IN THE SUPREME COURT OF TENNESSEE
SPECIAL WORKERS' COMPENSATION APPEALS PANEL
AT NASHVILLE
May 19, 2014 Session

DANNIE JOYNER[1] v. ERACHEM COMILOG, INC. ET AL.

Appeal from the Chancery Court for Humphreys County
No. 2008-CV-17 Robert E. Burch, Circuit Judge Sitting as Chancellor

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No. M2013-02646-SC-R3-WC - Mailed July 18, 2014
Filed September 22, 2014

The plaintiff sought workers' compensation benefits, alleging that he had developed diseases of the skin, lungs, and nervous system as a result of his exposure to nickel, cadmium, and manganese during his employment at a manufacturing facility owned by the defendant. The trial court found that the plaintiff had failed to prove by a preponderance of the evidence that his diseases were caused by exposure to these substances during his employment and entered judgment for the defendant. The plaintiff has appealed from the trial court's decision. Pursuant to Tennessee Supreme Court Rule 51, the appeal has been referred to the Special Workers' Compensation Appeals Panel for a hearing and a report of findings of fact and conclusions of law. We affirm the judgment of the trial court.

Tenn. Code Ann. § 50-6-225(e) (2008) Appeal as of Right;
Judgment of the Chancery Court Affirmed

CORNELIA A. CLARK, J., delivered the opinion of the Court, in which PAUL G. SUMMERS, SR. J., and E. RILEY ANDERSON, SP. J., joined.

Charles L. Hicks, Camden, Tennessee, for the appellant, Phillip G. Hollis, Administrator Ad Litem of the Estate of Dannie Joyner.

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[1] Dannie Joyner died while this case was pending in the trial court. The trial court entered an agreed order substituting his estate and personal representative as the plaintiff in this matter in accordance with Tennessee Rule of Civil Procedure 25. However, the trial court retained the style used on the complaint, and we do so as well.

Stephen B. Morton, Nashville, Tennessee, and Steven M. Loewengart and Kevin E. Hess, Columbus, Ohio, for the appellees, Erachem Comilog, Inc. and Commerce & Industry Insurance Company.

OPINION

I. Factual and Procedural Background

On January 30, 2008, Dannie Joyner ("Mr. Joyner") filed a complaint in the Chancery Court for Humphreys County seeking workers' compensation benefits.[2] The proof introduced at trial showed that Mr. Joyner, who was sixty-three-years old when the trial occurred, had worked thirty-three years at a manufacturing plant in New Johnsonville, Tennessee owned by Erachem Comilog, Incorporated ("Erachem") and its predecessor, Foote Mineral. At the New Johnsonville plant, ore from Gabon, Africa is processed into electrolytic manganese dioxide ("EMD") for use in alkaline batteries. The process involves mixing the ore with other materials, heating it, and bathing it with acids to produce plates or sheets of EMD, which are then ground into the end product—a fine powder that acts as the cathode in alkaline batteries.

Mr. Joyner was employed in a variety of jobs at the plant over his thirty-three-year career, including working in the water and sewer plants, in the ore room, and in the filter building. In 1994 or 1995, Mr. Joyner became a Certified Chemical Operator ("CCO") for Erachem. In this position, he was trained to "do everything"; however, he spent much of his time as a CCO in the ore room working in dusty conditions. His work required grinding up manganese. Patsy Joyner, Mr. Joyner's wife, testified that Mr. Joyner's clothing was "filthy" when he arrived home from work, so much so that she washed Mr. Joyner's clothing separately from the rest of the laundry.

Mr. Joyner testified that Erachem did not require ore room workers to wear respirators. Nevertheless, all of them did. In addition, Mr. Joyner testified that he wore all recommended protective equipment during the time he worked at the plant. Erachem stipulated that Mr. Joyner was exposed to manganese, cadmium, and nickel in the course of his employment at the plant.

[2] Mr. Joyner filed a request for assistance with the Department of Labor and Workforce Development on November 19, 2007. The request was denied, and the administrative process was declared to be exhausted on January 17, 2008.

Mr. Joyner testified that he started smoking when he was sixteen or seventeen years old and had smoked about one-half pack of cigarettes per day until 2003. Mrs. Joyner testified, however, that Mr. Joyner smoked a pack per day until 2006.

In 2002 or 2003, Mr. Joyner underwent coronary bypass surgery. Shortly thereafter, he noticed a loss of energy. In 2006, he began having episodes of urinary and bowel incontinence and also began suffering from dermatitis and mental problems, particularly with his memory. In November 2006, his treating physicians ordered an MRI of his brain due to a history of headaches. The results of that study were normal.

In January 2007, Mr. Joyner fell at his home and was thereafter temporarily unable to walk or speak. He was taken to St. Thomas Hospital, where he gave a history of having a similar episode about a week earlier. Mr. Joyner's condition improved, and he was released from the hospital a few days later. However, he subsequently developed breathing and balance problems. By the time of trial, Mr. Joyner had difficulty walking and used a wheelchair. Both Mr. Joyner and his wife testified that his breathing, balance, and memory problems had gotten worse after his employment at the New Johnsonville plant was terminated on November 26, 2006.

In February 2007, Mr. Joyner underwent gall bladder surgery at Vanderbilt University Medical Center. His attending physicians referred him to Dr. Saralyn Williams, an emergency medicine physician and board-certified toxicologist at Vanderbilt University Medical School, where she is an Associate Professor of Clinical Medicine, faculty physician, and attending medical toxicologist. Dr. Williams was one of Mr. Joyner's treating physicians from that point through the date of the trial.

Dr. Williams testified both by deposition and in person at the trial. In her June 8, 2009 deposition, she testified that she was asked to consult on Mr. Joyner's case because he had documented elevated levels of nickel and cadmium. Regarding the nickel levels, Dr. Williams testified that inhalational exposure to nickel can result in "delayed absorption over a period of time that exceeds [a person's] acute exposure. So to have an elevated exposure level several months after [Mr. Joyner] had last been exposed could be explained by continued exposures in the workplace that stopped but continued to have absorption from the lungs." However, Dr. Williams also testified that, based on skin patch testing, Mr. Joyner's dermatological symptoms were not caused by exposure to nickel.[3]

[3] At trial, Dr. Williams reiterated that she could not make a causal connection between Mr. Joyner's nickel exposure and his dermatitis.

Regarding Mr. Joyner's cadmium levels, Dr. Williams was able to rule out food, water, and outside activities, such as welding and jewelry making, as the source. Testing also showed that on February 26, 2007, three months after he left Erachem, Mr. Joyner's cadmium levels were measurable but within acceptable levels.

With regard to Mr. Joyner's pulmonary issues, Dr. Williams testified in her June 8, 2009 deposition that cadmium can cause both fibrosis and emphysema. On cross-examination she admitted that, with regard to Mr. Joyner's emphysema, "[i]t is difficult to discern out how much is related to his smoking history and how much is related to his cadmium [exposure]," and she agreed that it was possible that all of Mr. Joyner's pulmonary problems were related to smoking. Nevertheless, she stated on redirect examination that it was more probable than not that cadmium was a contributing factor to Mr. Joyner's pulmonary disease.

