must be considered. (EX 17 at 58).

Dr. Rosenberg testified about an article from *Chest*, the official journal of the American College of Chest Physicians, *Chronic Interstitial Pneumonia and Silicosis and Mixed-Dust Pneumoconiosis*. (EX 17 at 67). He testified that the article concluded that the "prevalence of chronic interstitial pneumonia in pneumoconiosis was approximately 12 percent on CT scan." (EX 17 at 72). Rosenberg acknowledged that the article states that chronic interstitial pneumonia in the lower lobes has been reported from 15.9 to 19.3 percent in autopsy cases of silicosis and coal workers' pneumoconiosis, although the dose-response relationships has not been established, and it reported that subjects engaged in some specific dusty occupations showed increased risk of idiopathic pulmonary fibrosis with an odds ratio ranging from 1.34 to 12.55. He quoted the article as concluding: "From these previous reports, it may be that chronic interstitial pneumonia has a pathogenic linkage with occupational exposure to some specific dusts, although prevalence seems much higher in the prevalence of nodular fibrosis (silicosis and mixed-dust pneumoconiosis.)" (EX 17 at 73).

Dr. Rosenberg concluded that Claimant has coal workers' pneumoconiosis but it does not cause any pulmonary impairment, because its extent is minimal. He agreed that Claimant had coal macules, but he reasoned that macules do not destroy lung tissue and do not cause gas exchange abnormalities. He offered the opinion that Claimant's macules had not progressed as he saw no change from the biopsy in 1992 to later when he had the lung transplant. (EX 17 at 30).

Dr. Spagnolo authored a report on June 4, 2006, and testified by deposition on April 26, 2011. (EX 3; EX 13). Dr. Spagnolo's report was a supplemental report as he had previously evaluated Claimant's pulmonary condition in a report dated October 14, 1995. This supplemental report was prepared in light of his review of a number of additional documents. He concluded from his review that Claimant suffers from a restrictive respiratory impairment caused solely by progression of his previously diagnosed idiopathic interstitial pulmonary

fibrosis. He opined that coal workers' pneumoconiosis was present but insufficient to have resulted in any respiratory impairment. (EX 3). Dr. Spagnolo is Board-certified in internal medicine and pulmonary medicine. (EX 6). He is a Professor of Medicine at George Washington School of Medicine and Health Services, Washington D.C. His current practice consists of seeing patients for pulmonary care, teaching, and presenting conferences. (EX 13 at 7, 8). He previously ran the pulmonary division of George Washington and he was on the editorial board of "Chest," the official journal of the American College of Chest Physicians from 2002 to 2006. (EX 13 at 8; EX 6). He testified to having an interest in interstitial disease over the years, and that he edited a book on interstitial disease. (EX 13 at 8).

Dr. Spagnolo testified that the histologic report from the lung transplant showed no description of coal dust-induced lung disease, as it did not describe coal dust macules adjacent to changes of interstitial pneumonia. (EX 13 at 15). Dr. Spagnolo testified that the focally calcified anthracosilicotic nodules in the multiple hilar lymph nodes can't be used to make a diagnosis of CWP as "[y]ou have got to see the CWP in the lung." (EX 13 at 16). He opined that the changes in lung tissue that are caused by coal dust are macular changes, forming nodular areas that lead to fibrosis, a scarring reaction, although not in every patient. (EX 13 at 18). Dr. Spagnolo believes Claimant's disease is a form of interstitial fibrosis related to hypersensitivity disease. (EX 13 at 24, 40). Dr. Spagnolo opined that the condition is not caused by coal dust because the biopsy did not show any remnant of coal dust macules. (EX 13 at 25). Dr. Spagnolo does not believe there is any established evidence of a relationship between coal dust exposure and interstitial fibrosis. (*Id.*). Dr. Spagnolo testified that the October 1992 biopsy showed a marked increase in lavage fluid from what is usually seen in idiopathic pulmonary fibrosis. He considered this to be an indication of a tremendous inflammatory reactive process. (EX 13 at 35).

Dr. Peter G. Tuteur authored a report evaluating Claimant's pulmonary condition at the request of Employer on March 21, 2011. (EX 9). The report considered medical records, pathology reports and deposition testimony provided to Dr. Tuteur by Employer. Dr. Tuteur is Board-certified in internal medicine and pulmonary medicine. He is presently Associate Professor of Medicine and Director of the Pulmonary Function Laboratory, Washington University School of Medicine, St. Louis, Missouri. (EX 10). He concluded that Claimant is totally disabled due to idiopathic usual interstitial pneumonitis, UIP, and that a specific etiological diagnosis cannot be ascribed to the UIP. He also diagnosed pneumoconiosis of insufficient severity and profusion to produce clinical symptoms or impairment of pulmonary function. Dr. Tuteur reported that UIP is a quite rare disease entity in general pulmonary practice. His understanding of the medical literature "suggests that it may be possible that the inhalation of coal mine dust may induce a UIP picture though this association is speculative and unconfirmed." Dr. Tuteur found Claimant's condition to be characterized by a slow development of progressive restrictive abnormality eventually leading to a requirement for supplemental oxygen. He noted that it is unusual for any form of UIP to have slow progression over nearly two decades but not unheard of, particularly if it is due to a post-inflammatory process. Dr. Tuteur testified that a reason for not considering the UIP to be caused by coal dust is that the UIP progressed to a restrictive abnormality, whereas the simple pneumoconiosis did not progress from 1992 to the autopsy, and both would have been expected to progress equally.

