

BEFORE THE ARKANSAS WORKERS' COMPENSATION COMMISSION

CLAIM NO. F007360

ALVIN LANSDELL,
EMPLOYEE

CLAIMANT

GEORGIA-PACIFIC CORPORATION,
SELF-INSURED EMPLOYER

RESPONDENT

OPINION FILED SEPTEMBER 3, 2003

Upon review before the FULL COMMISSION in Little Rock,
Pulaski County, Arkansas.

Claimant represented by HONORABLE GREGORY R. GILES, Attorney
at Law, Texarkana, Arkansas.

Respondent represented by HONORABLE MARK A. PEOPLES,
Attorney at Law, Little Rock, Arkansas.

Decision of the Administrative Law Judge: Affirmed as
modified.

OPINION AND ORDER

The claimant appeals an Administrative Law Judge's
opinion filed August 21, 2002. The Administrative Law Judge
found that Act 1281 of 2001 made substantive law changes to
the burden of proof for occupational disease and was to be
applied prospectively. The Administrative Law Judge
therefore found, "Claimant has failed to prove by clear and
convincing evidence that he sustained an occupational
disease which arose out of and in the course of his
employment." After reviewing the entire record de novo, the

Full Commission finds that our recent decision in a companion case, Sikes v. Georgia-Pacific Corporation, Workers' Compensation Commission F000657 (July 7, 2003), is controlling in this matter as to the appropriate burden of proof. We therefore find that the Legislature meant to apply Act 1281 retroactively, so that the "preponderance of the evidence" standard of Ark. Code Ann. § 11-9-601 (e) (1) (B) applies to the instant matter. The Full Commission further finds that the claimant failed to prove by a preponderance of the evidence that he sustained a compensable occupational disease. We therefore affirm, as modified, the opinion of the Administrative Law Judge.

I. HISTORY

Claimant Alvin Lansdell has been employed by the respondent, or its successor, since 1968. For approximately 30 years, the claimant worked inside in the wood room. In 1998, the claimant's job duties changed and he was required to rotate between the wood room and the purchased chips area. The claimant described the purchased chips area as an outside area where the employees unload hardwood and softwood chips from trailer trucks and hardwood chips from

railcars. Unloading of the railcars was sometimes delayed for weeks at a time because of the layout of the work area and the necessity of promptly moving the trailer trucks in and out. The claimant described the manner in which the railcars were unloaded as follows:

A. We have got a pit with rail tracks right across it. We push that railcar right across those tracks, over that pit, and we've got -- the doors will swing open on that railcar. We swing the doors open and ----

Q. They swing open from the outside, if you are looking at it, or is it underneath?

A. From underneath. The train opens from underneath and got us a shaker mounted up on top of the railcar and we've got to drop it down on the railcar and then we shake them.

Q. A shaker being a piece of equipment that actually attaches to it and shakes it?

A. Yes.

Q. Describe that for us. What does it look like when that happens, when the shaker is shaking the railcar?

A. Well, the shaker starts shaking the railcar and you've got your bottom doors open, the chips will just start drifting out, you know, falling out.

Q. Down into the pit?

A. Down into the pit, and we've got chains in the bottom of the pit pulling the wood chips out of the pit.

Q. When you first started working in purchased chips in 1998, when the shaker would shake a hardwood chip railcar, would the chips just fall to the bottom or was there any dust created?

A. It's a lot of dust coming up. When the chips fall through, dust will drift up, just clouds of dust.

Q. And was that -- does that happen every time?

A. Well, no, not every time. Your hardwood chips have a tendency to mold in them railcars. If they've brought them from Mississippi, Alabama, way long ways off, you store them in them railcars and it might take a month or two months to get up here and get shook out. That's whenever they really come out bad. Now, if they have just been cut two or three days ago, it wouldn't be any dust hardly in it.

The claimant testified that after he began working in the purchased chips area, he began to experience continuing flu-like symptoms. He said that on October 24, 1999, his crew unloaded a particularly bad load of hardwood chips from the railcars, and every man on the crew became ill. He did not go to the doctor that day, because he did not think he was sick enough to require medical attention. His previous episodes of headache and nausea had usually cleared up

within two or three days, but this time his symptoms persisted.

The claimant testified that he became so ill on Christmas Day that he was unable to breathe, and he called his doctor to obtain a prescription for antibiotics. On December 31, 1999, the claimant was admitted to Little River Memorial Hospital by Dr. Kevin Kleinschmidt with an admitting diagnosis of asthma and influenza. The claimant provided Dr. Kleinschmidt the following history:

One week history of non-productive cough with fever and chills, general malaise, myalgias, which he received some Cipro and then Cefzil on out-patient basis and cough medicine. Despite this his symptoms have become worse with increased shortness of breath and wheezing. He denies any nausea, vomiting, or diarrhea.

Pursuant to the claimant's request, he was discharged from Little River Memorial Hospital on January 2, 2000, and transferred to Wadley Hospital in Texarkana. Dr. Christopher Bailey admitted the claimant and assessed him with "respiratory distress with severe hypoxemia and atypical pneumonia as well as probable chronic bronchiectasis."

At Dr. Bailey's request, the claimant was examined by Dr. James Hurley. The claimant provided Dr. Hurley with the following history:

This is a 55 y/o patient admitted by Dr. Chris Bailey for referral from Ashdown with increasing shortness of breath and hypoxemia. He and his wife both apparently had a viral illness over the last couple of weeks and both thought they had the flu. His wife got better but the patient did not and in fact, on Christmas Day, he began having increasing shortness of breath. The wife had already started him on some Cipro and he was taken to the emergency room, I believe in Ashdown, where he received treatment for bronchitis.

