Josephine Chesson, et al. v. Montgomery Mutual Insurance Co., No. 97, September Term 2012, Opinion by Battaglia, J.

EVIDENCE – ADMISSIBILITY OF SCIENTIFIC EVIDENCE – GENERAL ACCEPTANCE TEST

In a Workers' Compensation case, Montgomery Mutual Insurance Company sought review of a decision by the Circuit Court for Howard County admitting testimony by Dr. Ritchie Shoemaker that exposure to mold caused neurocognitive and musculoskeletal symptoms based on a "differential diagnosis," which he referred to as a "Repetitive Exposure Protocol." The Repetitive Exposure Protocol involved removing patients from the contaminated area, treating them, then returning them to the subject building, where, Dr. Shoemaker testified, the symptoms would re-develop. The Court of Appeals held that Dr. Shoemaker's testimony was not admissible under *Frye-Reed*, reasoning that his methodology was flawed and not generally accepted because it failed to account for the levels of mold exposure. The Court, moreover, concluded that based on an examination of relevant scientific journal articles that the scientific community remained uncertain as to Dr. Shoemaker's techniques and conclusions.

IN THE COURT OF APPEALS OF MARYLAND

No. 97

September Term, 2012

JOSEPHINE CHESSON, ET AL.

v.

MONTGOMERY MUTUAL INSURANCE COMPANY

Barbera, C.J.
Harrell
Battaglia
Greene
Adkins
McDonald
*Bell,

JJ.

Opinion by Battaglia, J.

Filed: September 24, 2013

* Bell, C.J., now retired, participated in the hearing and conference of this case while an active member of this Court; after being recalled pursuant to the Constitution, Article IV, Section 3A, he also participated in the decision and adoption of this opinion.

When an expert opinion is offered to support the existence of new or novel scientific theory or methodology, "the basis of that opinion must be shown to be generally accepted as reliable within the expert's particular scientific field." *Reed v. State*, 283 Md. 374, 381, 391 A.2d 364, 368 (1978), citing *Frye v. United States*, 293 F. 1013, 1014 (1923). The conundrum presented in the instant Petition for Certiorari[1] involves the meaning of "general acceptance" in the context of what was offered as a "differential diagnosis"[2] that exposure to mold in a water-damaged office building allegedly caused non-respiratory neurocognitive and musculoskeletal symptoms.[3]

The general acceptance test imposes a significant gate-keeping role on the judge to determine whether a scientific theory or methodology should be admitted for consideration

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[1] We granted certiorari, 429 Md. 528, 56 A.3d 1241 (2012), to consider:

> Whether the Court of Special Appeals erred in finding that the opinions of Ritchie Shoemaker, MD, were not admissible under the Frye-Reed test.

[2] Differential diagnosis, a process critiqued in *Blackwell v. Wyeth*, 408 Md. 575, 613-18, 971 A.2d 235, 259-61 (2009) to prove that thimersol caused autism, was characterized in that case as "a process of elimination, [and] defined as, '[t]he process of weighing the probability of one disease versus that of other diseases possibly accounting for a patient's illness. The differential diagnosis of rhinitis (a runny nose) includes allergic rhinitis (hayfever), the abuse of nasal decongestants and, of course, the common cold.'" *Id*. at 611 n.22, 971 A.2d at 25 n.22, quoting Medicinenet.com, Differential Diagnosis Definition, http://medterms.com/script/main/art.asp?articlekey=2991 (last visited May 5, 2009).

[3] To be clear, respiratory illnesses identified with mold exposure (conceded by the parties as possible symptoms from inhaling mold spores) are not included in the spate of symptoms before us. Rather, the physician whose methods and theories are at issue in this case, Dr. Ritchie Shoemaker, identified numerous non-respiratory symptoms that he opined were caused by mold exposure, including memory loss and joint pain. The parties in this case have referred to these symptoms collectively as "neurocognitive and musculoskeletal symptoms" and we shall continue that appellation.

by the jury. *Blackwell v. Wyeth*, 408 Md. 575, 591, 971 A.2d 235, 245 (2009). The test originated in *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), and was adopted by this Court in *Reed v. State*, 283 Md. 374, 389, 391 A.2d 364, 372 (1978). Writing for the Court in *Reed*, Judge John C. Eldridge explained that a novel scientific technique may be admitted in evidence only after a judge determines that it is recognized as demonstrable, as opposed to unverified, by the relevant scientific community:

> "Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a *well-recognized* scientific principle or discovery, the thing from which the deduction is made must be *sufficiently established to have gained general acceptance in the particular field in which it belongs*."
>
> That is to say, before a scientific opinion will be received as evidence at trial, the basis of that opinion must be shown to be generally accepted as reliable within the expert's particular scientific field. Thus, according to the *Frye* standard, if a new scientific technique's validity is in controversy in the relevant scientific community, or if it is generally regarded as an experimental technique, then expert testimony based upon its validity cannot be admitted into evidence.

Id. at 381, 391 A.2d at 368, quoting *Frye*, 293 F. at 1014 (emphasis in original).

Determining whether a novel scientific theory is generally accepted in the relevant scientific community places the judge within the intersection of law and science. Unlike a trial, which involves a "quick and determinative" assessment of the evidence presented to determine whether guilt or liability is proven, the scientific inquiry "represents an ongoing

cycle, in which each inquiry into an observable phenomenon is but one aspect of an ongoing quest" for knowledge. *Blackwell*, 408 Md. at 581, 971 A.2d at 239.

Driving this quest for knowledge is the scientific method, "the analytical process by which a hypothesis is tested and analyzed and conclusions or theories are developed." *Id*. at 581, 971 A.2d at 239. Theories are developed and tested, to be disconfirmed or subjected to further scrutiny through critique and continued study. A theory's validity and reliability are measured by its ability to be replicated, so that "general acceptance" relates to that which survives scientific scrutiny:

> At the heart of this search for knowledge is the use of scientific method—or the analytical process by which a hypothesis is tested and analyzed and conclusions or theories are developed. This process has also been described as empirical study, that being study, "[f]ounded on practical experience, rather than on reasoning alone, but not established scientifically . . . [or] testing a hypothesis by careful observation, hence rationally based on experience." Stedman's Medical Dictionary 632 (28th ed. 2006) ("empiric"). In basic terms, the development of a theory, using the scientific method or empirical testing, follows characteristic steps:
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> > 1. Observations of some phenomenon are made. . . .
> > 2. Possible explanations (theories) are proposed for what is observed. . . .
> > 3. Hypotheses are logically derived from the theories . . . .
> > 4. Studies are designed to test the hypotheses. In essence, the study makes new observations that might disconfirm the hypothesis and thereby falsify the theory. Different theories have different implications and lead to different hypotheses. (Ideally, a study can be devised

> whose outcome will disconfirm one theory's hypotheses and not the other's. This is called a "critical experiment" because it permits a head-to-head test of two or more theories, and helps to determine which has done the best job of accounting for the relevant phenomena. Sometimes scientific controversies persist for a very long time because no commonly agreed upon critical experiment can be conducted.)
> 5. The results of such empirical tests lead to revision or abandonment of older theories or creation of still newer and hopefully better theories.
> 6. The process repeats itself as more empirical tests are conducted and theories undergo continued re-evaluation.