Dr. Repsher issued a report on June 7, 2006, after reviewing some of Claimant's medical records dating back to 1990 and 1991, as well as reports from Drs. Abrams, Renn, Jennings, Rose, Parker and Doyle. (EX 4). His impression was very mild simple coal workers pneumoconiosis of no clinical significance, very mild silicosis of no clinical significance and clinically significant UIP/IPF. Dr. Repsher concludes that Claimant does not suffer from either clinical or medical pneumoconiosis. He asserts that nothing in the published medical literature documents that exposure to coal mine dust can cause UIP/IPF. He disagreed with the opposite opinions of Drs. Jennings, Rose, Dauber, Parker, Davabhaktuni and Doyle because he found that their reports were not supported by specific citations or inductive logical reasoning.

Reports offered by Employer while the case was before Judge Leland were by Drs. Jerome Kleinerman, W.K.C. Morgan, Dr. Gregory Fino and Dr. Spagnolo.

Dr. Kleinerman issued a report on March 8, 1995, after reviewing the biopsy slides and other medical records. He testified by deposition on November 15, 1995. (DX 43; dep. of Kleinerman). Dr. Kleinerman is Board-certified in anatomic and clinical pathology. Dr. Kleinerman retired from the Director of the Department of Pathology at Case Western Reserve University in 1995. (dep. of Kleinerman at 5). He was involved in the early days of establishing the criteria for the federal black lung disability act. He chaired the committee that produced pathologic criteria for judging coal workers pneumoconiosis. (Id. at 7).

His review of the 1992 biopsy slides showed a minimal amount of simple pneumoconiosis but no evidence of nodular silicosis, complicated pneumoconiosis or conglomerate silicosis. He observed diffuse interstitial fibrosis which he opined is not caused by coal dust exposure, or developed from coal dust exposure. (Id. at 18). Dr. Kleinerman reviewed three articles referenced by Drs. Rose and Jennings; *Diffuse Interstitial Fibrosis In Nonasbestos Pneumoconiosis – A Pathological Study* by Dr. Honma; *Idiopathic Pulmonary Fibrosis*, by Dr. Iwai; and *Mineralogical Microanalysis of Idiopathic Pulmonary Fibrosis*, by Dr. Monso. He testified that the articles were not convincing that interstitial fibrosis can be caused, complicated or induced by coal mine dust exposure. He offered the opinion that the articles are preliminary, representing relatively small samples and do not identify a specific agent as part of the occupational exposure. (Id. at 42). Although he agreed that each of the articles were accepted for publication as peer reviewed articles, he considered them to be premature, unwarranted and not carrying supportive weight. (Id. at 43, 60).

Dr. W.K.C. Morgan was deposed on June 4, 1995. Dr. Morgan was a Professor of Medicine at Western Ontario University in 1995. His area of instruction was primarily chest diseases. (DX 46; EX 16 at 5) He has published studies on coal workers' pneumoconiosis including one with Dr. Lapp in 1975 looking at all aspect of the disease. (Id. at 9; EX 2). He authored a report dated July 23, 1995, on Claimant's pulmonary condition after a review of Claimant's medical records. Prior to his deposition he reviewed the report of Dr. Fino and the depositions of Drs. Jennings, Rose, Renn, Kleinerman and Devabhaktuni. (Id. at 15). Dr. Morgan testified that he does not believe that there is an association between Claimant's coal dust exposure and his interstitial pulmonary fibrosis. (Id. at 59, 72). He authored a study of coal miners with Dr. Lapp. The issue of association of coal mine dust and interstitial fibrosis was not considered by that study, but all the data was there and he believes that an excessive number of

interstitial pulmonary fibrosis cases would have been noticed. (*Id.* at 59). He did not recall that any of the 9,000 miners in the study had interstitial fibrosis. (*Id.*). Dr. Morgan reviewed the three articles referenced by Drs. Rose and Jennings, the studies by Dr. Honma, Dr. Iwai, and by Dr. Monso. He did not find their results to be convincing, as he considered them to be epidemiologically unsatisfactory. (*Id.* at 62 – 68). Dr. Morgan acknowledged that the three articles were probably peer reviewed, but he offered the opinion that not all peer review articles are correct. (*Id.* at 70).

