UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLUMBIA
Civil Division

DENICOLE YOUNG and VANESSA GHEE	)	
Plaintiffs,	)	
v.	)	**Civil Action No. 07cv0983 (ESH)**
WILLIAM F. BURTON and LEWIS & TOMPKINS, P.C.	)	
Defendants.	)	

MEMORANDUM OPINION AND ORDER

Plaintiffs Denicole Young and Vanessa Ghee have sued William F. Burton and Lewis & Tompkins, P.C., for legal malpractice based on their failure to file a timely personal injury lawsuit. The original lawsuit would have sought recovery for damages suffered by plaintiffs as a result of exposure to toxic mold while residing at the Stanton Glen Apartments. In order to succeed on their legal malpractice claim, plaintiffs must show that their attorneys' alleged negligence adversely affected their ability to benefit from an otherwise meritorious claim. *See Niosi v. Aiello*, 69 A.2d 57, 60 (D.C. 1949). To make their case, plaintiffs rely on the testimony of Dr. Ritchie Shoemaker as to the cause, nature, and extent of their injuries. Defendants have moved to exclude Dr. Shoemaker's testimony, arguing that his opinions are not based on a reliable methodology, and that regardless, Dr. Shoemaker did not follow his own methodology with respect to plaintiffs.

Based on the record herein, including the testimony presented at a *Daubert* hearing, the Court concludes that Dr. Ritchie Shoemaker's diagnosis of plaintiffs, as well as his opinions

relating to general and specific causation, are not sufficiently grounded in scientifically valid principles and methods to satisfy *Daubert*. Therefore, defendants' motion will be granted.

BACKGROUND

I. PLAINTIFFS

Plaintiffs moved into Apartment 2A at 3064 Stanton Road, S.E. on August 19, 2002. (Compl. ¶ 8.) They resided there for approximately thirty-four days, during which time plaintiffs contend they could smell noxious fumes from raw sewage. (Pls.' Opp'n at 5; Pls.' Ex. 5 [Ghee Dep.] at 252.) In early September 2002, while investigating the smell, plaintiffs climbed through a window of the adjacent apartment, Apartment 1A, and took photographs of the extensive visible mold growth in this vacant apartment. (Defs.' Mot. at 2; Defs.' Ex. 3 [Young Dep.] at 175-78; Pls.' Ex. 7 [Photographs].) Although plaintiffs are not sure exactly how long they spent in Apartment 1A, they estimate it was no longer than one or two minutes. (Defs.' Mot. at 2; Defs.' Ex. 3 at 178.) There was no documentation of any visible mold growth in plaintiffs' apartment (Daubert Hr'g Tr. ["Tr."] at 76:2-5, June 16, 2008), and plaintiffs do not believe the two apartments shared a common air source. (Defs.' Mot. at 2; Defs.' Ex. 1 [Ghee Dep.] at 452). On September 23, 2002, plaintiffs signed a lease agreement for a different unit in the apartment complex and immediately moved into the new apartment. (Pls.' Opp'n at 5; Defs.' Ex. 2 [Lease Agreement].)

Both plaintiffs submitted extensive medical records to document the health problems that they attribute to their mold exposure. Approximately two weeks after moving into the apartment, Vanessa Ghee visited George Washington University Hospital ("GWUH") on September 6, 2002. (Defs.' Ex. 4 [Ghee Medical Records] at 19.) She complained of a productive cough that

had lasted three weeks and indicated that she had experienced a similar cough three months prior to that visit. (*Id.*) She was diagnosed with viral bronchitis and was instructed to use a humidifier at home and to quit smoking. (*Id.* at 22.) When she returned to GWUH a week later on September 13, 2002, she was given Claritin and again instructed to stop smoking. (*Id.* at 27.) After moving out of the apartment, Ghee required medical care only intermittently. (Pls.' Ex. 11 [Ghee Medical Records].)

Denicole Young's medical records indicate significant medical problems prior to moving into the apartment. She was seen for bronchitis and sinusitis as early as December 10, 1996. (Defs.' Ex. 5 [Young Medical Records] at 642.) She was seen again for sinus congestion and cough on October 21, 1997 (*id.* at 632) and July 29, 1998 (*id.* at 609), and she complained of chronic fatigue on January 9, 1998 (*id.* at 611) and March 10, 2000. (*Id.* at 602). She was also seen many times during those years for complications from her sickle cell trait. Young went to GWUH with Ghee on September 6 and 13, 2002, and was also diagnosed with bronchitis, prescribed Claritin, and told to use her inhaler. (Defs.' Ex. 5 at 656-59.) Young's medical records from the September 13 visit indicate a past history of asthma (*id.*), although it is unclear exactly when she first received that diagnosis. In the months after moving out of the apartment, Young required a few medical visits for minor problems but was hospitalized for asthma exacerbation and pneumonia on April 15, 2003. She required intubation on three separate occasions during that hospital stay. (Pls.' Ex. 12 [Young Medical Records] at 983-94.) She had regular doctors' visits over the next two years relating to asthma, sore throats, coughing, allergic reactions, and swelling in her extremities. (*Id.* at 157-52, 150-48, 145-38, 134-33, 131-27, 123-

22, 118-16, 84-80, 75-74, 971-82, 924-35, 912-23, 899-911, 1000-22, 1055-70, 1086-94, 1308-13, 1326-30, 1332-38.)

II. DR. SHOEMAKER

Dr. Shoemaker received his doctorate from Duke University. (Pls.' Ex. 15 [Shoemaker CV] at 1.) He is currently a member of the American Medical Association, the American Society for Microbiology, the American Society of Tropical Medicine and Hygiene, the International Association for Chronic Fatigue Syndrome, and the Maryland Medical Chirurgical Association. (*Id.*) He has practiced as a licensed medical doctor in Pocomoke, Maryland since 1980 (Pls.' Ex. 14 [Shoemaker Aff.] ¶ 3) and has been the treating physician for over 4,700 patients whom he has diagnosed with ailments caused by exposure to water-damaged buildings. (*Id.* ¶ 5). He has also authored numerous publications and books, including *Mold Warriors*, which was published in 2005. (*Id.*)

A. Methodology

Dr. Shoemaker described his methodology for diagnosing cases of mold illness[1] as follows. He begins by following standard diagnostic procedures with new patients: first, he takes the patient's history, and second, he performs an examination of the area that is the subject of the patient's complaint. (Pls.' Ex. 14 ¶¶ 13-14.) Then, depending on the circumstances of the illness and if there is a temporal relationship that suggests that the patient was in a location where he may have been exposed to a possible environmental contaminant, Dr. Shoemaker will turn to his own differential diagnostic procedure for mold illness. (*Id.* ¶ 15.)

[1] "Mold illness" is a term coined by Dr. Shoemaker which he uses to describe an "acute and/or chronic, biotoxin associated illness caused by exposure to indoor environment of water-damaged buildings with resident toxigenic organisms." (Pls.' Ex. 55 [Shoemaker Report] at 6.)

That procedure involves a two-tiered analysis. (*Id.* ¶ 17.) To satisfy the first tier, all three of the following factors must be met: "(1) the potential for exposure; (2) the presence of a distinctive group of symptoms; and (3) the absence of confounding diagnoses and exposures." (*Id.* ¶ 18.) According to Dr. Shoemaker, the second tier acts as confirmation of the diagnosis arrived at in the first tier and requires that three of the following six factors be met: (1) HLA DR showing susceptibility to mold illness; (2) reduced levels of melanocyte stimulating hormone (MSH); (3) elevated levels of matrix metalloproteinase-9 (MMP9); (4) deficits in visual contrast sensitivity (VCS); (5) dysregulation of ACTH and cortisol; and (6) dysregulation of ADH and osmolality. (Defs.' Mot. at 6-7.) HLA DR refers to certain genes which Dr. Shoemaker believes are associated with a patient's susceptibility to mold illness. He claims there are certain versions of those genes, or genotypes, which render a patient more likely to have adverse health consequences from exposure to damp indoor environments. (Pls.' Ex. 14 ¶ 21.) VCS is a test of a patient's ability to detect certain visual patterns, which, in turn, is an indicator of neurologic functioning. (*Id.* ¶ 26.) The other four tests look at levels of certain hormones and enzymes in the blood which Dr. Shoemaker believes are altered by exposure to a biotoxin. (*Id.* ¶¶ 18-19.) Dr. Shoemaker refers to those hormones and enzymes as "biomarkers."

