

**IN THE SUPERIOR COURT OF THE STATE OF DELAWARE
IN AND FOR NEW CASTLE COUNTY**

HARRY WALKER,)
)
Employee Below/Appellant,)
) C.A. No. 08A-08-016 PLA
v.)
)
STATE OF DELAWARE,)
)
Employer Below/Appellee.)

**ON APPEAL FROM THE
INDUSTRIAL ACCIDENT BOARD OF THE STATE OF DELAWARE
IN AND FOR NEW CASTLE COUNTY
AFFIRMED**

Submitted: April 21, 2009
Decided: May 18, 2009

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ABLEMAN, JUDGE

I. Introduction

This is the Court's decision on an appeal from the Industrial Accident Board's ("the Board") denial of the Petition for Compensation Due filed by the appellant, Harry Walker ("Walker"). During the time period he was employed as a physical education instructor and paraprofessional at the Charter School ("Charter"), Walker began experiencing severe respiratory symptoms that treating physicians related to exposure to Curvularia, a mold that grows on plants and soil. Walker suffers from a rare reaction to a common substance: Curvularia is ubiquitous in the outdoor environment, and rogue spores frequently travel indoors as well.

Walker sought workers' compensation benefits on the basis that he suffered a bilateral lung injury due to Curvularia exposure occurring at Charter. After a lengthy hearing, the Board denied Walker's petition, holding that he failed to establish causation and that his condition therefore did not constitute a compensable occupational disease.

The Court concludes that substantial evidence supported the Board's determination that Walker had not met his burden of establishing causation. Accordingly, for the reasons set forth in this opinion, the Board's decision must be affirmed.

II. Factual and Procedural History

A. Factual Background

In the fall of 2005, Walker was hired as a physical education instructor and paraprofessional at Charter. This position continued Walker's life-long involvement in sports and fitness. He participated in the football, wrestling, and lacrosse teams in high school, played for his college's Division I rugby and lacrosse teams, and joined the Wilmington Men's Rugby Club after graduating from university. By all accounts, Walker was in good physical health when he started working at Charter School.

Walker's position at Charter encompassed a variety of responsibilities. His primary duty was to teach four gym classes per day. These gym classes were held outdoors during the fall and spring, but moved indoors for winter, starting around November. In addition, Walker supervised a homeroom and study hall periods, and would serve as a substitute teacher when a colleague was unavailable. According to Walker, his substitute teaching took him "all over the building," and brought him to Room 329, a physics classroom, several times a month.¹

In late November 2005, Walker began experiencing allergy-type symptoms, including coughing, sinus and nasal congestion, and a runny

¹ Indus. Accident Bd. Hr'g Tr., 101-02 [hereinafter Hr'g Tr.].

nose. Walker observed that his symptoms seemed to diminish when he was absent from the school building over the course of his weekends. Neither Walker's wife nor his son experienced any symptoms. Walker had observed mold and dampness in the school's basement locker room, where his office was located, and he became suspicious that his symptoms were related to these conditions. He visited his family doctor in January 2006 and was prescribed an inhaler, but his symptoms persisted.

Walker's condition worsened throughout spring 2006. Blood tests performed in March showed elevated levels of the antibody IgE and mildly elevated levels of white blood cells known as eosinophils. In April, he was admitted to the hospital and given a course of antibiotics to treat shortness of breath, chest pain, and a cough, which doctors attributed to bacterial community-acquired pneumonia. A CT scan revealed pulmonary infiltration. Walker was hospitalized a second time in early May 2006 for continued pulmonary symptoms.

During this time period, Walker began treatment with Drs. Stephanie A. Lee and Anand Panwalker, who are infectious disease specialists. Concerned that Walker might have cancer, Dr. Lee recommended a bronchoscopy to biopsy tissue from the area of infiltration revealed on the CT scan. During the procedure, a mucus plug was extracted from Walker's

airway. Walker's oxygen saturation level dropped during the bronchoscopy, and the procedure was aborted before a sample of Walker's lung tissue could be extracted. Nonetheless, testing was performed on the mucus plug, which was found to contain *Curvularia*.

The presence of *Curvularia* in the mucus plug, combined with the lack of a lung tissue sample, presented Walker's doctors with a diagnostic quandary. *Curvularia* is an extremely common mold, primarily found outdoors, where it feeds on plant proteins. It is not associated with indoor water damage. Although *Curvularia* is so prevalent in the outdoor environment that the medical experts for both parties agreed that it is "ubiquitous," it rarely causes invasive disease (i.e., fungal pneumonia) in humans. In addition, invasive *Curvularia* pneumonia would have been particularly unusual in a patient without some underlying condition causing him to be immune-compromised.

Without a tissue biopsy, doctors could not determine whether the *Curvularia* was causing invasive disease in Walker. His doctors, after consulting with a University of Pennsylvania infectious disease specialist for an additional opinion, continued to treat him for community-acquired pneumonia, which they considered to be a more likely cause of his severe symptoms than invasive *Curvularia*.

Following the bronchoscopy, Walker's symptoms abated somewhat. He did not return to Charter for the remainder of the 2005-2006 school year. Dr. Lee considered attempting another lung biopsy, but consultation with a pulmonologist convinced her that it would be very difficult to obtain tissue from the necessary location. Because Walker's condition appeared to be improving, he and Dr. Lee decided to forego both the biopsy and treatments with potent antifungal medications in favor of monitoring him.

In the wake of Walker's hospitalizations, both Charter's principal and the Red Clay Consolidated School District hired consultants to conduct mold studies at the school. The first study was performed by Harry Neill of One Source on May 17, 2006. One Source did not identify any *Curvularia* inside or outside the school, but did report dampness in the locker rooms, as well as mold in the faculty lounge and ceiling tiles in various locations. The second study, performed by Steven Woronicak of BATA Environmental Associates, was conducted on June 1, 2006. The BATA study identified a single colony of *Curvularia* in Room 329, a location not tested by One Source. Testing performed outside the school found no *Curvularia*.

Walker worked outdoors as a camp counselor during the summer of 2006, and his physical condition steadily improved. Walker recounts that he felt much better by late June 2006. He did not miss any work as a result of

health problems over the summer, although records indicate that he called Dr. Lee on July 6 and July 26 with complaints of fatigue, runny nose, chest tightness, and cough. Walker attributed those symptoms to allergies.