Dr. Gregory Fino testified by deposition on February 19, 1997, when this claim was before Judge Leland. (DX 47; (dep. of Fino at 17)<sup>3</sup>. Dr. Fino is Board-certified in internal medicine and pulmonary medicine. (*Id.* at 4). He examined Claimant in August 1995 as part of a pulmonary evaluation at the request of Employer. His deposition was taken after he reviewed Claimant's medical records from Jewish National Hospital; a medical report by Dr. Spagnolo; deposition testimony of Drs. Jennings, Rose, Morgan, Kleinerman and Devabhaktuni; and pathology reports from Drs. Naeye and Kleinerman. (*Id.* at 9). He also reviewed chest x-ray readings including an August 24, 1994 x-ray showing irregular opacities and an abnormality in lower zones consistent with a diffuse interstitial pulmonary fibrosis. (*Id.* at 10). He considered the histologic report of the 1992 biopsy showing coal workers pneumoconiosis in the upper and middle zones. (*Id.* at 12, 13). Dr. Fino testified that he has not seen a relationship between coal dust exposure and diffuse interstitial fibrosis in his practice, and he is not aware of any evidence to link coal dust exposure to IPF. (*Id.* at 16, 43). He mentioned textbooks by Drs. Churg and Green, by Morgan and Seaton, and by Dr. Parke as authoritative on diffuse interstitial fibrosis. (*Id.* at 44). Dr. Fino offered his view of the three articles referenced by Dr. Rose and Dr. Jennings when the case was before Judge Leland: *Diffuse Interstitial Fibrosis In Nonasbestos Pneumoconiosis – A Pathological Study* by Dr. Iwai; *Idiopathic Pulmonary Fibrosis*, by Dr. Honma; and *Mineralogical Microanalysis of Idiopathic Pulmonary Fibrosis* by Dr. Monso. Dr. Fino characterized the study by Dr. Iwai as a look at 1311 cases of idiopathic pulmonary fibrosis by autopsy results to determine if the disease could be associated with an occupation, and the study by Dr. Monzo as a look to see if there was an increase in silica exposure with fibrosis. He interpreted the studies' conclusions as not showing an association between IPF and occupation exposure. (*Id.* at 47). Dr. Fino also found that the study by Dr. Honma did not support a hypothesis that there is an increased incidence of pulmonary fibrosis as a result of coal workers' pneumoconiosis, and he asserted that the Dr. Honma article inaccurately referenced a passage in a textbook by Dr. Churg and Dr. F.H.Y. Green, *Occupational Pulmonary Pathology*, in that the article indicates that Dr. Green's textbook describes fibrosis and honeycombing associated with coal worker's pneumoconiosis, even though Dr. Green's textbook only discusses articles reviewing the association, it does not conclude that coal workers' pneumoconiosis causes the diffuse interstitial pneumoconiosis. Dr. Fino continued that Dr. Green's textbook states that medical information is insufficient to reach the conclusion that diffuse interstitial pneumoconiosis can be caused by coal dust exposure. (*Id.* at 49, 50). Dr. Fino testified further that he did not see any article from OSHA that found an increased risk of an IPF condition from coal dust exposure. (*Id.* at 50).

Dr. Fino reports that the 1992 biopsy report shows that the more serious fibrosis was located in the upper lung zones which, according to Dr. Fino, is atypical as IPF typically

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<sup>3</sup> EX 17 in the record before Judge Leland.

manifests itself initially in the middle lobe zones. Nevertheless, Dr. Fino did not consider that this indicates that the condition in the upper lung zone was not IPF, because the middle lobe notoriously either underestimates or overestimates lung disease. (*Id.* at 66).

After considering the opinions of all the physicians, it is determined that Claimant has met his burden of showing that his diffuse interstitial disease which necessitated a lung transplant was caused by his coal dust exposure. Dr. Jack Parker reported that the state of medical knowledge on the cause of diffuse interstitial pulmonary fibrosis is changing to show that its causes can be environmental. His opinion is consistent with the findings of Claimant's treating physicians, Dr. Jennings and Dr. Rose at National Jewish Hospital, and Dr. Dauber who treated Claimant at UPMC.

Drs. Jennings, Rose and Dauber are not only Claimant's treating physicians but have most impressive backgrounds in the treatment, study, and publication of articles involving interstitial pulmonary diseases and occupational exposure to coal mine dust. Dr. Jennings is presently the Clinical Director of the Interstitial Lung Disease Laboratory at the National Jewish Center for Immunology and Respiratory Medicine in Denver, Colorado. She is also an Assistant Professor of Medicine, Pulmonary Division, University of Colorado Health Sciences Center, Denver, Colorado. She was a staff member in the pulmonary branch of NIH for two years working exclusively in interstitial lung disease. She has worked at National Jewish Center for Immunology and Respiratory Medicine since 1993, exclusively in interstitial lung disease. She testified that National Jewish Hospital is a research center and one of the most well-known centers for the studies of interstitial lung disease. She sees patients from all over the country who suffer from interstitial lung disease including some who have worked in and about coal mines. Dr. Rose is a Professor of Medicine in the Division of Pulmonary and Critical Care at the University of Colorado, Professor of Medicine at National Jewish Health in the Department of Medicine's Division of Environmental and Occupational Health. She has a secondary academic appointment as Professor of Environmental and Occupational Health in the Colorado School of Public Health. She is active and involved in the Environmental and Occupational Health Section at American Thoracic Society. She is involved in American College of Occupational and Environmental Medicine. She has published chapters in a number of textbooks on hypersensitivity pneumonitis and on occupational lung disease. She has presented a number of case reports on occupational causes of interstitial lung disease in a review article in *Seminars in Respiratory and Critical Care Medicine*. She has a clinical practice at National Jewish Hospital in Denver, Colorado that is focused on occupational and environmental lung disease. She treats patients with interstitial lung disease, including those with diffuse interstitial fibrosis. She also treats coal miners with coal workers' pneumoconiosis. Her "practice has intensified its focus on the medical evaluation and management of miners, including coal miners." She sees on average ten miners a month at the clinic in Denver and sees between 35 and 120 miners at outreach clinics in distant rural areas of Colorado.