If a patient meets both tiers of this case definition, Dr. Shoemaker typically recommends treatment with Cholestyramine ("CSM"), a cholesterol-lowering drug which binds molecules in the intestinal track and prevents them from being absorbed into the body. (Defs.' Ex. 7 [Dr. S. Michael Phillips' Report] at 16.) Dr. Shoemaker uses CSM on an off-label basis, meaning he uses it for a purpose other than that for which it has been approved by the FDA. (*Id.* at 17.)

Dr. Shoemaker has published three peer-reviewed publications regarding mold illness. (Pls.' Ex. 16 [Shoemaker Mold Publications].) The first of these papers established the case definition for biotoxin illness by confirming a set of diagnostic criteria that was present in nearly all of the "cases" of biotoxin illness, and in virtually none of the "control" subjects. Ritchie C. Shoemaker, et al., *Sick Building Syndrome in Water Damaged Buildings: Generalization of the Chronic Biotoxin-Associated Illness Paradigm to Indoor Toxigenic Fungi*, in BIOAEROSOLS, FUNGI, BACTERIA, MYCOTOXINS AND HUMAN HEALTH: PATHOPHYSIOLOGY, CLINICAL EFFECTS, EXPOSURE ASSESSMENT, PREVENTION AND CONTROL IN INDOOR ENVIRONMENTS AND WORK, 66-77 (Eckhardt Johanning, ed., 2005). The second paper looked more closely at the changes in levels of certain biomarkers in biotoxin illness patients in response to treatment and re-exposure. Ritchie C. Shoemaker & Dennis E. House, *A Time-Series Study of Sick Building Syndrome: Chronic, Biotoxin-Associated Illness from Exposure to Water-Damaged Buildings*, 27(1) NEUROTOXICOLOGY AND TERATOLOGY 29 (2005). The third paper consisted of a double-blind, placebo-controlled study of the use of CSM to treat biotoxin illness and also reaffirmed his case definition. Ritchie C. Shoemaker & Dennis E. House, *Sick Building Syndrome (SBS) and Exposure to Water-Damaged Buildings: Time Series Study, Clinical Trial and Mechanisms*, 28(5) NEUROTOXICOLOGY AND TERATOLOGY 573 (2006). This third study was extremely limited; it looked at twenty-six subjects, only thirteen of whom participated in the placebo-controlled trial, and each subject served as his own control. *Id.* at 575-76.

In his studies, Dr. Shoemaker uses a five-step, repetitive exposure protocol to establish the cause of his subjects' illnesses. First, the patient is evaluated under the two tiers explained above and then diagnosed with mold illness. Second, the patient is treated with CSM and tested

to ensure that the biomarker levels have returned to normal. Third, the patient stops CSM treatment and stays away from the suspected mold environment to see if the illness returns when exposed to the variety of biotoxins which are ubiquitous in everyday life. If the patient's biomarker levels remain normal, this means that other exposures are ruled out as the source of the symptoms. Fourth, the patient then returns to the mold environment for no more than three days, and finally, the patient is re-tested to obtain final biomarker readings after having re-acquired the illness. (Pls.' Ex. 55 at 31-32.) By demonstrating that the abnormal levels of biomarkers are associated with the patient's presence in the suspected mold environment, Dr. Shoemaker claims that the illness was caused by exposure to that building.

B. Diagnosis of Plaintiffs

Plaintiffs visited Dr. Shoemaker on September 11, 2007, to obtain his expert opinion regarding the etiology of their symptoms. (Pls.' Ex. 55 at 1.) He spent roughly two hours with each plaintiff, during which time he took their medical histories and performed physical exams. (Pls.' Ex. 55 at 14.) He also performed a VCS test, pulmonary function, electrocardiogram, and pulse oximetry.[2] (*Id.*) At that time, he ordered that laboratory tests be conducted on plaintiffs' blood samples to determine plaintiffs' levels of the Tier 2 biomarkers. (*Id.*) However, even before he received the results of these tests, and thus with no information as to whether plaintiffs met the second tier of his diagnostic criteria, he concluded that "[b]oth Ms. Young and Ms. Ghee acquired a typical biotoxin-associated illness following exposure and re-exposure to the indoor

[2] Dr. Shoemaker requests that his patients complete a number of additional tests that he finds useful in making his diagnosis, all of which plaintiffs chose not to complete. These include an MR spectroscopy, which provides information about cognitive impairment; a pulmonary stress test, which determines O_2 max; and a stress echo, which measures pressure in the pulmonary artery circuit. (Tr. at 247:18-249:9.)

air environment of their townhouse at Apt 2A 3064 Stanton Rd SE, Washington, DC." (*Id.* at 1.) The September 2007 visit, which occurred five years after plaintiffs moved out of Apartment 2A, was the only time Dr. Shoemaker examined the plaintiffs. At some point after that examination, Dr. Shoemaker received the results of plaintiffs' blood tests, which he believes confirms his initial diagnosis. According to Dr. Shoemaker, Young had four of six abnormal blood test results, and Ghee had three of six (three being the minimum required to meet the second tier). (Pls.' Ex. 14 ¶¶ 103-04.) Both plaintiffs had mold susceptible HLA DR genotypes, and both had deficits in their VCS scores, although Dr. Shoemaker was unable to provide plaintiffs' actual results for the VCS test. (*Id.*; Tr. at 157:5.) In addition to those tests, Young's tests revealed MSH of 12 pg/ml and MMP9 of 565, and Ghee's test results revealed MSH of 18 pg/ml, all of which Dr. Shoemaker classifies as abnormal. (Pls.' Ex. 14 ¶¶ 103-04.)

Dr. Shoemaker did not perform his five-step protocol on plaintiffs, and indeed could not possibly have done so, as he first met them long after they left the suspected mold environment. Nor was he able to base his causation opinion on the plaintiffs' response to treatment, for both plaintiffs chose not to take the CSM that he had prescribed for them. (Tr. at 19:20-23.) However, he is of the opinion that now that he has proven the research model for mold illness in his 2006 publication, it is no longer necessary to follow the five-step protocol with new patients, because causation necessarily follows from his diagnosis. (Pls.' Ex. 14 ¶ 93.)

III. PROCEDURAL POSTURE

At the conclusion of discovery, defendants moved for a *Daubert* hearing, relying on the affidavits of two experts. According to their expert toxicologist, Dr. Scott Phillips, since there was no evidence as to the exact substance plaintiffs were exposed to or the level at which they

were exposed, formal toxicological causation analysis could not be performed. (Defs.' Ex. 6 [Dr. Scott Phillips' Report] at 23-24.) In addition, the tests Dr. Shoemaker uses to reach his diagnosis are experimental and "not generally accepted in the toxicology community." (*Id.* at 28-29.) Dr. Phillips explained the traditional causation analysis, comprised of the nine "Hill Criteria" that are necessary to establish a causal relationship between two things,[3] and using these criteria, he opined that "there is no support for a causal association between the dark material on the adjacent apartment walls and the Plaintiffs[']s health complaints." (*Id.* at 25-26.) Defendants' expert immunologist, Dr. S. Michael Phillips, walked through each of the Hill Criteria and explained how the facts of this case cannot support a finding of causation. (Defs.' Ex. 7 [Dr. S. Michael Phillips' Report] at 10-14.) He also faulted Dr. Shoemaker's conclusions on the grounds that "[b]iotoxins do not cause the spectrum of disease shown by Denicole and Vanessa"; that none of the laboratory criteria Dr. Shoemaker uses to arrive at his diagnosis has been "causally associated with specific biotoxin associated human illness"; and that "the medical community does not recognize" biotoxin-associated illness. (*Id.* at 15-17.) Also, according to Dr. Phillips, no actual exposure to mold has been demonstrated; neither plaintiff has any symptoms or test results that could be caused by biotoxins; and "allergies and infections may be plausible explanations of Denicole's major respiratory exacerbation" on April 15, 2003. (*Id.* at 17-18.)

