Granirer v Bakery, Inc.
2014 NY Slip Op 31141(U)
April 28, 2014
Supreme Court, New York County
Docket Number: 109334/2009
Judge: Saliann Scarpulla

[* 1]

SUPREME COURT OF THE STATE OF NEW YORK — NEW YORK COUNTY

PRESENT: Hon. Saliann Scarpulla PART 39
Justice

DAN GRANIRER and KEIKO HONDA, individually, and on behalf of their infant daughter MAYA HONDA-GRANIRER,

Plaintiffs,

- v -

THE BAKERY, INC.,

Defendant.

INDEX NO. 109334/2009
MOTION DATE
MOTION SEQ. NO. 002
MOTION CAL. NO.

The following papers, numbered 1 to ____ were read on this motion to/for ____

	PAPERS NUMBERED
Notice of Motion — Affidavits – Exhibits	
Answering Affidavits — Exhibits	
Replying Affidavits	

Cross-Motion: ☐ Yes ☒ No

Defendant's motion pursuant to CPLR 3212 for summary judgment against plaintiffs and dismissing the Complaint is decided in accordance with the attached Decision/Order.

FILED
MAY 01 2014
COUNTY CLERK'S OFFICE
NEW YORK

Dated: 4/28/14

Hon. Saliann Scarpulla, J.S.C.

MOTION/CASE IS RESPECTFULLY REFERRED TO JUSTICE FOR THE FOLLOWING REASON(S):

Check one: ☐ FINAL DISPOSITION ☒ NON-FINAL DISPOSITION

Check if appropriate: ☐ DO NOT POST ☐ REFERENCE

☐ SUBMIT ORDER/JUDG. ☐ SETTLE ORDER/JUDG.

SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK : IA PART 39

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DAN GRANIRER and KEIKO HONDA, individually, and on behalf of their infant daughter MAYA HONDA-GRANIRER,

Plaintiffs,

- against -

The Bakery, Inc.,

Defendant.

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THE BAKERY, INC.

Third-Party Plaintiff,

- against -

ARCADE CONTRACTING, INC. f/k/a ARCADE CONTRACTING AND RESTORATION, SUPER ROOFER EXTERIOR MAINTENANCE CORP. a/k/a SUPER ROOFER and FORT-CICA/ROOFING & GENERAL CONTRACTORS INC. a/k/a FORT-CICA,

Third-Party Defendants.

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THE BAKERY, INC.

Second Third-Party Plaintiff,

- against -

AURA GENERAL CONSTRUCTION CORP., and AURA HOME IMPROVEMENT, INC.,

Second Third-Party Defendants.

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SALIANN SCARPULLA, J.

DECISION/ORDER
Index No. 109334/09
Motion Seq. No. 002

Index No. 590528/11

Index No. 590646/11

I. Background

Plaintiffs Keiko Honda and Dan Granirer commenced this action individually, and on behalf of their infant daughter, Maya Honda-Granirer, as a result of personal injuries allegedly sustained from toxic mold exposure at the defendant cooperative corporation, The Bakery, Inc., located at 521 West 47 Street, New York, New York ("The Bakery"), where plaintiffs resided in apartment 4B. According to plaintiffs, heavy rains caused water intrusion into plaintiffs' apartment in October 2005. In February 2006, the water intrusion recurred, with the development of visible mold.

Repair work began in February 2006 and continued through March 2006. In May 2006, Maya, then 13 months old, became febrile and lethargic, with severe pain in her right wrist. Aff. of Daniel S. Moretti ("Moretti") in Supp., Ex. 16, Tr. 72:4-6; Aff. of Dr. Irene Grant ("Dr. Grant") in Opp., p. 3. On May 23, she was admitted to St. Lukes-Roosevelt Hospital, where she was diagnosed with pneumococcal meningitis and osteomyelitis. Moretti Aff. in Supp., Ex. 18. She was treated and eventually discharged on June 14, 2006. *Id.*, Ex. 19. Maya suffered permanent hearing loss in one ear. Aff. of Dr. Douglas Kerr ("Dr. Kerr") in Opp., p. 4. Plaintiff Keiko Honda, Maya's mother, resided with Maya while she was hospitalized, and after Maya was discharged, neither Maya nor Keiko returned to their apartment at The Bakery, other than Keiko returning periodically to retrieve clothing and personal items.