Dr. Dauber was a Professor of Medicine, Director of the Pulmonary Transplant Program and the Medical Director of the Center for Interstitial Lung Disease UPMC, before he retired from active practice in 2006. His position with UPMC involved starting up an interstitial lung disease program at UPMC. He spent the last four years of his career building the program, involving diagnosis and management of interstitial lung disease.

Drs. Jennings, Rose and Dauber compared Claimant's pulmonary disease symptoms and disease progression to studies of pulmonary fibrosis and found them to be atypical of Idiopathic Pulmonary Fibrosis but typical of fibrosis caused by coal dust. Initially, Drs. Dauber, Rose and Jennings all noted that the onset of Claimant's interstitial fibrosis was atypical as it commenced when he was in his mid-30s, whereas, according to Dr. Dauber, idiopathic pulmonary fibrosis is a disease that strikes older people, rarely before the age of 50. Their finding on the relevance of the onset of Claimant's interstitial fibrosis is consistent with studies of interstitial fibrosis in coal miners referenced here. Dr. Rose testified that an article by Drs. K. McConnochie, F.H.Y. Green, and V. Vallyathan et al., *Interstitial Fibrosis In Coal Workers – Experience in Wales and West Virginia*, has particular relevance to Claimant's case as the article showed that coal miners who developed interstitial fibrosis developed it at a statistically significantly younger age than what is reported as the mean age of diagnosis for people with non-occupational related idiopathic pulmonary fibrosis. (CX 1 at 21).

Second, the progression of Claimant's disease has not shown the typical acute exacerbation of idiopathic pulmonary fibrosis as Claimant's duration of survival is exceedingly unusual for idiopathic pulmonary fibrosis in that Claimant was treated for 16 to 18 years before the lung transplant, whereas median survival is only three years. Dr. Rose testified that a major finding of the study by McConnochie et al. is that coal miners with severe interstitial fibrosis had a fairly benign clinical course, that is, about two thirds of them survived for ten years, a significantly longer duration than for patients who have idiopathic pulmonary fibrosis. Also, a major finding of the article by Drs. Cohen, Patel and Green, *Lung Diseases Caused By Exposure To Coal Mine And Silica*, is that miners with severe interstitial fibrosis have a fairly benign clinical course, that is, about two thirds of them survived for ten years, significantly longer than for patients who have idiopathic pulmonary fibrosis. Thus, the studies by Dr. McConnochie et al. and by Dr. Cohen et al. show that age of onset and a relatively benign course are different in coal miners compared to patients with IPF.

Third, the physicians' interpretation of the pathology is consistent with the interstitial fibrosis being caused by coal dust exposure. Dr. Dauber's 2004 report stated that his review of the transplant records as well of the pathology of the lung tissue showed usual interstitial pneumonia, and showed dust deposition and anthracosilicotic nodules, confirming his clinical impression of pulmonary fibrosis caused by coal dust. Dr. Dauber testified:

He does have usual interstitial pneumonia, and since he was exposed to coal dust sufficiently to produce silicotic nodules and coal nodules in his lung, its clear that he got a lot of dust in the lung at the time that this other disease was developing.

(CX 12 at 27).

Dr. Dauber explained that the pathology from the transplant showed multiple hilar lymph nodes with focally calcified anthracosilicotic nodules, meaning that the regional lymph nodes were anthracotic due to the fact that the coal particles will transport through the lymphatics, then through the local lymph nodes and stay there. Typically, in idiopathic interstitial pneumonia, there is no enlarged lymph nodes. (EX 12 at 28).

Dr. Jennings testified that she based her finding that the interstitial fibrosis was caused by coal dust in part on the histology report from the biopsy as Dr. Waldren, the consulting pathologist, observed silicate deposits, dust macules and small airways disease. (DX 45; dep. of Jennings at 20). She opined that silicates were probably the cause, as she noted that the histologic evidence showed silicotic deposition. (*Id.* at 22). Specifically, she reported that the histologic findings included abundant polarized silicates within the alveolar space out of proportion, silicate crystals implicating the presence of the silicates in the pathogenesis of the large number of inflammatory cells within the lung, and silicate deposits observed within the areas of honeycomb, strongly indicating silicates in the pathogenesis of the fibrotic process. (*Id.* at 28). Dr. Jennings testified to the histologic findings that he found not to be typical of interstitial fibrosis: 1) changes in upper lobe were more severe than in lower lobe whereas in IPF, the disease begins in lower lobes; 2) number of cells within the lung were four times the number seen in patient with IPF who are non-smokers implicating irritants to the lung; 3) one never see silicates within areas of honeycombing in IPF.