In their opposition, plaintiffs argue that defendants' criticisms only amount to an attack on Dr. Shoemaker's conclusions, not his methodology, and therefore, defendants cannot prevail even if Dr. Shoemaker "draws *conclusions* from test methods and lab tests established for other

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[3] The nine Hill Criteria are: 1) strength; 2) consistency; 3) specificity; 4) temporality; 5) biological gradient; 6) plausibility; 7) coherence; 8) experiment; and 9) analogy. (Defs.' Ex. 6 at 25.)

purposes, and applies them to a different use." (Pls.' Opp'n at 27.) In making this argument, plaintiffs rely on Dr. Shoemaker's affidavit, in which he elaborated on his methodology and explained that he uses standard differential diagnostic procedures which are widely used and accepted in the scientific community. (Pls.' Ex. 14 ¶¶ 11-16.) Plaintiffs also submitted Dr. Shoemaker's peer-reviewed publications on "mold illness," along with numerous scientific papers explaining the human health effects of mold, in order to rebut defendants' contention that Dr. Shoemaker's testimony is not based on a scientifically valid methodology. (Pls.' Exs. 16-33.)

The Court granted a *Daubert* hearing, and both parties submitted direct testimony in the form of affidavits from their experts in advance of the hearing. During the hearing, held on June 16, 2008, Dr. Shoemaker was subjected to cross-examination, followed by the testimony of Dr. S. Michael Phillips. Based on this testimony, as well as the parties' prior submissions, the Court makes the following findings of fact and conclusions of law.

ANALYSIS

I. GOVERNING LEGAL STANDARDS

The admissibility of expert testimony in federal courts is governed by Federal Rule of Evidence 702, which provides:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert . . . may testify thereto in the form of an opinion or otherwise.

As explained by the Supreme Court, under Rule 702, "the trial judge must determine at the outset . . . whether the expert is proposing to testify to (1) scientific knowledge that (2) will assist the trier of fact to understand or determine a fact in issue." *Daubert v. Merrell Dow Pharms., Inc.*,

509 U.S. 579, 592 (1993). The first prong of the analysis "establishes a standard of evidentiary reliability," *id.* at 590, while the second prong "goes primarily to relevance." *Id.* at 591.

Testimony as to the nature, cause, and extent of plaintiffs' symptoms is clearly relevant to the final determination of liability and damages. Furthermore, such testimony involves medical and scientific matters which are beyond the ken of the average juror. Thus, the only inquiry is whether Dr. Shoemaker's testimony meets the standard for evidentiary reliability under the first prong of the *Daubert* analysis.

In performing its "gatekeeping" role, "the district court must focus 'solely on principles and methodology, not on the conclusions that they generate.'" *Ambrosini v. Labarraque*, 101 F.3d 129, 133 (D.C. Cir. 1996) (quoting *Daubert*, 509 U.S. at 595). In so doing, "the district court must engage in 'a preliminary assessment of whether the reasoning or methodology underlying the testimony is scientifically valid and of whether that reasoning or methodology properly can be applied to the facts in issue.'" *Id.* at 133 (quoting *Daubert*, 509 U.S. at 592-93). The Supreme Court suggested several factors to be used in making that assessment: "(1) whether the theory or technique can be (or has been) tested; (2) whether the theory or technique has been subject to peer review and publication; (3) the known or potential rate of error of the methodology; and (4) the general acceptance of the methodology." *Raynor v. Merrell Pharms. Inc.*, 104 F.3d 1371, 1375 (D.C. Cir. 1997). That list of factors "is 'flexible' and . . . neither necessarily nor exclusively applies to all experts or in every case." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 141 (1999). Nor is it a "definitive checklist" or test. *Daubert*, 509 U.S. at 593. The burden is on the proponent of the evidence to show that by a preponderance of

the evidence the opinions they seek to present are reliable. *Meister v. Med. Eng'g Corp.*, 267 F.3d 1123, 1127 n.9 (D.C. Cir. 2001).

II. APPLICATION OF *DAUBERT* TO TOXIC TORT AND MOLD CASES

Courts throughout the country have varied widely with respect to the level of certainty they require with respect to the issue of causation in toxic tort cases generally, and in mold cases specifically. *See* Jeffrey J. Hayward, *The Same Mold Story?: What Toxic Mold is Teaching us about Causation in Toxic Tort Litigation*, 83 N.C. L. Rev. 518, 536-38 (2005). One common method of attempting to demonstrate causation is showing a temporal relationship between exposure to a toxin and subsequent adverse health effects. While the circumstances of the exposure and the timing of the illness may be so compelling as to render further evidence of causation unnecessary, temporal association between exposure and illness, without more, is generally insufficient to establish causation. For example, the Fourth Circuit allowed testimony that relied heavily on temporality where the symptoms began shortly after the plaintiff started working with a toxic chemical, and where the plaintiff's symptoms increased or decreased depending on whether the plaintiff was at work or away from the job site. *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999). However, *Moore v. Ashland Chem.*, 151 F.3d 269 (5th Cir. 1998), represents a more traditional approach, in which the Fifth Circuit concluded that "[i]n the absence of an established scientific connection between exposure and illness, . . . the temporal connection between exposure to chemicals and an onset of symptoms, standing alone, is entitled to little weight in determining causation." *Id.* at 278. A district court judge in the Eastern District of Virginia applied that same logic to a mold case when he found that "[a]n

opinion based primarily, if not solely, on temporal proximity does not meet *Daubert* standards." *Roche v. Lincoln Property Co.*, 278 F. Supp. 2d 744, 764 (E.D. Va. 2003).

The most widely-used method of demonstrating causation in toxic tort cases is to present scientifically-accepted information about the dose-response curve for the toxin which confirms that the toxin can cause the health effects experienced by the plaintiff at the dosage plaintiff was exposed to. Indeed, "[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff's burden in a toxic tort case." *Mitchell v. GenCorp, Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) (quoting *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996)). Accordingly, the Fifth Circuit in *Moore* found an expert's testimony unreliable because he had no information about the level of plaintiff's exposure to the chemical solution and thus could not adequately support an assertion that the levels plaintiff was exposed to were sufficient to cause adverse health effects. 151 F.3d at 278.

In a similar vein, the court in *Cavallo v. Star Enterprise*, 892 F. Supp. 756 (E.D. Va. 1995), adopted the three-step methodology for toxicologists endorsed by the World Health Organization, which involves 1) evaluating the chemicals to which the individual may have been exposed and the concentrations of those chemicals in the air the individual breathed; 2) evaluating the level of exposure necessary to produce adverse health effects, according to the published scientific literature; and 3) combining the first two evaluations to estimate the likelihood that the individual actually suffered any of the harmful effects of the chemical in question. *Id.* at 764. That same court later required that any expert giving testimony as to

toxicology, even if not a toxicologist himself, must apply that same methodology in order to ensure reliability. *Roche*, 278 F. Supp. 2d at 754.[4]

Another issue that has affected the causation inquiry in many of the mold cases to date is whether the plaintiff had a proven allergy to the molds to which he or she was exposed. *See, e.g.*, *Roche*, 278 F. Supp. 2d at 751 (finding an expert's opinion that mold was the cause of an illness unreliable because the plaintiff was not allergic to the molds found in his apartment); *Flores v. Allstate Texas Lloyd's Co.*, 229 F. Supp. 2d 697, 702 (S.D. Tex. 2002) (finding testimony inadmissible in part because the medical expert had not based "his testimony on the results of any testing done to determine whether Plaintiffs [were] allergic to any specific type of mold found in their home"). In contrast, Dr. Shoemaker's theory of mold illness is based on the belief that patients have innate immune responses to mold, rather than acquired immune responses (*i.e.*, allergies), and as such, his methodology necessarily deviates from causation inquiries in prior mold cases. (Pls.' Ex. 14 ¶ 25.)