On January 6, 2000, Dr. Bailey performed a bronchoscopy; the pathology report dated the next day noted "many fungal organisms present." The bronchial washing pathology report noted "...a fibrinous background containing neutrophils and a few focally reactive columnar epithelial cells and squamous epithelial cells."

On January 10, 2000, the claimant was discharged from the hospital with a diagnosis of: "1. Respiratory distress; 2. Atypical pneumonia; 3. Severe hypoxemia; 4. Leukocytosis; 5. Hypokalemia; 6. Hyperglycemia; 7. Atherosclerotic coronary artery disease status post myocardial infarction about three years ago; 8. Chronic obesity; 9. Possible

obstructive sleep apnea syndrome; 10. Gastroesophageal reflux disease with possible chronic aspiration; 11. Possible chronic bronchiectasis." On January 12, 2000, Dr. Bailey issued a statement for the claimant's application for short-term disability benefits, identifying his diagnosis as, "1. Acute Respiratory Failure; 2. Hypoxemia; and, 3. Bronchiectasis." A subsequent certification by Dr. Bailey replaced the diagnosis of bronchiectasis with a diagnosis of atypical pneumonia.

Laboratory results from his hospital stay were sent to the Mayo Medical Laboratory for testing. A January 20, 2000, report from the Laboratory confirmed the presence of "aspergillus niger," "aspergillus terreus," and "penicillium sp" in the bronchial washings. Upon discharge, the claimant was placed on a home medical regime until he was released to return to work on February 7, 2000.

In August, 2000, the claimant again became ill and was hospitalized by Dr. Bailey. The claimant's admitting diagnosis included hemoptysis, bronchiectasis, hyperlipidemia and right upper lobe pneumonia. Dr. Bailey discharged the claimant on August 16, 2000, and listed a

history of aspergillus colonization in the discharge diagnosis. From this date forward, Dr. Bailey's diagnosis of the claimant included bronchopulmonary aspergillosis and bronchiectasis. Laboratory testing of the claimant during that hospitalization revealed no aspergillosis antibodies in the claimant's system, though it did reveal organizing pneumonia.

In a letter dated August 29, 2000, Dr. Bailey stated:

Mr. Lansdell has been seen by me since January 2000. He has bronchiectasis, Gastroesophageal Reflux Disease, Arteriosclerotic Coronary Artery Disease, and Bronchopulmonary Aspergillosis. He is currently receiving treatment for his Bronchopulmonary Aspergillosis with Itraconazole two 100 mg. po q day. It is quite possible that he may have been infected and/or colonized with Aspergillosis during his work experience since Aspergillus is a common organism found in the soil as well as in wood pulp. The patient reports that he is exposed to large quantities of wood pulp dust in the workplace. It is impossible to determine exactly where the infection was acquired. However, given the circumstances of his work environment, I have advised the patient to not return to the same work environment due to his pursuing treatment for Bronchopulmonary aspergillosis with Itraconazole two 100 mg per day. It would be preferable that the patient be removed from the high risk environment, if possible. (Emphasis added.)

In the Spring of 2001, the claimant began seeking treatment for hip pain. On March 8, 2001, Dr. Greg Smolarz

diagnosed the claimant with avascular necrosis of both hips. Dr. Smolarz indicated that this condition was more than likely related to the claimant's steroid treatment for his lung condition. In April of 2001, the claimant underwent a total hip replacement of his right hip. A subsequent IME report by Dr. Earl Peeples stated that it is reasonable to attribute the claimant's sudden development of avascular necrosis to his steroid treatment.

Mr. Lansdell claimed entitlement to worker's compensation, and a pre-hearing order was filed with the Commission on February 11, 2002. The respondent asked that the claimant undergo an independent medical examination, and the Commission directed the claimant to be examined by pulmonologist Dr. Nancy Rector in Little Rock. The Commission also allowed respondent to have a doctor of its own choosing, Dr. Gerald Kerby, present at the examination.

Dr. Rector and Dr. Kerby separately examined the claimant on June 15, 2001. With regard to her examination and findings, Dr. Rector noted in her report:

In summary, Mr. Lansdell is a longtime Georgia-Pacific employee who worked in the wood room and did not have regular exposure to the dust associated with emptying the chip cars. He is a

non-smoker. He had reported problems related to the dust associated with emptying the chip cars prior to the onset of his illness. He became ill over the Christmas Holiday in 1999. He assures me that he was working at that time and that they were unloading chips but I don't have that specific information. He was hospitalized on New Year's Eve with an acute respiratory illness characterized by dyspnea, wheezing, hypoxemia, but no pulmonary infiltrates. He subsequently did have Aspergillus identified in his sputum. He did not meet any of the other criteria for a diagnosis of allergic bronchopulmonary aspergillosis. He was treated short-term with prednisone and was on inhaled bronchodilators for obstructive airways disease. He had a second acute illness in August of 2000 characterized by hemoptysis and subsequently a right upper lobe pneumonia. I do not find information that confirmed he had Aspergillus in his sputum at that time, nor did he have positive Aspergillus titers or eosinophilia. He was placed on itraconazole, which is an antifungal treatment but is not actually thought to be effective for allergic bronchopulmonary aspergillosis. He subsequently developed hip pain in March of 2001 and was diagnosed with avascular necrosis. He really had fairly limited corticosteroid therapy throughout his course.

At the present time, Mr. Lansdell does have decreased breath sounds in his chest and does not do a very good forced expiratory maneuver, which may be patient effort issue. He had no coughing and no wheezing. His chest x-ray is normal, as is his chest CT. His resting room air oxygen saturation is 97%. His pulmonary function studies are abnormal and do show moderate airways obstruction. He is on low dose inhaled corticosteroid and as needed bronchodilator therapy. There is no question that this man, who is a non smoker, does have moderate obstructive airways disease. We have no pulmonary function

tests prior to his initial illness, however, I cannot confirm that allergic bronchopulmonary aspergillosis is the etiology of his obstructive airways disease. (Emphasis added.)