On May 30, 2006, Precision Consulting Inc. ("Precision Consulting") took swab samples from plaintiffs' apartment. Moretti Aff. in Supp., Ex. 22. Microscopic examination

of the samples identified: "Light" fungal structures of Hyphae, Basidiospores, Cladosporium, and Aspergillus/Penicillium; "Moderate" fungal structures of Ulocladium, and Hyphae; and both "Moderate" and "Heavy" fungal structures of Stachybotrys chartarum. *Id.*

On June 23 and 24, 2006, Environmental Consulting & Management Services, Inc. ("ECMS") inspected plaintiffs' residence and took swab and air samples, after which it issued an "Initial Microbial Sampling Report," dated July 5, 2006 (the "ECMS Report"). Moretti Aff. in Supp., Ex. 23. ECMS's interior swab sample identified "High" levels of Memnoniella and Stachybotrys, and "Medium" levels of Aspergillus/Penicillium and Pithomyces/Ulocladium. *Id.* at 3. The ECMS Report identified airborne mold concentrations in plaintiffs' living room, office, nursery, and bedroom, including "High" levels of the molds Stachybotrys and Memnoniella, and "Medium" levels of the molds Aspergillus/Penicillium and Pithomyces/Ulocladium. *Id.* at 2-3.[1]

On June 30, 2006, JLC Environmental Consultants, Inc. ("JLC") inspected plaintiffs' residence, taking swab and air samples, and issued a report, dated July 6, 2006 (the "JLC Report"). The JLC Report stated that the apartment "[w]indows had been open, but were closed at the onset of inspection." Moretti Aff. in Supp., Ex. 24, § 2.0. The JLC Report

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[1] Specifically, the Aspergillus/Penicillium concentration per unit area in the living room was 126 (total airborne concentration of 1,260 spores/m^3). The concentration per unit area in the nursery was 798 for Aspergillus/Penicillium and 378 for Stachybotrys (total airborne concentration of 3,150 spores/m^3). The office contained concentrations per unit area of 798 for Aspergillus/Penicillium and 840 for Stachybotrys (total airborne concentration of 3,480 spores/m^3). The second floor bedroom contained an Aspergillus/Penicillium concentration per unit area of 126 (total airborne concentration of 2,180 spores/m^3). The apartment exterior contained an Aspergillus/Penicillium concentration per unit area of 252 (total airborne concentration of 3,270 spores/m^3).

identified water damage with visible mold growth. A direct microscopic examination of fungal spores from swab samples identified "High" levels of Stachybotrys, "Medium" levels of Hyphal Fragment, and "Low" levels of Aspergillus/Penicillium, Cladosporium, and Pithomyces/Ulocladium. *Id.* at 3.0. The air samples detected various spores, including Aspergillus/Penicillium in the kitchen/dining room and hallway of 84 and 210 spores/m^3, respectively, as compared to 294 spores/m^3 from the sample taken outside. Both the ECMS and JLC Reports recommended containment and removal of the contaminated walls and ceilings, cleaning, vacuuming, and air filtration.

In November 2006, Keiko began to experience persistent muscle and joint aches. On December 5, 2006, Keiko returned to the apartment for approximately two hours, and the next day experienced flu-like symptoms, including vertigo and vomiting. These symptoms resolved the next day, but on December 11, 2006, Keiko experienced right arm sensory disturbance, which spread to her left arm and trunk, followed by paraplegia and bladder and bowel incontinence. She was admitted to the hospital on December 11 and was diagnosed with Transverse Myelitis ("TM"), a neurological disorder characterized by inflammation of the spinal cord. Keiko is now permanently paralyzed, and she suffers from various complications from her illness and paralysis.

