BEFORE THE INDUSTRIAL ACCIDENT BOARD
OF THE STATE OF DELAWARE

HARRY WALKER, Employee, v. STATE OF DELAWARE, Employer.	)	Hearing No. 1303216

Hrg officer rejects OD claim of curvularia fungal pneumonia

DECISION ON PETITION TO DETERMINE COMPENSATION DUE

Pursuant to due notice of time and place of hearing served on all parties in interest, the above-stated cause came, by stipulation of the parties, before a Workers' Compensation Hearing Officer on November 14, 2007, January 8, 2008 and January 23, 2008, in the Hearing Room of the Board, in New Castle County, Delaware.

PRESENT:

Natalie L. Palladino,
Workers' Compensation Hearing Officer

APPEARANCES:

Timothy E. Lengkeek, Attorney for the Employee

Dennis J. Menton, Attorney for Employer

NATURE AND STAGE OF THE PROCEEDINGS

Harry Walker ("Claimant") alleges that he developed invasive Curvularia fungal pneumonia as a result of his work at the Charter School for the State of Delaware ("Employer"). On May 30, 2007, Claimant filed a Petition to Determine Compensation Due seeking acknowledgement of the compensability of the Curvularia fungal pneumonia, with March 19, 2007 as the alleged manifestation date of injury. Claimant also seeks attorney's fees and medical witness fees. Employer disputes the diagnosis of Curvularia fungal pneumonia. Employer argues that Claimant suffers from allergic bronchial pulmonary Curvularia ("ABPC"), a genetically based condition that pre-disposes Claimant to allergically react to Curvularia. Further, Employer argues that Claimant's lung problems, which resulted from his ABPC, have no causal relationship to his work for Employer.

A hearing was held on Claimant's petition on November 14, 2007, January 8, 2008 and January 23, 2008. The parties stipulated that this matter could be heard and decided by a Workers' Compensation Hearing Officer, in accordance with 19 *Del. C.* § 2301B(a)(4). When hearing a case by stipulation, the Hearing Officer stands in the position of the Industrial Accident Board. *See* 19 *Del. C.* § 2301B. This is the decision on the merits.

SUMMARY OF THE EVIDENCE

Stephanie A. Lee, M.D., testified by deposition on behalf of Claimant (Claimant's Exhibit 1). Dr. Lee is board certified in infectious disease. Dr. Lee first saw Claimant in April 2006, when he was admitted to Wilmington Hospital with an acute onset of symptoms suggestive of pneumonia. His symptoms included a four-to-five day history of shortness of breath or difficulty breathing, fever, productive cough and pleuritic chest pain. Dr. Steve Cozamanis, a hospitalist for Christiana Care, admitted Claimant with an initial impression of

community-acquired pneumonia. Dr. Cozamanis wanted to rule out atypical pneumonia and environmental allergies. Claimant was seen in consultation by Dr. David M. Cohen, one of Dr. Lee's partners. Dr. Cohen noted in the "social history" of his report that Claimant was concerned that he acquired pneumonia from the school where he worked as a teacher because it was a moldy and dusty environment. During Claimant's hospitalization from April 20, 2006 through April 24, 2006, he was treated with an antibiotic, Levaquin, and given pain medication for the pleuritic chest pain. He was stabilized and discharged.

From May 1-11, 2006, Claimant was admitted to Christiana Hospital with similar symptoms. He was initially seen in consultation by Dr. Anand P. Panwalker, another partner of Dr. Lee. Claimant's symptoms were so severe there was a concern that he had an underlying lung malignancy. A bronchoscopy was ordered, but the procedure was stopped before completion because Claimant's oxygen saturation levels dropped. The biopsy portion of the procedure was not performed. However, a large mucus plug was removed. Some of Claimant's symptoms resolved after removal of the mucus plug, so there was no longer a concern that Claimant had cancer.

According to Dr. Lee, the mucus plug contained Curvularia, a very rare mold. However, Curvularia has rarely been reported to cause invasive diseases and there was no tissue biopsy to confirm that any invasive disease was present. Claimant was given Vorinconazole, an anti-fungal medicine. However, he developed anaphylaxis after two doses and the medication was stopped. Claimant was also being treated simultaneously for community-acquired pneumonia, which was thought to be the more likely cause of his symptoms.