Walker resumed his position at Charter in fall 2006. In November 2006, he began having breathing difficulties, chest pain, and coughing. His symptoms became increasingly severe during the winter months, although he did not seek treatment with Dr. Lee in the fall or winter. By March 2007, he was experiencing intense chest pain, running fevers, and coughing up blood. He was hospitalized on March 19, and a consulting pulmonologist recommended that a portion of his left lung be surgically removed.

On March 29, 2007, Walker underwent the lung resection surgery, which required removing one of his ribs and severing a nerve. A pathological examination of the resected lung tissue revealed the presence of a mold species consistent with *Curvularia*, although the specific mold could not be definitively identified.

After recovering from his surgery, Walker did not return to work at Charter, but has since found employment at another school. He has a thirteen-inch scar on his back from the operation, and experiences some difficulty with movement. He has not had a recurrence of the acute symptoms that arose during his time at Charter, but continues to experience

some respiratory symptoms following the surgery, which he attributes to the loss of a portion of his lung.

On May 30, 2007, Walker filed a petition to determine compensation due for bilateral lung injury caused by mold exposure. The Board held a hearing that spanned three days in November 2007 and January 2008.²

B. IAB Hearing

At the hearing, the Board heard extensive testimony: Walker described the course of his illness; Charter's president and facilities manager testified regarding air quality testing performed at Charter; Harry Neil and Steven Woronicak testified as industrial hygiene experts to explain the results of the two air quality tests; and medical experts for both sides offered their opinions as to diagnosis and causation.

A significant portion of the medical testimony revolved around a dispute over Walker's diagnosis. At issue was whether Walker's pulmonary illness was caused by invasive *Curvularia* fungal pneumonia (ICFP) or allergic bronchopulmonary Curvulariosis (ABPC). ABPC is a specific form of allergic bronchopulmonary mycosis (ABPM), an umbrella diagnosis for

² By stipulation, the hearings in this case were conducted by a Hearing Officer, pursuant to 19 *Del. C.* § 2301B. Under § 2301B, the Hearing Officer's decision is treated as the decision of the Board.

bronchopulmonary allergic reactions to various types of fungi. As previously discussed, invasive *Curvularia* is an extremely rare fungal pneumonia, which occurs when the *Curvularia* fungus actually invades and damages lung tissue. ICFP is often fatal. ABPC is a genetic condition that causes an allergic reaction to *Curvularia*.³ Unlike ICFP, ABPC does not result in fungal invasion of lung tissue or blood vessels, although both diagnoses are associated with pulmonary symptoms. ABPC patients generally respond well to treatment with steroids and antifungal medication.

To briefly summarize the experts' positions, Walker presented testimony from Dr. Lee and from Dr. Stephen Rodgers, who is board-certified in preventative, environmental, and occupational medicine. As Walker's treating physician, Dr. Lee diagnosed him with ICFP based upon the pathological analysis of his resected lung tissue that suggested invasive fungal infection, his lack of a history of adult allergy or asthma symptoms, and the fact that he did not present to her with wheezing or asthmatic symptoms. Dr. Rodgers opined that Walker had both ICFP and ABPC. Charter offered the testimony of Dr. Frederick C. Cogen, an allergist and

³ Much of the hearing testimony, as well as the decision below, often referred to Walker's diagnosis as ABPM. For clarity, especially given the proliferation of medical terminology necessarily involved in this case, the Court will refer to ABPC, the more specific diagnosis, wherever possible.

immunologist, who rejected the ICFP diagnosis offered by Walker's experts and opined that Walker presented a "classic case" of ABPC.

Dr. Cogen explained that ABPC is diagnosed where a patient satisfies three or four of eight criteria. His "absolute" certainty regarding Walker's diagnosis was based on the fact that Walker met all eight diagnostic criteria for ABPC. Specifically, Dr. Cogen identified the following findings as indicative of ABPC: (1) Walker's history of allergic reactions, including allergic rhinitis and asthma as a child, as well as an allergic reaction to one of the antifungal drugs with which he was treated for his *Curvularia*-related illness; (2) the existence of wandering and intermittent pulmonary infiltrates, as shown in CAT scans and multiple X-rays taken during Walker's acute illness; (3) elevated levels of eosinophils, indicative of an allergic reaction; (4) the presence of a mucus plug in Walker's airway; (5) symptoms of bronchiectasis, or dilation of the breathing tubes; (6) extremely high levels of IgE, a condition known as "hyper-IgE" that is characteristic of only three or four particular diseases, including ABPM; (7) Walker's strong reaction to several molds, including *Curvularia*, on a skin test; and (8) the presence of *Curvularia* mycelia in Walker's airway, detected during his bronchoscopy. Of these diagnostic criteria for ABPC, Dr. Cogen noted that several of Walker's symptoms—including elevated eosinophils, mucus plug formation,

and the appearance of infiltrates in multiple locations—would not be expected in a patient with ICFP. Furthermore, Dr. Cogen indicated that the persistence of some symptoms following Walker’s surgery suggested that he did not have ICFP, which would have resolved completely after the tissue resection.

The medical experts also presented divided conclusions regarding whether Walker’s illness was causally related to his employment. Both Drs. Lee and Rodgers opined that Walker’s illness was related to *Curvularia* exposure at Charter. Walker’s medical experts based their conclusion upon the discovery of a colony of *Curvularia* in Room 329, the evidence of mold in Walker’s mucus plug and lung tissue, and the pattern of Walker’s symptoms increasing when he worked indoors at Charter.

Dr. Cogen, however, testified that employment at Charter did not cause the ABPC with which he diagnosed Walker. Moreover, Dr. Cogen stated that ABPC is a genetic condition of the host patient and that the scientific literature indicates that it “has nothing to do with . . . exposure to any particular geographic site.”⁴ Because molds such as *Curvularia* are ubiquitous, Dr. Cogen testified, “no doctor can tell you where anybody picked up a mold that caused a real mold illness, and in this case there are

⁴ Hr’g Tr., 177, 198.

genetic tests that show that people like Harry Walker have a host problem that sets them up for this [reaction].”⁵

During his testimony, Dr. Cogen acknowledged that his report mistakenly stated that no *Curvularia* had been found in the Charter school building. He testified, however, that the test results identifying a single colony of *Curvularia* in Room 329 did not change his opinions as to Walker’s diagnosis or the causation of his ABPC.⁶ On cross-examination, he first suggested that his incorrect belief at the time he wrote his report that no *Curvularia* had been found in the building “had something to do with my conclusion [that Walker’s condition was not work-related].”⁷ He also explained that there is no identified “threshold” of mold exposure necessary to cause mold-induced illness and that two patients with allergies to the same substance may be triggered by different amounts of the allergen.⁸ However, Dr. Cogen continued to assert that his opinion remained

⁵ *Id.* at 211.