David L. Faigman, Michael J. Saks, Joseph Sanders & Edward K. Cheng, 1 Modern Scientific Evidence: The Law and Science of Expert Testimony, at 263-64 (2008[-2009]). Specifically, once a theory is conceived based on an observable phenomenon, a hypothesis, which is "[a] conjecture advanced for heuristic purposes, cast in a form that is amenable to confirmation or refutation by conducting of definable experiments and the critical assembly of empiric data," Stedman's [Medical Dictionary] 938 [(28th ed. 2006)], is developed, which defines the scope of an experiment. Studies then are designed to test the hypothesis and gather data:

> To real scientists a finding of fact is only as good as the methods used to find it. Scientific method is the logic by which the observations are made. Well designed methods permit observations that lead to valid, useful, informative answers to the questions that had been framed by the researcher. For scientists, the key word in the phrase "scientific method" is method. Methodology—the logic of research design, measures, and procedures—is the engine that generates knowledge that is scientific. While for lawyers

> and judges credibility is the key to figuring out which witnesses are speaking truth and which are not, for scientists the way to figure out which one of several contradictory studies is most likely correct is to scrutinize the methodology.

Faigman, *supra*, at 260 (emphasis in original). Once data is compiled, analysis occurs, from which conclusions are drawn; the hypothesis either remains viable or is disproven:

> Note that a hypothesis or a theory is never proven or confirmed to be true. Testing is capable only of disconfirming. But theories that withstand such attempts at falsification better and longer become accepted, at least until something better comes along. The opposite approach can readily be seen in non-scientific activities of numerous kinds, where investigators engage in a search for evidence that confirms their suspicions. This "confirmatory bias" is based on the erroneous assumption that a theory is confirmed by the accumulation of facts consistent with the theory . . . . It is the diligent search for inconsistencies, for falsification, that really puts a theory to the test. A theory that can withstand such scrutiny is one that deserves credence.

Id. at 264.

Blackwell, 408 Md. at 581-83, 971 A.2d at 240. Validity and reliability are the linchpins of the scientific method: validity, having been defined as "the extent to which something measures what it purports to measure," and reliability, characterized as "the ability of a measure to produce the same result each time it is applied to the same thing . . . consistency or reproducibility." *Id*. at 584, 971 A.2d at 240-41, quoting Faigman, *supra*, at 269.

In a courtroom, a judge or jury, obviously, is not able to replicate the scientific inquiry

and explore whether a novel scientific theory is reliable and valid and thereby demonstrable. The general acceptance test, along with the other *Frye-Reed* prongs, ensures that the trier of fact focuses on the "rendition of a judgment on the merits of the litigation" rather than on the drama of expert testimony that inures to courtroom presentations:

> ". . . *Frye* was deliberately intended to interpose a substantial obstacle to the unrestrained admission of evidence based upon new scientific principles. . . . Several reasons founded in logic and common sense support a posture of judicial caution in this area. Lay jurors tend to give considerable weight to 'scientific' evidence when presented by 'experts' with impressive credentials. We have acknowledged the existence of a '. . . misleading aura of certainty which often envelops a new scientific process, obscuring its currently experimental nature.'"

Reed, 283 Md. at 386, 388, 391 A.2d at 370, 371, quoting *People v. Kelly*, 549 P.2d 1240, 1245 (Cal. 1976), quoting in turn *Huntington v. Crowley*, 414 P.2d 382, 390 (Cal. 1966).

> The introduction of evidence based on a scientific process, not yet generally accepted in the scientific community, is likely to distract the fact finder from its central concern, namely the rendition of a judgment on the merits of the litigation. Without the *Frye* test or something similar, the reliability of an experimental scientific technique is likely to become a central issue in each trial in which it is introduced, as long as there remains serious disagreement in the scientific community over its reliability. Again and again, the examination and cross-examination of expert witnesses will be as protracted and time-consuming as it was at the trial in the instant case, and proceedings may well degenerate into trials of the technique itself. The *Frye* test is designed to forestall this difficulty as well.

Id. at 388-89, 391 A.2d at 371-72.

To ascertain general acceptance, this Court in *Reed* instructed that, absent taking

judicial notice, a trial judge considering whether a scientific technique is valid and reliable will not only consider testimonial evidence, but also should consider law journal articles, scientific journal articles that have reliability, and "other publications which bear on the degree of acceptance by recognized experts that a particular process has achieved":

> On occasion, the validity and reliability of a scientific technique may be so broadly and generally accepted in the scientific community that a trial court may take judicial notice of its reliability. Such is commonly the case today with regard to ballistics tests, fingerprint identification, blood tests, and the like. Similarly, a trial court might take judicial notice of the invalidity or unreliability of procedures widely recognized in the scientific community as bogus or experimental. However, if the reliability of a particular technique cannot be judicially noticed, it is necessary that the reliability be demonstrated before testimony based on the technique can be introduced into evidence. Although this demonstration will normally include testimony by witnesses, a court can and should also take notice of law journal articles, articles from reliable sources that appear in scientific journals, and other publications which bear on the degree of acceptance by recognized experts that a particular process has achieved.

Id. at 380, 391 A.2d at 367 (internal citations removed). "As long as the scientific community remains significantly divided, results of controversial techniques will not be admitted . . . ." *Id.* at 388, 391 A.2d at 371.

General acceptance does not equate to unanimity of opinion within a scientific community, nor universality, and is not subject to a quantum analysis. *See U.S. Gypsum v. Baltimore*, 336 Md. 145, 183, 647 A.2d 405, 424 (1994) (noting that general acceptance does not equate to universal acceptance, and determining that surface dust sampling to determine

asbestos contamination was generally accepted because the defendant "failed to direct this Court to any information which indicates that the divergence of opinion over the use of dust sampling amounts to the type of 'fundamental division in the scientific community' which necessitates the exclusion of such testimony." (internal citation omitted)). Even scientific techniques once considered to be generally accepted are excluded when subsequent scientific studies bring their reliability and validity into question and show a fundamental controversy within the relevant scientific community. *See Clemons v. State*, 392 Md. 339, 896 A.2d 1059 (2006). A trial judge also cannot admit expert testimony based on scientific methodology without consideration of whether the analysis itself is flawed and posits an "analytical gap." *Blackwell*, 408 Md. at 608, 971 A.2d at 255.

In *Reed*, in a rape trial, the State offered the expert testimony of Dr. Oscar Tosi concerning spectograms, a voiceprint recognition technique involving a spectograph, which measures the "acoustic energy of the human voice [in] three components – time, frequency, and intensity" to determine whether two voice recordings, a phone call in which the caller made admissions and the defendant's own voice, belonged to the same person. 283 Md. at 378, 399 A.2d at 366. The Court observed that prior to 1972, the reliability of voiceprint recognition was considered by the scientific community to be uncertain, noting that a scientific journal article authored by six scientists reviewing the technique, at the request of the Technical Committee on Speech Communication of the Acoustical Society of America, reported that "the voiceprint process was still in its experimental stage, and the reliability of

the conclusions based on the data obtained from the process was uncertain." *Id.* at 389-90, 391 A.2d at 372.