At trial, Dr. Williams reiterated her opinion that Mr. Joyner's pulmonary issues were caused by occupational exposures and not by smoking. She cited National Institute of Occupational Safety and Health ("NIOSH")[4] reports from 1994, which indicated that the workers at the New Johnsonville plant were exposed to levels of manganese that exceeded the threshold value limits, i.e., the permissible values for inhalation exposure. Based upon a report by pathologist Dr. Joyce Johnson, Dr. Williams testified that Mr. Joyner's primary pulmonary issues resulted from emphysema and scarring in his small airways. She stated that fibrosis[5] of the small airways (or bronchioles) cannot be caused by smoking. From a toxicology standpoint, such scarring can result from medications and inhalation agents. As such, she stated that Mr. Joyner's small airways fibrosis was more probably than not related to his inhalational exposures at work.

On cross-examination, Dr. Williams stated that Mr. Joyner's measurable cadmium level was in actuality "not moderately elevated in someone who [has] a history of smoking." She also testified that smokers have a higher risk for pulmonary disease when exposed to inhalation agents.

With regard to Mr. Joyner's neurological issues, Dr. Williams stated in her deposition that Mr. Joyner's issues were attributable to manganese exposure. She found that Mr. Joyner was exhibiting symptoms "similar to Parkinson's-like disease" which are consistent with chronic manganese exposure. Furthermore, Dr. Williams did not consider it dispositive that Mr. Joyner's MRIs did not show signs of manganism, as manganese is cleared fairly easily

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[4] NIOSH is also the federal agency responsible for certifying respirators.

[5] The record reflects that fibrosis means generally scarring of the lungs.

through a person's system[6] while the long-term effects can be permanent. Dr. Williams stated that a person can have chronic exposure to manganese, followed by a latency period, and subsequently develop a chronic disease related to the previous manganese exposure. She determined Mr. Joyner's neurological and physical impairment to be forty-three (43%) percent to the body as a whole.

On cross-examination, Dr. Williams testified that Mr. Joyner was the first case of manganism she had come across in her practice. She disagreed with the assertion of Dr. Herbert Schaumburg, who testified on behalf of Erachem, that a positive T1 MRI is necessary to diagnose manganism. Instead, Dr. Williams stated that a positive T1 MRI "[m]ay show in severe cases accumulation of radio densities in the brain that could be consistent with manganism; however, it does not necessarily diagnose manganism."[7] However, Dr. Williams conceded that patients typically do not experience a sudden degradation, such as Mr. Joyner exhibited, in cases of chronic manganism. Rather, the onset of manganism is generally gradual, and the initial stages can be more neuropsychiatric before Parkinson's-like symptoms are manifested.

At trial, Dr. Williams testified that "manganese was the culprit" for Mr. Joyner's neurological problems, and she referenced her referral of Mr. Joyner to Dr. Gary Duncan—a board certified neurologist—to corroborate her opinion regarding causation. She stated that documentation from NIOSH showing elevated manganese levels in the personal breathing zones of employees at the New Johnsonville plant was troubling. Dr. Williams testified that the threshold value limits for manganese exposure have been consistently lowered due to "increased data suggest[ing] increased risk to workers for neurotoxicity" as a result of manganese exposure.

On cross-examination at trial, Dr. Williams responded to questions regarding the lack of hyperintensity associated with manganism in Mr. Joyner's MRI results, and reiterated that a positive MRI is not determinative of manganism. As manganese "redistributes out of the body, [a person] loses that hyperintensity," resulting in a normal MRI; however, according to Dr. Williams, a person can still have manganism. In addition, Dr. Williams stated that manganism symptoms do not always recede when a person is removed from manganese exposure. Responding to a study stating that an MRI must return signs of manganese before

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[6] Since manganese is quickly cleared from the system, Dr. Williams testified that any signs of manganese in the MRI of Mr. Joyner's brain several months after he last worked at Erachem would only reflect recent dietary changes and would not reflect workplace exposures.

[7] Instead, Dr. Williams suggested a "[CT] scan with flouro-dopa, dopamine, could be more reliable in excluding Parkinsonism as the etiology since the clinical manifestations are very similar."

any symptoms of manganism occur, Dr. Williams stated that a negative MRI is consistent with Mr. Joyner's history insofar as he had been removed from manganese exposure. She also admitted that, in her deposition testimony, she had stated that the onset of manganism is typically gradual, and that Mr. Joyner's sudden onset of neurological dysfunction in January 2007 was more typical of a vascular episode.

Dr. Gary Duncan, a board-certified neurologist practicing at Vanderbilt University Medical School and Meharry Medical college, testified by deposition. He examined Mr. Joyner on two occasions in 2007, on referral from Dr. Williams. Like Dr. Williams, Dr. Duncan testified that Mr. Joyner had high blood levels of nickel and cadmium. In addition, he stated that manganese can accumulate in the brain and can cause neurological symptoms that resemble, but are not identical to, Parkinson's disease. He further testified that it was not unusual to have a normal MRI and blood levels after exposure to manganese has ended. Responding to inquiries about whether problems caused by manganese exposure can be permanent, he stated:

> They certainly can be, and they can even be progressive. They can improve to some degree . . . . It's thought that the cytotoxic or the cell killing ability of the manganese occurs when the manganese is in the cell and then the damage is done. There's very little recovery. Brain cells don't replicate themselves well at all and so . . . [if] parts of the brain die, then [the brain] will not improve even though the manganese goes away.

Dr. Duncan further stated that Mr. Joyner's symptoms were not otherwise explainable except by workplace exposure to manganese.

On cross-examination, Dr. Duncan testified that Mr. Joyner's case was the second case of alleged manganism with which he had been involved. He reiterated that, after manganese exposure ends, a patient's MRI and blood work may be normal even though the patient suffers from manganism. He testified that while a person removed from manganese exposure can improve, the symptoms may also persist or gradually worsen. Dr. Duncan also testified that he had personally read about or heard of a case of symptomatic nervous manganese where the MRI was normal.

However, Dr. Duncan acknowledged that his opinion regarding the cause of Mr. Joyner's condition was based solely on the subjective history Mr. Joyner provided to him and on Dr. Williams' opinion that Mr. Joyner suffered from industrial toxic exposure. Dr. Duncan further admitted having no objective medical evidence to support his opinion that Mr. Joyner's condition was related to industrial exposure.

Dr. Duncan agreed that a gradual onset of manganism is typical and conceded that Mr. Joyner's sudden and reversible nervous system compromise, as documented at the St. Thomas Emergency Room in 2007, was not consistent with manganism. Dr. Duncan stated, however, "that's not the history [Mr. Joyner] gave me." After having the history Mr. Joyner gave to St. Thomas hospital read to him, Dr. Duncan stated that the history was "certainly compatible with hypertensive complications occurring." Mr. Joyner testified that he had been diagnosed with hypertension while in his twenties.

Dr. Elisabeth D. Willers, a board-certified physician in internal medicine, pulmonary and critical care on the faculty at Vanderbilt University Medical School, testified by deposition on two occasions. Dr. Willers stated that Mr. Joyner's emphysema was multifactoral and related to his smoking and workplace exposures, and she further testified that "it is more probable than not that [Mr. Joyner's] exposures at his workplace environment . . . contributed to his shortness of breath and pulmonary problems." Dr. Willers specifically identified cadmium as a substance associated with the development of emphysema. She stated that it was her understanding that Mr. Joyner's chronic dermatitis had been caused by his exposure to nickel, but she admitted that the dermatological effects of nickel were outside her area of expertise and that her opinion would change upon learning that Dr. Williams did not link Mr. Joyner's dermatitis to nickel exposure.