Dr. Rector reviewed the claimant's x-rays from Wadley Hospital, and noted that the films of January, 2000, and the film of August 8, 2000, revealed no acute infiltrates but did show a flattened hemidiaphragm. A film dated August 14, 2000 revealed "an alveolar infiltrate involving the right mid lung". The final x-ray film, from April 8, 2001, revealed no acute infiltrates. The blood drawn from the claimant at the time of his independent medical examination tested less than .35 for aspergillus fungus, which is in the "absent or undetectable range".

The respondent chose as its expert for this examination Dr. Gerald Kerby, a professor in the Division of Pulmonary Diseases of the University of Kansas Medical Center. With regard to his examination and findings, Dr. Kerby reported:

...Mr. Lansdell has evidence of chronic obstructive pulmonary disease of moderate severity related to his prior history of cigarette smoking. In 12-99 and 1-00 he developed an acute exacerbation of chronic obstruction pulmonary disease treated with antibiotics and corticosteroids. In the course of his evaluation bronchoscopy was performed demonstrating a neutrophilic lavage and a few colonies of 3

environmental fungi. He had another episode of pneumonia with hemoptysis in 8-00 which also resolved with antibiotic therapy.

Allergic bronchopulmonary aspergillosis occurs in individuals who develop IgG and IgE antibodies against *Aspergillus*. It is characterized by fleeting, recurrent eosinophilic infiltrates, asthma and the development of bronchiectasis involving the central part of the tracheobronchial tree. Lab findings consist of eosinophils both in peripheral blood and bronchial secretions, the presence of IgE and IgG antibodies against *Aspergillus*, elevated total serum IgE and the presence of *Aspergillus* in large numbers in bronchial secretions. Mr. Lansdell meets virtually none of these criteria. The *Aspergillus* cultured on bronchiole alveolar lavage in 1-00 represent colonization of the tracheobronchial tree in someone with chronic bronchitis, particularly after prolonged antibiotic treatment.

In my opinion Mr. Lansdell does not meet criteria for a diagnosis of allergic bronchopulmonary aspergillosis and there is no evidence of [sic] that he suffers from any occupationally induced lung disease. (Emphasis added.)

The claimant submitted for his own medical evaluation to Dr. Andrew Prychodko, then an assistant professor in the Department of Occupational and Environmental Medicine at the University of Texas Health Center in Tyler, Texas. Dr. Prychodko stated in his report that the dust created from the *aspergillus* in the hardwood chip cars is "clearly an event that could cause direct serious harm to Mr. Lansdell

given his current sensitization, and must be avoided." Dr. Prychodko added, "I concur that Mr. Lansdell suffers from Bronchopulmonary Aspergillosis, which was proximately caused by intense fungal spore exposures to which he was subjected in the course and scope of his employment."

The parties deposed Dr. Prychodko on January 30, 2002, and his deposition was made a part of the record. At the time of his deposition, Dr. Prychodko had accepted the position of the Medical Director of the Center for Preventative Occupational and Environmental Medicine in the College of Medicine at the University of Arkansas for Medical Sciences. Dr. Prychodko is board certified in occupational medicine.

In his deposition, Dr. Prychodko testified that he agreed with both Dr. Kerby and Dr. Rector that the claimant does not satisfy the criteria for Allergic Bronchopulmonary Aspergillosis. However, Dr. Prychodko differentiated between Bronchopulmonary Aspergillosis with and without the allergic component. In reaching his conclusion that the claimant's pulmonary condition is related to his exposure to aspergillus in the workplace, Dr. Prychodko explained he

relied upon the fact that excessive amounts of organic material were in the air and that aspergillus fungal colonies were found in claimant's bronchial washings. Dr. Prychodko also relied upon the diagnosis of Allergic Bronchopulmonary Aspergillosis made by claimant's treating pulmonologist, Dr. Chris Bailey. In addition, Dr. Prychodko relied upon the fact that both Charles Adams and John Sikes have made claims for aspergillus-related lung conditions from their exposure to this fungus at Georgia-Pacific. Dr. Prychodko specifically stated, "...the cluster of incidents is an incredibly helpful adjunct tool in directing an injury." With regard to his diagnosis, Dr. Prychodko testified:

- A. ...With that analogy to the allergic bronchopulmonary aspergillosis, the presence of aspergillus and the presence of aspergillus hyphae, let's say, in the lung washings, tell me that there is bronchopulmonary aspergillosis; in other words, there is a growth of aspergillus in the bronchial washings.

That part about the allergic is a second question. So when you ask about A.B.A., you know, it's certainly a starting point to have the aspergillus in the lung. And having the aspergillus turn into hyphae is the -osis part of infection and allergic part. In other words, the body manifest [sic] immune

and antibody responses to that may or may not be represented in those findings that you ask.

Dr. Prychodko was then asked if the presence of aspergillus in the claimant's lungs meant there was a causal connection between the fungus and claimant's condition:

- A. I think that's a pretty close -- I think that's a pretty tight association. You know, the presence of his clinical history of experiencing respiratory difficulty and the presence of these plumes of mold spores, the reactive airway problem that he had, the obstruction, the acute onset obstructive changes where the airway spasmed out -- not the airway retention that you see in cigarette smoking emphysema, but just the broncho hyperreactivity, which can be caused by allergic bronchopulmonary aspergillosis. It can be caused by many kinds of irritants, chemical agents, mold agents. Cotton dust will -- I mean, there are all kinds of things that will cause a bronchial spasm without necessarily manifesting an Ig-E or antibody response.

