Fraser v 301-52 Townhouse Corp.

2008 NY Slip Op 10208

Decided on December 30, 2008

Appellate Division, First Department

Published by New York State Law Reporting Bureau pursuant to Judiciary Law § 431.

This opinion is uncorrected and subject to revision before publication in the Official Reports.

Decided on December 30, 2008
Mazzarelli, J.P., Friedman, Nardelli, Williams, Freedman, JJ.

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[*1]Colin Fraser, et al., Plaintiffs-Appellants,

v

301-52 Townhouse Corp., et al. Defendants-Respondents.

Jaroslawicz & Jaros, LLC, New York (Robert J. Tolchin of counsel), for appellants.
Schechter & Brucker, P.C., New York (Thomas V. Juneau, Jr. of counsel), for respondents.

Order, Supreme Court, New York County (Shirley Werner Kornreich, J.), entered July 9, 2007, which granted plaintiffs' motion for reargument and renewal of a prior order, same court and Justice, entered October 5, 2006, which, after a *Frye* hearing, granted defendants' motion to preclude plaintiffs from offering certain expert evidence at trial and granted defendants summary judgment dismissing plaintiffs' causes of action based on personal injury, and, upon reargument and renewal, adhered to the original determination, affirmed, without costs. Appeal from the aforesaid order entered October 5, 2006, unanimously dismissed, as academic, without costs.

Plaintiffs, former residents of a unit in the cooperative apartment building owned by defendant 301-52 Townhouse Corp., assert causes of action against defendants for, inter alia, personal injuries (specifically, respiratory problems, rash and fatigue) allegedly caused by dampness in the building and the mold infestations that allegedly resulted from such dampness. Upon defendants' motion seeking summary judgment and preclusion of plaintiffs' expert evidence purporting to establish that the building's alleged dampness and mold condition caused their health problems, the motion court directed that a *Frye* hearing be held to determine whether plaintiffs' causation theory was generally accepted as reliable within the relevant scientific community. After the *Frye* hearing, the court granted defendants' motion, precluding the expert evidence and dismissing the personal injury claims (other causes of action were severed for further proceedings). The court subsequently granted plaintiffs reargument and renewal, and, upon reargument and renewal, adhered to the prior determination. We now affirm.

Contrary to the dissent's contention, defendants' experts did deny that it is generally accepted within the scientific community that it has been established that indoor dampness and mold "cause" health problems like plaintiffs'. While there is general agreement that indoor dampness and mold are "associated" with upper respiratory complaints, defendants' experts took the position, consistent with the literature they submitted, that the observed association between such conditions and such ailments is not strong enough to constitute evidence of a causal [*2]relationship [FN1]. In other words, " *association' is not equivalent to causation*"' (Green, Freedman & Gordis, Reference Guide on Epidemiology, in Federal Judicial Center, *Reference Manual on Scientific Evidence*, at 336 [2d ed 2000] [emphasis in original]; *see also id.* at 348 ["Although a causal relationship is one possible explanation for an observed association between an exposure (to an agent) and a disease, an association does not necessarily mean that there is a cause-effect relationship"]). In this regard, even plaintiffs' main expert, Dr. Eckardt Johanning, testified that "association" is not the same concept as "causation." Given that plaintiff failed to demonstrate general acceptance of the notion that a causal relationship has been demonstrated between the conditions and ailments in question, Dr. Johanning's claim to have established causation in this case by means of "differential diagnosis" is unavailing (*see Marso v Novak*, 42 AD3d 377, 378 [2007] [expert's opinion as to causation, at which he arrived through differential diagnosis, was not admissible where the resulting conclusion was not accepted in the medical community]; *see also Lara v New York City Health & Hosps. Corp.*, 305 AD2d 106 [2003] [affirming preclusion of expert testimony that "relied solely on a theory . . . neither recognized nor accepted" in the medical community]). Thus, on the record presented to us, plaintiffs have failed to meet their burden of establishing general acceptance of the theory on which the specific claims at issue are based. We note that whether plaintiffs' theory of

causation is scrutinized under the *Frye* inquiry applicable to novel scientific evidence (*see Parker v Mobil Oil Corp.*, 7 NY3d 434, 446-447 [2006]) or under the general foundational inquiry applicable to all evidence (*see id.* at 447), the conclusion is the same: the proffered expert evidence must be precluded on the ground that the underlying causal theory lacks support in the scientific literature placed before us in the present record. We stress that our holding does not set forth any general rule that dampness and mold can never be considered the cause of a disease, only that such causation has not been demonstrated by the evidence presented by plaintiffs here.

Nothing said here "set[s] an insurmountable standard" (*Parker*, 7 NY3d at 447) for the reception of scientific evidence. In particular, we disclaim the suggestion attributed to us by the dissent that "*Frye* requires that the medical literature conclusively establish that an allegedly offending substance not only have the potential to cause illness but that it *always* causes illness" (emphasis in original). To be clear, the deficiency of plaintiffs' expert evidence is not that the medical literature fails to "conclusively establish" their causal theory or to show that indoor dampness and mold "*always* cause[] illness."[FN2] Rather, plaintiffs' expert evidence falls short [*3]because *none* of the medical literature in the record supports the stated position of plaintiffs' expert that the observed association between damp or moldy indoor environments and upper respiratory symptoms is strong enough to be considered, under generally accepted principles of scientific analysis, evidence that the former causes the latter. Aside from referencing two studies that Dr. Johanning mischaracterized as demonstrating a causal link, the dissent does not identify any study concluding that indoor dampness and mold have been shown to cause upper respiratory symptoms such as plaintiffs'[FN3]. Without any warrant in the scientific literature in the record, the dissent, like Dr. Johanning, simply asserts that "the association' between building dampness and illness is one of causation," thereby conflating the distinct concepts of association and causation.[FN4]

Even if it is assumed that plaintiffs' experts established the general acceptance of their view that indoor dampness and mold is capable of causing plaintiffs' health problems (general [*4]causation), the experts failed to specify the threshold level of exposure to dampness or mold needed to produce these effects. Without evidence that they were exposed to a level of dampness or mold sufficient to cause their alleged injuries (specific causation), plaintiffs cannot prevail on their personal injury claims (*see Parker*, 7 NY3d at 448 [plaintiff must show not only exposure to the toxin and that the toxin is capable of causing the particular illness alleged, i.e., general causation, but also that plaintiff was exposed to sufficient levels of the toxin to cause the illness, i.e., specific

causation]). It appears from plaintiffs' own literature that there is no standardized or recognized method of measuring "dampness," thus rendering it impossible for plaintiffs' experts to compare the level of dampness in plaintiffs' apartment to that in the studies (*cf. id.* at 449). Nor would plaintiffs' experts be able to make any reasoned comparison of plaintiffs' exposure to the by-products of dampness to those in other studies. While plaintiffs did offer a measure of the level of mold present in the apartment, their experts did not testify to any threshold level at which mold is capable of causing the injuries of which plaintiffs complain. Finally, while "it is not always necessary for a plaintiff to quantify exposure levels precisely or use the dose-response relationship" (*id.* at 448), we do not believe that, under the circumstances, plaintiffs' reliance on the method of differential diagnosis was an adequate substitute for quantitative proof.