Between 1971 and 1972, however, Dr. Tosi had authored a series of articles addressing voiceprint recognition study, which had served as grounds to admit expert testimony based on voiceprint recognition in other jurisdictions; the same six scientists, who had previously written on the scientific technique at the request of the Acoustical Society of America, published further observations in 1973, in which they remained uncertain of the technique's reliability and expressed concern over Dr. Tosi's lack of "an adequate scientific basis for estimating reliability in many practical situations . . . ." *Id.* at 390-91, 391 A.2d at 372-73, quoting Richard Bolt et al., Speaker Identification by Speech Spectograms: Some Further Observation, 54 J. Acoustical Soc'y Am. 531, 533-34 (1973). This article, as well as testimony during the *Reed* trial acknowledging a significant split in the scientific community as to the validity of the voiceprint process, led the Court to conclude that "the fundamental division in the scientific community . . . has continued without substantial abatement." *Id.* at 392, 391 A.2d at 373. To support this conclusion, the Court also reviewed decisions from other jurisdictions and legal commentary.

We subsequently have considered whether a method once considered generally accepted can lose that application in the relevant scientific community so that the theory returns to a disconfirmed state. In *Clemons v. State*, 392 Md. 339, 347, 896 A.2d 1059, 1064 (2006), testimony of an FBI expert in comparative bullet lead analysis (CBLA), "a three-step

process that involves the comparison of the elemental composition of bullets in an effort to determine whether different bullets originated from the same vat of lead," was offered by the State to prove that the accused Clemons, charged with four crimes including murder, was guilty. The CBLA was offered to show that the bullets in Clemons's gun matched a bullet found at the crime scene. The trial court, considering the CBLA expert's use of this technique for thirty years, admitted the testimony, and the Court of Special Appeals affirmed. We ultimately reversed, engaging in an in-depth review of the relevant scientific community's recent "attack" of the reliability of CBLA:

> Recently the assumptions regarding that uniformity or homogeneity of the molten source and the uniqueness of each molten source that provide the foundation for CBLA have come under attack by the relevant scientific community of analytical chemists and metallurgists.

Id. at 368, 896 A.2d at 1076. The "attack" was apparent from a progression of law review and scientific journal articles, which showed that CBLA's general and underlying assumption, that no two vats of lead were the same, was no longer generally accepted by the relevant scientific community. Thus, even a scientific technique once considered valid and reliable can lose its evidentiary admissibility when "fundamental assumptions underlying the process are not generally accepted by the scientific community." *Id.* at 372, 896 A.2d at 1079.

In *Blackwell v. Wyeth*, 408 Md. 575, 593-95, 971 A.2d 235, 246-47 (2009), the Blackwells brought a products liability action against Wyeth, Inc., alleging that the presence

of thimerosal[4] in its childhood vaccines caused autism and other neurological defects in the Blackwells' child. The Blackwells presented Dr. Mark Geier as an expert in epidemiology.[5] Dr. Geier's opinion as to causation was based on several of his scientific journal articles that linked thimerosal in vaccines as a cause of autism by using adverse event reports of vaccines containing thimerosal compiled in the Vaccine Adverse Effect Reporting System (VAERS)[6] and other third-party databases and, alternatively, he hypothesized that thimerosal in vaccines caused autism in certain genetically susceptible individuals. Wyeth moved to preclude the Blackwells' expert testimony under *Frye-Reed*.

During the *Frye-Reed* hearing in *Blackwell*, the trial judge reviewed several publications that questioned or rejected Dr. Geier's theory, including the National Academy of Sciences' Institute of Medicine (IOM)[7] 2001 Committee and 2004 Committee Reports,

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[4] Thimerosal "is an organic mercury based compound [that] has been used as a preservative in various vaccines and other biological and pharmaceutical products since the 1930's." *Blackwell*, 408 Md. at 577 n.2, 971 A.2d at 236 n.2 (alterations in original).

[5] Epidemiology, the trial judge in *Blackwell* opined, refers to "the science that studies the distribution of diseases within populations" and considered it to be the "single most relevant field of science to the general causation issue presented in this case, i.e., whether thimerosal-containing vaccines can cause autism." *Id.* at 600, 971 A.2d at 250.

[6] "The Vaccine Adverse Event Reporting System (VAERS) is a national vaccine safety surveillance program co-sponsored by the Centers for Disease Control and Prevention (CDC) and the Food and Drug Administration (FDA). VAERS is a post-marketing safety surveillance program, collecting information about adverse events (possible side effects) that occur after the administration of vaccines licensed for use in the United States." Vaccine Adverse Event Reporting System, http://vaers.hhs.gov/index (last visited August 8, 2013).

[7] "The National Academy of Sciences is a private, nonprofit, self-perpetuating society (continued...)

in which evidence regarding the alleged causal link between thimerosal-containing vaccines and autism was considered to be inadequate, in 2001, and favoring a rejection of that theory, in 2004. The 2004 IOM Committee Report criticized Dr. Geier's studies as "flawed methodologically," "uninterpretable," and "noncontributory," leading the trial judge to conclude that they were not generally accepted in the relevant scientific community. *Id.* at 603, 971 A.2d at 252. We affirmed, concluding that although the VAERS data was a potentially reliable source, Dr. Geier's conclusion and methodologies were not generally accepted, nor was the data gathered for the purpose of testing his hypothesis. Dr. Geier's theory of a direct link between thimerosal in vaccines and autism, thus, fell into an "analytical gap," making his theory unreliable, invalid, and not generally accepted, in addition to other flaws.

The Court also found Dr. Geier's alternative theory that the thimerosal vaccines caused autism in select genetically susceptible individuals, including the child at issue, not to be generally accepted, in part because the trial judge rejected what Dr. Geier purported to be a differential diagnosis, in "ruling out the potential causes until the most probable cause remains," because the tests used to rule out alternative causes of autism were not generally accepted in the medical community "as appropriate tests for either the work-up of a patient with autism or to determine the underlying cause of autism," and the differential diagnosis

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(...continued)
of distinguished scholars, created by congressional charter in 1863 to advise the federal government on scientific and technical matters." *Blackwell*, 408 Md. at 597 n.17, 971 A.2d at 248 n.17.

"failed even to consider the single most important alleged cause of autism," that being "a gene or series of interacting genes that have not yet been identified." *Id*. at 614-17, 971 A.2d at 259-60. Dr. Geier's failure to appropriately analyze genetic components in his alternative hypothesis led other courts, the trial judge and ultimately this Court, to determine that Dr. Geier's differential diagnosis was "fundamentally flawed," and therefore inadmissible. *Id.* at 616, 971 A.2d at 260.