⁶ *Id.* at 208-09.

⁷ *Id.* at 216.

⁸ *Id.* at 215-217.

unchanged and was “based on the fact that this disease is not related to any specific site.”⁹

C. Decision of the Board

In considering Walker’s Petition, the Board identified diagnosis and causation as the two central issues before it. The Board concluded that Walker suffered from ABPC that was not causally related to his employment. In rendering its decision, the Board accepted the testimony of Dr. Cogen over that offered by Drs. Rodgers and Lee.

As to Walker’s diagnosis, the Board was persuaded by the exact match between Walker’s symptoms and the diagnostic criteria for ABPC, as well as the fact that several of the ABPC criteria Walker displayed were not symptoms associated with ICFP. Furthermore, the Board noted that Dr. Cogen treated numerous patients with ABPM. By contrast, Dr. Lee had not treated any patients with ABPM since 2005. Dr. Lee dismissed Walker’s elevated eosinophil levels as insignificant and testified that she “did not know what to make of” Walker’s elevated IgE levels,¹⁰ while Dr. Cogen’s ABPC diagnosis accounted for both results. As to Dr. Rodgers’s opinion

⁹ *Id.* at 215.

¹⁰ Dep. Tr. of Dr. Lee, 48.

that Walker suffered from ABPC and ICFP simultaneously, the Board was persuaded by Dr. Cogen's testimony that the dual diagnosis was not consistent with Walker's symptoms and that there has never been a reported case of a patient with a normal immune system having both conditions at the same time.¹¹ Furthermore, the Board noted that Dr. Rodgers had never personally treated a patient with either ABPC or ICFP.

The Board continued by observing that even though ABPC is a "genetically based, pre-existing problem that predisposes [Walker] to allergically react to *Curvularia*," this fact does not necessarily mean that his condition is not compensable. Ultimately, however, the Board found that Walker had failed to meet his burden of establishing a causal connection between his work and his ABPC. Both Dr. Rodgers and Dr. Lee mentioned that the Charter school building was damp and musty, but the Board found that these conditions could not form a basis for a finding of causation, because *Curvularia* is not associated with indoor water damage. The Board acknowledged that Walker's medical experts had based their opinions on his increased symptoms while working inside at Charter, but found their testimony unpersuasive in light of the testimony from all of the medical

¹¹ Decision on Pet. to Determine Compensation Due, 17-19 (Indus. Accident Bd. Aug. 19, 2008) [hereinafter Bd. Decision].

experts that *Curvularia* is “present in much greater quantity in the general outdoor environment than [in Walker’s] indoor working environment.”¹² The Board therefore rejected the causation opinions offered by Walker’s medical experts and held that Walker had not established that his condition was work-related.

III. Parties’ Contentions

Walker’s appeal does not challenge the Board’s determination that his diagnosis was ABPC and not ICFP, but he alleges two points of error in the Board’s decision regarding causation. First, Walker contends that the Board’s conclusion that he had failed to establish causation was not supported by substantial evidence. Walker argues that he presented substantial evidence that his condition was work-related, and that Dr. Cogen’s testimony did not rebut that evidence. Walker also urges that Dr. Cogen’s opinion was unsupported by substantial evidence and should not have been accepted by the Board. Second, Walker asserts that the Board erred by misapplying the standard of review regarding causation to bar his claim based upon his genetic predisposition to ABPC.

¹² *Id.* at 20.

In response, Charter argues that the Board's decision was supported by substantial evidence and free from factual or legal errors. First, Charter asserts that the Board acted within its discretion when it credited Dr. Cogen's opinions regarding diagnosis and causation over those of Walker's medical experts. Furthermore, Charter submits that the opinions of Drs. Lee and Rodgers that Walker's condition was work-related were based upon inconsequential and insufficient evidence. Charter argues that the single colony of *Curvularia* found in Room 329 does not support causation, and that Walker presented no scientific literature or other medical evidence to contravene Dr. Cogen and establish that ABPC can be causally related to a specific location.

Charter also claims that Walker's medical experts misunderstood the alleged cycle of increasing symptoms during his indoor teaching period. Charter notes that Walker did report symptoms while he was working outside and off Charter's grounds, and argues that Walker's increased symptoms did not correspond with his increased work hours inside the Charter building during the winter, because he did not seek treatment with Dr. Lee from the summer of 2006 until the spring of 2007. In addition, Charter contends that Walker's pulmonary symptoms continued after he had separated from his employment at Charter in the fall of 2007.

IV. Standard and Scope of Review

Upon appeal from a decision of the Board, this Court's function "is confined to ensuring that the Board made no errors of law and determining whether there is 'substantial evidence' to support the Board's factual findings."¹³ Substantial evidence means "such relevant evidence as a reasonable mind might accept as adequate to support a conclusion."¹⁴ The "substantial evidence" standard requires "more than a scintilla but less than a preponderance of the evidence."¹⁵

The Court "does not weigh the evidence, determine questions of credibility, or make its own factual findings."¹⁶ These functions are reserved exclusively for the Board.¹⁷ The Court must afford "a significant degree of deference to the Board's factual conclusions and its application of

¹³ *Bermudez v. PTFE Compounds, Inc.*, 2006 WL 2382793, at *3 (Del. Super. Aug. 16, 2006).

¹⁴ *Anchor Motor Freight v. Ciabottoni*, 716 A.2d 154, 156 (Del. 1998).

¹⁵ *Breeding v. Contractors-One-Inc.*, 549 A.2d 1102, 1104 (Del. 1988).

¹⁶ *Hall v. Rollins Leasing*, 1996 WL 659476, at *2 (Del. Super. Oct. 4, 1996) (citing *Johnson v. Chrysler Corp.*, 213 A.2d 64, 66 (Del. 1965)).