Dr. Panwalker asked Claimant to return to the hospital later in May 2006, so that intravenous Amphotericin, a very toxic antifungal medication could be administered. Claimant

had to be admitted and monitored because the medication causes significant side effects. When Claimant returned to the hospital on May 12, 2006, he was feeling a little better. Dr. Lee wanted to have another bronchoscopy performed, but it was unable to be scheduled over the weekend. She did not want to admit Claimant and administer the toxic anti-fungal medications until she had proof of the disease, so she advised Claimant to continue taking his antibiotic medications and asked him to return the following Monday. In the meantime, Dr. Lee spoke to a pulmonologist who indicated that it would be difficult to obtain a biopsy from the area of Claimant's lung that appeared to be problematic. Therefore, when she spoke to Claimant the following Monday, they decided to monitor his symptoms and re-address the issue if Claimant's condition worsened. Claimant also saw an infectious disease physician at the University of Pennsylvania for a second opinion. The U-Penn physician agreed that Curvularia pneumonia is such a rare cause of invasive disease that Claimant most likely had community-acquired pneumonia.

Claimant was examined by the physicians at Dr. Lee's practice on May 17, 2006, May 22, 2006 and June 26, 2006. Claimant's condition was improving, so they decided not to pursue the tissue biopsy. Claimant worked outside as a camp counselor during the summer. He was slowly resuming his exercise regimen and playing lacrosse.

Dr. Lee did not see Claimant again until March 19, 2007, when he was re-admitted to the hospital with "the exact same symptoms." Claimant had returned to work as a teacher at the Charter School in the fall of 2006. He strongly believed that the musty, damp conditions at the school were the cause of his recurring problems. Claimant underwent a CT scan that showed the pneumonia was in the same location as it had previously occurred, which meant that it had never completely resolved. The condition of Claimant's left lung was so severe that a thoracic surgeon

had to remove a rib and the left upper lobe of his lung. Dr. Lee received a phone call from the pathologist who analyzed the lung tissue. The pathologist indicated that he found an invasive mold species consistent with Curvularia. There was a considerable degree of necrotizing granuloma formation and destruction. At that time, Claimant was diagnosed with Curvularia fungal pneumonia. According to Dr. Lee, it is a very rare diagnosis, there are only ten or twelve case reported in the medical literature. Further, it is usually found in immuno-compromised individuals and it is often fatal.

After the lung resection surgery, Claimant went to Dr. Lee's office for daily doses of intravenous Amphotericin. He was also referred for pain management treatment because he had severe pain in the area where the rib was removed that was not controlled with narcotics. Dr. Lee explained that removal of a portion of the lung resolved Claimant's pneumonia symptoms, but the surgery caused considerable nerve pain.

Dr. Lee reviewed the August 8, 2007 defense medical examination report of Dr. Frederick Cogen in which he concluded that Claimant suffered from an allergic bronchial pulmonary Curvularia ("ABPC"). Dr. Lee explained that ABPC is an allergic reaction to mold that, unlike Curvularia fungal pneumonia, does not invade and damage the lung tissue. If Dr. Cogen's diagnosis is correct, then Claimant would not have needed the lung resection. However, Dr. Lee disagrees with Dr. Cogen's diagnosis because Claimant did not present with wheezing, asthmatic-type symptoms, esinophiles were not present throughout the course of treatment, and Claimant had invasive disease.

Dr. Lee also reviewed the July 21, 2006 report prepared by BATTA Environmental Associates, Inc. *See* Claimant's Exhibit 2. The environment testing, which was performed approximately 40 days after Claimant's initial onset of symptoms, revealed the presence of

Curvularia in Room 329 at the Charter School. Dr. Lee testified that she found it "very interesting that Curvularia was found in the school where [Claimant] worked given the rarity." She believes that it is more likely than not that Claimant developed Curvularia fungal pneumonia from his exposure to mold at the school. Dr. Lee noted that Claimant had no allergic reactions as an adult prior to working at the school. As a gym teacher, Claimant spent most of the spring and fall days working outside. It was not until later winter, when Claimant was working in the building all day, that his symptoms occurred.