The case at hand originated in workers' compensation claims filed by six employees of the Baltimore Washington Conference of the United Methodist Church, Petitioners,[8] and was previously before us in *Montgomery Mutual Insurance Co. v. Chesson*, 399 Md. 314, 923 A.2d 939 (2007) (*Chesson I*). The six employees claimed that they had sustained physical injury, including neurocognitive and musculoskeletal symptoms, as a result of exposure to mold in the Baltimore Washington Conference's office. To prove causation, the employees proffered Dr. Ritchie Shoemaker as their expert. Montgomery Mutual, however, sought to exclude Dr. Shoemaker under *Frye-Reed*, arguing that his methodology to determine causation was not generally accepted in the relevant scientific community:

> Josephine Chesson, Martha Knight, Carole Silberhorn, Linda Gamble, Kenneth Lyons, and Connie Collins, were employees of the Baltimore Washington Conference of the United Methodist Church, and worked at the Church's offices located at 9720 Patuxent Woods Parkway, Columbia, Maryland. On November 18, 2002, several employees working in the office

[8] Josephine Chesson, Martha Knight, Carole Silberhorn, Linda Gamble, Kenneth Lyons, and Connie Collins, Petitioners, shall be referred to collectively as "the employees."

building noticed a foul odor emanating from the walls. A maintenance crew broke through an interior wall and discovered two types of mold, Aspergillus and Stachybotrys.

[The employees] each filed a claim with the Maryland Worker's Compensation Commission, alleging that they had sustained an accidental injury or occupational disease known as sick building syndrome due to mold exposure on November 18, 2002. *See* Md. Code (1999, 2006 Cum. Supp.) § 9-101 *et seq.* of the Labor and Employment Article. The Workers' Compensation Commission held a hearing and disallowed two of [the employees'] claims and awarded partial compensation to the remaining [employees] after finding accidental injury or occupational disease due to mold exposure.[2] Each [of the employees] filed a petition for judicial review in the Circuit Court for Howard County, *see* Md. Code (1999, 2006 Cum. Supp.) § 9-737 *et seq.* of the Labor and Employment Article, and a joint motion to consolidate the claims.

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[2] The Workers' Compensation Commission found that . . . Connie Collins and William Lyons suffered neither an accidental injury nor an occupational disease due to mold exposure. The Commission found that . . . Josephine Chesson, Martha Knight, and Carole Silberhorn suffered accidental injury due to mold exposure, and that . . . Linda Gamble suffered from an occupational disease and not accidental injury due to mold exposure.

The Circuit Court consolidated the claims.[3] Each [of the six employees] had been examined and treated by Dr. Ritchie Shoemaker, a licensed medical doctor and board-certified physician in the field of family medicine. Prior to trial, [Montgomery Mutual] filed a motion *in limine* seeking to exclude the testimony of Dr. Shoemaker on the grounds that his theories and methodologies for diagnosis regarding a causal connection between mold exposure and certain human health effects had not been generally accepted within the relevant scientific community.

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[3] The following cases were consolidated with the present case: 13-C-03-56904, 13-C-03-56955, 13-C-03-56956, 13-C-03-57033, 13-C-03-57043, 13-C-04-57483, 13-C-04-57784, and

13-C-04-60173.

Chesson I, 399 Md. at 318-19, 923 A.2d at 941. The Circuit Court Judge denied Montgomery Mutual's motion to exclude Dr. Shoemaker's testimony, without holding a *Frye-Reed* hearing. Dr. Shoemaker testified as to causation at trial and the jury returned verdicts in favor of each of the employees.[9] Montgomery Mutual appealed to the Court of Special Appeals, which affirmed the trial court's admission of Dr. Shoemaker's testimony without a *Frye-Reed* hearing. *Montgomery Mutual v. Chesson*, 170 Md. App. 551, 569-70, 907 A.2d 873, 884 (2006). We granted certiorari and determined that, "Dr. Shoemaker employs medical tests to reach a conclusion that is not so widely accepted as to be subject to judicial notice of reliability." *Chesson I*, 399 Md. at 332, 923 A.2d at 949 (footnote omitted). We ordered that the case be remanded to the Circuit Court, to hold a *Frye-Reed* hearing:

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[9] The jury found "a causal relationship between mold exposure and certain illnesses claimed . . .":

> The jury found that mold exposure on the date of November 18, 2002 caused a neuro-cognitive condition in Carole Silberhorn, a musculoskeletal and neuro-cognitive condition in Martha Knight, a musculoskeletal and neuro-cognitive condition in Josephine Chesson, an accidental injury that resulted in a respiratory condition in William Lyons, an accidental injury that resulted in a neuro-cognitive condition in Linda Gamble, and an accidental injury that resulted in a respiratory and neuro-cognitive condition in Connie Collins.

Montgomery Mutual v. Chesson, 399 Md. 314, 323 n.4, 923 A.2d 939, 944 n.4 (2007) (*Chesson I*).

> Accordingly, we remand this case for an evidentiary hearing to ascertain whether Dr. Shoemaker's methodologies used for diagnosis and theories regarding the causal connection between mold exposure and certain human health effects are generally accepted in the scientific community. The trial court is directed to make factual findings and conclusions and then to issue a *Frye-Reed* determination. If the trial court finds that Dr. Shoemaker's methods and theories satisfy the *Frye-Reed* test, the judgment should remain in effect. If the court finds to the contrary, the judgment should be vacated. Our remand is limited solely to this issue.

Id. at 336, 923 A.2d at 951.

The Circuit Court subsequently held a *Frye-Reed* hearing, in which the judge considered only whether the relevant scientific community generally accepted as reliable and valid Dr. Shoemaker's methodologies and theory that identified mold exposure as the cause for the neurocognitive and musculoskeletal symptoms allegedly suffered by the employees.

At the hearing, Dr. Shoemaker testified that the indoor air of a water-damaged building known to contain mold caused neurocognitive and muscuoloskeletal symptoms. He based his opinion on his application, beginning in 1998, of "Repetitive Exposure Protocol," a technique that he developed to study 101 individuals who worked or resided in forty buildings and complained of neurocognitive and muscuoloskeletal symptoms such as memory loss and muscle aching. To apply the technique, Dr. Shoemaker testified, he would identify the presence of mold in the building, through visual identification of mold, detecting a musty smell, or lab testing of a sample, such as a piece of drywall. With respect to the individuals, he would rely on a medical history and physical examination to rule out other

possible causes of the symptoms, according to a "differential diagnosis," based on his testimony. The individual would then be removed from the subject building and, for two weeks, receive a dosage of Cholestyramine,[10] which he opined could relieve symptoms related to mold exposure because he had earlier treated Chesapeake Bay watermen who had suffered severe diarrhea after exposure to fish with pfiesteria.[11] Dr. Shoemaker, then, testified that once a three-day break from Cholestyramine had been established, the individual would then be returned to the subject building for three days, during which, he opined, the individual would report that the symptoms had redeveloped, before the individual again was removed from the subject building and administered a second dosage of Cholestyramine.

Between 1999 and 2003, Dr. Shoemaker added, according to his testimony, a second tier of the technique, to bolster his opinion that mold exposure caused neurocognitive and musculoskeletal symptoms. Dr. Shoemaker testified that through blood testing and a visual contrast sensitivity test, he would test for six biological markers and, he opined, the presence of at least three indicated that mold exposure was the cause of the neurocognitive and

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[10] Cholestyramine has been defined as "an anion exchange [resin] used to bind dietary cholesterol and hence prevent its systemic absorption. Used to treat hypercholesteremia. Can bind many acidic drugs in the gastrointestinal tract and prevent their absorption." Stedman's Medical Dictionary 1674 (28th ed. 2006) ("Cholestyramine resin").