An additional ground for granting summary judgment dismissing the personal injury claims is that plaintiffs failed to offer a reliable measurement of the level of mold in the subject apartment. That is to say, even if plaintiffs' theory of causation satisfied the *Frye* test, the mold measurement they offered does not meet the standard of reliability set forth in the record and therefore fails to satisfy the post-*Frye* foundational inquiry into "whether the accepted methods were appropriately employed in a particular case" (*Parker*, 7 NY3d at 447, citing *People v Wesley*, 83 NY2d 417, 429 [1994])[FN5]. A textbook that plaintiffs placed into evidence at the hearing states that an estimate of average inhalation exposure should be based on sampling at least three times a day for at least three consecutive, representative days, with duplicate samples for all analyses (Macher, ed., *Bioaerosols: Assessment and Control*, at 5-10 [1999]). Plaintiffs' environmental expert, however, collected only two indoor air samples within a short time span on the same day, which, according to plaintiffs' own authority, was insufficient.

Finally, although defendants filed their motion for summary judgment 300 days after the filing of the note of issue, defendants have demonstrated good cause for the delay in that disclosure had been completed only two weeks before the motion was made. Accordingly, the motion was properly considered on the merits (*see Pena v Women's Outreach Network, Inc.*, 35 AD3d 104, 108 [2006]).

All concur except Mazzarelli, J.P. and Nardelli J., who dissent in a memorandum by Mazzarelli, J.P. as follows:

MAZZARELLI, J. (dissenting)

Plaintiffs allege that they suffered adverse health effects as a result of chronic water leaks into their cooperative apartment. The leaks began in 1996. In their bill of particulars plaintiffs

[*5]asserted that, as a result of the leaks, damp conditions prevailed in the apartment and promoted the generation of "toxic mold, toxic fungi, [and] other microbial life." They further claimed that the damp conditions caused them to suffer, among other things, repetitive upper respiratory infections, asthmatic symptoms, severe allergic reactions and allergy symptoms, rashes and fatigue. In addition, plaintiffs claimed certain cognitive and fertility problems, but subsequently withdrew those claims.

Plaintiffs identified five expert witnesses who they expected to testify at trial. Included among those experts was Dr. Eckardt Johanning, a medical doctor who had examined plaintiffs and who has extensively studied in the field of "health effects of microbiological exposure." The disclosure statement indicated that Dr. Johanning was expected to testify that the damp conditions inside the apartment exposed plaintiffs to "excessive and atypical microbiological contamination." Dr. Johanning was further expected to testify that such exposure can cause serious health effects in humans and that plaintiffs were harmed by the damp conditions in their apartment.

Defendants moved for summary judgment and, in the alternative, for preclusion of plaintiffs' medical experts, or a hearing pursuant to *Frye v United States* (293 F 1013 [DC Cir 1923]). With respect to summary judgment, defendants argued that plaintiffs could not prove that, generally speaking, "the presence of, or exposure to, mold in an indoor residential setting causes the types of ailments" alleged by plaintiffs. They argued that plaintiffs could not specifically prove they were exposed to "mycotoxins" in the apartment and that, even if they could, they could not prove when the exposure took place or the "dose or duration" of any such exposure.

With respect to preclusion, defendants specifically sought to bar testimony

> "to the extent the plaintiffs intend to have [their expert] witnesses provide scientific evidence' or opinions that: (a) the presence of, or exposure to, mold in an indoor residential setting causes the types of ailments for which [p]laintiffs are seeking money damages (such evidence' or opinions are not generally accepted as reliable by the scientific community); and (b) the presence of, or the exposure of [p]laintiffs to, mold in the Apartment caused [p]laintiffs' specific alleged injuries (such evidence' or opinions cannot be provided with a reasonable degree of medical certainty)."

Defendants submitted affidavits from three physicians in support of the motion. One of the affidavits, that of a neuropsychologist, became irrelevant once plaintiffs withdrew their claim that the damp condition in their apartment caused them to suffer cognitive deficits. The first was by Ronald E. Gots, M.D., Ph.D., a toxicologist who does not purport to have any expertise in mold sampling methodology. He devoted three paragraphs to critiquing mold sampling performed inside

the apartment and offered some generalities purporting to support his opinion that the sampling data "is not reliable for determining exposure." Dr. Gots failed to discuss any of the specific findings in any of the mold sampling reports exchanged by plaintiffs during discovery.

Dr. Gots opined that it is highly unlikely that the levels of mold humans can be exposed to in residential or commercial buildings can ever be enough to cause mycotoxicosis, that is, illness caused by the biochemical products produced by molds as part of their life cycle. However, he acknowledged that indoor mold can cause allergic effects "manifest[ed] primarily as [*6]respiratory allergies" and that, although uncommon, "[i]rritant effects may occur when there is significant mold growth (thousands of mold spores per cubic meter of air)." At best, he stated, household mold can cause allergic reactions in the 5% of people who are allergic to mold in the first place. Dr. Gots cited to two published scientific works to support his theory that any relation between building dampness and illness is essentially hypothetical. These were a 2002 paper by the American College of Occupational and Environmental Medicine (ACOEM) entitled *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, and a 2004 book entitled *Damp Indoor Spaces and Health*, which reflected findings of a study conducted by the Institute of Medicine of the National Academies (IOM).