Given the unique nature of his testimony, it is hardly surprising that Dr. Shoemaker has been challenged in numerous jurisdictions throughout the country. Plaintiffs assert that Dr. Shoemaker's testimony has been challenged under *Daubert*, *Frye*, and other standards over twenty times, and they claim that he has been permitted to testify "[t]he overwhelming majority

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[4] To be sure, not every court has required the same level of specificity with regard to exposure level. The *Westberry* court, for example, considered it sufficient that plaintiffs had shown both that inhalation of high levels of talc undisputedly could cause irritation of mucous membranes, and that plaintiff had been exposed to substantial levels of talc. 178 F.3d at 264. Similarly, the Supreme Court of Delaware affirmed the admission of expert testimony about mold-related injuries despite a lack of environmental testing during certain years that the plaintiff resided in the contaminated environment. *New Haverford P'ship v. Stroot*, 772 A.2d 792, 799 (Del. 2001). However, even in those and other similar cases, there has always been at a minimum confirmation of *some* exposure to mold or the toxin in question.

of the time." (Pls.' Opp'n at 32.) However, they have submitted exhibits documenting only five such cases, none of which was decided under *Daubert*. (Pls.' Exs. 47, 48, 49, 53, 54.) Furthermore, in only one of those cases did the court issue an opinion, and in that opinion, only two paragraphs were devoted to Dr. Shoemaker. *Colaianni v. Stuart Frankel Dev. Corp., et al.*, No. 2003 051245 NO, at 3-4 (Mich. Cir. Ct., Oakland County, May 29, 2007) (opinion and order granting in part and denying in part motion in limine). As a result, this Court cannot decipher the scope of Dr. Shoemaker's proffered testimony in those cases where he has been permitted to testify, nor can the Court evaluate the reasoning of those decisions. Furthermore, Dr. Shoemaker admits that this case is different from any other case where he has testified, because he has been unable to take any of the steps of his repetitive-exposure protocol, including treatment, which he relies on in determining causation. (Tr. at 105:23-25.) As such, none of the cases where Dr. Shoemaker's testimony was admitted is particularly informative.

Furthermore, his testimony has been excluded in a number of jurisdictions, including Virginia, Florida, and Alabama, as well as several cases that are remarkably similar to this one. (*See* Defs.' Mot. at 22-24.) A D.C. Superior Court judge excluded Dr. Shoemaker's testimony because neither his theory on the effects of indoor mold exposure nor his methodology in diagnosing the plaintiffs with chronic biotoxin-associated illness ["CBAI"][5] was generally accepted within the scientific community. *Wright v. Fort Lincoln Realty Co., et al.*, No. 03ca4555, at 2-4 (D.C. Sup. Ct. Oct. 15, 2007) (order granting motion in limine). The judge found that "Dr. Shoemaker failed to confirm that the patients were actually exposed to mold in

[5] Chronic biotoxin-associated illness is the name Dr. Shoemaker used for plaintiffs' condition before switching to "mold illness." (Tr. at 28:5-11.)

their indoor environments"; the general scientific community does not recognize Dr. Shoemaker's use of CSM to treat CBAI; and "some of the tests used by Dr. Shoemaker to diagnose the Wrights with CBAI are not generally used by or generally accepted by doctors to diagnose patients with mold-related illnesses." *Id.* at 5-6.

Even more recently, in May 2008, the Ohio Court of Appeals affirmed the trial court's grant of a motion to exclude Dr. Shoemaker's testimony. *Herzner v. Fischer Attached Homes, Ltd.*, No. CA2007-08-090, 2008 WL 2004473, at *3 (Ohio Ct. App. May 12, 2008). Importantly, Ohio's evidentiary standard for admissibility of expert testimony incorporates the teaching of *Daubert*. *Id.* at *1. Applying *Daubert*'s standard, the trial court offered a host of reasons for excluding Dr. Shoemaker's testimony. First, there was insufficient evidence demonstrating actual exposure to mold toxins. The environmental tests conducted on the apartment were completed three months after the plaintiff had moved out of the apartment, and they failed to demonstrate that the mold spores present in the apartment at that time were actually producing toxic byproducts. *Herzner v. Fischer Attached Homes, Ltd.*, No. 2004CVC00564, at 11-12 (Ct. of Common Pleas, Clermont County, Ohio, May 1, 2007). The trial court also found that there had been "inadequate testing to demonstrate a causal connection between exposure to mycotoxins and human health effects" and noted the "lack of peer-reviewed medical literature on 'mold illness' and its causes as defined by Dr. Shoemaker." *Id.* at 13. Furthermore, the court considered Dr. Shoemaker's differential diagnosis process to be unreliable, largely because his "use and interpretation of the laboratory results . . . is not widely recognized in the medical community." *Id.* at 19. On appeal, the appellate court concluded that "[t]he trial court's

thorough and well-reasoned analysis exposed numerous faults in the principles and methods utilized by Dr. Shoemaker to draw his conclusions." *Herzner*, 2008 WL 2004473, at *3.

For many of the same reasons cited by the courts in Ohio and D.C., as well as those set forth herein, this Court finds that Dr. Shoemaker's testimony as to the diagnosis of mold illness, general and specific causation, and the nature and extent of plaintiffs' injuries does not satisfy *Daubert*.

III. DIAGNOSIS

A. "Mold Illness" or "CBAI"

Differential diagnosis is a process by which a physician takes a patient's history, compiles all possible explanations for the symptoms complained of, and then rules out each explanation until only the most likely diagnosis remains. (Defs.' Ex. 19 [Dr. Scott Phillips Aff.] ¶¶ 17-18.) Dr. Shoemaker asserts that he conducted a differential diagnosis, and in the case of both plaintiffs, he determined that "mold illness" was the only possible explanation for their complaints. However, in order for his diagnostic process to be considered scientifically valid, the diagnosis must be one that is recognized by the scientific community.

Based on Dr. Shoemaker's testimony, the Court cannot conclude that "mold illness" is a generally-accepted illness in the medical community. First, he admits that no one outside his practice group has published any peer-reviewed articles on "mold illness," as defined by his two-tiered case definition. (Defs.' Ex. 9 [Shoemaker Dep.] at 51:18-22.) Second, he agrees that CBAI is not generally accepted by the medical community:

> Q: And CBAI, can we say that that's not a generally-accepted diagnosis?
>
> A: No argument about that.

(*Id.* at 196:13-15.)[6] Third, Dr. Shoemaker concedes that there is no formal code in the International Classification of Diseases (ICD-9-CM) for CBAI (*id.* at 196:16-21), and that his case definition for "mold illness" is not used in any medical school in the country. (Tr. 151:16-19.) And lastly, the tests that Dr. Shoemaker uses are not intended to test for "mold illness." (Defs.' Ex. 19 ¶ 14.) Therefore, as found in other recent cases, "mold illness," as defined by Dr. Shoemaker, is not a medically-accepted diagnosis. As such, any differential diagnosis which results in the conclusion that "mold illness" is the most likely explanation for the patients' illnesses is, by definition, unreliable.

B. Case Definition

1. Tier One

a. Plaintiffs' Potential for Exposure

Perhaps more importantly, even if "mold illness" were an accepted diagnosis, Dr. Shoemaker has not shown that plaintiffs meet his case definition. In the first tier of Dr. Shoemaker's case definition, the patient must have the potential for exposure to toxigenic organisms. However, as the court in *Herzner* pointed out, "[c]learly, a person cannot be made ill by mold toxins to which she has not actually been exposed." *Herzner*, No. 2004CVC00564, at 10. No environmental tests were conducted in plaintiffs' apartment to provide actual proof that plaintiffs did, in fact, inhale toxic substances when they resided there. Despite this absence of proof, Dr. Shoemaker attempts to show that plaintiffs had the requisite exposure in two ways, neither of which is convincing.