[11] Pfiesteria piscicida has been defined as "[a]n estuarine dinoflagellate having numerous life stages; afflicts many types of fish and other marine species; causes diverse symptoms in humans . . . ." Stedman's Medical Dictionary 1469 (28th ed. 2006) ("pfeisteria piscicida").

musculoskeletal symptoms. Through blood testing, he would test for: the presence of one of "fifty-four kinds of immune responses genotype, called HLA," a "reduction of an inflammation preventing hormone called . . . MSH," "elevated levels of an inflammation . . . enzyme called MMP9," the abnormal relationship of hormone ACTH to cortisol, and an abnormal relationship of hormone ADH to osmolality. He further testified that a visual contrast sensitivity exam would measure the sixth and final biological marker, a deficit in visual contrast, which has been defined as "[t]he ability to distinguish objects from the background in which they are located." Taber's Cyclopedic Medical Dictionary 520 (21st ed. 2009) ("contrast sensitivity"). Dr. Shoemaker testified that he would implement this second tier during the "Repetitive Exposure Protocol," before the individual was removed from the water-damaged building containing mold, and later at the final step of the technique, after the individual was removed from the water-damaged building.

Dr. Shoemaker referred to a number of articles during his testimony that reflected the causation theory that he posited, including two that he authored[12] and two others that purportedly supported his theory about neurocognitive and musculoskeletal symptoms;[13] the

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[12] Ritchie C. Shoemaker et al., *Sick Building Syndrome in Water Damaged Buildings: Generalization of the Chronic Biotoxin-Associated Illness Paradigm to Indoor Toxigenic Fungi*, Health Effects II – Toxicology & Neurological Effects, at 52-63 (2006); 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 Neurotoxicology & Teratology 1, 29 (2005).

[13] Edmond D. Shenassa et al., *Dampness and Mold in the Home and Depression: An*

(continued...)

latter two were the subject of the greatest scrutiny by the Court of Special Appeals in this case. Others that explored respiratory symptoms purportedly linked to mold exposure were also referred to by Dr. Shoemaker in his testimony.[14]

On cross-examination, Dr. Shoemaker admitted that he did not test any of the buildings, either the Baltimore Washington Conference's office or any others in which an individual resided or worked who underwent his "Repetitive Exposure Protocol," to determine the level of mold exposure that an individual working or residing therein would have experienced. According to Dr. Shoemaker, the identification of mold in a building, even by the presence of a musty smell alone, was sufficient to conclude that an individual

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(...continued)
Examination of Mold-Related Illness and Perceived Control of One's Home as Possible Depression Pathways, 97 Am. J. of Pub. Health 1893 (2007); and Luke Curtis et al., *Adverse Health Effects of Indoor Moulds*, 23 J. of Australasian C. of Nutritional & Envtl. Med. 3 (2004).

[14] Articles that were referenced by Dr. Shoemaker concerning respiratory symptoms purportedly from mold exposure in water-damaged buildings were: Carol Y. Rao et al., *Characterization of Airborne Molds, Endotoxins, and Glucans in Homes in New Orleans After Hurricanes Katrina and Rita*, 73 Applied Envtl. Microbiology 1630, 1630 (2007) (studying the level of mold exposure in water-damaged homes and commenting that "[m]uch evidence exists indicating that indoor exposures to molds contribute to occupant respiratory disease and symptoms"); J.H. Park et al., *Fungal and Endotoxin Measurements in Dust Associated with Respiratory Symptoms in a Water-Damaged Office Building*, 16 Indoor Air J. 192 (2006); Stephen J. Vesper et al., *Relative Moldiness Index as a Predictor of Childhood Respiratory Illness*, 17 J. of Exposure Sci. & Envtl. Epidemiology 1 (2006); Stephen J. Vesper et al., *Specific Molds Associated with Asthma in Water-Damaged Homes*, 48 J. of Envtl. & Occupational Medicine 852 (2006); and Je Hyeong Park et al., *Hydrophilic Fungi and Ergosterol Associated with Respiratory Illness in a Water Damaged Building*, 116 Envtl. Health Perspectives 45 (2008).

residing or working in that building inhaled mold, as part of a "chemical stew" that caused neurocognitive and musculoskeletal symptoms, without any further assessment of not only the level of mold, but also what other chemicals were contained in this "stew." He also acknowledged that the United States Department of Housing and Urban Development was developing "safe" and "dangerous" levels of mold standards for indoor buildings.

Montgomery Mutual countered by calling Dr. Hung Cheung, who was qualified as an expert in internal medicine, occupational medicine, environmental medicine, toxicology, and indoor air quality. Dr. Cheung testified that Dr. Shoemaker's "Repetitive Exposure Protocol" was not generally accepted as valid in the relevant scientific community, not only because it was experimental as well as controversial in its "second tier" of biological markers approach and use of drugs in an off-label fashion, as well as in its failure to account for stress levels in individuals, but also primarily because it failed to measure the levels of mold exposures by individuals in the water-damaged buildings. Mold exposure classified by Dr. Cheung as ranging from low to medium to high, would include consideration of the pathways that mold must travel to reach an individual, such as the building's ventilation system, walls, or ceiling, as well as the pressure or air flow of the building. Pressure and pathway interact with each other, he opined, to determine an individual's exposure: "If somebody is very close [to the mold spores]. . . you don't really need major pressure changes before that person is exposed, but if it's far away, there may be barriers to that person being exposed, or to the gradient. We also know that the distribution drops off significantly by the distance, from the

source."

Dr. Cheung testified that mold illnesses from inhalation have been documented in farm workers who shoveled mold-infested hay. An indoor environment, however, according to Dr. Cheung, does not contain that level of mold concentration, even in a water-damaged building. Specifically, Dr. Cheung testified:

> Q: So would you please go through some of these symptoms and tell me if that's a known or accepted human health effect of mold.
> A: I would say that the fatigue, memory loss, joint pain, muscle aches, confusion, weakness, depression I believe; and I believe disorientation – those are kind of musculoskeletal and neurocognitive things that in various studies have been looked at. So I would say no, they're not associated. That's not to say the others are; it really depends on dose and concentration, and various things.

With respect to the scientific literature related to the causation of neurocognitive and musculoskeletal symptoms by mold exposure, Dr. Cheung testified to a survey that he had commissioned relative to whether Dr. Shoemaker's diagnosis was generally accepted and found Dr. Shoemaker citing himself; a citation in an alternative medicine piece regarding "a novel hypothesis between, the interaction between obesity and toxins"; and a reference to Dr. Shoemaker in a book, *Nonhuman DNA Typing, Theory and Casework*. One other reference citing Dr. Shoemaker's work, according to Dr. Cheung, was in the negative and authored by Dr. Clifford Mitchell, chair of the Maryland Indoor Air Quality Task Force, who concludes that "there are no valid and reliable markers for mold exposure or health effects related to

mold" and "[t]here remains a lack of consensus regarding the systemic effects of mold exposure . . . ."