Dr. Gots also opined that under "causation analysis" plaintiffs could not prove their claim. He described the basic principle of such analysis as (1) identifying what is wrong with the patient, (2) whether the "agent at issue" can produce the given disorder, and (3) whether the agent did indeed cause the disorders at issue in the case. He claimed that plaintiffs could not have had an allergic reaction to mold because "RAST" blood testing performed on them, which looked for allergen-specific IgE-mediated antibodies, was negative. He discounted IgG testing results [FN1] as irrelevant to respiratory allergies and stated that, in any event, positive IgG results could not prove the time and place of exposure. He described the complaints of plaintiffs recorded in their medical records as subjective and not contemporaneous with medical visits. He further questioned why the symptoms persisted even after plaintiffs vacated the apartment. Dr. Gots concluded that plaintiffs' complaints had to have been related to conditions other than exposure to the damp conditions in the apartment. He did not offer any opinion as to what could have caused plaintiffs' symptoms.

The second affidavit was by S. Michael Phillips, M.D. He conceded that indoor mold can cause some of the symptoms asserted in plaintiffs' bill of particulars. For example, Dr. Phillips stated that plaintiffs "may have been exposed to molds and had minor allergic/irritant reactions resulting from such exposure." He further noted that "[e]pidemiologic studies indicate that the

presence of mold in indoor environments is associated with upper respiratory symptoms, cough, wheeze, asthma symptoms in sensitized asthmatic persons, and hypersensitivity pneumonitis in susceptible persons." He cited to one of the two scientific publications relied upon by Dr. Gots to support this conclusion. Dr. Phillips also stated that "some of [plaintiffs'] complaints are compatible with irritant or allergic reactions to molds."

However, Dr. Phillips concluded that plaintiffs could never prove that mold caused their symptoms because they could not establish that the level of mold in the apartment was sufficient to result in adverse health effects. However, much like Dr. Gots, Dr. Phillips failed to compare the actual mold measured by plaintiffs to what, in his expert opinion, would be a sufficient level to cause illness. Rather, he described the levels of "expected indoor molds" recorded inside the apartment as "modest," and asserted that "[n]o convincing evidence of high-level exposures via [*7]actual air sampling data was available in the materials available for my review." Also, similarly to Dr. Gots, Dr. Phillips noted that plaintiffs' "alleged allergic symptoms have more likely explanations than mold," and suggested that perhaps dust mites and/or plaintiffs' cats were the culprits. Finally, Dr. Phillips asserted that the ill effects of mold exposure are transitory and could not have persisted in plaintiffs after they moved out of the apartment.

In opposition to defendants' motion, plaintiffs submitted the affidavit of Dr. Johanning. In reviewing the state of scientific thought on the relationship between building dampness and illness, Dr. Johanning, who is Board certified in Family Practice and in Occupational and Environmental Medicine, focused on two "large-scale, peer-reviewed epidemiological studies." First, he discussed *Respiratory Morbidity in Office Workers in a Water Damaged Building*, commissioned by the National Institute of Health and published on line in January 2005. Second, he discussed *Home Dampness and Molds, Parental Atopy, and Asthma in Childhood: A Six-Year Population Based Cohort Study*. This was published in the March 2005 edition of the peer-reviewed journal *Environmental Health Perspectives*. Dr. Johanning represented that these two studies:

> "showed that the association' between damp buildings, mold, and respiratory morbidity, including new-onset asthma, is one of causation, a fact that had been apparent to clinicians for years. More importantly, they show that building dampness and mold cause permanent irritative and allergic-type problems, including new-onset asthma. Because these studies answered questions left open by the ACOEM and IOM papers **[FN1]**, they were widely publicized and discussed. Neither Dr. Gots nor Dr. Phillips are aware of these studies, or if they are, they chose not to reveal them to the Court, and instead assert that the biased ACOEM 2002 paper and the IOM 2004 paper are the only and final word' on the matter. They are not. Occupational and Environmental physicians involved in

> direct patient care and research disagree with the conclusions by scientists' with mostly theoretical or peripheral experience about these clinical matters. The defendants' experts' ignorance (or concealment) of the current relevant medical literature is no basis to exclude my testimony. These papers directly contradict the assertions of Drs. Gots and Phillips that irritative and/or allergic-type reactions caused by damp buildings are always transitory in nature."

Unlike defendants' experts' submissions, Dr. Johanning explained in detail the significance of the mold samplings taken by plaintiffs. For example, in attempting to discredit Dr. Gots's statement that indoor air sampling levels in the apartment were below outdoor levels, he pointed out that the indoor air samples were "approximately triple and five times higher than the outdoor sample in terms of levels." He further noted the significance of the fact that the indoor samples were dominated by Aspergillus versicolor, "an atypical, hydrophilic... mold not commonly found in the outdoor air in any significant concentration." According to Dr. Johanning:

> "From a medical perspective, its presence and predominance in the Fraser home was very significant because it reveals the presence of atypical species, meaning [*8]that our bodies are not used to breathing it in significant concentrations, and it or its by-products are therefore highly allergenic and irritative. This testing is indeed relevant from a health and exposure assessment perspective, as it is indicative of exposure by elevated levels of atypical molds, as a consequence of water events that preceded the testing."

Dr. Johanning further disputed Dr. Gots's statement that in order for mold to exert physical effects on a person that person must be one of the 5% of the general population who are generally susceptible. He explained that, contrary to Dr. Gots's position, mold irritation is not necessarily an allergic reaction but can come about because of chronic irritation caused by inhalation of mold. Accordingly, Dr. Gots's observation concerning the absence of elevated IgE levels was, Dr. Johanning observed, irrelevant. It was sufficient that Colin and Pamela Fraser

> "showed clear evidence of microbial specific IgG antibodies (typical in Type III or Type IV reactions) to a number of organisms commonly found in damp buildings. This means that their bodies produced antibodies in response to an exposure to these organisms prior to the testing and consistent with the patient's history and timeline of exposure."

Defendants did not submit any papers in reply to plaintiffs' opposition.

Without discussion of the parties' respective positions and submissions, the motion court denied the summary judgment motion and directed a hearing pursuant to *Frye v United States* (293 F 1013 [DC Cir 1923], *supra*). The court stated:

> "The submissions have raised an issue [of fact] as to whether the theory of plaintiffs - that mold in their apartment caused them respiratory problems - is generally accepted in the relevant scientific community and whether the methodology used by plaintiffs to measure the mold was within generally accepted scientific methods."