¹⁷ *Giofre v. C.G. Capital Group*, 1995 WL 264585, at *3 (Del. Super. Apr. 17, 1995), *aff'd*, 670 A.2d 1338 (Del. 1995) (TABLE).

those conclusions to the appropriate legal standards.”¹⁸ In reviewing the evidence, the Court must consider the record “in the light most favorable to the prevailing party below.”¹⁹ The Court reviews questions of law *de novo* to determine “whether the Board erred in formulating or applying legal precepts.”²⁰

V. Analysis

The issue facing the Board in this case was whether Walker’s condition constituted a compensable occupational disease. Delaware’s Workers’ Compensation Act defines “compensable occupational diseases” as “all occupational diseases arising out of and in the course of employment only when the exposure stated in connection therewith has occurred during employment.”²¹ In an occupational disease claim, the claimant must establish causation under the test set forth by the Delaware Supreme Court in *Anderson v. General Motors Corporation*,²² which provides:

¹⁸ *Bermudez*, 2006 WL 2382793, at *3 (citing 29 *Del. C.* § 10142(d)).

¹⁹ *Id.*

²⁰ *Id.*

²¹ 19 *Del. C.* § 2301(4).

²² 442 A.2d 1359 (Del. 1982) (per curiam).

[F]or an ailment or disease to be found to be a compensable occupational disease, evidence is required 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.²³

In *Anderson*, the Supreme Court held that an employee failed to establish that his employment at an automotive assembly plant had caused his allergic rhinitis, given that the treating physician testified that the employee-claimant's allergies were attributable to both household and factory dust, and natural pollen—in other words, “the stimuli [of] the everyday world.”²⁴ The Court held that medical expert testimony that the dust and fumes at the employer's plant “triggered” the employee's allergy-related breathing difficulties was insufficient to establish causation:

[E]ven if employee's ailment . . . were a pre-existing but latent condition, evidence of employee's physician that it was aggravated or “triggered” on the job is . . . insufficient [to establish causation]. The question is whether employee's ailment was occupational in nature; and simply because there was evidence to believe it had either been contracted or aggravated on his employer's premises is legally insufficient to find it to have been an occupational disease.²⁵

²³ *Id.* at 1361.

²⁴ *Id.*

²⁵ *Id.*

Of particular relevance to this case, the *Anderson* Court stated that its holding did not imply “that a person predisposed to a disability may, in no event, establish disability from an occupational disease.”²⁶ The standard for a compensable occupational disease could be met where there was evidence “of any incidence of [the disability to which the employee-claimant was predisposed] within the employee’s work force at the [workplace] or other evidence of a causal connection” between the employee’s work and his disability.²⁷ However, a condition will not be compensable unless the claimant can “establish by substantial competent evidence that his ailment resulted from the peculiar nature of the employment rather than from his own peculiar predisposition.”²⁸

Here, the Court concludes that substantial evidence supports the Board’s factual findings, and that the Board correctly applied the two-pronged *Anderson* test in determining that Walker failed to establish causation. Walker’s own experts testified that *Curvularia* is ubiquitous in the outdoor environment, and that rogue spores often enter the indoor

²⁶ *Id.*

²⁷ *Id.*

²⁸ *Anderson*, 442 A.2d at 1361.

environment.²⁹ Although the medical experts disagreed on the potential significance of the single colony of *Curvularia* identified in Room 329 of Charter, their testimony coincided with that of the industrial hygiene experts in noting that it was a “very small count”³⁰ not meriting remediation and did not suggest that Charter had a problem with excessive indoor *Curvularia*.

The only other evidence that might suggest a connection between Walker’s illness and his employment was his apparent cycle of increasingly severe symptoms during his time working indoors at Charter. The real crux of this appeal therefore concerns whether this cycle of symptoms, together with the presence of at least some *Curvularia* inside Charter, was sufficient evidence for Walker to satisfy *Anderson*’s causation standard. As will be discussed further *infra*, the Court finds that the Board properly exercised its discretion to accept Dr. Cogen’s expert opinion that these facts were not probative as to causation. The evidence presented to the Board made clear not only that exposure to *Curvularia* is a pervasive risk of everyday living, but also that Walker’s employment at Charter did not present any heightened or peculiar risk such that his ABPC could be considered occupational in origin.

²⁹ Hr’g Tr., 72, 162; Dep. Tr. of Dr. Lee, 58.

³⁰ Dep. Tr. of Dr. Lee, 61.

Walker raises several challenges to the Board's acceptance of Dr. Cogen's testimony, none of which have merit. First, Walker argues that Dr. Cogen's opinion was unsupported because his expert report erroneously stated that no *Curvularia* had been found inside Charter. During his hearing testimony, however, Dr. Cogen corrected this error and explained that the discovery of one colony of *Curvularia* in Room 329 did not change his opinion.³¹ Thus, the Court is satisfied that neither Dr. Cogen nor the Board were relying upon the incorrect information contained in the expert report.

Walker further claims that Dr. Cogen's testimony fails to adequately rebut the causation evidence presented by Walker's witnesses. Walker relies upon *O'Neal v. Diamond Fuel Oil*³² to attack the Board's acceptance of Dr. Cogen's testimony over that of his two medical experts, as well as its conclusion that his employment at Charter did not cause his ABPC. In *O'Neal*, the employee of an oil heater service company claimed that his cumulative employment-related exposure to a certain heating oil, known as heating oil No. 2, caused chronic interstitial nephritis, a serious kidney disease that required him to undergo a kidney transplant. The employee presented two medical experts who opined that the employee's kidney

³¹ Hr'g Tr., 208-09, 215.

³² 1998 WL 731562 (Del. Super. Aug. 20, 1998), *aff'd*, 734 A.2d 1060 (Del. 1999).

disease was caused by heating oil No. 2. His treating physician explained that the employee had been in excellent health prior to his kidney disease and had no other risk factors for kidney disease other than his exposure to heating oil No. 2. Her opinion as to causation was thus based upon the employee's medical records and self-reported history, and did not include any explanation of how the chemical composition of heating oil No. 2 could produce kidney failure. A second expert found causation based upon the employee's history, medical probability, and medical research literature relating kidney disease to exposure to hydrocarbons, including petroleum-based compounds. Although heating oil No. 2 was a hydrocarbon compound, none of the studies presented were related specifically to it.³³

The employer in *O'Neal* presented the expert testimony of a nephrologist, who indicated that there was "no way to tell any specific causes of the [employee's] condition from objective medical testing."³⁴ The employer's expert went on to state that, despite the opinion offered by the employee's expert, there was "no 'clear-cut' evidence in the literature that heating oil No. 2 . . . or any hydrocarbon has ever caused chronic interstitial

³³ *Id.* at *3.