On June 21, 2007, Michael McGuinness ("McGuinness"), a certified industrial hygienist and principal of RK Occupational & Environmental Analysis Inc. ("RK"), inspected plaintiffs' residence, taking air and swab samples, and issued a report, dated October 4, 2007 (the "RK Report"). Moretti Aff. in Supp., Ex. 25, ¶ 5. The air samples

detected various fungal structures, including Aspergillus/Penicillium as the dominant species at: 3,070 fungal structures/m^3 in the living room (with a total load of 5,270 fungal structures/m^3); 2,930 fungal structures/m^3 in the first floor bedroom (with a total load of 4,916 fungal structures/m^3); 1,010 fungal structures/m^3 in the first floor den (with a total load of 2,463 fungal structures/m^3); 2,670 fungal structures/m^3 in the second floor bedroom (with a total load of 7,846 fungal structures/m^3); and 4,020 fungal structures/m^3 in the second floor study (with a total load of 11,033 fungal structures/m^3). *Id.*, ¶ 3.1 and Table 1. The two outdoor air samples detected Aspergillus/Penicillium at 52 fungal structures/m^3 with a total load of 3,725 fungal structures/m^3, and 693 fungal structures/m^3 with a total load of 5,753 fungal structures/m^3. The dominant outdoor fungal structures were Cladosporium and Basidiomycetes *Id.* at Table 1.

RK also collected culturable fungi samples, which were incubated for two weeks and then examined, detecting various fungal structures, including dominant microbial colony forming units of Aspergillus and Penicillium. *Id.*, ¶ 3.2 and Table 2. Aspergillus and Penicillium were not detected in the outdoor culturable air samples. RK's surface wipe samples indicated the presence of Stachybotrys chartarum, Fusarium, Aspergillus niger, Aspergillus fumigatus, Trichoderma, Aureobasidium, Ulocladium, and Penicillium. *Id.*, ¶ 3.3. In his affidavit, McGuinness stated that, in the mold testing performed between July 2006 and December 2009, the following mold species, among others, were detected: Aspergillus ustus, Aspergillus versicolor, Eurotium, Stachybotrys chartarum, Trichoderma, and Memnoniella. McGuinness Aff. in Opp., pp. 3, 12. The RK Report concluded that the

mold sampling performed in plaintiffs' residence "indicate[d] a degraded indoor environment with widespread fungal contamination in the air and on surfaces in every room . . . tested in the home." *Id.*

In July 2006, plaintiffs commenced an action against, among others, The Bakery (Index No. 109915/2006), asserting claims for negligence, breach of the warranty of habitability, breach of contract, and injunctive relief. The three-count amended complaint in this action asserts one cause of action for negligence on behalf of Keiko, one cause of action for loss of consortium on behalf of Dan Granirer, and one cause of action for negligence on behalf of Maya.[2]

II. The Bakery's Motion and the Parties' Evidence

This summary judgment motion by The Bakery challenges plaintiffs' theory of causation, raised in the April 14, 2011 report of plaintiffs' expert, Dr. Kerr, and submitted by plaintiffs pursuant to their notice of expert exchange under CPLR 3101 (d). Moretti Aff. in Supp., Ex. 31. Dr. Kerr is a neurologist, neuroscientist, and professor, and his report is based upon physical examinations and laboratory studies of Keiko and Maya, and the reports of the mold experts. In his report, Dr. Kerr diagnosed Keiko's illness as transverse myelitis. He concluded that "Stachybotrys and Aspergillus are known to suppress the immune system

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[2] In addition to the above-captioned action and third-party actions, the Court's independent review of the County Clerk's records revealed an additional action commenced by Dan Granirer and Keiko Honda against various insurance companies under Index No. 114206/2006. By order dated April 21, 2009, that action was consolidated with plaintiffs' original action (Index No. 109915/2006), which is marked "active" in the County Clerk's records.

and the Aspergillus is known to directly cause TM by invasion into the CNS [central nervous system]," and that Keiko and Maya's illnesses "were caused by the mold exposure" in their residence. *Id.*, ¶ 7.

The Bakery now moves for summary judgment dismissing the amended complaint based upon lack of medical causation. Alternatively, The Bakery moves for an order precluding plaintiffs' expert witnesses from providing opinion evidence that: (1) exposure to mold causes the types of illnesses suffered by Keiko and Maya, because any such opinion is not generally accepted as reliable by the scientific community; and (2) Keiko and Maya's exposure to mold in their apartment caused their specific injuries, as any such opinion cannot be proven with a reasonable degree of medical certainty. If the preclusion motion is denied, The Bakery requests a hearing, pursuant to *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), to determine whether the causation theory proffered by plaintiffs' experts has gained general acceptance in the scientific community.