The *Frye* hearing was conducted on 10 days between July 27, 2005 and March 28, 2006. Dr. Johanning and Paul Ehrlich, M.D., a clinician specializing in pediatric allergies and asthma, testified on plaintiffs' behalf. Dr. Gots and Dr. Phillips testified for defendants. Plaintiffs placed in evidence nearly 40 articles, treatises and other published studies concerning the relationship between building dampness and mold and sickness in humans. Defendants placed approximately 15 such publications in evidence.

Dr. Johanning testified that the symptoms with which plaintiffs presented to him were caused by the damp conditions in the apartment. He stated that he utilized a differential diagnosis methodology, which he described as:

> "using a comprehensive occupational and environmental history, physical examination, laboratory tests, review of environmental data, looking at medical reports and test results from other providers, and looking at any information that can help me to rule in or out diagnosis or differential diagnostic considerations."

Dr. Johanning then explained that, in diagnosing Colin Fraser, he consulted the various environmental reports created by Olmsted Environmental Service, as well as medical reports generated by other medical providers who had examined or treated Mr. Fraser. He also viewed [*9]photographs showing stains and discoloration inside the subject apartment.

Dr. Johanning took his own history of Mr. Fraser. Mr. Fraser related that he never smoked or abused alcohol or drugs, and that he had never been exposed to organic dust or significant bioaerosols such as those associated with garden work, pesticides, heavy metals or chemicals. Although Mr. Fraser worked as a stamp broker, he denied working with wet or moldy stamps or working in anything but "clean" environments. Mr. Fraser filled out an eight-page questionnaire regarding his health. He reported that he felt generally better since vacating the apartment.

Dr. Johanning also performed a complete physical examination of Mr. Fraser. He ordered laboratory testing, the results of which revealed that Mr. Fraser was not suffering from an infection or any other identifiable condition. His total immunoglobin count was normal, indicating that he had an appropriate and normal level of immune parameters. However, Mr. Fraser had IgG subclass

abnormalities "which showed an altered immune response similar as it can be seen in people who have allergy." Dr. Johanning specified that these IgG levels (which he described as "striking") indicated "hypersensitivity to a number of fungi and bacteria, precisely . . . six out of eight; specifically, Micropolyspora, Thermoactinomyces, Alternaria, Aureobasidium, Phoma herbarum, Trichoderma." Two of those organisms, Micropolyspora, Thermoactinomyces, were described by Dr. Johanning as being more akin to bacteria than mold, and he testified that they were commonly found in people who are exposed to wet organic material, including wood. Dr. Johanning described all of these organisms as being capable of causing allergic, irritative and toxic reactions. Three of them, which are molds, were found in the apartment, according to laboratory reports reviewed by Dr. Johanning. The fact that the particular mold- and bacteria-antibody-specific IgG levels were high indicated to Dr. Johanning that Mr. Fraser had been exposed to those particular organisms for a lengthy period of time. Dr. Johanning stated that it was not necessary to perform skin prick testing to further confirm the significance of abnormal IgG results.

When asked to describe his diagnosis of Mr. Fraser, Dr. Johanning testified as follows:

> "Essentially, again, based on the history, the presentation, past medical history, family history, review of systems, the work history, the results of physical examination, laboratory test results as I outlined earlier, the environmental information, consultation reports from other specialists, I concluded, using a differential diagnosis approach, that the best explanation for Mr. Fraser's problem is the history of acute irritant allergic type reaction while he was living in his previous apartment at 301 East 52nd Street."

Regarding plaintiff Pamela Fraser, Dr. Johanning recounted a similar history related by the patient. The information elicited was also designed to rule out other possible causes of the reported symptoms including nasal problems, itchy and teary eyes, shortness of breath, burning sensation in the throat, sore throat, wheezing and tightness in the chest. IgG testing showed reaction to Micropolyspora, Thermoactinomyces and Trichoderma. Dr. Johanning testified that repeated general blood count and differential and platelet counts did not indicate any other medical problems. Accordingly, Dr. Johanning recorded in his records that, based on all the laboratory and clinical findings:

> "and the differential diagnosis approach, I conclude with a reasonable degree of medical certainty that Miss Fraser had a history of allergic and irritant type [*10]reactions while she was residing at her previous apartment which had water damage and microbial growth problems."

Finally, Dr. Johanning testified about his examination of the infant plaintiff, who, because of

her age, could not be subjected to the same diagnostic tests as her parents (including testing for IgG levels). The parents related a history of respiratory problems which dissipated after they vacated the apartment. Dr. Johanning concluded that, based upon his differential diagnosis approach, the child had respiratory problems which seemed to be ongoing and episodic and that, while she was too young to determine specific allergies, it was "reasonable to assume" that where she lived "caused some respiratory inflammation and allergic response."

Dr. Phillips testified that Dr. Johanning did not record the presence of IgE antibodies in any of the plaintiffs. While he described IgG as having the capability to cause certain diseases in people, he characterized it as "common" and "not unexpected." Moreover, he testified that IgG test results provide no clues as to when a person was exposed to a particular antigen, the length of exposure or the amount of exposure. He stated that approximately 10% of people have developed antibodies to mold, but less than 50% of those people have showed clinical problems related to mold.

Dr. Phillips asserted that IgG is not correlated in any way or related to an allergy. He criticized the manner in which Dr. Johanning tested for IgG, claiming that his technique would always yield a positive result. Moreover, because he did not see the presence in Dr. Johanning's report of any clinical symptoms associated with IgG exposure, he concluded that it was impossible to tell whether the conditions reported in plaintiffs' apartment contributed to any illness.

Subsequent to the *Frye* hearing, the court issued an order holding that "plaintiffs are precluded from introducing testimony demonstrating that mold caused their health complaints and plaintiffs' causes of action based upon personal injury are dismissed with prejudice." The order contained a lengthy recitation of facts that summarized in detail the initial mold sampling report secured by plaintiffs, the medical reports prepared by Dr. Johanning upon his examination of plaintiffs, the testimony of all the witnesses, and the scientific publications submitted by both sides. The court discredited the testimony of both Dr. Johanning and Dr. Gots as being compromised by their "strongly held views on the subject of mold and a stake in advancing those views." It credited the testimony of Dr. Phillips, whom it found to be "very impressive", and found that "plaintiffs failed to demonstrate that the community of allergists, immunologists, occupational and environmental health physicians and scientists accept their theory - that mold and/or damp indoor environments cause illness."