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[6] Similarly, in a *Frye* hearing held before the D.C. Superior Court on September 27, 2007, Dr. Shoemaker acknowledged the lack of consensus within the scientific community regarding the legitimacy of CBAI. *Wright*, No. 03ca4555, at 3.

First, Dr. Shoemaker believes that his case definition allows him to use the diagnosis of the disease as evidence of actual exposure. (*See* Pls.' Ex. 14 ¶¶ 18-19.) The flaw in his logic was succinctly explained by defense expert Dr. Scott Phillips:

> [T]he alleged symptoms and ailments are used in an attempt to explain that sufficient exposure and dose have occurred. Then, it is argued that exposure has now been shown to be sufficient, and this "proof of exposure" becomes a basis for explaining the cause of the symptoms and ailments. In short, the symptoms fundamentally become the basis for explaining themselves. Such circular reasoning is not scientifically or medically acceptable.

(Defs.' Ex. 19 ¶ 23.) In order for his methodology to be considered scientifically valid and reliable, Dr. Shoemaker must show *actual* exposure to toxins, and not mere *potential* for exposure.

Dr. Shoemaker's second argument is that because plaintiffs were exposed to a water-damaged building, it is "implausible" that plaintiffs would not have had any actual exposure to toxins, and so, in effect, potential for exposure is evidence of actual exposure. (Tr. at 60:23-61:5.) As evidence of exposure to a water-damaged building, Dr. Shoemaker relies on: 1) musty smells in plaintiffs' apartment; 2) visible mold growth in the neighboring apartment; and 3) a Department of Health letter pointing to musty odors in the basement of plaintiffs' building and visible mold growth on the walls of the utility room. (Tr. at 56:10-13, 57:17-25.) What he does *not* point to, because he cannot, is any sort of environmental test showing the presence of mycotoxins or other toxins in the air plaintiffs breathed while they resided in the apartment. However, Dr. Shoemaker considers it unnecessary to have any test results confirming what substances were present in either apartment, and whether those substances were actually producing toxins at the time plaintiffs resided there. With respect to the photographs of the

microbial growth in Apartment 1A, Dr. Shoemaker opined that "if you find such microbial growth, it is implausible that they would not be making toxigenic substances at some time," and thus, "the argument cannot be sustained that you must test for mycotoxins alone to show illness." (*Id.* at 60:23-61:5.)[7] He also considers it unnecessary to know the level of toxic substances to which plaintiffs were exposed because dose response is an invalid concept when discussing genetic susceptibility. He claims that even minimal exposure to a biotoxin for someone with a genetic susceptibility to mold illness can cause a large array of severe symptoms. (Pls.' Ex. 14 ¶ 131.) This reasoning permits Dr. Shoemaker to attribute any number of symptoms to a patient with a genetic susceptibility to mold who was exposed to a water-damaged building, without any information as to the type or amount of toxins she was exposed to.

These arguments are not scientifically valid. First, as explained in Section III(B)(2), the idea of a genetic susceptibility to mold induced illness is unsupported by the scientific literature. Dr. Shoemaker therefore cannot disregard the need for information as to dosage. Second, his methodology contravenes standard toxicology. As explained by defendants' toxicology expert, Dr. Scott Phillips, the more traditional, generally-accepted theory of causation involves the presence of a substance, the opportunity for contact between the patient and that substance, a known dosage of the substance, and an illness consistent with the substance at that dosage.

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[7] Importantly, that statement was made in reference to the visible growth in Apartment 1A. No such visible growth was documented in plaintiffs' apartment, Apartment 2A (Tr. at 61:6-15), and there is no indication that the two apartments shared a common air source. The apartment complex cannot be held liable for any injuries plaintiffs may have sustained while in Apartment 1A, as plaintiffs were trespassers at that time. *Firfer v. United States*, 208 F.2d 524, 528 (D.C. Cir. 1954) ("[A trespasser] must take the premises as he finds them, and cannot hold the owner to liability based upon negligence in failing to make the premises safe."). It is therefore significant that Dr. Shoemaker admits he cannot quantify what effects going into 1A may have had on plaintiffs, as opposed to just living in the adjacent apartment. (Tr. at 242:2-5.)

(Defs.' Ex. 6 at 17.) Because scientific studies do not yet exist that demonstrate what levels of toxins produced by water-damaged buildings are harmful to humans, and what illnesses they cause, that methodology cannot currently be applied to mold. The Institute of Medicine, in a paper cited by Dr. Shoemaker, concludes that the doses of toxins found in water-damaged buildings necessary to produce adverse health effects in humans have not yet been determined. (Pls.' Ex. 20 [Damp Indoor Spaces (IOM)] at 7.) Similarly, the New York City Department of Health issued a report entitled Guidelines on Assessment and Remediation of Fungi in Indoor Environments, which states that "it is not possible to determine 'safe' or 'unsafe' levels of exposure" to fungi. (Pls.' Ex. 22 [NYC Guidelines].) Without that information, Dr. Shoemaker's testimony about the health effects of any such "exposure" cannot possibly be anything other than conjecture. Even if such knowledge existed, Dr. Shoemaker would still be unable to offer any concrete evidence as to what substances existed at what levels. Thus, there is no basis upon which to conclude that plaintiffs' exposures were sufficient to account for the variety of symptoms they have experienced.

b. Presence of Distinctive Group of Symptoms

Dr. Shoemaker's diagnosis of mold illness requires that patients display a certain pattern of symptoms. He identifies eight organ systems which are relevant to the diagnosis, and a patient must present with chronic symptoms in four of those eight organ systems in order to meet the second requirement of the first tier of his case definition.[8] He acknowledges that no one else has

[8] The eight organ systems are: general, musculoskeletal, head, eye, respiratory, gastrointestinal, executive cognitive functioning, and neurologic. (Tr. at 41:18-44:11.) Curiously, Dr. Shoemaker's most recent scientific study, which he says reaffirmed his case definition, required that subjects have symptoms in at least five of ten organ systems. Shoemaker, *Sick Building Syndrome and Exposure to Water Damaged Buildings*, *supra*, at 575. Antiduretic

published on the use of four out of those eight organ symptoms as a diagnostic tool for mold illness:

> Q: There's no other publication that uses the four out of the eight symptoms that you've just identified to establish one leg of the mold diagnosis. Would you agree with that statement?
>
> A: I would agree that it's not been published.

(Tr. at 49:21-25.)

At the time of Dr. Shoemaker's examination, both plaintiffs had symptoms in at least four of those organ systems, and thus met the second component of Tier 1.[9] There are a number of problems with Dr. Shoemaker's reliance on those symptoms to conclude that plaintiffs are ill as a result of mold exposure. For one, plaintiffs' complex of symptoms did not begin immediately

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hormone and hypothalamic were the additional two organ systems, with headache and skin sensitivity being grouped into a "multifactorial; unique" organ system which takes the place of "head." *Id.*

[9] According to Dr. Shoemaker, plaintiff Young presented with: fatigue; weakness; aching; cramps; cramping of intrinsic muscles of hands and feet such that her digits assumed a claw-like posture; joint pains in feet, knees, and both hands; morning stiffness; unusual, sharp stabbing pain in side of chest and abdomen; headache; sensitivity to bright light; red eyes; tearing; profound shortness of breath; cough; sinus problems; abdominal pain with secretory diarrhea; difficulty handling abstract numbers in simple division, recent memory impairment; difficulty concentrating; decreased word finding; decreased assimilation of new knowledge; confusion; numbness and tingling in both feet; vertigo; tremors; mood swings; appetite swings; difficulty controlling body temperature; excessive thirst; frequent urination; increased susceptibility to static shocks; sensitivity to light touch. (Pls.' Ex. 55 at 15.)