On cross-examination, Dr. Lee agreed that a CT scan performed on December 1, 2006 showed considerable bronchiectasis and scarring, evidence of a serious infection. She also agreed that a pulmonary infiltrate shown on a CT scan and chest film may be caused by a non-infectious process such as an inflammatory or autoimmune disorder that causes tissue destruction. As far as Dr. Lee knows, however, Claimant's screening tests for autoimmune disorders were negative. Dr. Lee also agreed that when Claimant was first treated in March 2006 he had elevated levels of IgE and very mildly elevated levels of esinophiles, the type of blood cells that respond during an allergic reaction. However, the slightly elevated esinophiles were not found to be significant. She did not know "what to make of" the elevated IgE in terms of Claimant's presentation and diagnosis.

Dr. Lee does not see many patients with allergic bronchiopulmonary mycosis ("ABPM") because they are usually treated by allergists or pulmonologists. She last treated a patient with that condition in 2005 during her fellowship. She does not know how common ABPM is in our society.

Dr. Lee also agreed that Curvularia is normally found outside in the soil and on plants. It is essentially everywhere. Dr. Lee testified that only one colony of mold was found in Room

329 when BATTA performed its environmental testing on June 1, 2006. When 1Source performed environmental testing at the Charter School on May 16, 2006, it found no Curvularia mold inside or outside of the school. 1Source did not test Room 329. However, Dr. Lee was under the impression from the 1Source report and photographs of various areas of the Charter School that it had a significant mold problem and several areas of concern were identified. Further, it is unknown how much Curvularia is necessary to cause a negative health effect.

Steven Woronicak, an Indoor Air Quality Manager from BATTA Environmental Services, Inc. testified on behalf of Claimant. Mr. Woronicak was hired by the Red Clay Consolidated School District to perform environmental testing at the Charter School. He performed the testing on June 1, 2006. At that time, Mr. Woronicak knew that there was a general concern regarding rooms 329 and 118, although he did not know about Claimant's illness. When he arrived at the school, he was also asked to look at the teacher's lounge and the main office. Mr. Woronicak collected indoor and outdoor samples. The outdoor samples were for comparison purposes. He also performed a visual inspection. Mr. Woronicak's report lists the types of mold identified and the location where each type of mold was found. *See* Claimant's Exhibit 2. One colony of Curvularia, a very small amount, was found in Room 329. It was not found in the outdoor samples. Mr. Woronicak's visual inspection revealed some water stained ceiling tiles and other minor problems common for schools and other large buildings. *See* Claimant's Exhibit 3.

Mr. Woronicak testified that there was nothing of significant concern identified during the testing. No remediation was recommended. He just suggested that the school replace the damaged ceiling tiles. Mr. Woronicak testified that, according to industry standards, the test findings are concerning only if the level of mold found is 4 or 5 times greater than the outdoor

concentration. However, Mr. Woronicak is not a medical doctor, so he cannot identify the level of mold exposure necessary to cause disease.

With respect to the Curvularia, Mr. Worocinak testified that the one colony identified could have been a rogue spore brought into the building from the outside. Curvularia is commonly found in plant debris and soil. It is all over the place.

Claimant testified on his own behalf. He played football, lacrosse and wrestled for four years in high school. He then played rugby and summer league lacrosse for five years while attending college at the University of Delaware. After college, Claimant played rugby with the Wilmington Men's Rugby Club.

When Claimant was seven or eight-years-old, he had sinus problems, i.e., a runny nose for 2-3 months. He received some allergy shots. Once or twice a year, he also had hay fever symptoms. However, Claimant never had any problems with asthma or shortness of breath prior to working at the Charter School.

Claimant started working as a gym teacher at the Charter School in the fall of 2005. He also coached football, basketball and lacrosse. At that time, he was in excellent health. In the fall and spring, his gym classes were usually taught outside. His office was located in the boys' locker room, which is in the basement under the pool. It was very damp. Claimant also covered homeroom classes and study halls in various rooms. He specifically recalls covering the physics class in Room 329 on several occasions. Room 329 was memorable to Claimant because it contained robotics equipment. Claimant was asked to cover homeroom in room 329 when the physics instructor and some of his students were away at robotics competitions.

In November 2005, Claimant began having cold-like symptoms, including coughing, congestion, a runny nose and sinus pressure. The symptoms persisted until the spring of 2006.