The court summarized the scientific writings submitted by the parties, which it described as "peer-reviewed and published in journals generally accepted in the scientific community," and concluded that they:

> "demonstrate that, with the exception of one article, the scientific research has not established that indoor exposure to mold causes the symptoms for which the plaintiffs seek to recover in this action. Although some of the literature found that indoor mold exposure or dampness had an association' with transient upper respiratory problems in adults (symptoms similar to those of the common cold), or a strong association with asthma in children, these findings fall short of a finding of causation.'"

[*11]

The court then ruled that, even if plaintiffs had established at the hearing that, generally, there is a causal link between building dampness/mold and illness, the case could not go forward. According to the court:

> "It became clear at the hearing that plaintiffs wished to argue that moisture in the Fraser apartment caused them ill health. Plaintiffs contended that a damp indoor environment produced bacteria, mold, endotoxins, Beta Glucans, MVOCs and other toxic materials, which caused the Frasers' complained of symptoms. However, moisture, bacteria, endotoxins, MVOCs and Beta Glucans were never measured in the Fraser apartment. Moreover, the scientific literature and the testimony of Dr. Phillips established that two measurements for mold in a short time span, the method of measurement used here, was insufficient to give a valid mold reading. Then too, the hearing evidence demonstrated that: there are no standards for what amount of mold was excessive in terms of human health and the indoor environment; there are no generally accepted standards for measuring indoor airborne mold; there are no generally accepted standards for the acceptable amount of mold in indoor air; there are many types of mold, each of which have different or no health effects; there are no standard scientific definitions for "dampness" or "moisture"; skin prick tests for allergy, which were not done here, were deemed the most reliable way to test for allergy by the literature, Dr. Ehrlich, Dr. Gots and Dr. Phillips; and the IgE test performed on Colin and Pamela Fraser, which is related to allergies, did not show allergy to mold."

Plaintiffs moved to reargue and renew the order. They sought reargument based on a variety of asserted defects. This was granted, solely to modify the order to eliminate any reference to the manner in which air testing of plaintiffs' apartment was conducted, which the court recognized was improper in a *Frye* hearing. Renewal, based on the intervening Court of Appeals decision in *Parker v Mobil Oil Corp.* (7 NY3d 434 [2006]), was also granted. However, based on *Parker*, the court held that plaintiffs had failed to lay a proper foundation for their experts' testimony. The court determined that Dr. Johanning's differential diagnosis, which it defined as "a list of possible causes of a symptom," was an inadequate foundation for a finding of specific causation. The court wrote:

> "Dr. Johanning testified without underlying proof of causation or strong association, without proof of mold allergies, without reliable standards for measurement of mold exposure, and without measurements of mold by-products that plaintiffs' symptoms must have been caused by airborne mold and mold by-products. On the other hand, with respect to Mrs. Fraser, he failed to rule in cat and dust allergies."

The court further stated that *Parker* implies that only a "significant association" between a substance asserted to cause illness and illness itself is enough to pass the *Frye* test. It found that this is "consistent with Dr. Phillips' testimony that a strong association occurs all of the time."

The court acknowledged that some courts had found Dr. Johanning's differential diagnosis valid, but failed to provide any case citations. However, it stated that "other courts, which this court finds more persuasive, have disagreed," citing *Jazairi v Royal Oaks Apt. Assoc., L.P.* (217 Fed Appx 895 [11th Cir 2007]) and *Roche v Lincoln Prop. Co.* (278 F Supp 2d 744 [D [*12]Va 2003]). For a second time, the court granted summary judgment to defendants dismissing all of plaintiffs' personal injury claims.

Defendants' submissions on their original motion plainly did not, in the first instance, support the need for the *Frye* hearing directed by the motion court. Accordingly, I would reverse. Neither Dr. Gots nor Dr. Phillips ever stated in their respective affidavits that it is not generally accepted by scientists that indoor mold or damp conditions can cause the allergies and irritation experienced by plaintiffs. To the contrary, both stated, Dr. Phillips in unambiguous terms, that indoor mold and building dampness have the potential to cause the health conditions alleged by plaintiffs.

Defendants also failed to rebut Dr. Johanning's criticism of the scientific studies upon which they relied, nor did they even attempt to refute the studies cited by Dr. Johanning in his own affidavit. Defendants' failure to call into question the studies submitted by plaintiffs is most significant, because it left the court with two studies ostensibly doubting any link between indoor mold and illness and two which supported such a link. Even one of defendants' experts relied favorably on the IOM study - which defendants submitted in support of their motion - as supporting his statement that "the presence of mold in indoor environments is associated with upper respiratory symptoms, cough, wheeze, asthma symptoms in sensitized asthmatic persons, and hypersensitivity pneumonitis in susceptible persons."

The purpose of a *Frye* hearing is not to prove by any particular evidentiary standard that proposed scientific evidence is sound. Rather, it is to establish that a theory has gained general recognition in the scientific community. To be sure, the proponent of scientific evidence bears the

burden of establishing *Frye* admissibility (*Marso v Novak*, 42 AD3d 377, 378 [2007]). However, before that burden is ever imposed the party contesting the proffered evidence must first make a prima facie case that the theory has *not* gained general recognition in the scientific community (*see Middleton v Kenny*, 286 AD2d 957, 958 [2001]). Here, defendants' submissions failed to make a prima facie case that there is not a consensus in the scientific community that building dampness and mold can cause illness. In fact, their experts conceded this point.

As for the second issue defined by the motion court in its order directing a *Frye* hearing, defendants similarly failed to make a prima facie showing that the methodology by which plaintiffs measured mold in the subject apartment was not generally accepted by the relevant scientific community. Defendants' experts never identified how plaintiffs' mold-measuring methodology was "novel" such that plaintiffs should have been required to establish general acceptance. Indeed, in the original order to show cause submitted by defendants, they did not even seek a *Frye* hearing on this subject. Ultimately, the manner in which plaintiffs collected mold samples was a minor factor in the court's decision to preclude plaintiffs' experts. Indeed, in the decision on reargument the court modified the original order to eliminate any reference to the manner in which mold was sampled. Nevertheless, the court's directive that the *Frye* hearing include mold sampling methodology within its scope is indicative of the large gap between what defendants argued in their submissions and the issues that the court decided were the proper subject of a *Frye* hearing. As discussed below, this resulted in a great deal of confusion at the hearing as to what precisely was at issue.