Plaintiff Ghee presented with fatigue; aching; cramps; cramping of intrinsic muscles of hands such that thumbs assumed a claw-like posture; unusual, sharp stabbing pain in right lower back; headache; light sensitivity; red eyes; tearing; profound shortness of breath; sinus problems; joint pains in right knees; morning stiffness; difficulty handling abstract numbers in simple division; recent memory impairment; difficulty concentrating; decreased word finding; decreased assimilation of new knowledge; mood swings; night sweats; difficulty with temperature regulation; excessive thirst; frequent urination; increased susceptibility to static shocks; numbness and tingling in fingers and big toe on right foot. (*Id.* at 15-16.)

after exposure. Indeed, while living in the apartment, both plaintiffs complained only of respiratory symptoms. (Pls.' Ex. 10 [Ghee/Young Medical Records].) Second, the symptoms did not remain consistent over time. In November 2002, Young's medical records indicate that she reported feeling much better than she had in September. (Pls.' Ex. 12 at 0000168.) In virtually every medical record, Young reports slightly different symptoms, with many of her recurring symptoms, such as swelling in the extremities and rash, beginning many months after moving out of Apartment 2A. (Pls.' Ex. 12.) Furthermore, the vast majority of the symptoms Dr. Shoemaker reported for both plaintiffs five years after their supposed exposure are undocumented in any medical records that postdate their exposure in August-September 2002. (Pls.' Exs. 11, 12.) This is particularly evident with respect to Vanessa Ghee, whose brief medical records indicate only respiratory complaints and headaches, as opposed to the myriad of symptoms that Dr. Shoemaker attributed to her in 2007. (Pls.' Ex. 11.) There is simply no evidence that many of the symptoms Dr. Shoemaker reported existed at any time prior to his examination, and thus no evidence that those symptoms have been chronic in nature since plaintiffs' initial exposure to mold.

Furthermore, the suggestion that symptoms experienced five years after exposure to a biotoxin can be attributed to that biotoxin is unsupported by scientific literature. As defense expert Dr. S. Michael Phillips explained, "Dr. Shoemaker's findings in this case are . . . based on the false notion that biotoxins remain in the body for prolonged periods of time. This belief is misplaced and at variance with the known science of mycotoxin metabolism." (Defs.' Ex. 20 [Dr. S. Michael Phillips' Aff.] ¶ 23.) Rather, symptoms from exposure to mycotoxins are

"rapidly reversible" and should have remitted upon leaving the contaminated environment, "if that environment was causally related to symptoms," which did not happen here. (*Id.*)

Finally, Dr. Shoemaker is unable to determine which symptoms are actually attributable to the mold. Rather, he testified that roughly 75% of plaintiffs' symptoms are probably attributable to this mold exposure, although he cannot say which ones. (Tr. at 193:24-194:5.)[10] A diagnostic process which ultimately fails to determine which symptoms are components of the illness is inherently flawed and cannot be considered scientifically valid.

Ultimately, plaintiffs' symptoms have not had the longevity, consistency, and documentation necessary to support Dr. Shoemaker's diagnosis. Additionally, Dr. Shoemaker's assertions about the way symptoms of exposure to biotoxins present is unsupported by scientific literature.

c. Absence of Confounders

The third element of the first tier of Dr. Shoemaker's diagnostic protocol is that there be an absence of confounding diagnoses and exposures. This requirement fulfills the critical purpose of a differential diagnosis, which is to conclude that only the chosen diagnosis could be

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[10] THE COURT: Can you say to a degree of medical certainty that 75 percent of these [symptoms] were caused by the exposure [Ghee] suffered in that building?

THE WITNESS: Yes.

THE COURT: But you can't identify which ones, correct?

THE WITNESS: That's correct.

Id.

responsible for the symptoms presented. Nevertheless, Dr. Shoemaker glosses over the explanation of how he ruled out all potential confounding explanations for plaintiffs' symptoms.

At numerous points in the record Dr. Shoemaker brushes off discussion of confounding diagnoses as almost irrelevant. For instance, his report merely asserts that "[t]hey had no confounding medical illnesses or environmental exposures, as confirmed by a collection of medical records forwarded to [him] before their office visit." (Pls.' Ex. 55 at 2.) He later states that nothing other than mold illness causes patients to present with chronic symptoms in four separate organ systems. (Tr. at 51:2-17.) Similarly, in reference to patients with potential confounders such as diabetes, hypertension, smoking, anxiety, or allergies, he states that "the grouping of symptoms [his] patients have with mold illness are different and the lab abnormalities that those other patients have are different." (*Id.* at 34:15-25.) However, he does not elaborate on exactly what the symptoms or abnormalities would look like in patients with those diseases. In his affidavit, he contends that "[p]otential confounders, such as allergy to trees, dander and grasses, for example, never give any abnormalities" on his Tier 2 tests like MSH and VCS. (Pls.' Ex. 14 ¶ 103.) However, the requirement that there be no confounders is part of Tier 1 of Dr. Shoemaker's case definition and should therefore be satisfied before any blood test results are known. It is insufficient for him to rely on Tier 2 results to justify his findings with respect to Tier 1.

In *Herzner*, the trial court judge pointed out that Dr. Shoemaker "does not explore the possibility that Herzner's symptoms could have been caused by several different ailments." *Herzner*, No. 2004CVC00564, at 20. In this case, Dr. Shoemaker briefly addressed this issue by saying that in his studies he "looked at [the subjects] for symptoms to try to sort out is smoking a

confounder for mold illness, or do people actually have two things . . . ?" (Tr. at 52:18-21.) In short, although he seems to be claiming that he considered the possibility that there may be more than one cause for plaintiffs' symptoms, he provided no specific testimony as to plaintiffs, who appear to have a host of possible confounders, and he does not explain why it is implausible that several simultaneous conditions may have contributed to their symptoms.

The one potential confounder Dr. Shoemaker addresses at any length is Young's prior diagnosis of asthma. However, he manages to use that potential confounder to support his "mold illness" diagnosis. He asserts that "the fact that she, after this exposure, . . . has countless visits in 2003, '04, '05, and '06 for asthma-related conditions is consistent with the hypothesis that this exposure to the water damaged building made her lung condition much worse." (*Id.* at 214:14-18.) Rather than acknowledging that Young's asthma-related symptoms may, in fact, have been caused by the asthma, which she apparently had prior to moving into the Stanton Glen Apartments, rather than the mold, he claims that because her asthma got worse after 2002, she must be a "mold illness" patient. (*Id.* at 214:19-24.)

Overall, Dr. Shoemaker failed to adequately demonstrate his methodology for "ruling out" other possible explanations for plaintiffs' symptoms.

2. Tier 2

Even if Dr. Shoemaker could show that plaintiffs met the first tier of his diagnostic process, his assertion that plaintiffs meet the requirements of his second tier is based on a methodology that is not generally accepted in the scientific community. The first, and most fundamental, flaw in Dr. Shoemaker's Tier 2 analysis is that not one of his Tier 2 biomarker tests (VCS, MSH, MMP9, ADH, ACTH) is generally accepted or clinically-validated for the purpose

of diagnosing "mold illness." Indeed, the laboratory which performs Dr. Shoemaker's tests for MSH, Laboratory Corporation of America ["LabCorp"], includes the following disclaimer regarding the test: "the results should not be used as a diagnostic procedure without confirmation of the diagnosis by another medically established diagnostic product or procedure" (Defs.' Ex. 14 [LabCorp MSH Test for Young]), and the test for MMP9 includes a similar admonition. (Defs.' Ex. 13 [Quest Diagnostic MMP-9 Test for Ghee].) Furthermore, Dr. Shoemaker admits that none of the tests he uses can affirmatively show that a person is ill because of exposure to a water-damaged building. Rather, they can only show that an inflammatory response is present in the patient, which says nothing about the cause of that response. (Pls.' Ex. 14 ¶¶ 23, 25, 28, 30, 32, 34.)