So the fact that he had a clinical exposure history of problems and documented bronchial spasm obstructive problems, washings of organisms that we don't necessarily expect to see in the lung -- I think that's all one continuous process that within medical -- within reasonable medical probability, that relates together.

In spite of the criteria for a specific diagnosis of allergic bronchopulmonary aspergillosis, Dr. Prychodko

testified that claimant's good health prior to being moved to the purchased chip yard, where aspergillus fungus was found in high concentrations, and claimant's pulmonary problems after being moved to the purchased chip yard, show a direct line of causation between claimant's work environment and his lung condition.

On cross-examination, Dr. Prychodko clarified his diagnosis of the claimant as reactive airways bronchial hyperactivity with the presence of bronchopulmonary aspergillosis. According to Dr. Prychodko, the bronchial hyperactivity is the obstructive change on the pulmonary function test and is what caused claimant's need for hospitalization and medical treatment.

At the hearing held before the Commission on May 16-17, 2002, the claimant contended that he sustained a compensable injury in the form of an occupational disease to his lungs, and that treatment for the disease caused him to develop avascular necrosis in his right hip, as a compensable consequence of his lung condition. The claimant contended that he was entitled to several periods of temporary total disability compensation, in addition to reasonably necessary

medical treatment for his lung condition and his hip condition. The claimant contended that the legislative change of 2001 to Ark. Code Ann. § 11-9-601, wherein the burden of proof for occupational disease claims was lowered from clear and convincing evidence to a preponderance of the evidence, is applicable to this claim.

The respondent contended that the appropriate burden of proof was clear and convincing evidence, but also that the claimant could not prove by either clear and convincing evidence or by a preponderance of the evidence a causal connection between the claimant's work and his condition. The respondent contended that the medical care received by the claimant was not reasonable and necessary. The respondent further contended that if the claim was found to be compensable, respondent was entitled to an offset for disability benefits received by the claimant and that any attorney fees should be based on the net TTD award, after the offset was applied.

Dr. Kerby testified at the hearing on behalf of respondent. When asked where aspergillus is found, he replied, "Everywhere. It's on the floor here, it's on the

table here, it's all over. It is ubiquitous. It is one of the environmental fungi that exists almost everywhere in the world." Dr. Kerby defined allergic bronchopulmonary aspergillosis as follows:

A. Allergic bronchopulmonary aspergillosis is a disease of the lungs which occurs in people who develop a sensitivity to the fungus. Most micro-organisms, including fungi, can act as antigens and the body can react to antigens by forming antibodies. If the antibodies and the antigens complex, it can then result in disease.

Q. Antibodies? You are talking about white blood cells?

A. No, these are proteins within the serum of the blood. They are produced by cells call [sic] lymphocytes and when a lymphocyte is exposed to an antigen, it may produce an antibody. It is the way we defend ourselves against infections, so people who get a flu shot have antibodies against influenza, people that get measles vaccinations have antibodies against measles. The antibodies are -- God maybe set them up to defend people against infection, however, in the course of reacting with an antibody against an antigen, it may result in disease. Now, there are three classes of antibodies in the blood. There is one of them, the so-called IgE class, which is the antibody of immediate hypersensitivity, it's the antibody involved in hayfever, allergic sinusitis, often in asthma, and in allergic skin diseases such as eczema which occur in children. Milk allergy causing eczema is mediated by an IgE antibody directed against milk protein. In the case of allergic aspergillosis, people develop IgE antibodies and when they are then re-exposed to aspergillus, it can result in release of mediators

from cells called mast cells. The most important mediator is histamine, and histamines, which we block with antihistamines, that's why we take them for sneezing and that sort of thing, in the lung results in bronchial restriction, which is one of the features of asthma, results in the production of fluid and mucus into the airway, and it can result in inflammation and the kind of inflammation that results from IgE antibody complexes is mediated by a class of white cells called eosinophils, so most eosinophilic inflammatory reactions in the body are mediated by IgE antibodies. So, one of the abnormalities in allergic aspergillosis is that people develop IgE class antibodies against the fungus. In addition, most people with allergic aspergillosis also develop antibodies of the so-called IgG class, and this is gammagobulin. IgG class antibodies when they complex with an antigen such as aspergillus, triggers a series of inflammatory reactions which are often mediated by a cell line called lymphocytes, and as they complex with the antigen they trigger a system in the body called the complement system. The complement system is there to destroy bacteria and viruses and other things that are infected.

So, if you are immunized against influenza and you have IgG antibodies and you are exposed to influenza, this triggers a series of chemical steps which results in destruction of the virus. The same thing occurs if you get a pneumonia shot against pneumococcus and you get pneumococcal pneumonia, the IgG antibodies trigger a series of things which end up with complement attaching the pneumococci and destroying them. So, the system is put there to defend against infection, but it can also produce disease as well. The IgG antibody complexes against aspergillosis results in destruction of the wall of the airway. The aspergillus is inhaled into the lung, it sits there in the airway, and if there are antibodies

formed in the wall of the airway where the fungus sits, it results in destruction of the lining of the airway and this results in dilation in the formation of the sacs and cylinders where there is normally a straight tube, and we call that disease bronchiectasis. Ectasis means dilated bronchus. So, the features of bronchopulmonary aspergillosis is that you get an asthmatic reaction with the IgE antibody class, and this is wheezing, bronchospasm, mucus plugs, and the typical finding that you see in asthma. The body reacts forming eosinophils and eosinophils are usually elevated in the blood and they are elevated in the airway fluid if you sample the airway fluid. There is an elevated level of IgE antibodies in the blood and this is manifested both by a specific IgE directed against aspergillus, which can be tested for. One of the problems in differential diagnosis is that people who are allergic have high total IgEs, but unless they are allergic to aspergillus they don't have IgE antibodies against aspergillus. So one of the features of the - the other thing that happened with the IgE antibodies is that the eosinophils sometimes infiltrate in the periphery of the lung where the air sacs are and this results in what is called an eosinophilic pneumonia, that is you see a shadow on x-ray that looks like pneumonia.