A. The Bakery's Medical and Scientific Literature

The Bakery submits the following 8 medical and scientific articles and position papers.

1. Chitra Krishnan et al., *Transverse Myelitis: Pathogenesis, Diagnosis and Treatment*, Frontiers in Bioscience 9, 1483-1499 (May 1, 2004).

This article, co-authored by Dr. Kerr, summarizes "recent classification and diagnostic schemes, which provide a framework for the acute management of patients with TM," and it reviews "current concepts on the natural history, immunopathogenesis and treatment

strategies for patients with TM." Moretti Aff. in Supp., Ex. 32, p. 1483. The article states that TM is "on a continuum of neuroimmunologic disorders," and that there is "an acquired alteration of the innate or acquired immune system, resulting in dysfunction and/or cellular injury to cells within the nervous system." *Id.* at 1484. According to this article, TM "may be post-infectious . . . , suggesting that the infectious agent triggers breakdown of immune tolerance of self antigens." *Id.* It states that, "in TM patients, it is likely that there is abnormal activation of the immune system resulting in inflammation and injury within the spinal cord." *Id.* at 1491. The article states that "very little is known definitively about the inciting cause of inflammation in the CNS of patients with TM," that "nothing is currently understood about the mechanisms of tissue injury in this inflammatory disease" (*id.*), and that, "[al]though the causes of TM remain unknown, recent advances have suggested specific cytokine derangements that likely contribute to sustained disability." *Id.* at 1494.

2. Douglas A. Kerr, *Immunopathogenesis of acute transverse myelitis* (2002).

In this article, Dr. Kerr examines "recent evidence that shed light on the immunopathogenesis of ATM [acute transverse myelitis] and, where applicable, related neuroinflammatory disorders." Moretti Aff. in Supp., Ex. 33, p. 339. Dr. Kerr states that "[i]t is unclear what are the triggers and effector mechanisms resulting in neural injury," but that "studies point to a variety of humoral and cellular immune derangements that potentially result in neuronal injury and demyelination." *Id.*

3. Adam I. Kaplin, et al., *Diagnosis and Management of Acute Myelopathies*, The Neurologist, vol. 11, no. 1 (January 2005).

This article, co-authored by Dr. Kerr, "summarize[s] recent classification and diagnostic schemes, which provide a framework for the diagnosis and management of patients with acute myelopathy," and it "review[s] the state of current knowledge about the epidemiology, natural history, immunopathogenesis, and treatment strategies for patients with TM." Moretti Aff. in Supp., Ex. 34, p. 2. Like the 2004 article co-authored by Dr. Kerr, this article concludes that "[al]though the causes of TM remain unknown, recent advances have suggested specific cytokine derangements that likely contribute to sustained disability due to injury of motor, sensory, or autonomic neurons within the spinal cord." *Id.* at 15.

4. *New York State Toxic Mold Task Force Final Report to the Governor and Legislature*, New York State Department of Health and Department of State (December 2010).

This report summarizes the findings of a task force charged with "assessing and measuring, based on scientific evidence, the adverse environmental and health effects of mold exposure . . . ." Moretti Aff. in Supp., Ex. 41, p. 10. The report acknowledges that "mold has been recognized nationally and at the state and local level as a potential public health problem," but states that "[e]vidence for associations between non-respiratory effects of mold exposures in buildings is much more limited and generally does not allow clear conclusions to be drawn one way or the other." *Id.* at 11, 29, 38; *see also id.* at 28 ("[s]cientific evidence for associations between indoor exposures to mold toxins [also called mycotoxins] and adverse health effects in building occupants is inconclusive"). This report

also acknowledges that *in vitro* studies and studies using laboratory animals "indicate[s] that microbial toxins can cause adverse effects to many body organs or systems, including . . . immune [and] neurological," but explains that "existing animal study results are largely based on high-dose, single exposure or short-term repeated experiments using exposure routes that may not be relevant to indoor air exposures." *Id.* at 28. The report further acknowledges that "more serious fungal infections mostly occur in people with conditions that substantially suppress immune function," and that "[s]usceptibility to these opportunistic infections depends more on the host's immune status than on the level of mold spore exposure, so that severely immune compromised patients are at risk from background exposure levels common in outdoor air." *Id.* at 31. The report recognizes hospital construction as a risk factor for opportunistic fungal infection, but otherwise concludes that "the published scientific literature did not find evidence of fungal infectious disease risk specifically attributable to mold growth in damp buildings." *Id.*

5. *Damp Indoor Spaces and Health*, Institute of Medicine (2004).