Dr. Jennings sent the histologic slides to Dr. Honma in Japan because he has extensive experience in silicosis-induced lung disease. Dr. Honma reported finding silicate mineral mixed with the carbonaceous dust, and he was of the opinion that the biopsy was consistent with silica-diffuse interstitial lung disease. (*Id.* at 24).

Dr. Rose found the transplant pathology to be consistent with findings from the 1992 biopsy pathology in that it showed interstitial lung disease in a UIP pattern progressing to end-stage fibrotic lung disease with areas of pigmentation and some coal macules. She concluded that the lung transplant pathology showed end-stage lung disease in a UIP pattern with findings of air-way-centered injury that were consistent with an inhalation exposure, which she opined with a reasonable degree of probability was Claimant's coal dust exposure. (CX 1 at 33).

These opinions on causation by Drs. Parker, Jennings, Rose and Dauber are credited over the physicians consulted by Employer, that is, the physicians who evaluated Claimant's pulmonary condition in this modification proceeding, Drs. Renn, Rosenberg, Spagnolo and Tuteur, as well as those physicians, Drs. Morgan, Fino, Repsher, Kleinerman, and Spagnolo, who reported on the cause of Claimant's pulmonary condition when the case was before Judge Leland.

Dr. Renn testified that his reading of the medical literature including the statement on idiopathic pulmonary fibrosis by the American Thoracic Society in 2000 showed no support for a finding that idiopathic pulmonary fibrosis is caused by coal dust exposure. (EX 11 at 26, 29). Dr. Rosenberg rejected the idea that coal dust could cause diffuse interstitial pulmonary fibrosis. He described the pathology of coal workers pneumoconiosis as beginning as coal macules, evolving into micronodules, then into macronodules and then potentially progressive massive fibrosis. He contrasted this pathology with that of idiopathic pulmonary fibrosis being a diffuse fibrosis that spreads out and forms honeycombing and destruction of capillary bed. (EX 17 at 20). He testified that the research done over the years shows no evidence that coal dust causes the pathologic findings and clinical correlation of IPF. (EX 17 at 20, 21). He noted that the American Thoracic Society issued a statement in 2000 about diagnosis and treatment of

idiopathic pulmonary diagnosis, wherein it listed potential causative factors but coal dust exposure was not one of those listed. (EX 17 at 21).

Dr. Rosenberg does not deny that the McConnochie et al. study shows that autopsies of the coal miners it studied saw IPF at a greater rate than in the general population, as the study found that around 15% of the miners had changes consistent with IPF as seen by pathologic tissue. However, Dr. Rosenberg questions whether generalized statements on the association of coal miners and IPF can be made as the study's samples were "selected pathology specimens," not randomly selected. (EX 17 at 25). Dr. Rosenberg quoted the article as stating, "there is no doubt that interstitial pulmonary fibrosis occurs in coal miners but it is unknown whether this is related to coal dust inhalation." (*Id.*) Dr. Rosenberg considers the value of the McConnochie et al. study to be raising an issue, but that more information must be gathered over time to prove the hypothesis it raised. (EX 17 at 26). Dr. Rosenberg also rejects the findings by Drs. Cohen, Patel and Green in *Lung Disease Caused by Exposure to Coal Mine and Silica Dust*, because the article is based on the McConnochie et al. study. (EX 17 at 50-54).

Dr. Rosenberg acknowledged that the duration of Claimant's condition is atypical since the ATS provides that IPF usually has only a three to five year survival period. Dr. Rosenberg interpreted a 2011 update by ATS on idiopathic pulmonary fibrosis, dated March 15, 2011, that changed some of the recommendations for diagnosis including diagnosis of patients younger than age 50 at time of presentation, as showing that patients with IPF under 50 years of age are rare. (EX 17 at 55, 57). The update changed the diagnostic criteria to state that accuracy depends on multidisciplinary approach, and that domestic and occupational, environmental exposures, connective tissue diseases and drug toxicity must be considered. (EX 17 at 58). Nevertheless, the ATS did not affect Dr. Rosenberg's opinion as he responds that the disease course is variable, and some patients do live longer. (EX 17 at 22, 23).

Dr. Spagnolo testified that Claimant's pulmonary disease was not caused by coal dust because the 1992 biopsy and the histologic report of the lung transplant did not evidence it, as the biopsy did not show any remnant of coal dust macules in association with fibrosis, and the histologic report of the lung sections described no coal dust macules adjacent to interstitial pneumonia. (EX 3 at 15, 25). Dr. Spagnolo also considered that granulomas were observed and granulomas are not seen in coal workers pneumoconiosis. (EX 3 at 15). Dr. Spagnolo noted the existence of focally calcified anthracosilicotic nodules in the multiple hilar lymph nodes, but opined that they can't be used to make diagnosis of CWP. "You have got to see the CWP in the lung." (EX 3 at 16). Dr. Spagnolo finds the evidence of a relationship between coal dust exposure and interstitial fibrosis to be speculative.