Dr. Kerby said this was a rare disease - "I've probably seen more than 10 and less than 20 cases in my whole life."

Dr. Kerby testified that in his opinion, within a reasonable degree of medical certainty, the claimant does not suffer from allergic bronchopulmonary aspergillosis, aspergilloma, or invasive aspergillosis. Dr. Kerby further testified that the claimant's hospitalizations for his

respiratory condition did not result in whole or in part from claimant's exposure to aspergillus while on the job.

When Dr. Kerby was asked about the claimant's physical findings on the x-ray performed at Wadley Hospital in January of 2000, Dr. Kerby acknowledged that the claimant presented with inflammation and bronchiectasis, or thickened bronchial walls which could be related to allergic aspergillosis. However, Dr. Kerby opined that this was a non-specific finding which could be related to any number of conditions.

Dr. Kerby explained the claimant's bronchoscopy findings on January 9, 2000, of "diffuse tracheo-bronchitis with mucopurulent exudate" as evidence of inflammation with puss and mucus lining the windpipe. The Bronchoalveolar lavage results from this same hospitalization revealed a class of white cells called neutrophils "that are commonly seen with acute infections." However, as explained by Dr. Kerby, this test did not reveal the presence of any eosinophils, which he testified are always present in the bronchial spaces in cases of allergic bronchopulmonary aspergillosis.

Dr. Kerby attributed the presence of aspergillus in the claimant's bronchial airway in January of 2000 to the fact that the claimant had just been treated with antibiotics for a bacterial infection. With bacteria suppressed, the fungi which are normally present in the body are allowed to grow, and thus appear in greater amounts. Dr. Kerby explained that this fungal colonization is expected after a treatment of antibiotics and thus is typically ignored.

According to Dr. Kerby, the CT scan did not show any shadowing consistent with pneumonia in the periphery of the lung, and no bronchiectasis or dilated airways due to destruction. Dr. Kerby explained that dilated airways or bronchiectasis are two other major criteria of a diagnosis of allergic bronchopulmonary aspergillosis.

With regard to the claimant's hospitalization in August of 2000 for right middle lobe pneumonia, Dr. Kerby testified that such pneumonia is not related to aspergillus exposure. While fungi can sometimes cause pneumonia, there was no evidence of fungal pneumonia in the claimant. As for the claimant's April 8, 2001, hospitalization at St. Michael's Hospital, Dr. Kerby described the claimant's x-ray findings

as revealing evidence of anatomical emphysema, which is most commonly caused by smoking.

Dr. Kerby acknowledged that the headaches, fever, and flu-like symptoms claimant suffered after his October 24, 1999, exposure to "bad" mold dust were consistent with Organic Toxic Dust Syndrome, or OTDS. It is a self-limited illness, typically resolving within eight to sixteen hours, for which there is no treatment.

Dr. Kerby was asked about Dr. Prychodko's diagnosis of hypersensitivity pneumonitis. Dr. Kerby explained that the claimant does not have the positive serum IgG class antibodies necessary for this condition, nor does he have interstitial fibrosis on high resolution CT, meaning he does not meet the criteria for a diagnosis of hypersensitivity pneumonitis. Dr. Kerby was also asked about Dr. Prychodko's diagnosis of "bronchopulmonary aspergillus", and Dr. Kerby testified that medical science does not recognize any such disease without the "allergic" component.

Dr. Kerby was asked about the coincidental timing and relationship of the claimant's exposure to aspergillus and his subsequent illness, to which he responded:

A. Everybody gets sick that is exposed to something in a day, week, month, or year beforehand. Possibly it is related, but more frequently it is coincidental.

Q. You are speaking in the abstract? When you are saying possibly related, you are not referring to Mr. Lansdell's case, are you?

A. I think some of the symptoms of the nausea, feeling bad, fever, could well have been related to the mycotoxin exposure from the aspergillus in the dust that he breathed.

Q. You are talking about the symptoms in October?

A. The symptoms in October. I think with his obstructive lung disease, that the non-specific effects of any kind of dust could have caused some increased breathing difficulties. People with pre-existing lung disease don't tolerate dusty environments as well as other. They cough more, have more mucus, and they may get more short of breath, but that is a non-specific effect of any dust, not because it has aspergillus in it.

Q. The fact that he breathed in dust of any kind in September or October, did that result in his hospitalization beginning 12-31-99?

A. No.

Q. Would he have been hospitalized irrespective of whether or not he breathed in that dust?

A. I think if he got in for acute influenza A at Christmas of 1999, on top of his obstructive lung disease, the same thing would have happened.

Q. There has been some suggestion that removing Mr. Lansdell from exposure in the purchased chips area of the mill improved his condition. Would

you agree that there is a cause and effect in removing him from the purchased chips area and the improvement in his condition?

A. What time period are we talking about?

Q. When he went back to work, I think after his August, 2000 hospitalization, he was allowed to not have to rotate into the purchased chip area and his condition improved somewhat. Do you agree that there is a cause and effect relationship between the removal of his exposure to these great clouds of aspergillus?

A. I think that in somebody with obstructive lung disease such as he had, that they may have more symptoms of cough and perhaps slightly increased shortness of breath with exposure to any kind of dust, and that decreasing dust exposure might be associated with fewer symptoms.

Dr. Kerby testified that it was his opinion, given within a reasonable degree of medical certainty, that the claimant would have been hospitalized and suffered the same medical problems even if he had not been exposed to aspergillus in the workplace.