The Bakery submits a portion of this article, which found evidence of a causal relationship between the presence of mold and respiratory symptoms, but insufficient evidence to determine whether an association exists between mold and immune diseases. Moretti Aff. in Supp., Ex. 42, pp. 253-54. However, these conclusions were "not applicable to immunocompromised persons, who are at increased risk for fungal colonization or opportunistic infections." *Id.* at 254.

6. Bryan D. Hardin et al., *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, American College of Occupational and Environmental Medicine (October 27, 2002).

In this statement, the American College of Occupational and Environmental Medicine ("ACOEM") found that, "[e]xcept for persons with severely impaired immune systems, indoor mold is not a source of fungal infections." Moretti Aff. in Supp., Ex. 43, p. 476. The ACOEM reiterated this conclusion in a position statement dated February 2011. *Id.*, Ex. 44, at 5.

7. Robert K. Bush et al., *The medical effects of mold exposure*, Journal of Allergy and Clinical Immunology, vol. 117, no. 2 (February 2006).

This position paper states that, "[g]enerally, host factors, rather than environmental exposure, are the critical factor in the development of opportunistic mold infection in immunocompromised individuals because exposure to potential fungal opportunistic pathogens (e.g., *Aspergillus* species) is ubiquitous in normal outdoor and indoor environments." Moretti Aff. in Supp., Ex. 45, p. 328. It further states that: the published literature concerning evidence of "significant altered immunity expressed as either immunodeficiency or auto-immunity . . . is of particularly poor quality and should not be relied on as scientifically valid"; that "even the most intense form of airborne mold exposure is not a recognized cause of secondary immunodeficiency in human subjects"; and that "[e]xposure to molds and their products does not induce a state of immune dysregulation (e.g., immunodeficiency or autoimmunity)." *Id.* at 330.

8. Stephen Vesper et al., *Development of an Environmental Relative Moldiness Index for US Home*, American College of Occupational and Environmental Medicine, vol. 49, no. 8 (August 2007).

In this study, dust samples were taken from 1,096 homes, which found Stachybotrys chartarum in 35 percent of the homes and species of Aspergillus in 12 to 90 percent of the homes. *Id.*, Ex. 46, p. 831.

B. The Bakery's Expert Affidavits and Reports

1. Dr. Gerr

Dr. Frederic E. Gerr, M.D. ("Dr. Gerr") is a practicing physician and professor with expertise in occupational and environmental health, epidemiology, and internal medicine. Gerr Aff. in Supp., ¶ ¶ 1-3. Based upon the medical literature submitted by The Bakery, Dr. Gerr concludes that there is no accepted medical basis for Dr. Kerr's conclusion that Keiko's TM was caused by mold exposure. *Id.*, ¶ 16. Dr. Gerr claims that, in addition to the lack of authoritative medical evidence to link mold in otherwise healthy people to TM, there is no support for the conclusion that mold exposure suppresses the immune system. *Id.* Dr. Gerr states that "the only generally accepted medical support for Dr. Kerr's poposition that 'Aspergillus [is] known to directly cause TM by invasion into the CNS [central nervous system]' exists in the specific, narrow context of central nervous system complications of the disease *Aspergillosis*, a systemic infection caused by the Aspergillus fungus." *Id.*, ¶ 13. Dr. Gerr claims that there is no medical evidence that Keiko had aspergillosis or any known immunosuppression. *Id.*

Dr. Gerr maintains that Keiko's medical records, including an examination of cerebral spinal fluid obtained from a spinal tap, demonstrate that her neither her brain nor her spinal cord (the two components of the CNS) were invaded by Aspergillus or any other mold. *Id.*, ¶¶ 14-16. According to Dr. Gerr, if the molds found in plaintiffs' apartment "were capable of suppressing the immune systems of healthy people, a large number of otherwise healthy people in the United States would have suppressed immune systems, which is not the case." *Id.*, ¶ 8.