However, on Sunday evenings Claimant usually felt better. Claimant's wife and son did not have any symptoms. Claimant first sought treatment for the symptoms with his family doctor, Dr. Charles R. Sharbaugh, in January 2006. Dr. Sharbaugh thought Claimant's symptoms might be related to acid reflux, allergies or exercise induced asthma. He prescribed an inhaler. Claimant's condition worsened, so in March 2006, he sought treatment with Dr. Bernard F. King at the recommendation of his wife and a friend. Dr. King sent Claimant to the emergency room because he thought Claimant had pneumonia. The emergency room physician diagnosed community acquired pneumonia or environmental allergies.

On April 20, 2006, Claimant was admitted to the hospital and given antibiotics. In May 2006, he was re-admitted to the hospital. When a bronchoscopy was performed, Claimant coughed up some mucus that contained Curvularia fungus. Dr. Panwalker recommended that he take Voriconazole. He started taking the medication via a PICC line, but it caused hives and swelling. The medication was discontinued and he was given an IV antibiotic. Claimant did not return to work at the Charter School during the remainder of the 2005-2006 school year.

During the summer of 2006, Claimant worked outside at St. Anthony's summer camp. He testified that he did not have any significant symptoms during the summer. However, Claimant's medical records apparently reflect that he called Dr. Lee on July 6, 2006 and July 26, 2006 to report complaints of coughing, fatigue and a runny nose. Claimant could not recall making those phone calls.

In the fall of 2006, Claimant returned to work at the Charter School. In December 2006 he started having significant symptoms once again. Claimant's symptoms in 2006 were the same as they had been in 2005, except they were worse. He started coughing up blood and Dr. Lee advised him to go directly to the hospital. Claimant was admitted to the hospital and some

additional testing and a bronchoscopy was performed. A few days later, Claimant was advised that a portion of his left lung needed to be removed. According to Claimant, the surgeon found quite a bit of damage and fungus in his left lung. He was diagnosed with fungal pneumonia.

In April and May 2007, Claimant was recovering from the surgery. His breathing was better, but he had extreme pain in the area where his rib was removed. Three nerve blocks were performed at Brandywine Pain Management. The nerve blocks were helpful. Claimant has a 13 inch scar on his back and has some difficulty moving, but he is "trying to get back in the game." He never returned to work at the Charter School because he knew that it made him sick. He is now working at Thomas Edison Charter School. His symptoms have not recurred.

Dr. Stephen J. Rodgers testified on behalf of Claimant. Dr. Rodgers is board certified in preventative medicine, environmental and occupational medicine. Dr. Rodgers opined that Claimant had reactive airway disease and a Curvularia fungal infection, both causally related to his work at the Charter School. He explained that the two conditions are not mutually exclusive, so he essentially agrees with Dr. Cogen's diagnosis of APBC and Dr. Lee's diagnosis of Curvularia fungal pneumonia.

Dr. Rodgers noted that Claimant had elevated levels of IgE and esonophiles, suggesting an allergy. Dr. Sharbaugh and Dr. King also suspected that Claimant had some type of an allergy. However, the later records document pleural pain and abnormalities on the chest x-rays and CT scans that are evidence of infection from the mold. Fungal pneumonia is rare, in general. It can occur in healthy individuals, but more frequently occurs in people with immune problems such as individuals who are HIV positive or undergoing chemotherapy. Dr. Rodgers is not aware of any studies regarding fungal pneumonia in patients with a normal immune system.

Dr. Rodgers causally relates Claimant's fungal pneumonia to his work because there is testing showing that Curvularia was present in his workplace in Room 329 and the bronchosccopy and post-surgical pathology findings are consistent with that diagnosis. Further, Claimant's history of symptoms, i.e., the evidence that he allergically reacted to his indoor work environment at the Charter School, is evidence of ABPC. Dr. Rodgers testified that Curvularia is a common outdoor fungus associated with vegetation, but it can also be found indoors.