After Mr. Joyner exhibited a decline in pulmonary function in March 2011, Dr. Willers ordered a CT scan, which showed evidence of progressive fibrosis in his lungs. Dr. Willers subsequently ordered a surgical lung biopsy,[8] which showed severe emphysema, multifocal honeycombing, central aveolar fibrosis and bronchiolectasis (small airways scarring), as well as the presence of amorphous polarizable material.[9] Dr. Willers testified

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[8] Mr. Joyner's surgical lung biopsy on April 11, 2011, entailed biopsies of two different lobes of his right lung. The upper right lobe wedge biopsy "showed severe emphysema, bronchiolectasis, which is small airways scaring, with peribronchiolar metaplasia, foci of subpleural fibrosis." The right lower lobe biopsy "showed severe emphysema, multifocal honeycombing . . . central alveolar fibrosis, with bronchiolectasis and peribronchiolar metaplasia . . . as well." Hereinafter, the findings from these separate lung biopsies are referenced together unless otherwise noted.

[9] A defense expert who testified at trial described the meaning of this term as follows:

> Amorphous means no structure. Crystal and silica has structure. Just like a diamond has edges, silica in the lungs or when you inhale it has a crystal form. Amorphous does not. It's only crystalline silica that damages the lungs. Amorphous materials are nontoxic. OSHA has determined that. Amorphous polarizable particles are found in— in—it's used for fillers in cosmetics. It's used for fillers in paint. It's actually a silica—it's a silica—a non—excuse me—an amorphous material like this is used in cigarette paper, it's used in

(continued...)

that the biopsy "clearly showed . . . evidence of fibrosis . . . , and even further clarified that fibrosis to be an airway fibrosis, which is very consistent with an inhalation injury." Based on the location and concentration of scarring in the small airways, as well as the presence of polarizable materials, Dr. Willers opined that the inhalation component of Mr. Joyner's lung condition was not consistent with tobacco use or smoking.

On cross-examination, Dr. Willers stated that this was the first case of alleged cadmium poisoning she had seen. Dr. Willers admitted that her opinions about exposure at the New Johnsonville plant were entirely derived from Mr. Joyner's representations to her and to the other treating physicians. Dr. Willers was not aware of any validated data correlating cadmium levels and smoking history, although she was aware cadmium levels can be elevated in smokers. However, she was unaware of the exact levels that correlate with smoking history, and she was unable to say whether Mr. Joyner's cadmium levels were actually normal for a cigarette smoker. In addition, Dr. Willers testified that there was no way to determine the degree to which cadmium exposure, rather than smoking, contributed to Mr. Joyner's emphysema, and she conceded that it was possible that Mr. Joyner's emphysema was caused entirely by his smoking.

On cross-examination in her supplemental deposition, Dr. Willers testified that pulmonary fibrosis can be associated with cadmium exposure according to the medical literature. She disagreed with Drs. Rosenberg and Tomashefski, both of whom testified on behalf of Erachem, that Mr. Joyner suffered from interstitial fibrosis, stating that the CT and biopsy results did not support such a diagnosis. While reiterating that Mr. Joyner had "an inhalational injury that cannot be explained by smoking," Dr. Willers acknowledged that she did not know the source of the injury, nor had she seen any evidence suggesting Mr. Joyner was exposed to a specific level of cadmium at his work place.

Dr. Joyce E. Johnson, a board-certified anatomic and clinical pathologist specializing in cytopathology and a professor at Vanderbilt University Medical School, testified by deposition. Dr. Johnson had reviewed the tissue samples from Mr. Joyner's lung biopsy. Her findings were bronchiolectasis, which she defined as "a dilation or enlargement of the diameter of the small airway, with scarring in the wall and inflamation." Dr. Johnson testified that the majority of Mr. Joyner's pulmonary scarring was found in the small airways, and she noted that primary small airways scarring is an uncommon finding in lung biopsies, and is consistent with an inhalation injury. Dr. Johnson explained that small airways injury is indicative of an agent or agents being inhaled through the airways and landing on, or

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[9](...continued)
filters for cigarettes, and this is something that is nontoxic and has no significance in this situation, in this case.

targeting, the last part of the airway branching tree. Dr. Johnson opined that small airways scarring is not caused by smoking or emphysema.

Furthermore, Dr. Johnson stated that Mr. Joyner's small airways fibrosis was "by far more severe" than his interstitial fibrosis. Mr. Joyner's alveolar tissue (gas-exchanging units) showed minimal fibrosis, ruling out a separate list of diagnostic possibilities that cause fibrosis in the alveolar walls. Dr. Johnson testified she was "ninety-nine percent" positive her findings were a result of a "continuous injury" caused by something Mr. Joyner inhaled. Based on the location and concentration of the fibrosis, Dr. Johnson stated that what she observed was not Usual Interstitial Pneumonia (UIP).

Dr. Johnson noted that Mr. Joyner also suffered from emphysema and agreed that the most common cause of the condition is cigarette smoking. Dr. Johnson testified that emphysema is a separate process affecting different parts of the lung than those affected by his inhalation injury.

On cross-examination, Dr. Johnson testified that smoking may have some impact on interstitial scarring but that it is not an initiator. She admitted that she had never treated a patient with pulmonary conditions allegedly caused by exposure to cadmium, nickel, or manganese.

Dr. Herbert Howard Schaumburg, a board-certified neurologist, testified at trial on behalf of Erachem. Dr. Schaumburg had been the head of the neurology department at Albert Einstein College of Medicine of Yeshiva University in New York for over twenty years, and he had co-authored the first textbook of neurotoxicology. Over his career, Dr. Schaumburg had seen more than twenty patients with manganese poisoning and had been consulted in ten other cases. Dr. Schaumburg testified that industrial manganese poisoning in North America is extremely rare; however, he had seen patients with industrial manganese poisoning in India.

Dr. Schaumburg testified that, while Mr. Joyner had symptoms consistent with manganism, Mr. Joyner's negative MRIs, his removal from exposure prior to exhibiting symptoms, and the acute onset of his neurological symptoms all demonstrate he was not suffering from manganese poisoning. Dr. Schaumburg testified that, by definition, a patient must have an abnormal MRI when he is exhibiting manganism symptoms. According to Dr. Schaumburg, the presence of manganese in an MRI is blatantly obvious, "like a searchlight." Neither the November 2006 MRI of Mr. Joyner's brain, taken while he was still working at Erachem, nor the January 2007 MRI from St. Thomas, taken after the onset of Mr. Joyner's significant neurological symptoms, showed signs of manganese. In Dr. Schaumberg's opinion, the negative MRIs were dispositive that Mr. Joyner was not suffering from

manganism. He cited a recent medical journal article which supported his assertion that the presence of manganese on MRI was essential to a diagnosis of manganism.

In addition, Dr. Schaumburg testified that manganism has a slow, "insidious onset," and therefore manganism patients do not tend to end up in the emergency room for treatment. Mr. Joyner's acute onset of neurological symptoms and abrupt improvement, as documented in his St. Thomas medical records, was inconsistent with manganism. Instead, Dr. Schaumburg testified that the MRI report produced the day after Mr. Joyner was admitted to the St. Thomas Emergency Room showed symptoms consistent with a vascular cause, such as a small stroke. Dr. Schaumburg further stated a small stroke would be consistent with Mr. Joyner's history of hypertension and specific symptoms such as his altered mental status and inability, as opposed to an impaired ability,[10] to speak.