2. Dr. Phillips

Dr. S. Michael Phillips, M.D. ("Dr. Phillips") is a practicing physician and professor with expertise in allergy, immunology, internal medicine, and epidemiology. Phillips Aff. in Supp., ¶¶ 1-4. Based upon the same medical literature, Dr. Phillips concludes that "[t]here is no authoritative literature to suggest that inhalation of mold, including Stachybotrys and Aspergillus, in the residential environment suppresses the immune system or causes bacterial meningitis or TM, as described by Dr. Kerr." *Id.*, ¶ 20. Dr. Phillips also states that there is no evidence that Keiko or Maya suffered from immunodeficiency or had infections caused by Aspergillus, and that there is "no general acceptance in the medical community for the statement that mold can cause systemic infections in otherwise healthy people like Keiko and Maya." *Id.*, ¶ 22.

According to Dr. Phillips, "[t]here is no general acceptance in the medical community for the proposition that exposure to mold can cause bacterial meningitis, NMO, or TM in otherwise healthy individuals such as Keiko and Maya. *Id.*, ¶ 20. He also states that "[t]here

is no authoritative literature to suggest that inhalation of mold, including Stachybotrys and Aspergillus, in the residential environment suppresses the immune system or causes bacterial meningitis or TM, as described by Dr. Kerr." *Id.*

Dr. Phillips states that, if Keiko and Maya were allergic to the molds at issue, at most, they could have suffered allergy-like symptoms as a result of mold exposure, but that there is no evidence of mold allergies. *Id.*, ¶ 20. Dr. Phillips maintains that "[e]xposure to molds as described here does not suppress the immune system," and that Keiko and Maya were never "diagnosed with any disease that would commonly be associated with mold exposure." *Id.*, ¶ 24.

3. Dr. Snyder

Dr. David H. Snyder, M.D. ("Dr. Snyder") is a practicing physician and professor with expertise in neurology and demyelinating diseases such as TM. Snyder Aff. in Supp., ¶¶ 1-2. Dr. Snyder reviewed Keiko's medical records, which indicate that no final diagnosis was agreed upon by her treating physicians, but included the diagnoses: myelitis, neuromyelitis optica, transverse myelitis, and a monophasic type of transverse myelitis of idiopathic etiology. *Id.*, ¶ 4; Moretti Aff. in Supp., Ex. 21. According to Dr. Snyder, "[t]he term 'idiopathic' by definition means from an unknown cause," which he claims is the best description for Keiko's illness because the extensive hospital testing failed to demonstrate an etiological cause. Dr. Snyder Aff. in Supp., ¶ 13.

Dr. Snyder states that, at the time Keiko was hospitalized, "no infectious process involving the spinal cord occurred," and that her "clinical course and type of treatments she

received are additional evidence that neither aspergillosis or any specific infection involving the spinal cord was present." *Id.* Dr. Snyder claims that Keiko's laboratory studies and serology test results were within normal limits, and that her cerebral spinal fluid did not evidence inflammation or infection. *Id.*, ¶ 10. Her blood cultures were also negative for evidence of infection. *Id.* Dr. Snyder claims that he "performed a comprehensive medical literature search regarding the medical issues addressed in this case." *Id.*, ¶ 5. Although he fails to identify any medical literature that he reviewed, he states that he could not find any articles or case reports to support the conclusion that "mold causes TM in otherwise healthy people like [Keiko] Honda." *Id.*, ¶ 14.

4. Dr. Shapiro

Dr. Eugene Shapiro, M.D. ("Dr. Shapiro") is a professor of pediatrics, epidemiology, and investigative medicine, who has also been involved in the diagnosis, care, and treatment of children who suffered from meningitis. Shapiro Aff. in Supp., ¶¶ 2-3. Dr. Shapiro also did not identify any medical literature that he reviewed, but claims that there is no support in the scientific literature for Dr. Kerr's contention that mold exposure causes or contributes to the development of bacterial meningitis or immune system suppression. *Id.*, ¶ 5.