Dr. Tuteur diagnosed two different histologic processes: very early mild and physiologically insignificant simple pneumoconiosis in the upper lobes; and a process predominantly in the lower lobes of greater severity and clinical significance, and characteristic of usual interstitial pneumonitis, UIP. Dr. Tuteur rejected coal dust exposure as the cause of the UIP. He offered that the medical literature "suggests that it may be possible that the inhalation of coal mine dust may induce a UIP picture though this association is speculative and unconfirmed." He contends that "the coal mine dust induction of UIP is not rigorously scientifically proven, nor has it been studied in a systematic way."

The physicians whose reports Employer offered before Judge Leland all diagnosed diffuse interstitial fibrosis as the cause of Claimant's pulmonary condition and they all opined that it was not caused by coal dust exposure. The physicians, Drs. Kleinerman, Morgan, and Fino, rejected coal dust exposure as a cause because they do not believe that there is an association between coal dust exposure and diffuse interstitial fibrosis. All three physicians reviewed the three medical articles referenced by Dr. Rose and Jennings as support of their opinions on causation, *Diffuse Interstitial Fibrosis In Nonasbestos Pneumoconiosis – A Pathological Study* by Dr. Honma; *Idiopathic Pulmonary Fibrosis* by Dr. Iwai; and *Mineralogical Microanalysis of Idiopathic Pulmonary Fibrosis*, by Dr. Monso. All three physicians rejected the conclusions of the articles as epidemiologically unsatisfactory.

As previously stated herein, Judge Leland credited the opinions of Drs. Rose and Jennings that there is an association between Claimant's interstitial fibrosis and his coal dust exposure over the contrary opinions of Drs. Kleinerman, Morgan, Fino, and Spagnolo. His award of benefits was ultimately affirmed by the Benefits Review Board but reversed by the Fourth Circuit Court of Appeals. The Fourth Circuit referred to the aforesaid three articles as "flawed" and held that no reasonable mind could have interpreted and credited the medical opinions as the ALJ did.

Claimant's request for modification of the denial asserted a mistake of fact in the earlier denial of benefits by contending that medical science has progressed in its understanding of the etiology of Claimant's pulmonary disease since the hearing on his claim on February 25, 1997. Claimant also pointed to the West Virginia Workers' Compensation Commission proceeding where the Commission referred Claimant's case for independent medical evaluations from Drs. Doyle and Parker, both of whom found that Claimant's condition was related to his interstitial pulmonary fibrosis. Claimant also obtained a report from his treating pulmonary specialist, Dr. James Dauber stating that Claimant's interstitial pulmonary fibrosis was caused by coal dust exposure. Dr. Dauber's report stated:

Reports over the last 15 years from four separate countries have confirmed the finding of UIP in the lungs of coal miners with heavy exposure to coal mine dust. Green and Vallyathan summarized these reports in a chapter of the book "The Pathology of Occupational Lung Disease" that was edited by Drs. Churg and Green and published in 1998. Green and Vallyathan concluded that "it seems reasonable to consider that the pigmented form of fibrosis (the pattern seen in [Claimant's] biopsy) to be caused by exposure to coal mine dust." They also pointed out the presence of a UIP pattern in autopsied lungs from coal miners is approximately 10 times higher than in the general population.

(DX 103)

Thus, evidence is available that was not a part of the record before Judge Leland that shows coal dust exposure can and did cause Claimant's interstitial pulmonary fibrosis. Dr. Rose reported that her findings on coal dust being the cause of Claimant's pulmonary condition was corroborated by the study by McConnochie et al. Dr. Rose considered that the McConnochie et al. study discovered that the prevalence of diffuse pulmonary fibrosis at autopsy of the studied

miners was substantially higher than would be expected, and documented that the age of onset and relatively benign course of diffuse pulmonary fibrosis was different in coal miners compared to people with IPF. Dr. Dauber testified that he found the Dr. McConnochie et al. study to be very helpful in making a causal relationship between coal dust exposure and UIP.

Employer argues that the McConnochie et al. study does not support a finding that coal dust exposure causes interstitial fibrosis. Employer references the deposition of Dr. Rosenberg where Dr. Rosenberg quotes the McConnochie et al. study as stating, “[t]here is no doubt interstitial pulmonary fibrosis occurs in coal miners, but it is unknown whether this is related to coal dust inhalation.”