Dr. Schaumburg also testified that symptoms of manganism tend to improve over time after exposure to manganese has ended. Mr. Joyner's worst symptomatic episode, the January 2007 event, occurred two months after he had stopped working at Erachem. Dr. Schaumburg stated that, unlike a material such as asbestos, there is no delayed toxicity with manganese exposure.[11] Dr. Schaumburg opined that it was "fundamental" that a patient's symptoms had to arise during his period of exposure.

Dr. Schaumburg examined Mr. Joyner almost three years prior to the trial at a hotel near Mr. Joyner's home. He watched Mr. Joyner walk and talk. He noted that, as Mr. Joyner relaxed, his speech and movements became easier. He observed that Mr. Joyner had an "action tremor," which occurs while moving but disappears at rest.

Dr. Schaumburg also visited Erachem's plant in New Johnsonville. He noted that there were no reports of other employees with alleged manganism and that Mr. Joyner's urinalysis and blood testing while working at the plant showed normal levels of manganese. These facts led him to conclude that because "there's no manganese in his urine, none in his blood, and none in his brain[,] [i]t can't be manganism."

During cross-examination, Dr. Schaumburg agreed that Mr. Joyner had neurological symptoms compatible with manganism and that Mr. Joyner had been exposed to manganese in the course of his employment. However, Dr. Schaumburg explained, elevated exposure

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[10] Dr. Schaumburg testified manganism causes an impaired ability to speak but does not lead to an inability to speak, such as Mr. Joyner exhibited during his emergency room visit.

[11] In contrast, Dr. Williams opined that there could be a latency period between the date of manganese exposure and the development of symptoms.

to manganese does not equate to toxicity. Responding to questions concerning NIOSH studies in 2007-2008 which found that CCOs at Erachem were overexposed to manganese by three separate standards, Dr. Schaumburg stated that the average person can tolerate "a lot of exposure to manganese . . . and not get sick."

Additionally, Dr. Schaumburg opined that the lasting effects of manganism only persist after exposure to manganese ends in cases of "overwhelming exposure." In his experience, he had only seen such cases in India and Taiwan. Additionally, Dr. Schaumburg testified that the effects of manganism were not permanent in North American cases unless the patient also had a pre-existing liver problem. Dr. Schaumburg stated that Mr. Joyner exhibited a degree of disability and symptoms consistent with North American cases in which the patient improved after exposure ended. He therefore considered the fact that Mr. Joyner's symptoms did not improve after his exposure ended to further support his opinion that manganism was not present. Dr. Schaumburg again reiterated his opinion that Mr. Joyner had suffered from strokes too small to be visible on an MRI.

Finally, Dr. Schaumburg admitted that he only interacted with management when he visited Erachem's New Johnsonville plant and that he did not discuss potential neurological concerns with any of the CCOs or similar employees. He was unable to testify as to which buildings he visited, nor was he familiar with the processes at the plant, and he stated that Erachem had advance notice of his visit.

Dr. David Masur, a Ph.D. in neuropsychology and a professor of neurology at Albert Einstein College of Medicine,[12] testified at trial on behalf of Erachem. As part of his practice, Dr. Masur evaluates whether a patient's thought pattern is consistent with toxic exposure. He had evaluated one previous patient with manganism. Dr. Masur examined Mr. Joyner at the same hotel as Dr. Schaumburg and reviewed his medical records. Mr. Joyner had a history of anxiety for many years, which Dr. Masur stated can produce cognitive impairment over time. Dr. Masur observed that Mr. Joyner was also depressed.

Dr. Masur testified that Mr. Joyner's anxiety had an overriding impact on his neurological symptoms. During his examination, Mr. Joyner's memory was consistent with a person suffering from anxiety issues. Dr. Masur stated that Mr. Joyner had difficulty with his memory at the beginning of the exam, but as he relaxed, his condition improved. Dr.

[12] Dr. Masur defined neuropsychologists as psychologists concerned with how the brain is working. Neuropsychologists "use different kinds of tests in order to be able to examine individuals that may have complaints or problems with things like memory, concentration, language ability, attention . . . . [Neuropsychologists] utilize these tests in order to evaluate these complaints" and make a causation diagnosis.

Masur also noted that persons who have undergone bypass surgery, such as Mr. Joyner, often experience memory problems that can be exacerbated by anxiety issues. Like Dr. Schaumberg, Dr. Masur stated that the manifestations of manganism improve when the exposure ceases. As that was not the case with Mr. Joyner, Dr. Masur stated that Mr. Joyner's psychological manifestations were not consistent with manganism.

On cross-examination, Dr. Masur testified that about twenty percent of his practice consisted of litigation and forensic type matters, reviewing fifteen to twenty cases per year. He typically testifies in court about three times per year. Dr. Masur reiterated that Mr. Joyner's neurological problems were inconsistent with manganism, noting that Mr. Joyner's performance improved during Dr. Masur's examination as his anxiety lessened. "Brain injury of any kind does not produce [a] kind of pattern where in a short period of time an individual performs poorly and subsequently gets better," as was the case in his examination of Mr. Joyner. The short time lapse in Mr. Joyner's improvement suggested he was not cycling through good days and bad days, as is associated with brain injury. Dr. Masur noted that, after Mr. Joyner's anxiety level diminished, his cognitive function was close to normal, which was not consistent with brain injury from toxicity. Therefore, Dr. Masur stated that Mr. Joyner's cognitive problems were caused by anxiety and depression.

David M. Rosenberg, a board-certified pulmonologist, also testified at trial on behalf of Erachem. In addition to his medical degree, Dr. Rosenberg holds a master's degree and is board-certified in occupational medicine. After finishing his medical training at Duke University, Dr. Rosenberg spent three years at the National Institute of Health, where he completed his pulmonary fellowship and was "actively involved doing research in interstitial fibrosis." In addition to practicing as a pulmonologist for over thirty years, he is one of about three hundred "NIOSH B Readers,"[13] doctors certified to evaluate and interpret x-rays for the presence of dust-related disorders and occupational-related conditions. Dr. Rosenberg reviewed Mr. Joyner's medical records and examined Mr. Joyner on one occasion.

Based on the examination and Mr. Joyner's medical records, Dr. Rosenberg testified that Mr. Joyner suffered from interstitial fibrosis (or UIP), not small airways disease. To establish small airways disease, a patient must exhibit (1) wheezing, (2) airflow obstruction, and (3) air trapping, none of which he found to be present in Mr. Joyner. Instead, Dr. Rosenberg stated that Mr. Joyner's airways disease relates to his interstitial fibrosis, as it is common for the latter to involve the small airways. Interstitial fibrosis can result from a variety of causes, not just occupational exposure, according to Dr. Rosenberg. For example, a 2011 study by the American Thoracic Society listed cigarette smoking first among various

[13] Dr. Rosenberg testified that of the three hundred certified B Readers, only approximately one hundred are active pulmonary doctors or nonradiologists.

risk factors for interstitial fibrosis. In that regard, Dr. Rosenberg stated that the presence of polarizable particles cited by Mr. Joyner's physicians as evidence of an inhalation injury are actually consistent with, and attributable to, Mr. Joyner's smoking history.