According to Dr. Shapiro, in 2000, the Prevnar vaccine, which contains 7 strains of pneumococcus bacterium, was introduced and routinely administered to children to prevent serious pneumococcal infections. *Id.* Dr. Shapiro maintains that the Prevnar vaccine did not contain strain 19A of pneumococcus, which became one of the most common causes of bacterial meningitis in children in the United States. *Id.* Dr. Shapiro concludes that,

therefore, Maya's meningitis was caused by strain 19A of the pneumococcus bacterium. *Id.* Dr. Shapiro claims that Maya had a normally functioning immune system that was neither suppressed nor compromised, and that her blood tests did not reveal any fungal infections or other objective findings of mold exposure. Dr. Shapiro concludes that mold played no role in Maya's illnesses. *Id.*, ¶ 9.

5. Dr. Nass

The Bakery submits the examination report of Dr. Ruth Nass, M.D. ("Dr. Nass"), of New York University's Child Study Center, dated January 29, 2011. Moretti Aff. in Supp., Ex. 35. Dr. Nass assessed Maya and, as of the date of the report, concluded that Maya is functioning normally despite having had pneumococcal meningitis. Dr. Nass stated that she knows of no medical literature associating mold exposure with pneumococcal sepsis, meningitis, or osteomyelitis, and that Maya's illness was not related to mold exposure.

6. Dr. Block

The Bakery submits the neurologic examination report of Dr. Jerome M. Block, M.D., P.C. ("Dr. Block"), dated December 16, 2010. Moretti Aff. in Supp., Ex. 36. Dr. Block examined Keiko and reviewed her medical history, concluding that she suffered from multiple sclerosis or neuromyeltitis optica. *Id.* at 16. Dr. Block states that he "can understand the temptation to ascribe [Keiko's] problem to mold-induced neuro immunodeficiency," but that he has "not found documentation of such in [his] studies or personal experience." *Id.*

C. Plaintiffs' Medical & Scientific Literature

Plaintiffs submit 45 exhibits of articles and position papers concerning the adverse health effects of molds on humans and animals. Pertinent, representative portions of these exhibits are summarized below.

1. Luke Curtis et al., *Adverse Health Effects of Indoor Moulds*, Journal of Australasian College of Nutritional & Environmental Medicine, vol. 23, no. 1, 3-8 (April 2004).

This article was presented by the American Academy of Environmental Medicine ("AAEM") "to describe the current knowledge of adverse health effects of indoor mould." Plaintiffs' Ex. 3, p. 1. The AAEM states that "[i]ndoor airborne mould exposure frequently causes adverse human health effects with injury to and dysfunction of multiple organs and system including: 1) respiratory, 2) nervous, 3) immune, 4) haematological systems and 5) the skin. Indoor mould is also a common cause of life-threatening systemic infections in immuno-compromosed patients." *Id.* According to this article, "[t]here are no official standards, at this time, for indoor airborne fungus concentrations. However, indoor fungal levels above a range of 150 to 1,000 colony-forming units per cubic meter of air (cfu/m^3) are considered to be sufficient to cause human health problems." *Id.* The AAEM identifies several toxic chemicals produced by fungi, called "mycotoxins," including carcinogens, immunosuppressives, and protein inhibitors produced by Aspergillus, Penicillium, and Stachybotrys. *Id.* at 2.

Under the heading "Immunological Changes," the AAEM explains that "[f]ungal exposure can alter immunological parameters," causing "higher serum levels of IgG, IgA and

IgM antibodies to common fungi, trichothecenes and satratoxins." *Id.* at 3. The article states that "[h]uman cell line studies have also found that many mycotoxins can suppress T cell, B cell and NK activity at serum concentrations similar to those found in indoor mould exposed patients. Thus, airborne exposure to mycotoxin is seen to cause harmful effects to the immune system." *Id.*

2. *Molds and Mycotoxins (Toxic Molds) in Human Health*, American Academy of Environmental Medicine (March 1, 2008).