However, the November 6, 2008 article by Drs. Cohen, Patel and Green, *Lung Disease Caused by Exposure to Coal Mine and Silica Dust*, to a large extent, references the McConnochie et al. study to support its conclusions that diffuse interstitial fibrosis “is a recognized type of pneumoconiosis following exposure to coal dust, silica, and dusts containing a mixture of minerals (mixed dust pneumoconiosis).”<sup>4</sup> The article describes the clinical features of interstitial fibrosis in coal miners as being associated with irregular opacities on x-ray, diffusion impairment, hypoxemia, a restrictive pattern, and possible clinical, radiological, and functional characteristics mimicking IPF. (*Id.*) The article comments that important reasons for distinguishing diffuse interstitial fibrosis due to an occupational exposure from IPF include the occupational form of diffuse fibrosis having a much more benign clinical course than IPF and requiring different medical management. It reads that a major finding of the McConnochie et al. study is that miners suffering with severe interstitial fibrosis have a relatively benign clinical course. The article also discusses findings of the McConnochie et al. study showing workers exposed to coal mine, silica and silicate dusts consistently show a prevalence of interstitial coal miners at autopsy between 15 and 20 per cent compared to 1.8 per cent in the general population, a major difference between IPF and interstitial fibrosis in workers exposed to coal mine, silica and silicate dusts. Thus, although Dr. Rosenberg accurately quotes the study as stating that it is unknown whether the interstitial pulmonary fibrosis occurring in coal miners is related to coal dust inhalation, Dr. Cohen, and Dr. Green found the study to be sufficiently reliable to provide support for their positions that workers exposed to coal mine, silica and silicate dusts consistently show a prevalence of interstitial coal miners at autopsy between 15 and 20 per cent, much higher than the general population; and support the position that a difference between IPF and miners suffering with severe interstitial fibrosis is that the miners have a relatively benign clinical course.

Dr. Morgan testified that there is no evidence to link coal dust exposure to IPF. However, Dr. Morgan is the co-author of *Occupational Lung Diseases*, Drs. Morgan and Seaton, Third Edition, 1994, which referenced the McConnochie study’s recognition of an occupational interstitial disease. Under the heading *Interstitial Fibrosis*, the text, *Occupational Lung Diseases*, stated, “[i]nterstitial fibrosis was found in the lungs at autopsy of approximately 16% of Welch and West Virginia coal workers. It appears to run a longer clinical course than nonoccupational idiopathic interstitial disease. The reasons for its development in coal workers are not known.”<sup>5</sup>

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<sup>4</sup> *Lung Disease Caused by Exposure to Coal Mine and Silica Dust*, by Drs. Cohen, Patel and Green, p. 655.

<sup>5</sup> *Occupational Lung Diseases*, p. 147.

Dr. Fino testified that there is no evidence to link coal dust exposure to IPF. In support he mentioned textbooks by Dr. Parkes, by Drs. Morgan and Seaton, and by Drs. Churg and Green, as being authoritative on diffuse interstitial fibrosis. However, to the contrary, the textbook by Dr. Parkes, *Third Edition of Occupational Lung Disorder*, by Dr. W. Raymond Parkes, published in 1994, referenced the McConnochie study and it observed that “[a]n interesting point to emerge from the study of McConnochie et al. (1988) is the survival after diagnosis was significantly longer in miners with DIPF than reported in some of the earlier series of cases of idiopathic DIPF in the general population.”<sup>6</sup> (CX 1 at 26-28). Dr. Parkes’ text employed the McConnochie study in its chapter, *Diffuse interstitial pulmonary fibrosis with coal and other carbonaceous pneumoconiosis*, to point out that “[a] combined study of the lungs of coal miners from South Wales and West Virginia (USA) suggested that the overall incidence of DIPF of moderate or severe degree is approximately 5 per cent (milder forms accounting for a further 10 to 12 per cent) and pigmented fibrosis appeared to be associated with continuing exposure to coal-mine dust after it had been recognized radiologically.”<sup>7</sup> Dr. Fino quotes the textbook by Dr. Churg and Dr. F.H.Y. Green, *Occupational Pulmonary Pathology*, as teaching that there is insufficient information to come to the conclusion that coal dust exposure causes diffuse interstitial fibrosis. However, Dr. Green is one of the authors of the McConnochie study and an author, along with Drs. Cohen and Patel, of *Lung Disease Caused by Exposure to Coal Mine and Silica Dust*, which explicitly states that diffuse interstitial fibrosis is a recognized type of pneumoconiosis following exposure to coal dust, silica, and dusts containing a mixture of minerals.

The Fourth Circuit Court of Appeals’ January 23, 2004 decision held that it was error for the ALJ to find the qualifications of Drs. Rose and Jennings to be superior to those of Drs. Kleinerman, Renn, Morgan and Fino because although Drs. Rose and Jennings have experience with IPF, the relevant question is whether Claimant’s IPF is a chronic dust disease of the lungs arising out of coal mine employment, and it is error to credit their opinions over the opinions of Drs. Kleinerman, Renn, Morgan and Fino without considering their vast experience in researching, diagnosing and treating diseases that meet the regulatory standard of chronic dust disease of the lung arising out of coal mine employment.

Initially, the testimony of Dr. Rose shows that she does have extensive experience in the treatment and research of occupational lung disease as well as interstitial fibrosis. Further, the qualifications of Dr. Green and Dr. Parker are extensive in the treatment and study of pulmonary disease caused by coal dust as well as the study of diffuse interstitial fibrosis.