Dr. Rosenberg testified that neither cadmium nor nickel caused Mr. Joyner's pulmonary issues. Cadmium levels twenty-five to one hundred times greater than normal exposure levels are necessary "to either cause emphysema or in rare situations a mild fibrosis." Dr. Rosenberg described the cadmium levels at the plant where Mr. Joyner worked as "minuscule" and not at the level that could cause Mr. Joyner's current lung conditions. In addition, Dr. Rosenberg testified that even if cadmium exposure reaches over a hundred times greater than acceptable standards, overexposure does not cause the type of honeycombing scarring found in Mr. Joyner.

Dr. Rosenberg additionally testified that "[t]he medical literature indicates that . . . patients who get cadmium-related lung disease have had extremely high concentrations of exposure to cadmium, reflected in high levels of urinary cadmium." Dr. Rosenberg further specified that patients suffering from cadmium toxicity do not start experiencing a reduction of pulmonary functions until urine cadmium levels reach around fifty. Cadmium has a half life of decades,[14] meaning a patient suffering from overexposure would have elevated levels of urine cadmium even after the exposure ended.

The highest level of urine cadmium exhibited by Plaintiff was 1.2.[15] Dr. Rosenberg noted that Mr. Joyner's cadmium levels were consistent with his smoking history, as smokers have higher levels of cadmium and nickel than the general population due to the cadmium and nickel found in cigarettes. Dr. Rosenberg also cited the results of a study of workers in a high cadmium work environment (an alkaline battery manufacturer) which demonstrated that, even in areas with elevated cadmium levels, workers' cadmium levels from smoking far exceeded cadmium levels from the work environment. Finally, Dr. Rosenberg pointed out that Mr. Joyner had not developed kidney disease, which necessarily precedes lung disease caused by extremely high cadmium exposure.

Dr. Rosenberg also testified that occupational exposure to nickel could not have caused Mr. Joyner's pulmonary issues or his dermatitis. Unlike cadmium, nickel is cleared from the body within hours of exposure. Any nickel found in tests from 2007 could not be

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[14] Dr. Rosenberg disagreed with Dr. Williams that the half life of urine cadmium depended on when the exposure took place.

[15] Mr. Joyner's levels of urine cadmium from February 2007 through September 2007 were zero, 1.2, and zero. Dr. Rosenberg stated that these findings did not demonstrate persistent levels of elevation.

attributable to workplace exposure, because Mr. Joyner stopped working at Erachem in November 2006. Dr. Rosenberg also stated that nickel does not cause lung disease. With regard to Mr. Joyner's dermatitis, Dr. Rosenberg testified that the negative patch test was dispositive that nickel did not cause Mr. Joyner's dermatological issues.

On cross-examination, Dr. Rosenberg stated that he testifies as an expert once or twice per year, and that ninety-five percent of the time he testifies for defendants. Dr. Rosenberg acknowledged that smoking could not cause the honeycombing in Plaintiff's lungs and that patients can have peripheral scarring from small airways disease. After acknowledging that nickel exposure can cause dermatitis, Dr. Rosenberg reiterated that Mr. Joyner's skin condition is not work related, as dermatitis related to an allergic reaction disappears and does not persist in the system.

Dr. Joseph F. Tomashefski, Jr., a board-certified anatomic and clinical pathologist, testified for Erachem by deposition. At the time of his deposition, Dr. Tomashefski served as Chairman of the Department of Pathology at MetroHealth Medical Center in Cleveland, Ohio, and had practiced pulmonary pathology for over thirty years.[16] Dr. Tomashefski stated that he had reviewed Mr. Joyner's medical records and viewed both the photographs and the actual slides of the biopsies of Mr. Joyner's lungs prior to his deposition. Based on these materials, Dr. Tomashefski opined that Mr. Joyner suffered from UIP—one of the major forms of interstitial fibrosis—with an additional finding of "mixed panlobular and centrilobular emphysema."

Dr. Tomashefski disagreed with Dr. Willers's statement that "central alveolar fibrosis is not consistent with interstitial fibrosis," stating,

> Fibrosis in the centri-alveolar region still would qualify as interstitial fibrosis, as that air space fibrosis gets converted into the connective tissue structure of the lung. . . . [F]ibrosis is extending from the pleura [the outer membrane that covers the lung] into the central regions of the lung. So it's a distribution and a pattern of scarring. And this pattern, by the way, is typical of the disease condition [UIP].

Dr. Tomashefski opined that Mr. Joyner's pattern of pulmonary fibrosis was consistent with a disease known as idiopathic pulmonary fibrosis, which can minimally be caused by asbestos-related occupational lung diseases or disease due to inhalation of

[16] Dr. Tomashefski clarified during cross-examination that he was not board-certified as a pulmonologist, but as a clinical and anatomic pathologist. His focus had been on pulmonary pathology for over thirty years.

"silicates." However, Dr. Tomashefski stated that UIP and Mr. Joyner's pattern of fibrosis cannot be caused by nickel, cadmium or manganese. In addition, Dr. Tomashefski confirmed the "honeycombing" described by Mr. Joyner's treating physicians, but stated that such honeycombing is common with UIP and refers to end-stage interstitial fibrosis.

Dr. Tomashefski testified that Mr. Joyner's "elevated" cadmium levels were "entirely compatible" with his smoking history, explaining that the levels were actually at the low-end of the range typically seen in cigarette smokers. While Dr. Tomashefski agreed that nickel and cadmium can produce lung injury at high exposure levels, he saw no evidence of such elevated exposure levels at the New Johnsonville plant. Dr. Tomashefski stated that if high levels of cadmium exposure were present at the plant, he would expect to see elevated levels in other workers, which was not the case here. Dr. Tomashefski further stated that the injury produced by elevated cadmium exposure looks nothing like the pattern of UIP scarring Mr. Joyner exhibited. Rather, elevated cadmium and nickel exposure causes diffuse alveolar damage, which was not found on Mr. Joyner's biopsy, nor was it mentioned in any of his medical records. While Dr. Tomashefski agreed that Mr. Joyner's airway injuries were not consistent with tobacco use, he stated that those injuries were not inhalation related either. Rather, the changes seen in Mr. Joyner's airways were part of a more diffuse interstitial fibrosis.

In addition, Dr. Tomashefski testified that the polarizable materials shown in Mr. Joyner's lung biopsy represent silicates, which are frequently seen in long-term tobacco smokers. He also stated that manganese, cadmium, and nickel are not polarizable materials. In Dr. Tomashefski's opinion, the presence of polarizable particles consistent with smoking exposure was merely an incidental finding that "in no way indicate[d] occupational exposure as a cause of his fibrosing lung disease."

Dr. Tomashefski testified that smoking caused Mr. Joyner's emphysema. Emphysema is a destructive process in which lung tissue is destroyed and the small air spaces of the lung coalesce to produce greatly enlarged air spaces without associated fibrosis. Dr. Tomashefski testified that of the three metals Mr. Joyner alleged to have caused his injuries, only cadmium has been associated with emphysema. Dr. Tomashefski emphasized that workers who developed emphysema secondary to cadmium exposure had been exposed to high levels of cadmium in the workplace and had high levels of cadmium in their blood and urine. However, Mr. Joyner's cadmium levels, as revealed in his blood and urine testing, were not high enough to cause emphysema. Because emphysema is also caused by the cadmium in tobacco smoke, Dr. Tomashefski testified that Mr. Joyner's emphysema was likely related to tobacco smoke and not cadmium exposure in the workplace.