Most importantly, Dr. Cheung testified regarding the absence of any study utilizing the scientific method that confirmed the relationship of mold exposure to neurocognitive and musculoskeletal symptoms. He cited the "considerable debate" about the potential for mold exposure in office buildings to cause "toxic effects," including neurocognitive and musculoskeletal symptoms that had been identified in the Final Report of the Maryland State Task Force on Indoor Air Quality Report, of which he was a member:

> [T]here was considerable scientific debate . . . about the potential for mold and toxic effects at levels that we normally see in office buildings; and then it goes on:
>
> > The consensus among the Task Force and most health professionals that: Mold growth in buildings can have adverse health consequences, normal background levels of mold can be found in all buildings; there is inadequate base of scientific knowledge at this time to set health-based mold standards for buildings because of uncertainties about the levels of mold, the relationship between exposure and different health effects and differences in susceptibility from person to person.

Dr. Cheung further testified that in 2001, the National Institute for Occupational Safety published *The Role of Stachybotrys Mycotoxins in Building-Related Illness*,[15] which

[15] Elena H. Page & Douglas B. Trout, *The Role of* Stachybotrys *Mycotoxins in Building-Related Illness*, 62 Am. Indus. Hygiene Ass'n J. 644 (2001).

concluded that there was "inadequate evidence to support a causal relationship between symptoms or illness among building occupants, and exposure to mycotoxins." He then referred to *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, which was an evidence-based statement by the American College of Occupational and Environmental Medicine,[16] and testified that the article summarized a number of animal studies involving different mold concentrations and concluded that it was "highly unlikely" for a level of mycotoxins from mold in an indoor environment to be sufficiently high to cause human health effects.

Dr. Cheung, then, referenced the findings of the Committee on Damp Indoor Spaces and Health of the Institute of Medicine (IOM Committee) that there was insufficient or inadequate evidence to support an association between damp indoor environments—which include mold—and fatigue, neuropsychiatric, cancer, and immune diseases, as well as between the presence of mold, alone, and these symptoms.

After the IOM Committee Report, subsequent journal articles, Dr. Cheung testified, demonstrated that the level of exposure in a building was not associated with human health effects, including the American Academy of Asthma, Allergies and Immunology's *The Medical Effects of Mold Exposure*,[17] which stated that "[t]he occurrence of mold-related

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[16] Am. C. of Envtl. Med., *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, 45 J. of Envtl. Med. 470 (2003).

[17] Robert K. Bush et al., *The Medical Effects of Mold Exposure*, 117 J. of Allergy & (continued...)

toxicity from exposure to inhaled mycotoxins in non-occupational settings is not supported by current data, and its occurrence is improbable." Occupational settings, Dr. Cheung opined, referred to "an industry where you are routinely exposed to high amounts of molds, such as compost workers, mushroom workers, [and] farmers . . . ." Another article referenced by Dr. Cheung during his testimony, published by the American College of Medical Toxicology in 2006, provided: "With respect to mycotoxins in the indoor air, exposure modeling studies have concluded that even in moldy environments, the maximum inhalation dose of mycotoxins is generally orders of magnitude lower than demonstrated threshold for adverse health effects."

Finally, Dr. Cheung proffered a study that considered alternative potential causes for "sick building syndrome" in a water-damaged building, other than mold exposure, *Building Health: An Epidemiological Study of "Sick Building Syndrome" in the Whitehall II Study*,[18] which, he testified, concluded that "[t]he physical environment of the office building appeared to be less important than features of the psychosocial work environment [in] explaining differences in prevalence of symptoms."

After the *Frye-Reed* hearing, the Circuit Court issued written Findings of Fact and, thereafter, its Memorandum Opinion and Order. The Circuit Court Judge concluded that Dr.

(...continued)
Clinical Immunology 326 (2006).

[18] A.F. Marmot et al., *Building Health: An Epidemiological Study of "Sick Building Syndrome" in the Whitehall II Study*, 63 Occupational Envtl. Med. 283 (2006).

Shoemaker's "Repetitive Exposure Protocol" was a differential diagnosis, which itself was "reliable" and "properly performed," bringing it within general acceptance of the relevant scientific community. Because Dr. Shoemaker applied his technique to the employees at issue and found that they contracted these symptoms as a result of mold exposure, the Circuit Court held that the prior trial verdicts, entered in favor of the employees, remained intact:

> This Court has reached the following conclusions about whether the differential diagnosis process followed by Dr. Shoemaker was properly performed and reliable:
>
> > (1) The medical community is the relevant scientific community to determine whether differential diagnosis is a well recognized and generally accepted procedure.
> > (2) Differential diagnosis is well recognized and generally accepted in the medical community.
> > (3) Two of the well recognized methods to establish the presence of toxicity and an exposure are the presence of black or visible mold and an odor. Dr. Shoemaker verified the presence of visible mold and an odor in the building where [the employees and others] were exposed.
> > (4) There is no requirement that toxic mold alone be identified or determined to be in sufficient quantity to be the pollutant. In establishing an exposure, the medical doctor must consider the combination or chemical mix present in any particular water damaged building for a finding of general and specific causation.
> > (5) Dr. Shoemaker was aware of the exposure to microbial growth and proliferation before meeting with the claimants. He . . . met with the claimants for purposes of diagnosis and treatment rather than litigation. Dr. Shoemaker's expertise in treating patients exposed to toxicity dates back to

1980, and is not litigation-driven.

(6) Dr. Shoemaker took a detailed medical history and conducted a physical examination of [appellees] as a part of his routine diagnostic process.

(7) Dr. Shoemaker determined that there was a cohort of fifty (50) employees who were exposed. Of that number, eleven (11) employees were determined not to be ill and therefore were not tested. Dr. Shoemaker found twenty-two (22) employees who showed evidence of illness that could be ascribed to the exposure. Seventeen (17) other employees were examined and tested by other health care providers. Where a number of people become ill following an exposure, the temporal relationship between the exposure and illnesses becomes stronger and furnishes compelling evidence of causation.

(8) Dr. Shoemaker relied upon strong evidence of the temporal relationship between the onset and exacerbation of symptoms from exposure and the improvement and remission of symptoms upon treatment and removal from exposure. He also relied upon the reported relapse of symptoms in three (3) of the claimants following their exposure to boxed files that had been removed from the vacated office building.

(9) Dr. Shoemaker, after performing a differential diagnosis, was able to reach a final diagnosis to a reasonable degree of medical certainty as to the claimants. In doing [so], he relied upon evidence of an exposure, the presence of multiple health symptoms, and diagnostic studies and blood tests, to rule in probable causes and rule out alternative causes of the claimants' illnesses. The Tier Two (2) studies confirmed the final diagnosis.

Based on the foregoing conclusions, this Court holds that the differential diagnosis performed by Dr. Shoemaker was

> reliable and admissible to prove general and specific causation of the claimants' illnesses. Because this Court finds that Dr. Shoemaker's proper use of differential diagnosis, a methodology generally accepted by the relevant scientific community, satisfies the *Frye-Reed* test, the judgment shall remain in effect.

Montgomery Mutual filed a timely appeal to the Court of Special Appeals, which reversed in a reported opinion, 206 Md. App. 569, 51 A.3d 18 (2012). The intermediate appellate court concluded that there existed a continued division in the relevant scientific community relative to any causal connection between inhalation of mold in a water-damaged building and neurocognitive and musculoskeletal symptoms, including "sources that support and oppose Dr. Shoemaker's theories and methodologies, and at least one that recognizes the relevant scientific field is undecided," and held that "Dr. Shoemaker's theories and methodologies with regard to exposure to water damaged buildings, and the human health effects suffered by appellees, are not generally accepted in the relevant scientific community." *Id.* at 607, 51 A.3d at 41.