The Administrative Law Judge filed an opinion on August 21, 2002, and found, "Claimant has failed to prove by clear and convincing evidence that he sustained an occupational disease which arose out of and in the course of his employment." The claimant appeals to the Full Commission.

II. ADJUDICATION

A. Burden of Proof

Ark. Code Ann. § 11-9-601 formerly provided:

(e) (1) (A) "Occupational disease", as used in this chapter, unless the context otherwise requires, means any disease that results in disability or death and arises out of and in the course of the occupation or employment of the employee, or naturally follows or unavoidably results from an injury as that term is defined in this chapter.

(B) However, a causal connection between the occupation or employment and the occupational disease must be established by clear and convincing evidence.

Act 1281 of 2001 (effective July 1, 2001) substituted "a preponderance of the" for "clear and convincing" in (e) (1) (B). The Administrative Law Judge found in the present matter, "Act 1281 of 2001 made substantive law changes to the burden of proof for occupational disease claims and is thus to be applied prospectively." The Administrative Law Judge therefore found, "Claimant must establish the compensability of his occupational disease claim by clear and convincing evidence; the law in effect at the time claimant's claim arose."

The Administrative Law Judge erred as a matter of law. We considered this same issue in a companion case, Sikes v. Georgia-Pacific Corporation, Workers' Compensation

Commission F000657 (July 7, 2003), and we adopt the reasoning of Sikes in the present matter. The Full Commission therefore reverses the Administrative Law Judge on this issue, and we find that the Legislature meant for Act 1281 to apply retrospectively. The instant claimant must establish a causal connection between his occupation or employment and the occupational disease by a preponderance of the evidence.

B. Whether the claimant's disease arose out of his employment

The Administrative Law Judge found that the claimant failed to prove by clear and convincing evidence that he sustained an occupational disease which arose out of and in the course of his employment. The Full Commission finds that the claimant failed to establish by a preponderance of the evidence a causal connection between his occupation or employment and the occupational disease. We therefore affirm, as modified, the opinion of the Administrative Law Judge.

To prevail on his claim, the claimant must prove by a preponderance of the evidence a causal connection between

his employment and his medical problems. Ark. Code Ann. § 11-9-601 (e) (1) (B). Determination of a causal connection in the instant matter depends on our weighing the competing opinions of the physicians who have evaluated the claimant's condition. The Commission's authority to resolve conflicting evidence extends to medical testimony, and the Commission is entitled to judge the weight and credibility of a medical opinion. Maverick Transp. V. Buzzard, 69 Ark. App. 128, 10 S.W.3d 467 (2000).

We first note that in terms of qualifications alone, the opinions of Dr. Rector and Dr. Kerby are entitled to greater weight than that of Dr. Prychodko, given Dr. Kerby's impressive qualifications, and his and Dr. Rector's status as board certified in pulmonary disease. In previous cases involving pulmonary disease, the Commission has given the opinions of pulmonologists greater weight than the opinions of other physicians. See, e.g., Steve Tibbett v. Agricultural Productivity Co., Full Commission Opinion filed March 11, 1998 (E510708). Dr. Prychodko is not a specialist in pulmonary medicine, but rather in occupational and environmental medicine.

Far more important to the Commission's review, however, is the extensive testimony of Dr. Prychodko and Dr. Kerby as to how they reached their respective opinions and conclusions. A review of his deposition testimony reveals that Dr. Prychodko based his conclusions primarily, if not exclusively, on the coincidental timing between the onset of the claimant's symptoms and his exposure to aspergillus. At no point in his testimony, nor in his written reports, did Dr. Prychodko explain any physiological mechanism by which aspergillus could have caused the claimant's medical difficulties. Dr. Prychodko acknowledged that claimant does not meet the diagnostic criteria of allergic bronchopulmonary aspergillus, but he in essence testified that the mere presence of aspergillus was sufficient to prove a causal connection to the claimant's medical difficulties.

Dr. Kerby, on the other hand, offered credible testimony as to why aspergillus was present in the claimant's body. He explained that aspergillus is commonly found throughout nature. He explained that antibiotics, such as those used by the claimant at the onset of his flu

symptoms, could sufficiently rid the body of bacteria so as to allow the growth and colonization of a fungi such as aspergillus. He explained that the presence of fungal colonization in a patient taking antibiotics is normal and is typically ignored. He detailed the diagnostic criteria for the three illnesses which can be caused by aspergillus exposure, and he testified that the claimant has none of these illnesses. Neither the claimant nor Dr. Prychodko offered any evidence to contradict this testimony.

Likewise, Dr. Kerby offered a credible, detailed explanation of the physiological mechanism by which claimant's symptoms began, and a credible, detailed explanation of why the medical evidence does not support a causal connection between the claimant's medical problems and his aspergillus exposure. Dr. Kerby concluded that while the claimant may have suffered short-term problems due to organic toxic dust syndrome, his long-term lung and hip problems, medical treatment and hospitalizations are attributable to at least three non-work related conditions: influenza, which both the claimant and his wife apparently suffered from immediately prior to his initial

hospitalization; pneumonia, for which he sought hospitalization in August, 2000; and his preexisting smoking-related illnesses, which exacerbated the other conditions. Dr. Prychodko attempted to rebut Dr. Kerby's opinion that the claimant suffers from smoking-related illnesses, but Dr. Kerby credibly testified that Dr. Prychodko reached that conclusion by a misinterpretation of the claimant's lab results. Overall, we find Dr. Kerby's conclusions to be plausible and credible, and we find that neither Dr. Prychodko nor the claimant have offered a credible alternative theory.