Dr. Tomashefski agreed that Mr. Joyner suffers from bronchiolectasis, a widening or an expansion of the caliber of the lumen of the small airways, which "may or may not be associated with fibrosis of the airway wall." Dr. Tomashefski cited both diffuse interstitial fibrosis, which produces traction bronchiolectasis, and emphysema as causes of bronchiolectasis that do not include fibrosis of the airway wall. Dr. Tomashefski further stated that bronchiolectasis is a secondary manifestation of interstitial fibrosis and emphysema, and that traction bronchiolectasis is a common finding in patients with interstitial fibrosis. Dr. Tomashefski believed diffuse interstitial fibrosis and emphysema to have caused Mr. Joyner's bronchiolectasis.

Dr. Tomashefski further stated that his finding of peribronchiolar metaplasia is frequently associated with the diagnosis of UIP. Dr. Tomashefski described peribronchiolar metaplasia as "a proliferation of cells occurring around the smallest air conducting tubes" in the lung "which pass air from the environment into the deep recesses of the lung." Peribronchiolar metaplasia can be seen in 50% to 60% of all cases of UIP. Dr. Tomashefski clarified that when peribronchiolar metaplasia is associated with UIP, "it is not related to inhalational injury to the small airway, but it's part of the fibrosing process of [UIP]."

On cross-examination, Dr. Tomashefski testified that manganese, cadmium, nickel, and other heavy metals can cause permanent lung damage when inhaled. However, there is little evidence that heavy metals cause small airways fibrosis. Additionally, the relevant level of exposure has more to do with intensity than duration. Dr. Tomashefski further stated that treatment of small airways scarring (the disease advanced by Mr. Joyner's treating physicians) involves first removing the patient from exposure, which causes the patient's toxicity levels to diminish. However, Dr. Tomashefski admitted that "once you have fibrosis remodeling, that will not be reversed." He also acknowledged that his report does not indicate the levels of exposure Mr. Joyner encountered at the New Johnsonville plant.

While Dr. Tomashefski stated that small airways fibrosis "certainly can be caused by smoking," he agreed that smoking does not cause the type of small airways scarring and inflamation seen in Mr. Joyner, nor will smoking cause honeycombing. However, Dr. Tomashefski reiterated his disagreement with the treating physicians' emphasis on small airways inhalation injury. In response to a question concerning Dr. Johnson's testimony that essentially every small airway had some degree of scarring, he stated, "[t]here may have been a small amount of scarring in many of the airways." However, contrary to Dr. Johnson, Dr. Tomashefski stated that he did not believe the majority of the scarring to be found in the small airways, and he reiterated that honeycombing is an "end-stage form of interstitial fibrosis." In general, however, Dr. Tomashefski agreed with Dr. Johnson's analysis that "[f]ibrosis that involves the airway would not be included in interstitial disease."

Dr. Tomashefski reiterated that manganese, cadmium, and nickel are not polarizable and that in over thirty years of reviewing polarizable materials he had not encountered a single reference to these three metals producing polarizable materials in the lung. However, he stated that specifically identifying the polarizable materials present in Mr. Joyner's biopsy would require an elemental analysis.

In rebuttal, Mr. Joyner testified that Bobby Cantrell, a co-worker at Erachem, had experienced many of the same symptoms as Mr. Joyner, and he stated that other workers also had similar symptoms including shaking, difficulty walking, and slurred speech. Several other Erachem employees had rashes in the early 1990's.

Medical reports concerning Mr. Joyner from 1989 and 1995, made in conjunction with pre-placement exams at the New Johnsonville plant, also mention some interstitial markings. An X-ray report from 1989 showed "[m]inimal increase in interstitial markings . . . since July 1985 suggesting minor chronic interstitial pneumonitis." A report dated January 16, 1995, noted "[m]inor interstitial prominence in the lung bases likely due to chronic bronchitis or chronic interstitial pneumonitis." Additionally, an April 2006 certification by Mr. Joyner's health care provider under the Family and Medical Leave Act stated that "to avoid possible contact with irritants [Mr. Joyner] could need to work outside of such an environment" and recommended that he "needs to stay away from chemicals."

Matthew Kosmider, the plant manager at Erachem's New Johnsonville plant, testified both at trial and by deposition.[17] Mr. Kosmider acknowledged that the plant had received

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[17] During the deposition of Mr. Kosmider, Mr. Joyner's counsel referred to Material Safety Data Sheets (MSDS) regarding toxins present at the New Johnsonville plant, which were prepared by the company from which the materials were purchased or by Erachem's parent company, Emaret. These documents were produced by Erachem during discovery. The trial court discounted the value of these materials, however, noting that it was unclear if they were written by doctors and, if so, upon what evidence their conclusions were based.

According to the MSDS, electrolytic manganese dioxide (EMD) may aggravate pre-existing respiratory issues, and persons with a history of neurologic or pulmonary disease may have a greater risk of developing symptoms resulting from overexposure. The MSDS showed the primary route of exposure is inhalation and that inhalation exposure to EMD primarily targets the lungs and the Central Nervous System. Discussing symptoms, the MSDS states, "Chronic exposure can result in conjunctivitis from irritant dusts and dermatitis. Chronic inhalation of manganese containing dust can result in Central Nervous System disorders *that may appear after 6 months to 2 years of elevated exposure.* The symptoms . . . resemble Parkinson's Disease." (Emphasis added). The MSDS goes on to list potential symptoms including: sleep disturbances, diminished fine motor coordination, "manganic psychosis," slow speech and stammering, salivation, and gait disturbances.

(continued...)

NIOSH complaints regarding concerns of exposure to chemicals—especially manganese—in late 2007. Mr. Kosmider also acknowledged a discussion with Mr. Joyner regarding his health concerns prior to his termination. Mr. Kosmider stated that the predominant metals extracted during production at the plant are cobalt, nickel, and copper. He also testified that certain CCO jobs require the employee to wear a respirator. However, this only became a requirement in the "last couple of years."

At the conclusion of the proof, the trial court took the case under advisement, and it issued its decision as a written memorandum. The trial court found that Mr. Joyner had not sustained his burden of proof that any of the three conditions from which he suffered were caused by exposure to toxins in his workplace. Judgment was entered in accordance with those findings. This appeal followed. Mr. Joyner contends that the evidence preponderates against the findings of the trial court on the causation issues.

II. Analysis

Appellate review of decisions in workers' compensation cases is governed by Tennessee Code Annotated section 50-6-225(e)(2) (2008), which provides that appellate courts must "[r]eview . . . the trial court's findings of fact . . . de novo upon the record of the trial court, accompanied by a presumption of the correctness of the finding, unless the preponderance of the evidence is otherwise." As the Supreme Court has observed many times, reviewing courts must conduct an in-depth examination of the trial court's factual findings and conclusions. Wilhelm v. Krogers, 235 S.W.3d 122, 126 (Tenn. 2007).

Additionally, when expert testimony differs, the trial judge maintains discretion to accept the opinion of one expert over another. Hinson v. Wal-Mart Stores, Inc., 654 S.W.2d 675, 676-77 (Tenn. 1983). When the trial court has seen and heard the witnesses,

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[17](...continued)

A similar MSDS for manganese dioxide states that inhalation of the substance may increase incidences of pulmonary infections and may cause emphysema. In addition, the MSDS states that chronic exposure to manganese dioxide can lead to manganese poisoning (or manganism).