³⁴ *Id.*

nephritis.”³⁵ Perhaps as a result of this position, the employer’s expert did not obtain a medical history, nor did he familiarize himself with the composition of heating oil No. 2.

The Board concluded that the employee had failed to establish causation under *Anderson*, stating that the employee’s experts could only “suggest” a link between hydrocarbon exposure and chronic kidney disease, but did not present any “studies that are even close to being definitive.”³⁶ The Board held that the employee had not established the existence of a “recognizable link” between his kidney disease and “some distinct feature of his job.”³⁷

This Court reversed the Board’s decision, and the Delaware Supreme Court affirmed. As the Supreme Court explained:

It is important to note at the outset that the circumstances of this case do not present, in the usual context, conflicting expert opinions each supported by substantial evidence from which the Board would be free to accept one expert’s opinion over another. Employer’s expert . . . did not opine as to the cause of [Employee’s] kidney disease nor testify that chronic exposure to heating fuel oil #2 was not a substantial cause of the disease. He merely testified to his belief that no one could opine that

³⁵ *Id.*

³⁶ *Id.* at *4.

³⁷ *Id.*

[Employee's] exposure was more probable than not the cause of his kidney condition.³⁸

Thus, the Board could not rely upon this testimony of employer's medical expert to negate "the opinions of two qualified experts, one of whom was the treating physician."³⁹ The Board's decision was also flawed in requiring that the employee present "definitive" evidence of causation, because the legal standard under *Anderson* does not require such a high level of certainty.

Viewing the testimony in light of the different questions of causation presented by the two cases, it becomes clear that this case involves "the usual context" not presented in *O'Neal*, in which the Board must weigh "conflicting expert opinions each supported by substantial evidence" and choose which opinions to credit. Although there are superficial resemblances to the facts of *O'Neal*, Walker's case is distinguishable.

O'Neal did not address the activation or aggravation of symptoms arising from a pre-existing genetic condition. The causation issue in *O'Neal* revolved around whether the employee had presented sufficient evidence linking the origin of his disease to heating oil No. 2, a chemical to which he had only been exposed while in the workplace. Notably, the employer's

³⁸ 734 A.2d at 1064 (citations omitted).

³⁹ *Id.* at 1065.

Material Safety Data Sheet indicated that heating oil No. 2 posed a hazard of degenerative kidney changes if inhaled for prolonged periods of time in high concentrations, which the Supreme Court considered “some evidence of causation.”⁴⁰

By contrast, there is no dispute in this case that Walker’s symptoms arose because he was exposed to Curvularia. There is also no dispute that Walker was exposed to at least some Curvularia while working at Charter. At issue here is whether Walker’s exposure to Curvularia in the workplace was causative in the sense that it produced Walker’s ABPC as a “natural incident” of his employment and presented a “hazard distinct from and greater than the hazard attending employment in general,” given the ubiquity of the mold, the fact that ABPC is a genetic condition, and medical literature suggesting that ABPC is never caused by a particular location.

Unlike the employer’s medical expert in *O’Neal*, Dr. Cogen provided an opinion based on his review of Walker’s history and medical records, as well as applicable medical literature, that workplace exposure to Curvularia did not cause Walker’s ABPC. Dr. Cogen also testified, based upon peer-reviewed scientific literature, that ABPC cannot be caused by any specific location, because it is a genetic condition of the host. Walker’s medical

⁴⁰ *Id.* at 1064.

experts did not present any scientific literature or other medical evidence to support that ABPC could be caused by exposure to *Curvularia* at a particular location. Indeed, because Dr. Lee did not diagnose ABPC, her opinion as to causation would apply only to ICFP, a diagnosis that the Board properly exercised its discretion to reject. The Board did not, as in *O'Neal*, erroneously require the employee to present “definitive” proof of causation. Rather, it chose to credit the testimony of Dr. Cogen over that of Walker’s medical experts. The Board, as trier of fact, is free to resolve conflicts between experts’ opinions by accepting the opinion of one expert over another, provided that both opinions are supported by substantial evidence.⁴¹ This Court will not disturb the Board’s choice.

Walker also posits that the Board could not accept Dr. Cogen’s opinion as to causation because it failed to offer an alternative explanation for the pattern of Walker’s symptoms, which increased during periods when he worked inside the Charter building and lessened when he was away from the school. Walker’s argument draws upon *San Juan v. Mountaire Farms*, in which this Court reversed the Board’s decision to accept the testimony of an employer’s expert that a claimant’s wrist disease was not occupational.

⁴¹ See, e.g., *DiSabatino Bros. v. Wortman*, 453 A.2d 102, 106 (Del. 1982); *San Juan v. Mountaire Farms*, 2007 WL 2759490, at *3 & n. 29 (Del. Super. Sept. 18, 2007).

The employee had presented medical testimony, supported by scientific literature, that the repetitive minimal wrist trauma associated with the employee's work de-boning chickens caused his disease. The employer's expert contested that repetitive trauma could have caused the wrist disease, but "could offer no other basis for causation" and never opined as to whether the usual stress and strain of employment was a substantial factor in the development of the disease, which was the applicable test of causation.⁴²

Neither *San Juan* nor any other authority imposes a requirement that an employer's expert must offer an alternative explanation for all of a claimant's symptoms or the pattern of their occurrence. In *San Juan*, the employer's expert failed to provide *any* opinion as to causation,⁴³ and thus there was no alternative theory of causation for the Board to accept. In the case at bar, Dr. Cogen not only opined that Walker's illness was not work-related, but provided an alternative explanation as to causation: he explained that Walker's ABPC is a genetic condition, and is never caused by a particular location.

⁴² *San Juan*, 2007 WL 2759490, at *3-4.

⁴³ *Id.* at *3 ("[T]hough the Board was free to accept [the opinion of the employer's expert] about etiology, the Board was not free to accept his opinion in isolation of all other evidence, especially where [he] could offer no opinion as to what caused the claimant's . . . disease.").