On cross-examination, Dr. Rodgers agreed that he initially diagnosed Claimant with "reactive airway disease" or asthma. It was not until Dr. Rodgers reviewed Dr. Cogen's report and the medical literature cited by Dr. Cogen that he labeled Claimant's condition as APBC. Dr. Rodgers has never personally treated any patients with APBC or fungal pneumonia. He did not review any medical literature prior to offering his initial opinion that Claimant had reactive airway disease and fungal pneumonia. However, Dr. Rodgers explained that "reactive airway disease" is simply a less precise way to describe APBC. Claimant responds to Curvularia with an allergic or asthma-like reaction, i.e., the reactive airway disease.

After reviewing the literature cited by Dr. Cogen, Dr. Rodgers noted that one of the papers had been criticized after its publication. Dr. Cogen cited a 2002 position paper from the American College of Occupational Medicine ("ACOM"), which stated that molds do not produce toxins and it is unlikely that humans would be injured by mold. However, a 2007 New York Times or Wall Street Journal article noted that there was internal strife at ACOM about the article. Further, the doctors who wrote the article were involved in the defense of mold cases. According to Dr. Rodgers, there is also a 2007 article from the International Journal of Occupational and Environmental Health that criticizes several ACOM position papers over the years, including the 2002 ACOM position paper cited by Dr. Cogen. Specifically, it states that

the 2002 paper was not evidenced based, i.e., the research cited did not, overall, support the proposition. Specifically, rodent studies were used to support the proposition that mold does not cause human injury. And again, it was noted that the there was a conflict of interest because the doctors who wrote the article were testifying on behalf of the defense in mold exposure cases.

Frederick C. Cogen, M.D., testified on behalf of Employer. Dr. Cogen is board certified in immunology and allergy. *See* Dr. Cogen's Curriculum Vitae, Employer's Exhibit 1. He examined Claimant on August 8, 2007 at Employer's request. He also reviewed Claimant's pertinent medical records, the BATTA and 1Source reports, and Dr. Lee's deposition transcript. Dr. Cogen disagrees with the diagnosis of invasive fungal pneumonia. He believes that Claimant has a classic case of ABPC. Further, Dr. Cogen opined that Claimant's ABPC is not causally related to his workplace.

Dr. Cogen is currently treating approximately 25-27 patients with ABPM. He is familiar with the medical literature regarding the condition; the criteria for diagnosing the condition; and treatment of the condition. Dr. Cogen explained that ABPM is never related to the workplace, home or any other environment. It is a condition unique to the host. Some individuals are simply genetically programmed to be more susceptible to ABPM. APBC usually responds well to treatment with steroid and anti-fungal medications. There are several criteria relevant to a diagnosis of ABPM. The patient must meet at least 3-4 criteria to make a diagnosis. However, according to Dr. Cogen, Claimant meets all of the criteria.

First, Claimant has a history of allergic reactions. He received allergy shots as a child and more recently had an allergic reaction to anti-fungal medication. Second, Claimant has lingering pulmonary infiltrations that have affected more than one area of his lungs. Claimant's x-rays and CT scans showed problems in the left upper and lower lobes. Third, Claimant had a

mucus plug, which is a symptom of ABPM but not pneumonia. Fourth, Claimant had elevated esonophiles during his initial hospitalization in 2006. Dr. Cogen explained that elevated esonophiles are a response to an allergen, they are not a sign of fungal infection of fungal pneumonia. Fifth, Claimant continued to have lingering symptoms and the lung resection surgery did not resolve Claimant's symptoms. Claimant made several phone calls to Dr. Lee in July 2006 regarding ongoing symptoms. Further, in October 2007, approximately six months after his lung resection surgery, Claimant reported to Dr. Goodill that he was coughing, wheezing and sleeping sitting up. Dr. Cogen also noted that fungal pneumonia does not cause wheezing. Sixth, and most importantly, the December 2006 CAT scan showed bronchiectasis or dilation of the breathing tubes, which is caused only by ABPM. Seventh, in March 2007, Claimant's IgE level was 514. The normal level is 13.2. An elevated IgE level is characteristic of ABPM, but not pneumonia or even normal allergies. Eighth, Claimant reacted to several molds and strongly to Curvularia upon skin testing. Further, Curvularius was cultured after it was removed from Claimant's airway during the May 2006 bronchoscopy.