Similar materials for manganous oxide state that it can aggravate pre-existing respiratory infections, and, as in the case of EMD, persons with a history of neurologic, pulmonary, or liver disease may have a greater risk of developing symptoms of overexposure, with damage to the lungs and central nervous system similar to that listed for EMD above.

Each MSDS referenced above states that the minimum respiratory protection in areas exceeding OSHA permissible conditions is a negative pressure air purifying respirator.

considerable deference must be afforded the trial court's factual findings. Tryon v. Saturn Corp., 254 S.W.3d 321, 327 (Tenn. 2008). No similar deference need be afforded the trial court's findings based upon documentary evidence such as depositions. Glisson v. Mohon Int'l, Inc./Campbell Ray, 185 S.W.3d 348, 353 (Tenn. 2006). However, reviewing courts afford no presumption of correctness to a trial court's conclusions of law. Seiber v. Reeves Logging, 284 S.W.3d 294, 298 (Tenn. 2009).

The issue in this case is whether the evidence preponderates against the trial court's finding that Mr. Joyner failed to prove that exposure to manganese, cadmium, and/or nickel at the New Johnsonville plant caused his neurological, dermatological, and pulmonary diseases.[18] Generally, the Workers' Compensation laws should be "liberally construed to promote and adhere to the [purposes of the Workers' Compensation] Act of securing benefits to those workers who fall within its coverage." Martin v. Lear Corp., 90 S.W.3d 626, 629 (Tenn. 2002). Nonetheless, an employee must prove each element of his worker's compensation claim by a preponderance of the evidence. Elmore v. Travelers Ins. Co., 824 S.W.2d 541, 543 (Tenn. 1992). As such, a workers' compensation claimant must establish by expert medical evidence the causal relationship between the alleged injury and the claimant's employment activity, "'[e]xcept in the most obvious, simple and routine cases.'" Cloyd v. Hartco Flooring Co., 274 S.W.3d 638, 643 (Tenn. 2008) (quoting Orman v. Williams Sonoma, Inc., 803 S.W.2d 672, 676 (Tenn. 1991)).

While causation cannot be based on speculative or conjectural proof, a claimant in a workers' compensation action does not have to demonstrate causation as an absolute certainty, as medical proof can rarely be certain. Clark v. Nashville Mach. Elevator Co., 129 S.W.3d 42, 47 (Tenn. 2004). Evidence that employment could have caused the injury is sufficient to establish a prima facie case that the injury arose out of employment, and, if the employer does not produce evidence to the contrary, the employee has established by a preponderance of the evidence that a workers' compensation award is appropriate. Id. at 49. All reasonable doubts as to the causation of an injury and whether the injury arose out of the employment should be resolved in favor of the employee. Phillips v. A&H Constr. Co., 134 S.W.3d 145, 150 (Tenn. 2004); Reeser v. Yellow Freight Sys., Inc., 938 S.W.2d 690, 692 (Tenn. 1997).

[18] Although Mr. Joyner also listed a second issue in the section of his brief titled "Statement of Issues" concerning the trial judge's ruling on Erachem's motion to strike Dr. Williams's testimony relative to cobalt, Mr. Joyner failed to provide any argument or citation to the record regarding this issue in the argument section of his brief. The issue is therefore waived. See Tenn. R. App. P. 11, 27(a)(7); see also Lovlace v. Copley, 418 S.W.3d 1, 33, n.17 (Tenn. 2013); Sneed v. Bd. of Prof'l Responsibility, 301 S.W. 603, 615 (Tenn. 2010).

Mr. Joyner contends that he developed three medical conditions as a result of his exposure to three specific substances at Erachem's plant in New Johnsonville, Tennessee. Specifically, Mr. Joyner alleges that: (1) exposure to nickel caused chronic dermatitis; (2) exposure to nickel and cadmium aggravated his smoking-related emphysema and caused small airways scarring of the lungs; and (3) exposure to manganese caused various neurological symptoms, including short-term memory loss, slurred speech, and unsteadiness of gait. As a general matter, it is undisputed that Mr. Joyner was exposed to nickel, cadmium, and manganese in the course of his employment. The dispute in this appeal centers on whether the proof in this record preponderates against the trial court's finding that Mr. Joyner failed to prove that his work place exposure to these substances caused the conditions for which he now seeks workers' compensation benefits.

1. Dermatitis

First, we address the trial court's finding that Mr. Joyner failed to establish by a preponderance of the evidence that his dermatological condition was caused by exposure to nickel in his workplace. The evidence concerning Mr. Joyner's chronic dermatitis is straightforward and convincing. Every physician who testified on the subject agreed that the negative patch test administered on Dr. Williams's order eliminated nickel as the cause of his chronic dermatitis. The evidence does not preponderate against the trial court's finding that Mr. Joyner failed to prove that his dermatological condition was caused by his employment.

2. Pulmonary Conditions

Second, we address the trial court's finding that Mr. Joyner failed to establish by a preponderance of the evidence that his pulmonary disease was caused by exposure to cadmium, among other toxins, in his workplace. All of the medical witnesses agreed that Mr. Joyner had emphysema. All also agreed that this condition was related, at least in part, to his forty-year history of cigarette smoking. In addition, all of the medical experts with knowledge of cadmium levels in smokers agreed that Mr. Joyner's level of cadmium was consistent with his history of smoking. Nevertheless, Drs. Williams, Willers, and Johnson opined that cadmium exposure demonstrated that Mr. Joyner's workplace had contributed to his emphysema, but they could not estimate the extent of that contribution. Drs. Rosenberg and Tomashefski, on the other hand, testified that the emphysema was entirely caused by smoking.

Drs. Williams, Willers, and Johnson also opined that Mr. Joyner suffered from small airways disease. They stated that this condition was not caused by smoking but by an inhalational injury. These doctors further pointed to the presence of amorphous, polarizable

substances detected by Mr. Joyner's lung biopsy as evidence of an inhalation injury. However, Dr. Willers, the pulmonary specialist, was not able to state which substance inhaled by Mr. Joyner had caused the small airways scarring.

Drs. Rosenberg and Tomashefski disagreed with the diagnosis of small airways disease. These experts opined that Mr. Joyner suffered from UIP in addition to emphysema. Both doctors testified that UIP was associated with cigarette smoking, but its actual cause is unknown. Dr. Rosenberg found the results of Mr. Joyner's CT scan to be consistent with UIP. He noted that the records of various clinical examinations of Mr. Joyner did not reveal wheezing, airflow obstruction, or air trapping in the lungs all necessary prerequisites to a diagnosis of small airways disease. Dr. Tomashefski, too, found evidence of UIP in his examination of the lung biopsy slides. He also considered the presence of polarizable particles on the CT scan of Mr. Joyner's lungs to be irrelevant because cadmium, nickel, and manganese are not polarizable materials. He attributed the presence of these particles to Mr. Joyner's smoking.

As mentioned above, a trial court generally has the discretion to choose which expert to accredit when there is a conflict of expert opinions. Johnson v. Midwesco, Inc., 801 S.W.2d 804, 806 (Tenn. 1990); Kellerman v. Food Lion, Inc., 929 S.W.2d 333, 335 (Tenn. 1996). In evaluating conflicting expert testimony, a trial court may consider, among other things, "the qualifications of the experts, the circumstances of their examination, the information available to them, and the evaluation of the importance of that information by other experts." Orman, 803 S.W.2d at 676. When a trial court has heard witnesses through live testimony, considerable deference is given to the trial judge's assessment of expert credibility. See Tryon, 254 S.W.3d at 327.