Plaintiffs met their burden of establishing the admissibility of evidence that the conditions in their apartment caused their illness. The conclusion by the motion court, adopted by the majority, that plaintiffs failed to establish at the hearing that exposure to building dampness and mold can cause illness is based on too restrictive an application of *Frye*. *Frye* hearings are to test [*13]the reliability of novel scientific evidence (*Parker v Mobil Oil Corp.*, 7 NY3d at 446. *Frye* itself held that "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." (*Frye*, 293 F at 1014). As Judge Kaye stated in her concurrence in *People v Wesley* (83 NY2d 417, 439 [1994]), *Frye* "emphasizes counting scientists' votes, rather than on verifying the soundness of a scientific conclusion."

Courts have warned against an over-restrictive use of *Frye*. For example, the Court of Appeals stated in *Parker* that

> "As with any type of expert evidence, we recognize the danger in allowing unreliable or speculative information (or junk science') to go before the jury with the weight of an impressively credentialed expert behind it. But, it is similarly inappropriate to set an insurmountable standard that would effectively deprive toxic tort plaintiffs of their day in court. It is necessary to find a balance between these two extremes."

(7 NY3d at 447). The Second Department, in reversing a preclusion order after a *Frye* hearing, observed that:

> "[t]he trial court, while purporting to credit the deductive reasoning of the plaintiff's experts, apparently believed that the *Frye* test could only be satisfied with medical texts, studies, or other literature which supported the plaintiff's theory of causation under circumstances virtually identical to those of the plaintiff. However, the *Frye* test is not that exacting."

(*Zito v Zabarsky*, 28 AD3d 42, 46 [2006]).

Here, the majority has "set an insurmountable standard." (*Parker*, 7 NY3d at 447). It essentially posits that in a case such as this, *Frye* requires that the medical literature conclusively establish that an allegedly offending substance not only have the potential to cause illness but that it *always* causes illness. Indeed, the motion court, in interpreting *Parker* to require at least a "significant association" between an allegedly harmful substance and illness, endorsed Dr. Phillips's position that a "strong association occurs all of the time." This is far too rigorous an application of *Frye*.

In this case, plaintiffs submitted at least 20 peer-reviewed publications describing an association or strong association between building dampness and mold and the type of irritative symptoms described by plaintiffs. Collectively, these studies establish that the "association" between building dampness and illness is one of causation, not one of coincidence. In his testimony, Dr. Phillips stated that an "association" is probative of nothing. In other words, he explained that if one observes a man in a black suit get struck by a car, and observes the same thing a few blocks later, it would not be logical to conclude that one should not wear a black suit while crossing the street. "Association," however, is a continuum, which spans from the coincidence described in the above scenario to unquestionable causation. The evidence submitted by plaintiffs here, while perhaps not establishing that building dampness always causes illness, is far closer to the causation end of the continuum than the coincidence end.

Moreover, it is not plaintiffs' contention that building dampness and mold *always* cause [*14]illness, and that is not required. Rather, plaintiffs claim, and the literature confirms, that more than an outlying segment of the scientific community has concluded that there is evidence that building dampness and mold have *the potential* to cause allergic and irritative reaction in sensitized people. Plaintiffs simply seek an opportunity to prove to a jury that the dampness and mold in their apartment caused their symptoms.

Indeed, Dr. Phillips's testimony concerning the causal relationship between building dampness and illness reveals that he considers the "association" described in the literature submitted by plaintiffs as being on the causation end of the continuum discussed above. Dr. Phillips stated that because science has only identified an "association" between dampness and illness, a doctor treating a patient complaining of mold-related illness must perform a complete evaluation of the patient and his environment to confirm his claim. In other words, he said that because science has not established that mold always causes illness, the doctor may not simply accept that the patient is sick from mold. In his practice, Dr. Phillips has treated "thousands" of patients complaining of respiratory problems associated with a damp building. He testified that when a patient presents with such a complaint:

> "[y]ou evaluate the patient, you try to see how ill they are, what the clinical manifestations are. You try to establish the presence or absence of mold sensitization. I give them instructions in terms of what they can do to control, for example, the moisture, the dehumidification.
>
> ***
>
> "Because excess moisture increases growth of mites and mold and bacteria and other things. So high amounts of moisture is an adverse environment in which that patient is going to live. You are going to try to help them in any way you can. You give them proper medicine and test them if in fact they are sensitive to mold, and in some cases do desensitization shots."

This is the precise approach Dr. Johanning took with plaintiffs. It recognizes that building dampness can cause illness but that the link between the two is not so consistent that a doctor can dispense with a detailed examination into whether it did in fact cause illness. In employing this approach, Dr. Phillips recognizes that the theory that there is a link between building dampness and illness is not the type of "theoretical speculation or...scientific hunch'" that *Frye* hearings are designed to weed out (*Zito v Zabarsky*, 28 AD3d at 46).

Finally, to the extent that this Court has in recent history precluded expert testimony under *Frye*, it has based such decisions upon a complete *absence* of literature or studies supporting the claim (*see e.g. Marso v Novak*, 42 AD3d 377 [2007], *supra*; *Lara v New York City Health & Hosps. Corp.*, 305 AD2d 106 [2003]; *Selig v Pfizer, Inc.*, 290 AD2d 319 [2002], *lv denied* 98 NY2d 603 [2002]; *Stanski v Ezersky*, 228 AD2d 311 [1996], *lv denied* 89 NY2d 805 [1996]). Here, a *plethora* of peer-reviewed articles supports plaintiffs' claim.

The motion court was correct in stating that *Parker v Mobil Oil Corp.* required it not only to consider the general question of whether the link between building dampness and illness is generally accepted, but also that a scientific foundation existed for plaintiffs' experts' conclusion that plaintiffs were sickened by the conditions in their apartment. However, *Parker*'s applicability here is limited to that general proposition. Indeed, there is no basis for the motion court's statement that "[t]here is a striking similarity between the testimony of plaintiffs' experts [*15]and the vague expert testimony rejected by the Court of Appeals in *Parker*."