Additionally, the idea that levels of these biomarkers five years after an exposure is in any way related to that exposure is unsupported by generally accepted science. Defendants' expert immunologist, Dr. S. Michael Phillips, explained, for example, that "ACTH rises in the body within minutes of the stress and falls in hours after the stress. . . . [I]n the light of the short biological half-life of ACTH, the measurement of ACTH taken years after a putative exposure could not be relevant to that exposure." (Defs.' Ex. 20 ¶ 20.) Dr. Phillips made similar assertions with respect to C4a, another biomarker Dr. Shoemaker looks at which, although not one of his Tier 2 tests, he nonetheless uses to confirm his diagnosis. Ghee tested within the normal range for C4a, but Young was outside the normal range. (Pls.' Ex. 14 ¶ 103.)[11] Dr. S. Michael Phillips explained that "C4a is an activation marker, which rises in seconds or minutes and falls to

[11] Dr. Shoemaker defines "normal" as less than 2830 ng/ml. Ghee tested at 2694 ng/ml, while Young had a C4a result of 10,935 ng/ml. (*Id.*)

baseline levels with[in] hours after the activation stimulus." (Defs.' Ex. 20 ¶ 21.) Based on this testimony, which the Court credits, testing for these biomarkers five years after an exposure cannot possibly reflect the effects of that exposure.

Furthermore, Dr. Shoemaker's use of HLA DR genotypes to determine mold susceptibility is completely unsupported by the scientific literature. HLA DR genes are found on Chromosome 6, and "are associated with the success or failure to clear illnesses from the body." (Pls.' Ex. 14 ¶ 20.) Dr. Shoemaker believes that certain of these genes can cause people to be susceptible to "mold illness." He estimates that 24% of the population has one mold susceptible HLA DR haplotype, which would make them more likely to develop "mold illness" after exposure to mold toxins. Additionally, 4% of the population has one of what Dr. Shoemaker calls the two "dreaded" haplotypes, so named because those patients have the worst clinical outcomes in response to mold exposure. The theory of a genetic basis for "mold illness" is critical to Dr. Shoemaker's theory, for it allows him to explain how plaintiffs' extensive symptoms can arise from a brief or mild exposure without applying the theory of a dose-response relationship. (*Id.* ¶ 131.) However, with respect to the HLA DR gene, Dr. S. Michael Phillips explained:

> It is associated with various genetic linkages and diseases but has never been shown to be important in biotoxin injury. There have never been any controlled prospective studies indicating that any specific markers in the HLA or HLA-DR loci code can be linked with any mold-associated disease. In fact, Dr. Shoemaker's "dreaded haplotypes" have previously never been linked with any mold-associated illness.

(Defs.' Ex. 20 ¶ 16.) Furthermore, even aside from HLA DR, "[t]here are no accepted genetic markers for susceptibility to mold or toxin induced diseases." (Defs.' Ex. 7 at 17.) Thus, the inclusion of a diagnostic criteria based on genetics is entirely without merit.

Finally, the parameters Dr. Shoemaker has set to determine what constitutes an "abnormal" test result on these Tier 2 tests are not universally accepted in the scientific community. Indeed, they are not even recognized by the labs which he uses to perform the tests. Dr. Shoemaker defines "normal" test results for MSH as 35-81 pg/ml and for MMP9 as 0-332. LabCorp, the lab which runs the MSH tests, recently changed its normal range from 35-81 pg/ml to 0-40 pg/ml. (Pls.' Ex. 55 at 28.) Additionally, the two labs Dr. Shoemaker regularly uses have different normal ranges for MMP9. Quest Laboratories agrees with Dr. Shoemaker that normal is 0-332, but LabCorp sets 0-983 as normal. (Defs.' Ex. 9 at 95:16-98:21.) Given that the two national laboratories that run tests on Dr. Shoemaker's blood samples disagree as to what constitutes a "normal" test result, it is impossible to conclude that Dr. Shoemaker's method of assessing abnormalities in certain biomarkers is generally accepted by the scientific community. Furthermore, if LabCorp's normal ranges are applied here, neither plaintiff has three abnormal test results, and thus, neither plaintiff meets the diagnostic criteria for Tier 2 of Dr. Shoemaker's case definition. (Tr. at 166:8-13.)

Ultimately, Dr. Shoemaker diagnosed plaintiffs with a condition that is not recognized in the scientific community. In doing so, he used circular reasoning to work backwards from diagnosis to proof of exposure, he failed to explain exactly which symptoms comprise that diagnosis, and he did not rule out confounding diagnoses. His methodology in arriving at his diagnosis of "mold illness" is therefore unreliable.[12]

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[12] Interestingly, Dr. Shoemaker does not believe his opinion was significantly affected by the five-year gap between plaintiffs' exposure and their arrival in his office. He claims that both the symptoms and the biomarker abnormalities persist over time and thus would likely have been the same very shortly after exposure as they are now. (*Id.* at 147:15-22.) The only change in his diagnosis that would occur if it had been done five years earlier is that he could have had the

IV. CAUSATION

In a toxic tort case, "[t]he plaintiff must show that the toxicant in question is capable of causing the injury complained of (general causation) and must further prove that the toxicant in fact did cause that injury in the present case (specific causation)." Hayward, *supra*, at 533. General causation must be affirmatively proven before specific causation can be shown. *See Raynor*, 104 F.3d at 1376 ("testimony on specific causation had legitimacy only as follow-up to admissible evidence that the drug in question *could* in general cause birth defects") (emphasis in original). Plaintiffs have failed to sustain their burden as to both.

A. General Causation

Satisfying the general causation inquiry in this case requires a showing that the substance plaintiffs were exposed to is capable of causing the illness they experienced.[13] The first hurdle plaintiffs must overcome is that there is no way of knowing what "substance" the plaintiffs were in fact exposed to, as Dr. Shoemaker freely admits he does not know what molds or bacteria were present in plaintiffs' apartment in 2002, or what toxic substances were being produced at the time. (Defs.' Ex. 9 at 203:13-206:6.) Dr. Shoemaker attempts to overcome this hurdle by referring to the "substance" in question as simply a water-damaged building. However,

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apartment tested to determine what toxins were actually present while plaintiffs lived there and thus would have had a greater level of confidence regarding the substances they were exposed to. (*Id.* at 147:23-148:5.) Even if such a test could have been done, the Court cannot credit his "mold illness" as a diagnosis or his conclusions regarding plaintiffs' diagnosis.

[13] Plaintiffs spend a significant portion of their Opposition citing authorities in support of the contention that mold can cause human illness. (Pls.' Opp'n at 12-21.) In so doing, plaintiffs misconstrue the nature of the general causation inquiry. Whether mold can cause any illness in humans contributes nothing to the much more specific discussion of whether toxins produced by a damp indoor environment are capable of causing the numerous, multi-system symptoms experienced by plaintiffs.

defendants' toxicology expert exposed the fallacy of referring to unspecified environmental conditions as the "substance" in view of the need to identify specific toxins and connect them to specific symptoms. (Defs.' Ex. 6 at 17.)

However, if one takes a broad view of "substance" to include "water-damaged building," and if one accepts "mold illness" as a real disease, the question that remains is whether it is generally accepted in the scientific community that exposure to a water-damaged building causes "mold illness." Even the studies cited by Dr. Shoemaker fail to establish such a connection. For example, the Environmental Protection Agency's 2004 paper, produced with the University of Connecticut, recognizes that "the notion that indoor mold growth can lead to significant toxicity in occupants of 'moldy buildings' has been very controversial in the scientific literature and likely will remain so for the foreseeable future." (Pls.' Ex. 21 [EPA/Connecticut Guidance] at 28.) Furthermore, those papers which affirm the potential for toxic effects as a result of mold exposure refer primarily to upper and lower respiratory tract symptoms (and occasionally to other symptoms such as fatigue, nausea, and headaches), but not to the multi-system symptoms that Dr. Shoemaker attributes to "mold illness." (Pls.' Ex. 22; Pls.' Ex. 23 [CDC 2005] at 24.) The Center for Disease Control also pointed out that "[the Institute of Medicine] found inadequate or insufficient evidence for a link between exposure to damp indoor environments and molds with a variety of conditions that have been attributed to toxicity." (Pls.' Ex. 23 at 24.) It is thus clear that at the present time, the scientific community is not in agreement with Dr. Shoemaker about the wide-ranging effects of exposure to non-specific toxins from water-damaged environments.