The experts who testified for Mr. Joyner—Drs. Williams, Willers, and Johnson—all had excellent credentials and all held academic appointments at Vanderbilt University School of Medicine. Drs. Williams and Willers were also treating physicians and therefore had more extensive direct contact with Mr. Joyner. Of these three physicians, only Dr. Williams testified in person at trial for the Plaintiff.

Drs. Rosenberg and Tomashefski also had excellent credentials. Dr. Rosenberg, who testified at trial on Erachem's behalf, had earned a Masters degree in occupational medicine, in addition to board-certification as a pulmonary physician. Of more direct relevance to his testimony in this case, Dr. Tomashefski had performed extensive research concerning pulmonary fibrosis and had published peer-reviewed papers on the subject. He also relied on specific articles and texts to support his opinions in this case. Dr. Tomashefski was board-certified in anatomic and clinical pathology, served as Chair of the Department of

Pathology at MetroHealth Medical Center, and had practiced pulmonary pathology for over thirty years.

Although causation is a close question, the proof does not preponderate against the trial court's finding that Mr. Joyner failed to carry his burden. The qualifications and testimony of Drs. Rosenberg and Tomashefski were more specifically related to the causation of Mr. Joyner's pulmonary condition than those of the Vanderbilt physicians. We note that all physicians agreed that the two substances, cadmium and nickel, alleged to be the cause of Mr. Joyner's emphysema and other pulmonary problems are present in cigarette smoke. The test results showed that the levels of those substances in Mr. Joyner's system were consistent with the levels to be expected from his forty-year smoking habit.

In Tennessee, an employer takes an employee "as he is and assumes the risk of having a weakened condition aggravated by some injury which might not hurt or bother a perfectly normal, healthy person." Blalock v. Williams, 483 S.W.2d 578, 579 (Tenn. 1972) (internal quotation marks omitted) (quoting Swift & Co. v. Howard, 212 S.W.2d 388, 391 (Tenn. 1948)). However, the plaintiff must provide material evidence that his disability resulted from aggravation of a pre-existing weakness. Id. Despite some evidence of interstitial scarring dating back to 1989, Mr. Joyner did not advance any evidence that the interstitial scarring referred to by Erachem's experts could have been an extension of previous interstitial scarring exacerbated by occupational exposure. Regarding his emphysema, Mr. Joyner's treating physicians acknowledge that smoking can cause emphysema and that cadmium likely contributed to his worsening condition; however, aside from supporting these claims with a single elevated cadmium level consistent with his smoking history, Mr. Joyner presented no evidence that his workplace exposure exacerbated any existing pulmonary conditions.

In addition, Mr. Joyner's physicians pointed to polarizable materials as evidence that Mr. Joyner suffered a work-related inhalation injury. However, Dr. Tomashefski testified that none of the three metals alleged to have caused Mr. Joyner's pulmonary issues are polarizable. Considering the record as a whole, we are unable to conclude that the evidence preponderates against the trial court's finding that Mr. Joyner failed to sustain his burden of proving that his pulmonary condition was caused or exacerbated by his employment.

3. Neurological

Finally, we address the trial court's finding that Mr. Joyner failed to sustain his burden of proof that a causal connection existed between his neurological symptoms and his exposure to manganese in the workplace. Once again, a comparison of the relative qualifications of the expert witnesses is useful. Dr. Williams is a board-certified emergency

physician and toxicologist. She has broad knowledge concerning the toxic effects of various substances on the human body. However, Mr. Joyner's case was her first encounter with a case of neurological damage allegedly caused by manganese exposure. Dr. Duncan, a board-certified neurologist, had one previous case of manganese exposure. In contrast, Dr. Schaumberg had seen and diagnosed twenty such cases and had been consulted in ten others. He was the editor of a text on neurotoxicology and had headed a laboratory devoted to the study of the effect of chemicals on the nervous system. Thus, his background is more specifically addressed to the relationship between Mr. Joyner's neurological symptoms and his manganese exposure.

Dr. Duncan and Dr. Schaumberg agreed that manganism is an insidious, gradual disease. While Dr. Duncan originally opined that Mr. Joyner's problems were related to manganese exposure, he retreated from that position after learning of the acute onset of symptoms that led to Mr. Joyner's January 2007 hospitalization. He described those events as being inconsistent with the history given to him by Mr. Joyner and inconsistent with a diagnosis of manganism.

Dr. Schaumberg testified that the lack of manganese on a T1 MRI was dispositive on the subject of manganism. He supported his testimony with references to recent medical journal articles. His conclusion that Mr. Joyner was not suffering from manganism was based upon Mr. Joyner's negative MRIs, his removal from exposure to manganese, and the acute onset of his symptoms. Dr. Duncan did not entirely disagree with Dr. Schaumberg's opinions, but he believed that symptoms of manganism might continue even after manganese had been removed from the brain by normal biological processes.

Dr. Masur, a neuropsychologist, testified that Mr. Joyner's history of anxiety and depression, as well as his recent bypass surgery, were the more likely causes of his memory problems.

While all the experts agreed that Mr. Joyner's symptoms were consistent with manganism, the proof does not preponderate against the trial court's finding that Erachem's experts effectively eliminated a causal connection between Mr. Joyner's neurological issues and his workplace exposures. Mr. Joyner had an MRI in November 2006, while he was still working at Erachem and still being exposed to manganese. That study was normal. His most dramatic and severe episode of neurological symptoms occurred in January 2007, two months after he left Erachem. An MRI taken at that time was also normal. We find, as did the trial court, that Dr. Schaumberg's description of the indicators of manganism is compelling.

As was the case with his pulmonary issues, Mr. Joyner did not advance any evidence that exposure to manganese exacerbated his pre-existing neurological issues such as anxiety

and depression. See Blalock, 483 S.W.2d at 579. Therefore, we conclude that the evidence does not preponderate against the trial court's finding that Mr. Joyner's neurological problems were not caused by manganism.

III. Conclusion

We affirm the judgment of the trial court that Mr. Joyner was unable to sustain his burden and prove by a preponderance of the evidence that his skin, pulmonary, and neurological symptoms were a result of exposure to metals or chemicals in the workplace. Costs are taxed to the Estate of Dannie Joyner, and its surety, for which execution may issue if necessary.

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CORNELIA A. CLARK, JUSTICE

IN THE SUPREME COURT OF TENNESSEE
AT NASHVILLE

DANNIE JOYNER v. ERACHEM COMILOG, INC., ET AL.

Chancery Court for Humphreys County
No. 2008-CV-17

No. M2013-02646-SC-WCM-WC - Filed September 22, 2014

Judgment Order

This case is before the Court upon the motion for review filed by Dannie Joyner pursuant to Tennessee Code Annotated section 50-6-225(e)(5)(A)(ii), the entire record, including the order of referral to the Special Workers' Compensation Appeals Panel, and the Panel's Memorandum Opinion setting forth its findings of fact and conclusions of law.

It appears to the Court that the motion for review is not well taken and is, therefore, denied. The Panel's findings of fact and conclusions of law, which are incorporated by reference, are adopted and affirmed. The decision of the Panel is made the judgment of the Court.

Costs are assessed to the estate of Dannie Joyner and his surety, for which execution may issue if necessary.

It is so ORDERED.

PER CURIAM

Clark, J., not participating