In *Parker*, the plaintiff, a former gasoline station attendant, claimed to have developed acute myelogenous leukemia as a result of exposure to benzene contained in gasoline. The defendants sought to dismiss the case on the theory that the plaintiff could not establish a causal link between the exposure and his illness. The Court of Appeals held that a traditional *Frye* analysis was unnecessary because the plaintiff's scientific theory was not "novel." However, it further held that the trial court still had a gatekeeping role of ensuring the reliability of the proposed scientific evidence. In *Parker*, that required ensuring that the plaintiff's experts could demonstrate the threshold of exposure to benzene below which leukemia would not occur, as well as the exposure level to which the plaintiff was subjected. The Court rejected the plaintiff's experts' opinions because they failed to offer any scientific measure of the level of the plaintiff's exposure in other than the most general and conclusory terms.

In contrast to *Parker*, here plaintiffs are not claiming that they were harmed by the *toxic* effects of mold. Rather, they claim to have been sickened by those properties of mold and building dampness which have an irritative and allergic effect. Accordingly, as Dr. Johanning explained, ascertaining the specific levels of a particular mold in a building is not determinative of whether the mold caused irritative or allergic effects. This is because, he explained, as long as a person has become sensitized to the mold, he or she may react to a small amount of exposure. In any event, Dr. Johanning objectively determined that there was sufficient mold in the apartment for plaintiffs to have become sensitized. Specifically, he viewed photographs demonstrating the significant mold

growth in the apartment. More importantly, he relied on the Olmsted report showing levels of atypical organisms existing in the apartment as high as five times the levels in which they are normally encountered outdoors. This was in sharp contrast to the facts in *Parker*, where the record was devoid of any specific articulation of the plaintiff's exposure.

In any event, even if quantifying mold levels was critical to plaintiffs' case, *Parker* does not help defendants here.
The holding in *Parker* put rest to the notion that to establish an appropriate reliability foundation, plaintiffs in a toxic tort case must establish precisely quantified exposure levels or a dose-response relationship, provided, the Court wrote, that "whatever methods an expert uses to establish causation are generally accepted in the scientific community" (7 NY3d at 448). Here, Dr. Johanning's differential diagnosis satisfied that test. Differential diagnosis has been recently accepted by the Fourth Department as a generally accepted method for establishing specific causation in mold cases. That court found in *B.T.N. v Auburn Enlarged City School Dist.* (45 AD3d 1339 [2007]), a case involving atypical mold in a school building, that a differential diagnosis was an adequate basis for opining that the mold caused the plaintiffs' symptoms.

Here, defendants never argued in their initial motion papers that the differential diagnosis performed by Dr. Johanning was not a generally accepted methodology. Moreover, to the extent that the motion court can be read as holding that differential diagnosis is not a generally accepted methodology in mold cases, that was patently unfair. The order directing the *Frye* hearing cannot possibly be read to include within its scope the issue of whether differential diagnosis is generally accepted in such cases.

Dr. Johanning's differential diagnosis was scientifically valid and the motion court articulated no basis for concluding otherwise. A differential diagnosis has been described as "a patient-specific process of elimination that medical practitioners use to identify the most likely' [*16]cause of a set of signs and symptoms from a list of possible causes" (*Ruggiero v Warner-Lambert Co.*, 424 F3d 249, 254 [2d Cir 2005][internal quotation marks and citation omitted). Indeed, *Jazairi v Royal Oaks Apt. Assoc., L.P.* (217 Fed Appx 895 [11th Cir. 2007], *supra*), one of the cases upon which the motion court relied in rejecting Dr. Johanning's differential diagnosis approach, noted that "[t]he record reflects that differential diagnosis is widely accepted by the medical community" (*id.* at 898).

Here, Dr. Johanning specifically ruled in the damp conditions in the subject apartment to be the cause of plaintiffs' symptoms, based not only on the history related by plaintiffs, but also on specific immunological markers which demonstrated lengthy exposure by plaintiffs to specific

organisms related to irritants that were found to be inside the apartment in levels greater than outdoors. In addition, he ruled out all other causes, such as smoking, other allergens and irritants unrelated to mold or building dampness, or even other possible dampness-related conditions such as those related to Colin Fraser's vocation as a stamp broker.

In contrast, the 11th Circuit in *Jazairi* rejected the differential diagnosis (also performed by Dr. Johanning) because he:

> "apparently did not conclude that [the plaintiff] suffered symptoms due to exposure to any of the molds that were present in her apartment. To the extent that Dr. Johanning was prepared to testify that the mold in [the plaintiff's] apartment caused her conditions, Dr. Johanning's testimony would have been based *solely* on temporal proximity and anecdotal evidence." (Emphasis added)(*Jazairi*, 217 Fed Appx at 898).

In other words, the differential diagnosis in *Jazairi* was not one at all, because it was based on no objective medical data and because it ruled nothing in and nothing out. Here, that was far from the case. Indeed, the motion court's statement that "Dr. Johanning's opinion was based solely on temporal proximity to mold and anecdotal evidence" is plainly contradicted by his testimony.

Moreover, nearly all of the factors which the motion court identified as demonstrating that Dr. Johanning's opinion had no reliable scientific foundation were erroneous. First, the court stated that Dr. Johanning testified "without underlying proof of causation or strong association." As discussed above, however, plaintiffs established that it is generally accepted that building mold and dampness can generally cause illness. The court also stated that Dr. Johanning testified "without proof of mold allergies." However, Dr. Johanning did base his conclusion on plaintiffs' physiological reaction to mold and other dampness-related organisms. While this may not have been the "traditional" IgE-mediated allergy, which the court was focused on, the fact that diagnostic tests revealed an IgG-mediated response to dampness-related irritants was probative of a causal link between the conditions in the apartment and plaintiffs' symptoms. Moreover, while defendants tangentially questioned the reliability of IgG readings in their initial motion and at the hearing, they did not ask for a ruling that using IgG testing for diagnostic purposes in mold cases is not generally accepted, nor can the order directing the hearing be interpreted as requiring plaintiffs to establish the reliability of such testing.

Second, the court treated as fatal plaintiffs' failure to measure for moisture and non-mold by-products of moisture identified by Dr. Johanning as contributing to illness, such as endotoxins, mycotoxins, or Beta-D-glucans. However, such measurements were not critical to Dr. Johanning's

differential diagnosis because, as he testified, the level of organisms sufficient to sensitize plaintiffs could vary significantly depending on the individual. In any event, Dr. [*17]Johanning did have objective evidence of significant mold growth in the apartment from the photographs he viewed and the Olmsted report. Moreover, the fact that there were significant water intrusions into the subject apartment was apparently never in dispute.