It is not enough for the claimant to prove that he was sick and that he was exposed to aspergillus at his workplace. He must also prove a causal connection between the aspergillus and his medical problems. Dr. Prychodko's opinion of a causal connection is based on coincidence and timing. Dr. Kerby, however, offered a credible, detailed explanation of why the claimant became sick, and why his sickness is not causally related to his aspergillus exposure. The claimant has offered no other evidence to show a causal connection to his employment, and no evidence to

credibly contradict the opinion of Dr. Kerby. We must therefore find that claimant has failed to prove by a preponderance of the evidence a causal connection between his employment and his medical condition, and that he has therefore failed to prove an occupational disease arising out of and in the course of his employment.

III. CONCLUSION

Based on our de novo review of the entire record, the Full Commission finds that Act 1281 of 2001 was remedial or procedural legislation. As such, the Legislature intended for Act 1281 to operate retrospectively, so that the claimant must prove a causal connection between his occupation or employment and an occupational disease by a preponderance of the evidence, pursuant to Ark. Code Ann. § 11-9-601(e)(1)(B). The Administrative Law Judge erred as a matter of law in finding that Act 1281 was to operate prospectively by applying the "clear and convincing evidence" standard in the original statute. However, we find that the claimant failed to prove by a preponderance of the evidence that he sustained an occupational disease which arose out of and in the course of his employment. The Full

Commission therefore affirms, as modified, the opinion of the Administrative Law Judge. This claim is denied and dismissed.

IT IS SO ORDERED.

OLAN W. REEVES, Chairman

Commissioner Turner concurs in part and dissents in part.

CONCURRING AND DISSENTING OPINION

_____ While I agree with the finding in the principal opinion that Act 1281 of 2001 must be applied retroactively, I respectfully dissent from the finding that claimant failed to prove by a preponderance of the evidence that he sustained a compensable pulmonary injury.

Claimant worked approximately 30 years in respondent employer's wood room before rotating his duties between the wood room and the purchased ship areas every two months. As a wood room operator, claimant ran the barking drums and the chipper. In the purchased chip area, claimant worked as a wood prep operator. Shortly after working in the purchased chip area, claimant began having flu-like symptoms, "couldn't get a deep breath, kept a

headache, nausea, and it just kept getting worse." Claimant stated that the dust and mold from working with the wood chips caused his current lung problems. Respondent contends that claimant cannot show a causal connection between his work and his subsequent pulmonary difficulties.

Claimant had a resolved blood clot from 1995 and a history of smoking, which he discontinued in 1976. The medical records do not indicate a history of any lung complaints prior to this incident.

Claimant testified that he and several other employees informed the plant superintendent that they were becoming ill from the work. He believed October 24, 1999 was the date of injury based on an incident where his entire crew became ill while working, "...that day we had some extremely bad chip cars, extremely bad, and we all four got sick." Claimant stated that when they shook the cars, there was a cloud of green smoke from all the mold. He stated that he, and the other crew members, immediately experienced chest tightness, coughing, headache and shortness of breath.

Claimant was admitted to Little River Memorial Hospital on December 31, 1999, exhibiting symptoms of

shortness of breath and acute stress, "in general this well-developed, well-nourished, white male who is alert and oriented x3 who appears to be very ill, short of breath, unable to sit straight, leaning forward and is very dyspneic on minimal exertion or walking." He had decreased lung air flow and was transferred to Wadley Regional Medical Center with diagnoses of atypical pneumonia, hypoxemia and respiratory distress.

On January 1, 2000, claimant received a bronchoscopy with bronchoalveolar lavage and transbronchial lung biopsy and bronchial brushings. While no evidence of fungal infection was found in claimant's left lung lingual, there were many fungal organisms, as well as atypical squamous metaplasia, present in his left lower lobe. (Record, pp. 97-98).

Dr. Prychodko is Medical Director of the Center for Preventive Occupational and Environmental Medicine in the College of Medicine at the University of Arkansas for Medical Sciences. Additionally, he is board certified in Occupational Medicine by the American Board of Preventive Medicine and holds a Juris Doctor in an effort to examine

the interplay between the legal and medical aspects of occupational diseases. Dr. Prychodko also worked as a staff physician in the Department of Occupational and Environmental Medicine for the University of Texas Health Center where he met claimant. He noted (in part) on May 2, 2001:

In the vast majority of his nearly 33 years of employment there, [claimant] worked in the "wood room" as an operator. This area had relatively filtered ambient air, and Mr. Lansdell never had any respiratory problems working there. Indeed, he was in his usual good health until 1999, when he noted breathing difficulties while working in the "purchase chip" department.

Approximately two years ago, new work practices were introduced where [claimant] would have to rotate from the wood room to the purchase chip area on a 2-monthly rotation schedule. It was after this change in work practice was instituted that he developed respiratory health problems.

The purchase chip area involves direct exposure to circulating air that has directly swept through wood chips and contains airborne fungal spores. Wood chips are often stored in rail cars. There are approximately 40 - 60% chips required for the fine printing paper product manufactured at this facility. The longer the wood chips stay in the rail car, the more time there is for mold growth to take place and result in release of mold spores into the surrounding ambient atmosphere.

It is widely apparently known at that plant, and among its employees, that the turnover time for railcars containing wood chips is too long.

In his January 30, 2002, deposition, Dr. Prychodko added the following:

I was impressed that this gentleman had worked for a number of years at the facility and done reasonably well. I think it was in the wood chip room area; and there was a change in job practices, which involved his rotating to the - I think - purchased chip area, on a two-month interval.

And he began experiencing respiratory problems coincident to this change in occupational work schedule, work practices. In combination with that history, I was also impressed with the fact that there was apparently a delay in the turnaround time of the hard wood chip rail cars.