The employees, in seeking a reversal of the Court of Special Appeals' decision, maintain that the scientific literature they introduced constituted a "reasonable quantum of support" sufficient to establish Dr. Shoemaker's technique and causation theory as generally accepted in the relevant scientific community. Montgomery Mutual contends, however, that the literature, as well as the testimony presented at the *Frye-Reed* hearing, demonstrate that "there is no reliable evidence that any expert, other than Dr. Shoemaker, would reasonably rely upon Dr. Shoemaker's methods to form their own opinions on causal

relationship . . . ."

In testing the general acceptance in the relevant scientific community, methodology is a key component: the primary fundamental flaw in Dr. Shoemaker's methodology, according to Dr. Cheung, is a failure to account for the level of mold exposure in his "Repetitive Exposure Protocol." In determining whether Dr. Shoemaker's "differential diagnosis" and causal theory are generally accepted, we begin by observing that even Dr. Shoemaker, in his study associating mold exposure in water-damaged buildings to neurocognitive and muscuoloskeletal symptoms, acknowledged as a study limitation that "[e]xposure to specific agents was not demonstrated." 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 Neurotoxicology & Teratology 29, 29 (2005). He further stated that prior to 2005, his causal theory linking indoor air of water-damaged buildings with mold and neurocognitive and musculoskeletal symptoms was controversial and not generally accepted:

> The human health risk for chronic illnesses involving multiple body systems following inhalation exposure to the indoor environments of water-damaged buildings (WDBs) has remained poorly characterized and the subject of intense controversy.

Id.

> Several studies have investigated complaints of SBS[19] in populations occupying WDBs, but none have provided evidence sufficient to conclude that illness was firmly associated with exposure to biological agents.

Id. at 31.

Prior to 2005, the literature concerning a link between mold exposure and neurocognitive and musculoskeletal symptoms indicate, at best, uncertainty. These articles, however, make clear the importance placed by the relevant scientific community on the level of mold exposure.

In 2001, a literature review discussed studies published between 1994 and 1998 that involved questionnaires and self reports of health effects related to water-damaged homes or offices. Elena Page & Douglas Trout, *The Role of* Stachybotrys *Mycotoxins in Building-Related Illness*, 62 Am. Indus. Hygiene Ass'n J. 644 (2001). To establish a causal link, the authors posited that measuring the level of mold exposure is required, as opposed to the mere identification of mold on the wall of a building. *Id.* at 646 ("Identification of mycotoxin on a wall or in an air duct demonstrates only a potential for exposure and does not alone provide evidence of exposure, much less evidence linking reported symptoms to the fungi or fungi

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[19] The acronym "SBS" refers to "Sick Building Syndrome," which "refers to a combination of ailments associated with exposure to modern buildings that lack proper ventilation. The World Health Organization has identified sick building syndrome as an excess of irritation of the skin and mucous membranes and other symptoms, including headache, fatigue, and difficulty concentrating." *Chesson I*, 399 Md. at 318 n.1, 923 A.2d at 940-41 n.1, citing World Health Organization Regional Office for Europe, "Indoor air pollutants: exposure and health effects," EURO Reports and Studies No. 78, p. 23-26 (1983), available at http://whqlibdoc.who.int/euro/r&s/EURO_R&S_78.pdf.

products."). They ultimately concluded that there was insufficient evidence to establish a causal link:

> In summary, review of this related literature reveals evidence of clinical illness (in humans and animals) caused by ingestion of significant quantities of mycotoxin-contaminated foodstuffs. Illness associated with less well-defined (likely inhalation and/or dermatologic) bioaerosol exposures in agricultural or industrial environments has also been reported. However, the relevance of these findings to the indoor (nonindustrial) environment is unclear.
>
> * * *
>
> This review of the literature indicates that *there is inadequate evidence to support the conclusion that exposure to mycotoxins in the indoor (nonindustrial) environment is causally related to symptoms or illness among building occupants*.

Id. at 647 (emphasis added).

In 2003, the American College of Occupational and Environmental Medicine published an "evidence-based statement" discussing the state of scientific knowledge of correlations between mold exposure by inhalation and health problems. Am. C. Of Envtl. Med., *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, 45 J. of Envtl. Med. 470 (2003). In what appears to be in line with Dr. Cheung's testimony during the *Frye-Reed* hearing, the authors cautioned that human health effects may be linked to mycotoxins produced by mold, but only at a high "dose":

> If mycotoxins are to have human health effects, there must be an actual presence of mycotoxins, a pathway of exposure from source to susceptible person, and absorption of a toxic dose over

> a sufficiently short period of time. As previously noted, the presence of mycotoxins cannot be presumed from the mere presence of a toxigenic species . . . . For toxicity to result, the concentration and duration of exposure must be sufficient to deliver a toxic dose. What constitutes a toxic dose for humans is not known at the present time . . . .

Id. at 474. Several animal studies, involving the exposure of mice and rats to high or low concentrations of mycotoxins, led the authors to conclude that humans will not likely inhale a "toxic dose" of mycotoxins in a water-damaged building: "[l]evels of exposure in the indoor environment, dose-response data in animals, and dose-rate considerations suggest that delivery by the inhalation route of a toxic dose of mycotoxins in the indoor environment is highly unlikely at best, even for the hypothetically most vulnerable subpopulations." *Id*. at 475-76. No causal relationship had been established between "indoor airborne levels of microorganisms" with "building-related symptoms":

> Sick building syndrome, or non-specific building-related illness, represents a poorly defined set of symptoms (often sensory) that are attributed to occupancy in a building. Investigation generally finds no specific cause for the complaints, but they may be attributed to fungal growth if it is found. The potential role of building-associated exposure to molds and associated mycotoxins has been investigated, particularly in instances when *Stachybotrys chartarum* (aka *Stachybotrys atra*) was identified. . . . Recent critical reviews of the literature concluded that indoor airborne levels of microorganisms are only weakly correlated with human disease or building-related symptoms and that a causal relationship has not been established between these complaints and indoor exposures to *S. chartarum*.

Id. at 473-74 (footnotes omitted).

In 2004, the Committee on Damp Indoor Spaces and Health of the Institute of Medicine (IOM Committee) published a study investigating the relationship between damp indoor environments and illnesses. The IOM Committee summarized its findings "regarding the association between health outcomes and . . . the presence of mold or other agents in damp indoor environments" by noting that there was insufficient or inadequate information "to determine whether damp indoor environments or the agents associated with them are related to" non-respiratory symptoms. Comm. on Damp Indoor Spaces & Health, Inst. of Med., *Damp Indoor Spaces and Health* 10, tbl. ES-2 (2004) (finding inadequate or insufficient evidence to establish any association between exposure to damp, indoor environments and fatigue, gastrointestinal tract problems, neuropsychiatric symptoms, among other symptoms).[20]

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[20] The 2004 IOM Committee on Damp Indoor Spaces and Health Report, as well as the American College of Occupational and Environmental Medicine's evidence-based statement, *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, gained further support in Robert K. Bush et al., *The Medical Effects of Mold Exposure*, 117 J. of Allergy & Clinical Immunology 326, 329 (2006):

> Only certain mold species produce specific mycotoxins under specific circumstances. Importantly, the mere presence of such a mold should not be taken as evidence that the mold was producing any mycotoxin. For a toxic effect to occur in a subject, (1) the toxin must be present, (2) there must be a route of exposure, and (3) the subject must receive a sufficient dose to have a toxic effect. In the nonoccupational setting the potential route of exposure is through inhalation. Mycotoxins are not volatile and, if found in the respirable air, are associated with mold spores or particulates. They are not cumulative

(continued...)