Moreover, the emphasis Walker places on cycle-of-symptoms evidence as it pertains to causation is misplaced. Because he has conceded that ABPC is the correct diagnosis, even if his apparent cycle of symptoms demonstrates that employment at Charter triggered, activated, or aggravated his ABPC, this evidence would not suffice to establish causation.

To explain why this is so, it is necessary to clarify how Walker's genetic predisposition to react allergically to *Curvularia* affects the standard for establishing causation. The Board's decision, quoting *Reese v. Home Budget Center*, stated that "[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."⁴⁴ The Board therefore concluded that "if Claimant's working conditions, in combination with his genetic predisposition [to ABPC], *triggered* his lung problems then his condition is a compensable work injury."⁴⁵

In its original context, the quoted language from *Reese* referred to the compensability of psychiatric symptoms related to an employee's physical

⁴⁴ Bd. Decision, 19 (quoting *Reese v. Home Budget Ctr.*, 619 A.2d 907, 910 (Del. 1992)).

⁴⁵ *Id.* (emphasis added).

injuries sustained in an identified work accident.⁴⁶ Walker notes that, notwithstanding the particular facts of *Reese*, it has been cited by the Board for the principle that an employee is entitled to compensation if his employment triggered or aggravated a pre-existing condition even in decisions involving occupational diseases not traceable to an identifiable accident—as in the Board’s decision in this case.⁴⁷ Walker therefore urges that *Reese* applies to his case, and that he is entitled to compensation because, even accepting Dr. Cogen’s diagnosis of ABPC, he has presented substantial evidence that exposure to Curvularia at Charter triggered or aggravated his condition.

Contrary to Walker’s argument, *Reese* is inapposite. The issue in *Reese* was “the extent of compensability for the non-physical or psychological consequences of a physical injury and, more importantly, the required nexus between the two.”⁴⁸ In explaining its holding that a pre-existing disease or infirmity did not disqualify a workers’ compensation claimant if employment aggravated or triggered the condition, the Delaware Supreme Court stated: “If [an] injury [caused by a work-related accident]

⁴⁶ *Reese*, 619 A.2d at 910.

⁴⁷ Docket 12 (Appellant’s Reply Br.), at 11.

⁴⁸ *Reese*, 619 A.2d at 909.

serves to produce a further injurious result by precipitating or accelerating a previous, dormant condition, a causal connection can be said to have been established.”⁴⁹ It was unnecessary to “quantify causation [by requiring a showing that employment was a ‘substantial cause’ of the aggravation or triggering of the pre-existing condition] where . . . the claim for compensation is *linked to a specific undisputed work related accident.*”⁵⁰

As the language of *Reese* indicates, its holding applies in typical “eggshell plaintiff” (or, more properly, “eggshell claimant”) situations, where a chain of causation can be established because an underlying work-related accident triggered or aggravated the pre-existing condition. But however it has been subsequently applied—or misapplied—by the Board in later cases, *Reese* does not supplant the causation test set forth in *Anderson* for occupational diseases that are unconnected to work-related accidents. Walker’s condition, unlike that of the employee in *Reese*, is not related to an identifiable accident, and *Reese* is therefore inapplicable.⁵¹

⁴⁹ *Id.* at 910.

⁵⁰ *Id.* (emphasis added).

⁵¹ See also *Spencer v. E.I. DuPont de Nemours & Co.*, 2005 WL 388264, at *3 (Del. Super. Feb. 11, 2005) (“The standard of causation for an occupational disease is different from that applied to an injury allegedly caused by an identifiable industrial accident or an employment-based aggravation of a pre-existing condition.”).

Because *Anderson* supplies the proper standard, evidence that Walker's employment at Charter triggered or aggravated his disease would be insufficient to establish causation. In its analysis, the Board found that Walker's employment at Charter did not trigger his condition. Walker argues, and the Court agrees, that an allergic reaction to a particular substance requires not only a genetic pre-disposition in the host, but exposure to the allergen. If, for instance, Walker spent his entire life in the polar regions, where Dr. Cogen indicated that *Curvularia* is not found,⁵² it stands to reason that his ABPC would never have become symptomatic. Because exposure to the allergen must occur at *some* physical location, it is not strictly true that an allergic reaction to even a ubiquitous substance is never "related" (in the lay sense of the term) to a geographic location or locations, although it may be difficult or impossible to pinpoint where exposure occurred when the allergen is, like *Curvularia*, a common substance.

The Board's "triggering" analysis appears to blend the concepts of triggering and causation. In explaining why it concluded that Charter did not "trigger" Walker's ABPC, the Board noted that *Curvularia* is ubiquitous in the outdoors, that only a single colony was identified in testing of the

⁵² See Hr'g Tr., 211.

school building, and that the damp and musty conditions at Charter were irrelevant to the question of whether Walker was exposed to Curvularia, as it is not associated with dampness. The Board's decision did not explain why it concluded that Walker's ABPC was not triggered by his employment at Charter given that Walker lacked any history of adult allergy or pulmonary problems prior to his working at Charter and that, even if they did not abate completely, his symptoms significantly decreased in severity when he was away from the school. Together with the presence of at least some Curvularia within the school, these facts suggest that working inside the Charter building may have triggered Walker's ABPC. The Board's failure to either explain or explicitly reject Walker's argument regarding his cycle of symptoms and lack of prior ABPC reactions renders unclear the basis for its conclusion that employment at Charter did not trigger Walker's disease.

This lack of clarity may stem from the Board's error in attempting to apply *Reese*. *Reese* and *Anderson* present irreconcilable rules regarding whether evidence of triggering or aggravation alone can satisfy causation, yet the Board identified both *Reese* and *Anderson* as applicable precedents. The Board correctly found that Walker had not established causation under *Anderson*. Then, in what appears to be an attempt to ensure that this conclusion would not conflict with *Reese*—which provides that evidence

that employment triggered a latent condition is sufficient to establish causation—the Board determined that Charter could not have triggered Walker’s ABPC, without addressing Walker’s evidence to the contrary.