They normally should be processed within - I recall it was like ten days and normally have two of three turnarounds per month, as far as these hard wood chips that would be brought in. And they were staying in the yard for 20, 25 days, basically allowing accumulation, perhaps, of the mold. This is what I'm gathering from the information. (Deposition, pp. 8-9).

Dr. Prychodko ultimately opined that claimant's pulmonary problems were causally related to the overexposure to fungal spores at work.

Based on the above evidence, I find that claimant has met his burden of proof and, accordingly, the opinion of the Administrative Law Judge should be reversed.

SHELBY W. TURNER, Commissioner

Commissioner Yates concurs in part and dissents in part.

CONCURRING AND DISSENTING OPINION

I respectfully concur in part and dissent in part from the majority opinion. Specifically, I concur in the finding that the claimant failed to prove that he sustained an occupational lung disease. However, I must dissent from the majority's finding that the provisions of Act 1282 of 2001 are to be applied retroactively, as was stated in my dissent in Sikes v. Georgia Pacific Corporation, Full Workers' Compensation Commission Opinion filed July 7, 2003 (E200657). It remains my opinion that the provisions of Act 1281 of 2001 are to be applied prospectively, as opposed to retroactively, which a majority of the Commission has found.

In 2001, the General Assembly amended Ark. Code Ann. §11-9-601. Act 1281 of 2001 substituted "a preponderance of the" for "clear and convincing." Now occupational disease claims carry the same burden of proof as all other workers' compensation claims. Act 1281 of 2001 was both enacted and went into effect subsequent to the date the claimant's claim arose, as well as, subsequent to the date the claimant filed his claim. Thus, when the claimant's claim arose and as of the date the claimant filed

his claim, the Act placed a higher burden of proof upon the claimant. This higher burden of proof may allow the respondent to successfully resist the claim. If the new lower burden of proof is to be applied retroactively, then, in my opinion, the respondent's ability to successfully defend the claim may be removed. If the burden of proof is now retroactively lowered from clear and convincing evidence, or evidence so clear, direct, weighty and convincing, to a preponderance of the evidence or evidence of greater convincing force, then the respondent faces a prejudicial change in position.

Generally, the law in effect at the time of an injury governs the disposition of the case for the duration of the claim. Arkansas State Police v. Welch, 28 Ark. App. 234, 772 S.W.2d 620 (1989); Chism v. Phelps, 228 Ark. 936, 311 S.W.2d 297 (1957); Lucus v. Handcock, 266 Ark. 142, 583 S.W.2d 491 (1979). In Arkansas State Police v. Welch, supra, the Arkansas Court of Appeals stated:

It is well settled that changes in statutes relating only to remedies or procedural matters are generally held to be immediately applicable to existing causes of action and not just to those which may accrue in the future unless a contrary intent is expressed in the

statute. Fowler v. McHenry, 22 Ark. App. 196, 737 S.W.2d 663 (1987). However, any changes in statutes relating to vested rights are characterized as substantive and require application of the law as it existed at the time the claimant sustained a compensable injury. See id. A vested right exists when the law declares that one has a claim, or that one may resist enforcement of a claim. Forrest City Mach. Works, Inc. v. Aderhold, 273 Ark. 33, 616 S.W.2d 720 (1981).

In 1987, the workers' compensation act was amended to state that "... the commission shall weigh the evidence impartially and without giving the benefit of the doubt to either party." In determining whether this amendment was substantive or procedural, the Arkansas Court of Appeals stated in Fowler v. McHenry, 22 Ark. App. 196, 737 S.W.2d 663 (1987); "As we have seen, however, the burden of proof has always rested upon the claimant and this rule was not affected by the amendment." The Court concluded that a claimant did not have a vested right in the procedure the Commission uses to weigh the evidence and thus the amendment was not found to be substantive in nature. Accordingly, the Court applied the procedural change in the law retroactively. Although the Court in Fowler stated, "We

note that even if the amendment had changed the burden of proof, the amendment still might be fairly characterized as procedural," I find this statement in Fowler to be mere dicta and is not controlling in the case presently before the Commission. First, the amendment did not change the burden of proof, so the Court did not address or analyze a change in the burden of proof in a procedural or substantive light. Second, the amendment addressed the procedure for weighing the evidence - not the actual weight of evidence the claimant must produce in order to prevail.

All statutes are to be construed as having only a prospective operation, unless the purpose and intention of the legislature to give them a retrospective effect is expressly declared or is necessarily implied from the language used. See, Gannett River Pub. V. Ark. Dis. & Disab., 304 Ark. 244, 801 S.W.2d 292 (1990). A strict rule of construction applies to remedial statutes which do not disturb vested rights, or create new obligations, but only supply a new or more appropriate remedy to enforce an existing right or obligation. These types of statutes should be given a retrospective effect whenever that seems to have been the intention of the legislature. When

construing remedial legislation, one should give appropriate regard to the spirit which prompted the legislation's enactment, the mischief it sought to abolish and the remedy proposes. See, Aluminum Co. of America v. Neal, 4 Ark. App. 11, 626 S.W.2d 620 (1982).

It is clear from a review of Act 1281 of 2001 that the legislature did not intend a retrospective application. The changes brought on by Act 1281 of 2001 did not change the procedure or manner in which the Commission is to view the evidence - it changed the weight the evidence must carry. Act 1281 of 2001 lowered the burden of proof, and thus removed a vested right and valid defense of the respondent. See, Wall v. Doctor's Hospital, Full Commission Opinion filed December 12, 1990 (D502329) (1986 amendment requiring objective and measurable findings to support an impairment rating is substantive and cannot be retroactively applied.) Therefore, I find that Act 1282 of 2001 made substantive changes to the workers' compensation act and may not be applied to this claim retroactively.

Therefore, I must respectfully concur in part and dissent in part from the majority opinion.

JOE E. YATES, Commissioner