As for the court's statement that Dr. Johanning failed to rule in cat and dust allergies as a possible cause of Pamela Fraser's symptoms, there is no evidence in the record that she had cat allergies. Dr. Johanning did note that she had a reaction to dust mites. However, he stated that it was "slight," which hardly suggests that he would have ever ruled it in as the most likely cause of her symptoms. Moreover, there is no requirement that the proponent of expert scientific testimony prove the ultimate theory of the case during a *Frye* hearing. Rather, he or she must only establish the basic reliability of the methodology utilized to reach that conclusion. As we recently held, any further challenges to an expert's methodology and/or conclusions above and beyond its basic reliability are more "properly the subject of cross-examination at trial, as they go to credibility and to the weight to be given to the evidence" (*Nonnon v City of New York*, 32 AD3d 91, 108 [2006], *affd* 9 NY3d 825 [2007]).

Here, Dr. Johanning's use of the methodology of differential diagnosis was reliable as it was based on the confirmed presence of dampness and mold in plaintiffs' apartment and the presence in plaintiffs' systems of antibodies to organisms typically associated with mold and dampness. Moreover, plaintiffs exhibited symptoms consistent with dampness-related illness, and related no other source of exposure to dampness-related antigens. Once Dr. Johanning established this baseline of reliability, defendants were relegated to challenging his conclusions before the trier of fact.

Finally, plaintiffs not only met their burden at the hearing in this case, they did so in the face of substantial confusion fostered by the motion court. The order directing the hearing was vague and overbroad in terms of what plaintiffs were required to establish. Moreover, the order required them to prove the general acceptance of mold sampling methodology when not even defendants had asked for such relief. Because of the court's ambiguous order, a significant portion of the hearing was devoted to argument between the parties and the court over precisely what was at issue. For example, the parties and the court differed over whether plaintiffs were required to establish the general acceptance of a causal link between damp buildings and sickness or, more specifically, mold in general. They argued about whether plaintiffs were required to identify a specific "disease"

caused by the conditions in their apartment, or merely the presence of physical symptoms. They also debated whether plaintiffs were required to establish the general acceptance of differential diagnosis as a methodology for establishing specific causation.

As a result, the scope of the hearing was continuously defined and re-defined over its course. This left plaintiffs at sea, without the ability to divine the path the court required for them to satisfy their burden. In spite of this confusion, plaintiffs established the reliability of their experts' opinions. Nevertheless, the motion court usurped the function of the jury here and became the finder of fact, not as to whether or not plaintiffs' theories and evidence satisfied the *Frye* and *Parker* tests, but of the ultimate question as to whether defendants were responsible for [*18]plaintiffs' injuries. Accordingly, I would reverse the orders appealed and reinstate plaintiffs' personal injury claims.

THIS CONSTITUTES THE DECISION AND ORDER
OF THE SUPREME COURT, APPELLATE DIVISION, FIRST DEPARTMENT.

ENTERED: DECEMBER 30, 2008

CLERK

Footnotes

Footnote 1: For example, a review of the relevant scientific literature published by the Institute of Medicine of the National Academies, *Damp Indoor Spaces and Health* (National Academies Press 2004), concluded that there was "sufficient evidence of an association" between upper respiratory (nasal and throat) tract symptoms, on the one hand, and damp indoor environments and the presence of mold, on the other hand, but found that it could *not* be said that there was "sufficient evidence of a causal relationship" between any set of health outcomes and such conditions (*id.* at 253-254 [Tables 5-12 and 5-13]).

Footnote 2: Obviously, there is no rule that a jury may hear only theories that are either "conclusively establish[ed]" by the scientific literature or unanimously supported by the scientific authorities. Further, we do not suggest, nor did the motion court suggest, that a substance cannot be considered the cause of a health outcome unless the substance "always" causes that health outcome.

Footnote 3: Contrary to Dr. Johanning's assertion, neither of the two studies referenced in the first excerpt from his opposition affidavit quoted by the dissent reached the conclusion that a causal relationship has been demonstrated between indoor dampness and mold and the upper respiratory symptoms of which plaintiffs complain. The first study referenced in the excerpt from Dr.

Johanning's affidavit (Cox-Ganser, et al., *Respiratory Morbidity in Office Workers in a Water-Damaged Building*, 113 Environmental Health Perspectives 485 [2005]) concluded only that "[o]ccupancy of the water-damaged building was *associated* with onset and exacerbation of respiratory conditions" (*id.* at 485 [emphasis added]). The authors of *Respiratory Morbidity* acknowledged that the "major limitation" of the study, which was based on voluntary responses to a questionnaire, was "the possible influence of participation bias" (*id.* at 490). The other study referenced in the quoted excerpt from Dr. Johanning's affidavit (Jaakkola, et al., *Home Dampness and Molds, Parental Atopy, and Asthma in Childhood: A Six-Year Population-Based Cohort Study*, 113 Environmental Health Perspectives 357 [2005]) focused on risk factors for the development of childhood asthma, and is therefore of little relevance to this case, which does not involve a child suffering from asthma.

Footnote 4: We have no argument with the dissent's statement that " [a]ssociation' . . . is a continuum . . . span[ning] from . . . coincidence . . . to . . . causation." This observation is of little help to plaintiffs, however, because the dissent points to nothing in the record, other than Dr. Johanning's unsupported assertions, that justifies the conclusion that the observed association between the conditions and ailments in question is strong enough to constitute evidence of causation.

Footnote 5: Although the *Frye* inquiry and the foundational inquiry are distinct, they may proceed simultaneously (*see People v Wesley*, 83 NY2d at 436 n 2 [Kaye, Ch. J., concurring]).

Footnote 1: "IgE" and "IgG" refer to immunoglobulin types which reflect the level of antibodies developed by the body in response to exposure to antigens such as allergens and foreign organisms. IgG is a delayed marker, which indicates that the antibody to the inducing matter was developed over an elongated period of time. This contrasts with IgE, which is indicative of an immediate allergic reaction to a foreign body.

Footnote 1: That is, the papers discussed by Dr. Gots in his affidavit.

Return to Decision List