Although the Board’s conclusion that Charter did not trigger Walker’s ABPC is unclear, this does not undermine the Board’s overarching no-causation finding. Walker does not contest the diagnosis of ABPC, and the Board accepted Dr. Cogen’s testimony that ABPC is a disease of the host that results from a genetic predisposition. When Dr. Cogen repeatedly asserted that ABPC is never “related” to a location, he was stating, in non-legal terms, that ABPC is never *caused by* a particular place or environment, even if, logically speaking, it seems ABPC must be triggered or aggravated by exposure to Curvularia. Thus, at the most, the presence of a colony of Curvularia in the Charter building, Walker’s lack of previous reactions during adulthood, and his apparent cycle of increasing symptoms during his indoor time at Charter provide evidence that Walker’s employment triggered or aggravated his ABPC. But *Anderson* makes clear that evidence of triggering or aggravation, without more, is an insufficient basis upon which to find that Walker’s ABPC is compensable.

Walker claims Charter’s position that “ABPC is never related to any geographic place” leads to the incorrect conclusion that an allergic reaction

to a workplace irritant would never be compensable. Although the law in this area is somewhat muddled, the Court agrees with Walker that *Anderson* leaves open the possibility that an allergic condition could constitute a compensable occupational disease; nevertheless, in this particular case, Walker has not met his burden in establishing causation.

In *Smith v. Service Tire Truck Center, Inc.*, this Court interpreted *Anderson* to mean that “when an allergy is triggered by the employment of one who is predisposed to that allergy, it is insufficient to establish compensability.”⁵³ The *Smith* Court emphasized language in *Anderson* requiring that an occupational disease “be one which is commonly regarded as natural to, inhering in, an incident and concomitant of, the work in question. There must be a recognizable link between the disease and some distinctive feature of the claimant’s job, common to all jobs of that sort.”⁵⁴ Thus, the Court affirmed the Board’s decision to deny compensation to the employee of a tire repair center whose employment triggered a previously latent allergic reaction to latex.⁵⁵

⁵³ 2000 WL 145817, at *4 (Del. Super. Jan. 19, 2000).

⁵⁴ *Id.* at *4 (quoting *Anderson*, 442 A.2d at 1360).

⁵⁵ *Id.* at *1, 4.

Smith suggests that an employee predisposed to an allergy or allergy-related disease whose illness is triggered or aggravated by his employment can never establish causation under any circumstances. It appears, however, that the meaning of “compensable occupational disease” under *Anderson* would encompass allergy-related conditions that are triggered or aggravated by “a hazard distinct from and greater than the hazard attending employment in general” and peculiar to the nature of the employment.⁵⁶ Such a rule would comport with the language of *Anderson* and with the policy of the Delaware courts to liberally construe the workers’ compensation scheme to carry out the goals of compensation without transforming it into a system of general health insurance.⁵⁷ Given that the facts of *Anderson* involved a claim for allergic rhinitis, *Anderson*’s insistence that a person predisposed to

⁵⁶ This approach would be consistent with the rule adopted in New York after *Air Mod Corp. v. Newton* and *Anderson*, which looked to that jurisdiction in formulating Delaware’s causation test for occupational diseases. See *Anderson*, 442 A.2d at 1360-61; *Air Mod Corp. v. Newton*, 215 A.2d 434, 442 (Del. 1965) (quoting *Harman v. Republic Aviation Corp.*, 82 N.E.2d 785 (N.Y. 1948)). New York permits compensation for “[s]evere allergies and other reactions arising from exposure to substances in the workplace” where they constitute a workplace accident. *Bruse v. Holiday Inn*, 790 N.Y.S.2d 765, 766 (N.Y. App. Div. 2005). If the condition is a gradual injury that accrued over a reasonably definite period of time, the claimant cannot establish causation absent a showing that the injury resulted from “unusual environmental conditions or events assignable to something extraordinary” at the workplace. See, e.g., *In re Johannesen v. N.Y. City Dep’t of Hous. Preservation and Dev.*, 638 N.E.2d 981, 985 (N.Y. 1994); *Adams v. Univera Health Care/Excellus*, 807 N.Y.S.2d 749, 750 (N.Y. App. Div. 2006).

⁵⁷ See 19 Del. C. § 2304; *Duvall v. Charles Connell Roofing*, 564 A.2d 1132, 1134 (Del. 1989).

a disability is not necessarily barred from establishing a compensable occupational disease suggests that *Smith* may construe the holding in *Anderson* too narrowly.

Nonetheless, this case is not one in which the Court confronts such a close question of *Anderson*'s scope and application. Walker's allergy is to *Curvularia*, a mold that both scientific literature and his own expert witnesses describe as ubiquitous. Walker did not present evidence suggesting that the triggering of his latent condition during the time of his employment at Charter was a natural incident of his employment such that his work at Charter posed a hazard "distinct from and greater than the hazard attending employment in general." There was, for example, no evidence of an increased incidence of ABPM among other Charter employees or of indoor *Curvularia* levels at Charter excessively higher than those ordinarily found outdoors. The presence of a single colony of *Curvularia* is far from sufficient to support a causal connection, particularly in light of testimony from several experts for both parties, including Dr. Lee, that rogue spores often end up inside buildings.⁵⁸ Although the mold tests at Charter failed to find *Curvularia* outdoors on the school grounds, it is not clear whether the air testing was performed in an area with significant plant life and it is

⁵⁸ Hr'g Tr., 71-72, 75-76, 248-49, 262-63; Dep. Tr. of Dr. Lee, 58.

undisputed that outdoor *Curvularia* levels generally are much higher than those found in Room 329. Thus, even an expansive reading of *Anderson* allowing for the possibility that an allergy could constitute a compensable occupational disease does not suggest that the Board erred in finding that Walker failed to establish causation.

Walker next argues that the Board's decision conflicts with *Chrysler Motors Corp. v. Taylor*⁵⁹ by impermissibly basing a no-causation finding on the fact that his illness "*could have* occurred outside of the workplace."⁶⁰ Walker further relies on *Taylor* as holding that a cycle of symptoms that worsen while the employee is in the workplace and abate when the employee is away from work is evidence of the occupational nature of a disease. The Court considers both arguments to distort the reasoning of *Taylor*. The holding in *Taylor* is more limited than Claimant suggests, and it is inapposite here.

Taylor involved a claim for occupational asthma brought by an automobile plant worker who was exposed to various chemical fumes while working in different plant departments. The employee was awarded benefits for a compensable occupational disease after presenting the Board with

⁵⁹ 1992 WL 354212 (Del. Super. Nov. 2, 1992).