Dr. Cogen agreed that Claimant's pathology report noted some features characteristic of invasive fungal pneumonia. For example, granulomas may occur in either fungal pneumonia or APBM. However, Dr. Cogen explained that when a patient has a true case of invasive fungal pneumonia, one will find fungus all over the place, for example, invading the blood vessels. It is often lethal. Therefore, Dr. Cogen believes that the pathology report suggests that Claimant has ABPM not fungal pneumonia.

According to Dr. Cogen, there are ten cases of ABPC cited in the medical literature, none of which document a case of infective pneumonia from Curvularia in someone with a normal immune system. However, ABPC has been shown to cause skin boils, wound infections, etc.

There is also a case where ABPC caused an abscess in the lung of an immune compromised person. Yet, despite Dr. Rodgers' testimony that fungal pneumonia and ABPC are not mutually exclusive, Dr. Cogen has never seen anyone who had both conditions simultaneously.

Dr. Cogen agreed with Dr. Rodgers' testimony that there was a 2007 editorial article from the International Journal of Occupational Health criticizing the ACOM organization and the authors of the 2002 ACOM article based on alleged conflict of interest/bias issues. However, according to Dr. Cogen, the 2002 ACOM article is evidenced based and there has been no contradictory evidence presented to refute the 2002 conclusions. Further, regardless of the validity of the 2002 ACOM paper, this case is not about mold toxicity. The true issue is whether Claimant suffered from ABPM or pneumonia. The seven articles that Dr. Cogen initially referred to are about ABPM, they have nothing to do with invasive fungal pneumonia.

Harry M. Neal from 1Source Safety and Health, Inc., testified on behalf of Employer. Mr. Neal is a certified industrial hygienist. He has been the vice president of 1Source since 2000. In May 2006, Mr. Neal was asked by Dr. Ron Russo, the principal of the Charter School. to perform some air quality testifying. Mr. Neal was informed that Claimant had pneumonia related to Curvularia fungus. When assessing the school, he was specifically looking for Curvularia, but also generally examining the building for moisture issues.

On May 16, 2007, Mr. Neal collected dust, air and surface samples to look for Curvularia. Mr. Neal inspected several areas of the Charter School, including the office in the boys' locker room, the shower area, the faculty lounge and room 327. *See* Employer's Exhibit 2 (map of the Charter School identifying areas that Mr. Neal inspected). He did not obtain any samples from Room 329. Mr. Neal found no Curvularia as a result of any of the air samples, dust samples and surface swab samples. There was some visible mold growth on ceiling tiles

and a 20 linear foot area behind the cove base in the faculty lounge. *See* Employer's Exhibit 3 (Mr. Neal's June 12, 2006 report with attached photographs of water damage and mold growth). The dust samples also contained some normal plant molds. The air samples were consistent with the outdoor findings.

Mr. Neal opined that all visible mold growth should be abated according to the EPA standards. However, the spot areas he identified and photographed are normal for older school buildings like the Charter School and do not reflect the overall condition of the school.

Further, Mr. Neal explained that Curvularia is not related to water damage. It is generally an outdoor fungus that lives on grass and plants, although a dry spore is sometimes brought into a building by air currents.

Mr. Neal agreed that he is not a medical doctor. He does not know how much mold one must be exposed to or how long an exposure must be before an individual will suffer an injury. However, he testified that a finding of one colony of any fungus "does not raise a red flag." It is insignificant.

Brain Moore testified on behalf of Employer. Mr. Moore is the Facilities Manager for Red Clay Consolidated School District. According to Mr. Moore, the Charter School is a tenant in the building that co-exists with several other schools. Mr. Moore was advised by the chief custodian at the building that 1Source performed some air quality testing. Mr. Moore then notified BATTA that it would like some testing performed. The chief custodian showed Steven Woronicak from BATTA around the building and pointed out potential problem areas.

Ronald Russo, the President of the Charter School, testified on behalf of Employer. According to Mr. Russo, Claimant was hired as a physical education instructor and para-professional for the 2005-2006 school year. He continued to work as a physical education

instructor and coach during the 2006-2007 school year, although Claimant stopped working in April 2007. Mr. Russo reviewed Claimant's schedule and determined that he worked primarily in the gym/locker room area during the 2005-2006 school year. He also had study halls in rooms 306A, 321 and 327, but not in room 329. From September 2006 through April 2007, Claimant worked primarily in the gym and room 306A, not room 329. According to Mr. Russo, Claimant would only have worked in room 329 if he was asked to cover a physics class which was regularly held in that room.