Secondly, the record supports Dr. Parker’s testimony that it has slowly become more broadly accepted that interstitial pulmonary fibrosis can be caused by occupational dust exposure. A comparison between Dr. Fino’s February 10, 1997 deposition testimony with that of Dr. Rose’s July 6, 2011 testimony shows this change. Dr. Fino testified that he had not seen any articles out of NIOSH that have shown an increased risk of a development of an IPF condition associated with exposure to coal mine dust. (DR. 47; dep. of Fino at 50). Dr. Rose testified four years later that NIOSH as well as NIH, National Institute of Health, are increasingly recognizing that diffuse interstitial fibrosis with irregular linear opacities and

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<sup>6</sup> *Lung Disease Caused by Exposure to Coal Mine and Silica Dust*, pp. 367, 368.

<sup>7</sup> *Id.* p. 367.

without classical findings of nodular opacities on the imaging is occurring in this country and elsewhere. She pointed out that the NIH's website mentions that coal dust exposure is associated with risk for IPF. (CX 1 at 25; att. 6).

Moreover, there is substantial support for the proposition that coal dust exposure can cause diffuse interstitial fibrosis not only by Claimant's treating physicians, Drs. Rose, Jennings and Dauber, by the physicians who provided reports for the West Virginia Workers' Compensation Office, Drs. Parker and Doyle, but also by Drs. Green and Cohen, both of whom have excellent qualifications in occupational and pulmonary medicine. See the Department of Labor's preamble to its revised regulations where Dr. F.H.Y. Green's article, *Coal Workers' Pneumoconiosis and Pneumoconiosis Due to Other Carbonaceous Dusts* from the textbook, *Pathology of Occupational Lung Disease* by Drs. A. Chung and FHY Green is referenced. See FEDERAL REGISTER, Vol. 65, No. 245, p. 79943.

Lastly, the clinical evidence supports the findings of Claimant's treating physicians that his diffuse interstitial pneumoconiosis is caused by his coal dust exposure. 20 C.F.R. § 718.104(d). Dr. Jennings reported that the histologic findings from Dr. Waldren included abundant polarized silicates within the alveolar space out of proportion, silicate crystals implicating the presence of the silicates in the pathogenesis of the large number of inflammatory cells within the lung, and silicate deposits observed within the areas of honeycomb, strongly indicating silicates in the pathogenesis of the fibrotic process. Dr. Honma, who has extensive experience in silicosis-induced lung disease, found the biopsy to be consistent with silica-diffuse interstitial lung disease. Claimant's work history of extensive exposure to sandstone in the drilling and long wall work is consistent with silicosis-induced lung disease. Claimant's age of 39 years was considered by his treating physicians to be way out of range of the age of a person treated for idiopathic pulmonary fibrosis, as it strikes people as they age. Dr. Dauber reasoned that Claimant's duration of survival was exceedingly unusual for idiopathic pulmonary fibrosis.

#### CONCLUSION

Accordingly, Claimant has met his burden of establishing a mistake in a determination of fact by establishing a total pulmonary disability caused by coal dust exposure. Claimant's request for a modification is granted and he is found to be entitled to benefits under the Act.

#### ONSET DATE

Benefits commence in a miner's claim on the date the medical evidence first establishes that he became totally disabled due to pneumoconiosis, or if such a date cannot be determined from the record, the first day of the month in which the miner filed his most recent claim. 20 C.F.R. § 725.503 (2010); *Carney v. Director, OWCP*, 11 B.L.R. 1-32 (1987); *Owens v. Jewell Smokeless Coal Corp.*, 14 B.L.R. 1047 (1990). As Claimant ceased all employment on April 30, 1994, on the advice of Dr. Jennings, that date is considered to be onset of his total disability.

ATTORNEY'S FEE

Claimant's counsel shall file within 30 days of the date of issuance of this Decision and Order with this Office and with opposing counsel, a petition for a representative's fees and costs in accordance with the regulatory requirements set forth at 20 C.F.R. § 725.366 (2010). Director's Counsel shall file any objections with this Office and with Claimant's counsel within 20 days of receipt of the petition for fees and costs. It is requested that the petition for services and costs clearly provide (1) counsel's hourly rate with supporting argument or documentation; (2) a clear itemization of the complexity and type of services rendered; and (3) that the petition contains a request for payment for services rendered and costs incurred before this Office only as the undersigned does not have authority to adjudicate fee petitions for work performed before the district director or appellate tribunals. *Ilkewicz v. Director, OWCP*, 4 B.L.R. 1-400 (1982).

ORDER

IT IS HEREBY ORDERED that Consolidation Coal Company shall pay all augmented benefits to which Theodore M. Latusek, Jr. is entitled under the Act, commencing April 30, 1994.

**A**  
THOMAS M. BURKE  
Administrative Law Judge

**NOTICE OF APPEAL RIGHTS:** If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board. To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board. After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed. At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481. If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).