⁶⁰ Docket 6 (Appellant's Opening Br.), at 10.

medical testimony that her asthma related to her exposure to various chemicals at work, as indicated by the fact that her breathing problems arose when she was at work and dissipated when she was not in the plant. The Board approved Taylor’s claim for occupational asthma, and Chrysler challenged the compensation award on the basis that Taylor “could not specify what particular substance at the plant had caused her asthma and therefore could not prove that it resulted from exposure at the plant and not from some other source.”⁶¹

Taylor came before this Court on a motion to affirm the Board’s decision to grant compensation. The Board had accepted medical expert testimony offered by the employee to show that her illness was more likely than not caused by exposure to chemicals at her workplace, a conclusion that was supported by cycle-of-symptoms evidence.⁶² This Court therefore affirmed on the ground that the employee had presented substantial evidence that her workplace had caused her asthma, even without identifying the specific chemical or irritant that caused her condition. Thus, *Taylor* does not stand for the proposition that an employer’s medical expert testimony that a disease “could have” been caused by a non-occupational source is never

⁶¹ 1992 WL 354212, at *1.

⁶² *Id.* at *3.

sufficient to support a no-causation finding. Rather, the holding in *Taylor* reiterates the Board's discretion to choose between diverging expert opinions, provided they are both supported by substantial evidence.

Furthermore, *Taylor* addresses occupational asthma, a different condition from Walker's ABPC. As its name suggests, occupational asthma is a condition *induced* by workplace exposure to a substance or substances. Unlike ABPC, occupational asthma can arise in a patient without a genetic predisposition to an asthmatic reaction. In *Taylor*, the employee's asthma was caused by exposure to chemicals she only encountered at the workplace. The difference in diagnoses, and thus in causation, is crucial. Because the Board in *Taylor* accepted the occupational asthma diagnosis given by the employee's expert and did not address the compensability of a latent condition or a disease to which the employee was genetically predisposed, its discussion of cycle-of-symptoms evidence does not apply here.⁶³

⁶³ For similar reasons, the Court rejects Walker's efforts to compare his case to *Lewis v. State*, Hr'g No. 1285928 (Indus. Accident Bd. Dec. 13, 2007). In *Lewis*, the claimant's experts opined that multiple unidentified irritants in the workplace caused her to develop occupationally-induced reactive airway disease syndrome. In part because the irritants were not specifically identified, the claimant's causation argument depended heavily upon cycle-of-symptoms evidence suggesting that her symptoms worsened when she was in the workplace. The Board in *Lewis*, unlike in this case, accepted the opinions of claimant's experts and held that "Claimant's [reactive airway disease syndrome] was *actually produced or created by exposure in the workplace* and, as a progressive disease . . . then left her increasingly reactive to other stimuli outside the workplace." *Id.* at 39 (emphasis added). In this case, however, the Board accepted expert testimony that ABPC is never "produced" or "created" by a particular location.

Finally, Walker claims that the Board's decision mischaracterized his medical experts' testimony such that reversal is merited. According to the Board's decision, Dr. Lee and Dr. Rodgers testified that generally damp and moldy conditions inside Charter supported their opinions that Walker's illness was work-related. Walker submits that although Dr. Lee testified to the existence of damp, moldy conditions at Charter, that testimony was not the basis of her opinion regarding causation. In addition, Dr. Rodgers did not mention damp or moldy conditions at all in relating his causation opinion. Walker argues that the Board's mischaracterization of crucial portions of his experts' opinions requires reversal under *Hinckle v. Shorts Enterprises, Inc.*⁶⁴

In *Hinckle*, this Court reversed the Board's denial of a workers' compensation claim where the Board stated, contrary to fact, that the employee's medical expert had not opined that the employee's injury was work-related.⁶⁵ Because the misstatement went to "the crucial part" of the expert's testimony, and because the employer's no-causation argument was not supported by substantial evidence, reversal was merited.⁶⁶

⁶⁴ 2004 WL 1731142 (Del. Super. July 28, 2004).

⁶⁵ *Id.* at *3-4.

⁶⁶ *Id.*

To the extent the Board mischaracterized statements made by Drs. Lee and Rodgers regarding damp conditions at Charter as relating to causation, the Court does not consider the misstatement to be of the same magnitude as the error in *Hinckle*. Although the Board ultimately rejected their opinions, its decision acknowledged that Walker's medical experts related his illness to Charter based upon his apparent cycle of symptoms and the presence of *Curvularia* in the school building. The Board's decision therefore correctly stated "the crucial part" of Walker's experts' opinions as to causation; the fact that the Board accepted Dr. Cogen's opinion instead is not an error, but rather an example of the Board carrying out its duty to resolve conflicts in testimony.

VI. Conclusion

The Court expresses its sympathy for the medical ordeal Walker has endured. Anyone hearing his nightmarish story would be hard-pressed not to notice that his symptoms seemed correlated to his time spent indoors at Charter. But the Board credited expert testimony that essentially restated the well-known adage: correlation is not causation. Even accepting that Walker's ABPC symptoms were triggered by exposure to *Curvularia* inside the Charter school building, his disease was not caused by that exposure and therefore is not occupational within the meaning of *Anderson*. Although the

Board erred in attempting to apply *Reese* to this case, that error is harmless and could only have worked in Walker’s favor. The Board properly applied the applicable causation test under *Anderson*, and its conclusion as to compensability was supported by substantial evidence. The Court therefore affirms the Board’s decision on different legal grounds.⁶⁷

For the foregoing reasons, the decision of the Board denying Walker’s Petition for Compensation Due is hereby **AFFIRMED**.

IT IS SO ORDERED.

Peggy L. Ableman, Judge

Original to Prothonotary

cc: Timothy E. Lengeek, Esq.
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Lauren C. McConnell, Esq.

⁶⁷ See, e.g., *Chrysler Corp. v. Alston*, 702 A.2d 925, 1997 WL 597120, at *2 (Del. Sept. 22, 1997) (TABLE) (“In affirming a decision of the Board, the Superior Court may provide legal reasoning different from that of the Board, so long as the Superior Court does not fall ‘into the error of weighing the evidence, determining questions of credibility and making factual findings and conclusions.’” (citation omitted)); *Smith*, 2000 WL 145817, at *3.