When Mr. Russo learned of Claimant's fungal pneumonia, he called 1Source to schedule the inspection. Mr. Russo's primary concern was the gym and locker room area. Mr. Russo was also aware that there was some prior water damage in the faculty lounge as a result of the refrigerator and air conditioning unit leaking. Mr. Russo asked 1Source to look at Room 327 because there were some stained ceiling tiles in that room. Once Mr. Neal issued his findings, steps were taken to remediate the problems identified by 1Source.

Mr. Russo also notified Red Clay Consolidated School District about Claimant's illness and concerns. Red Clay indicated that it wanted to perform its own inspection. Mr. Russo thought it was a good idea for the additional testing to be performed because BATTA could inspect different areas and cover as much of the building as possible. Mr. Russo received a copy of the BATTA report.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Compensability

The Delaware Workers' Compensation Act provides that employees are entitled to compensation "for personal injury or death by accident arising out of and in the course of

employment."[1] Because Claimant filed the current Petition, he has the burden of proof.[2] In this case, Claimant alleges that he sustained invasive Curvularia fungal pneumonia as a result of his work for Employer. Employer disputes the diagnosis of fungal pneumonia and argues that Claimant has ABPC, a condition that is causally unrelated to his work. Thus, the issues that must be decided at this time are: (1) the diagnosis of Claimant's condition; and (2) its causal relationship, if any, to Claimant's work for Employer. Claimant has the burden of proving his injury and causation by a preponderance of the evidence.[3]

Since Claimant is alleging an occupational disease, compensability is predicated on a showing that "the employer's working conditions produced the ailment as a natural incident of the employee's occupation in such a manner as to attach to that occupation a hazard distinct from and greater than the hazard attending employment in general." *Anderson v. General Motors Corp.*, Del. Supr., 442 A.2d 1359, 1361 (1982); *Air Mod Corporation v. Newton*, Del. Supr., 215 A.2d 434, 442 (1965). I must, therefore, determine whether Claimant has established that it is more likely than not that he contracted invasive Curvularia fungal pneumonia as a natural incident of his teaching and coaching duties at the Charter School, attaching to his occupation a hazard distinct from and greater than the hazard attending employment in general. For the reasons that follow, I hold that Claimant has failed to meet that burden.

To begin, I found Dr. Cogen's opinion persuasive that Claimant suffers from ABPC and did not have invasive Curvularia fungal pneumonia. The doctors agreed that invasive fungal pneumonia is a rare condition that generally occurs only in immuno-compromised individuals

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[1] 19 *Del. C.* § 2304.

[2] 29 *Del. C.* § 10125(c).

[3] *See Goicuria v. Kauffman's Furniture*, Del. Super. Ct., C.A. No. 97A-03-005, Terry, J., 1997 WL 817889 at * 2 (October 30, 1997), *aff'd*, 706 A.2d 26 (Del. 1998).

and if often fatal. Although Claimant had some symptoms that are common to both fungal pneumonia and ABPC, Dr. Cogen clearly explained how Claimant met all of the criteria relevant to a diagnosis of ABPC. Further, several of the criteria relevant to the diagnosis of ABPC are not symptoms of pneumonia, such as the mucus plug, elevated esonophiles, the bronchioectasis demonstrated by CAT scan, the elevated IgE level, the allergic reaction to Curvularia on skin testing, and the wheezing and ongoing symptoms Claimant reported to Dr. Goodill after his lung resection surgery.

Dr. Rodgers agrees that Claimant has ABPC. Although Dr. Rodgers initially diagnosed Claimant with reactive airway disease, he explained that it was simply a less precise way to describe ABPC. According to Dr. Rodgers, Claimant responds to exposure to Curvularia with an allergic or asthma-like reaction. Dr. Rodgers agreed that the elevated levels of IgE and esonophiles initially found in March 2006 suggested that Claimant had an allergy. Dr. Rodgers also noted that Dr. Sharbaugh and Dr. King, Claimant's primary care physicians, initially suspected that Claimant was suffering from allergies or asthma.