Absent a consensus in the medical community about the health effects of exposure to mold, Dr. Shoemaker is left with only his own most recent peer-reviewed publication on "mold

illness" to demonstrate general causation. However, defendants correctly highlight several deficiencies in this study. For one, the study was far too limited to stand alone as proof of general causation; only twenty-six subjects participated in the study, and the double-blinded, placebo-controlled clinical trial involved only thirteen of those subjects. (Defs.' Reply at 4.) Furthermore, at the time of publication, LabCorp had already changed its "normal range" for the MSH blood test, such that Dr. Shoemaker's diagnostic criteria were no longer in accordance with medically accepted standards. (Defs.' Reply at 4; Tr. at 164:11-17.) Additionally, in the introduction to his third article, even Dr. Shoemaker acknowledges that "[t]he hypothesis that chronic exposure to the indoor environments of water-damaged buildings (WDB) causes a multi-system illness, often referred to as "sick building syndrome" (SBS), remains controversial." Shoemaker, *Sick Building Syndrome and Exposure to Water Damaged Buildings*, *supra*, at 574.[14] Given these substantial limitations and his own admission that a causal link is not generally-accepted, this single study cannot serve to establish general causation.

B. Specific Causation

[14] The D.C. Superior Court pointed to similar language in Dr. Shoemaker's second peer-reviewed publication on "mold illness" in finding a lack of evidence as to general causation. In the abstract to that paper, he stated: "[t]he human health risk for chronic illnesses involving multiple body systems following inhalation exposure to the indoor environments of water-damages buildings (WDBs) has remained poorly characterized and the subject of intense controversy." Shoemaker, *A Time-Series Study*, *supra*, at 29. In his affidavit, Dr. Shoemaker objects to the D.C. Superior Court's use of that sentence to discredit his theory. He argues that "[s]aying why a paper is going to be written is standard practice; citing the reason for the paper as the same as the conclusion is illogical." (Pls.' Ex. 14 ¶ 105.) However, given the obvious limitations of his third paper, this study is hardly sufficient to transform his theory from "controversial" to generally-accepted.

In the absence of sufficient proof of general causation, it goes without saying that plaintiffs cannot establish specific causation. But even if they could, plaintiffs fail to offer any evidence of specific causation.

In his studies, Dr. Shoemaker has utilized a repetitive exposure protocol ["REP"] to demonstrate causation. By showing that his study participants get better with treatment, remain healthy without treatment when away from the water-damaged building, and then experience an almost immediate return of symptoms when they return to the building, he is able to rule out other environmental exposures as the source of his patients' illnesses. (Pls.' Ex. 55 at 31-32.) Dr. Shoemaker even advises his patients that with respect to proving their mold injury claims, "the most unbeatable evidence is your response to treatment and re-exposure in the 5-step repetitive exposure protocol." (Defs.' Ex. 21 [Dr. Shoemaker Website FAQ] at 3.) However, because plaintiffs moved out of their suspected mold environment five years before they went to see Dr. Shoemaker, there was no way for him to re-create the conditions that existed there five years earlier so that plaintiffs could return to that environment to determine what would happen to their symptoms. More importantly, as explained by the court in *Herzner*, Dr. Shoemaker's use of the REP to establish causation "is supported by nothing other than a temporal relationship." *Herzner*, No. 2004CVC00564, at 26. Drawing conclusions about causation from temporality is a common logical fallacy known as *post hoc ergo propter hoc* (after the fact, therefore because of the fact), and is as unpersuasive in the courts as it is in the scientific community. *See, e.g.*, *Rolen v. Hansen Beverage Co.*, 193 F. App'x 468, 473 (6th Cir. 2006) ("Expert opinions based upon nothing more than the logical fallacy of *post hoc ergo propter hoc* typically do not pass muster under *Daubert*.").

Furthermore, although Dr. Shoemaker prescribed Cholestyramine (CSM) to both plaintiffs, neither plaintiff followed his advice, so he was unable to see how they responded to treatment. (Tr. at 19:20-23.) Importantly, Dr. Shoemaker admits that he has never before testified in a case where the plaintiffs had not at least taken the prescribed medication and shown improvement. (*Id.* at 105:23-25.) Even in other cases where he first met the plaintiffs years after their potential exposure, he had still treated them with CSM, observed their symptoms improve, and then stopped CSM treatment and demonstrated that their condition again deteriorated.[15] (*Id.* at 106:23-107:3.) This case thus lacks any of the steps which Dr. Shoemaker himself has relied upon in the past to draw a direct link between an exposure and an illness.

Because he was unable to complete any part of his REP, Dr. Shoemaker claims that merely by diagnosing plaintiffs with "mold illness," he has established evidence of causation. He asserts that because the research model for his case definition was proven in his most recently-published study, causation is established. And "once established, causation does not have to be re-invented for each repeat case." (Pls.' Ex. 14 ¶ 109.) This assertion is entirely without merit. In actuality, the results from his third paper merely support "the general hypothesis that SBS [Sick-Building Syndrome] is associated with exposure to WDBs [water-damaged buildings]." Shoemaker, *Sick Building Syndrome and Exposure to Water Damaged Buildings*, *supra*, at 583. In other words, Dr. Shoemaker himself reports that it confirms a

[15] Dr. Shoemaker claims his diagnosis is actually strengthened by the fact that plaintiffs have not received any treatment for their symptoms. He states that "[t]he fact they didn't get treatment from 2002 until when their lab database was accumulated in 2007 assists [him] because it shows that the lab abnormalities which should be durable without treatment are indeed durable" (Tr. at 198:7-11), and thus are consistent with his illness model. However, this runs directly counter to Dr. Shoemaker's own statement on his website that response to treatment is the best evidence of mold illness. (Defs.' Ex. 21 at 3.)

"*general* hypothesis" (one that finds no support outside of Dr. Shoemaker's research group), not proof of *specific* causation for every future patient. Indeed, as pointed out by defendants' expert immunologist, Dr. Shoemaker's assertion that he need not determine causation for future patients is contrary to accepted medical principles. (Defs.' Ex. 19 ¶ 30 ("for each new case, one must evaluate each patient on a case-by-case basis . . . to determine the most likely diagnosis and ultimately causes for that disease process").)

Given that Dr. Shoemaker arrives at his opinions as to both general and specific causation based on novel and unaccepted theories and methodologies, plaintiffs cannot sustain their burden under *Daubert* as to causation.

V. NATURE AND EXTENT OF PLAINTIFFS' INJURIES

Because the Court finds "mold illness" to be an unaccepted diagnosis, any testimony as to the nature or extent of plaintiffs' injuries relating to that illness is necessarily unsupported by reliable scientific evidence. Dr. Shoemaker himself admits that without any knowledge of how each plaintiff would respond to treatment, he cannot offer an opinion as to the permanency of their symptoms. (Defs.' Ex. 9 at 36:11-19 ("I can't give permanency in this case, because she hasn't even taken the first intervention that can correct this illness.").) And similarly, he cannot say which of plaintiffs' symptoms were caused by exposure to the damp environment of the apartment. (Tr. at 193:24-194:5.) Therefore, based on Dr. Shoemaker's own admissions, his testimony in these areas would be nothing other than speculation.

CONCLUSION

For the foregoing reasons, defendants' motion to exclude the opinion testimony of plaintiffs' expert, Dr. Ritchie Shoemaker, is **GRANTED**. A status hearing is set for July 31, 2008, at 11:00 a.m.

/s/
ELLEN SEGAL HUVELLE
United States District Judge

Date: July 22, 2008