Dr. Shoemaker contended that during and after he was in the process of publishing his 2005 article, *A Time-Series Study of Sick Building Syndrome*, *supra*, a watershed occurred and the relevant scientific community, thereafter, generally accepted his "Repetitive Exposure Protocol" and causal theory, as evidenced by those publications proffered during the *Frye-Reed* hearing and relating to neurocognitive and musculoskeletal symptoms: Luke Curtis et al., *Adverse Health Effects of Indoor Moulds*, 23 J. of Australasian C. of Nutritional & Envtl. Med. 3 (2004) and Edmond D. Shenassa et al., *Dampness and Mold in the Home and Depression: An Examination of Mold-Related Illness and Perceived Control of One's Home as Possible Depression Pathways*, 97 Am. J. of Pub. Health 1893 (2007).[21]

(...continued)

> toxins, having half-lives ranging from hours to days depending on the specific mycotoxin. Calculations for both acute and subacute exposures on the basis of the maximum amount of mycotoxins found per mold spore for various mycotoxins and the levels at which adverse health effects are observed make it highly improbable that home or office mycotoxin exposures would lead to a toxic adverse health effects.
>
> Thus we agree with the American College of Occupational and Environmental Medicine evidence-based statement and the Institute of Medicine draft, which conclude that the evidence does not support the contention that mycotoxin-mediated disease (mycotoxicosis) occurs through inhalation in nonoccupational settings.

[21] Before us in oral argument, counsel for the employees was asked whether Dr. Shoemaker's methodologies were taught in medical school or located in a medical textbook, to which he replied that Dr. Shoemaker is referenced in *Harrison's Manual of Internal Medicine*, regarding his treatment of Pfiesteria. A review of *Harrison's* reveals that under "Pfiesteria," recommended treatment involves Cholestyramine, without reference to Dr. (continued...)

Adverse Health Effects of Indoor Moulds, *supra*, summarized the results of select clinical studies and acknowledged a link existed between mold exposure and "multi-system adverse effects." This link, however, was unsupported by the medical literature, and, even more compelling to the present case, advised that in diagnosing and treating mold-related health problems, the level of exposure, as part of a "careful environmental and medical history" was essential:

> **Diagnosis and Treatment of Mould Related Problems**
> A careful environmental and medical history is an essential first step in evaluating a patient for mould-related health problems. . . . Environmental sampling for viable spores, total spores, and mycotoxins in the air and dust can provide important exposure information.

Id. at 3. For the purposes of general acceptance in the relevant scientific community, this article does not support a causal connection between mold exposure and neurocognitive and musculoskeletal symptoms.

Dampness and Mold in the Home and Depression, supra, studied a potential link between mold and depression based on survey data from eight European cities. The study resulted in an association between living in a mold-exposed environment and depression, but the authors acknowledged that causation was neither established nor supported by the study: "However, such an association may not be causal, but rather attributable to residual confounding by variables that were either assessed with error or not measured in our study.

(...continued)
Shoemaker or his "Repetitive Exposure Protocol."

. . . . The cross-sectional design of our study does not allow [an] inference regarding causality." *Id*. at 1897, 1898.

Another article published in 2007 and referenced by Dr. Cheung, Clifford S. Mitchell et al., *Current State of the Science: Health Effects and Indoor Environmental Quality*, 115 Envtl. Health Perspectives 958 (2007), supports the conclusion that Dr. Shoemaker's theory about mold exposure and neurocognitive and musculoskeletal symptoms remains unverified, controversial, and not generally accepted within the relevant scientific community. The article addressed specifically the issue of airborne mold exposure and non-respiratory symptoms, by first recounting the 2004 IOM Committee Report, *supra*, indicating that there is no established causal link between mold exposure and non-respiratory health problems, *id*. at 961 ("One area in which the IOM panel felt evidence was insufficient to conclude whether an association or causal relationship concerned molds and a number of systemic conditions alleged to be related to mycotoxins."), and then concluding that the scientific community had not reached a consensus on whether mold exposure can produce non-respiratory symptoms. The article observed that even after Dr. Shoemaker's studies, a lack of consensus in the relevant scientific community remained:

> Despite the absence of validated markers of exposure, efforts have been made to understand the relationship between mold exposures and chronic non allergic health effects. There have also been trials of empiric therapies for treating mold-exposed individuals, including patients treated with cholestyramine (Shoemaker and House 2005). *There remains a lack of consensus regarding the systemic effects of mold exposure*.

Id. at 962 (emphasis added).

We would note that other jurisdictions have determined that Dr. Shoemaker's theory, based on his "Repetitive Exposure Protocol," is neither generally accepted nor reliable. *See Young v. Burton*, 567 F. Supp. 2d 121, 130-31 (D.D.C. 2008) (also listing Virginia, Florida, and Alabama as jurisdictions rejecting Dr. Shoemaker's theory). In *Young v. Burton*, 567 F. Supp. 2d 121 (D.D.C. 2008), the United States District Court for the District of Columbia ordered that Dr. Shoemaker's expert testimony be excluded under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993),[22] because the court could "not conclude that 'mold illness' is a generally accepted illness in the medical community" and any diagnosis resulting from Dr. Shoemaker's program is unreliable. 567 F.Supp.2d at 131. His theory was not generally accepted in part because there is no classification for mold illness in the World Health Organization's International Classification of Diseases, no one other than Dr. Shoemaker has published any peer-reviewed articles on the illness as defined by his "differential diagnosis," and in his deposition for that case, Dr. Shoemaker conceded that chronic building-associated illness is not a generally accepted diagnosis in the medical community:

> Q: And [Chronic Building Associated Illness], can we say that that's not a generally-accepted diagnosis?

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[22] In *Blackwell*, we considered opinions of various federal jurisdictions that employ *Daubert*, rather than *Frye*, to observe "what they have opined . . . when they are speaking about reliability." 408 Md. at 605, 971 A.2d at 253. The reliability of a scientific technique at issue is also a consideration under *Frye*.

A: No argument about that.

Id.

Dr. Shoemaker's technique, which reflects a dearth of scientific methodology, as well as his causal theory, therefore, are not shown to be generally accepted in the relevant scientific community; we affirm the Court of Special Appeals' judgment.

JUDGMENT OF THE COURT OF SPECIAL APPEALS AFFIRMED. COSTS IN THIS COURT AND THE COURT OF SPECIAL APPEALS TO BE PAID BY PETITIONER.