Dr. Rodgers opined that Claimant also suffered from invasive Curvularia fungal pneumonia, as evidenced by the abnormalities shown on Claimant's diagnostic studies and the pathology report. However, I found Dr. Cogen's testimony persuasive that the diagnostic and pathology findings were more suggestive of ABPC. Dr. Cogen articulately explained why he felt that the pathology, despite some features characteristic of both invasive fungal pneumonia and ABPC, was more consistent with a diagnosis of ABPC. Further, Dr. Cogen noted that Claimant's December 2006 CAT scan showed bronchiectasis, a condition that is caused only by ABPC. Finally, Dr. Cogen also testified that there are no known cases of a non-immuno-compromised patient that concurrently had ABPC and invasive fungal pneumonia.

Dr. Lee disagreed with the diagnoses of ABPC because Claimant did not present with wheezing, asthma-type symptoms; esonophiles were not present throughout the course of Claimant's treatment; and he had invasive disease. However, Dr. Lee agreed that when Claimant initially presented in March 2006 he had mildly elevated esonophiles that were dismissed as an insignificant finding and she "did not know what to make of" the elevated IgE. Dr. Lee also agreed that pulmonary infiltrates shown on CAT scans or chest x-rays can be caused by non-infectious processes such was inflammation or autoimmune disorders that cause tissue destruction. Finally, Dr. Lee agreed that she has not treated a patient with ABPM since 2005, during her fellowship, and does not know how common ABPM is in the general population.

For all of these reasons, I found Dr. Cogen's opinion that Claimant suffers from ABPC, and did not have invasive Curvularia fungal pneumonia, the most persuasive. That finding, however, does not necessarily mean that Claimant's lung problems were causally unrelated to his work. According to Dr. Cogen and Dr. Rodgers, ABPC is a genetically based, pre-existing problem that predisposes Claimant to allergically react to Curvularia. However, "[a] pre-existing disease or infirmity, whether overt or latent, does not disqualify a claim for workers' compensation if the employment aggravated, accelerated, or in combination with the infirmity produced the disability."[4] Thus, if Claimant's working conditions, in combination with his genetic pre-disposition, triggered his lung problems then his condition is a compensable work injury.

In this case, however, I did not find sufficient evidence from which to conclude that Claimant's working conditions at the Charter School triggered his ABPC to become symptomatic. Claimant testified that his work environment, especially the area where his office

[4] *Reese*, 619 A.2d at 910.

was located, was very damp and musty. Further, Dr. Lee and Dr. Rodgers based their causation opinions on Claimant's history of increased symptoms after working indoors at the Charter School.

However, all of the expert witnesses agreed that Curvularia, the particular type of mold that allegedly caused Claimant's injury (whether the invasive Curvularia fungal pneumonia or ABPC) is a ubiquitous mold normally found outdoors in the soil and on vegetation. As Mr. Worocinak and Mr. Neal explained, a rouge spore is sometimes brought into a building from outside. However, Curvularia is not an indoor mold associated with water damage. The environmental testing performed by BATTA and 1Source confirmed that there were some areas of the Charter School where prior water damage had occurred and mold was present. However, 1Source found no Curvularia at the Charter School and BATTA found only one colony in Room 329.

In light of the evidence that Curvularia is a ubiquitous outdoor mold, and only one colony was identified indoors at the Charter School during environmental testing, I did not find the opinions of Dr. Lee and Dr. Rodgers convincing that the damp, moldy indoor working conditions at the Charter School caused Claimant's injury. Claimant is alleging injury due to an outdoor mold, which by all accounts, is present in much greater quantity in the general outdoor environment than his indoor working environment. Accordingly, I reject the causation opinions of Dr. Lee and Dr. Rodgers and hold that Claimant has failed to meet his burden of demonstrating a work related injury.

STATEMENT OF THE DETERMINATION

FOR THESE REASONS, I DENY Claimant's May 30, 2007 Petition to Determine Compensation Due.

IT IS SO ORDERED THIS 5th day of August 2008.

INDUSTRIAL ACCIDENT BOARD

Natalie L. Palladino

Natalie L. Palladino,
Workers' Compensation Hearing Officer

Mailed Date:
8/6/08

OWC Staff