The AAEM board of directors approved this article, which states that "[i]t is commonly recognized that a large body of medical literature and extensive clinical experience indicates that sufficiently high exposures to *indoor airborne* mold can lead to disease in otherwise healthy individuals." Plaintiffs' Ex. 6 (emphasis in original). The AAEM states that "[e]xposure to significant levels of indoor mold can cause acute or chronic *dysfunction or injury to all organ systems* including the respiratory, neurological, cardiovascular, genitourinary, gastrointestinal, musculoskeletal, immune (through both immediate and non-IgE mechanisms) and hematological systems." *Id.*

3. Kay H. Kilburn, *Indoor Mold Exposure Associated with Neurobehavioral and Pulmonary Impairment: A Preliminary Report*, Archives of Environmental Health, vol. 58, no. 7, 390-398 (July 2003).

This article summarizes a study of 65 adult patients who were exposed to molds in their homes. The study concludes that "[i]nhaled mycotoxins, liberated from indoor mold growth, caused brain impairment and neurological symptoms." Plaintiffs' Ex. 7, p. 396. It states that "[t]richothecenes are epoxides that covalently adduct deoxyribonucleic acid,

ribonucleic acid, protein, and microtubules of nerve axons, providing mechanisms for lung, brain, and immune system toxicity." *Id.*

4. Enusha Karunasena et al., *Building-Associated Neurological Damage Modeled in Human Cells: A Mechanism of Neurotoxic Effects by Exposure to Mycotoxins in the Indoor Environment*, Mycopathologia, 377-390 (2010).

This study purports to demonstrate that "neurological system cell damage can occur from satratoxin H exposure to neurological cells at exposure levels that can be found in water-damaged buildings contaminated with fungal growth." Plaintiffs' Ex. 10, p. 377. It states that "[e]pidemiological studies conducted at office buildings contaminated with *Stachybotrys chartarum* demonstrated abnormal respiratory activity, elevated white blood cell counts, and central nervous system (CNS) disorders." *Id.* at 378. The study also states that "[e]xposure to mycotoxins has been shown to result in a decrease in hematopoiesis and symptoms associated with mycotoxin exposure include inflammation of the skin, diarrhea, hemorrhage, emesis, and nervous system abnormalities." *Id.*

5. Ebere C. Anyanwu et al., *Mycotoxins and Antifungal Drug Interactions: Implications in the Treatment of Illnesses Due to Indoor Chronic Toxigenic Mold Exposures*, The Scientific World Journal, 167-177 (2004).

This paper "review[s] the structure and functions of mycotoxins, their relationships, and the implications in the treatment of fungal infections." Plaintiffs' Ex. 13, p. 168. It states that "[m]ycotoxins are toxic chemicals released by indoor toxigenic molds. Under physiological conditions, they undergo metabolic processes to produce yet other toxic chemical compounds known as volatile organic compounds (VOCs)." *Id.* The paper states

that Aspergillus produces the mycotoxins aflatoxin and Sterigmatocystin. *Id.* Aspergillus and Penicillium produce, among others, the mycotoxin "gliotoxin (cytotoxic and immunosuppressive effects)." *Id.* Stachybotrys chartarum produces the mycotoxin Satratoxin H, which is "implicated in very high cytotoxicity and several environmental allergic reactions." *Id.* The paper identifies effects of "chronic exposures" to toxigenic molds as including, among others, brain disease, immune suppression, and organ damage. *Id.* at 171. The paper also states that "[n]eurological effects of mycotoxins are those effects on the structure or functioning of the central nervous system," resulting from "exposure to toxigenic molds" and causing "generalized damage to nerve cells (neuropathy), injury to axons (axonopathy), destruction of the myelin sheath (myelinopathy), or neurobehavioral dysfunction." *Id.* at 172.

6. *Transverse Myelitis Fact Sheet*, National Institute of Neurological Disorders and Stroke (2012).

This document states that "[r]esearchers are uncertain of the exact causes of transverse myelitis," but that "[t]he inflammation that causes such extensive damage to nerve fibers of the spinal cord may result from viral infections or abnormal immune reactions." Plaintiffs' Ex. 15, p. 2. "Cases in which a cause cannot be identified are called *idiopathic*." *Id.*

7. Anu Jacob et al., *An Approach to the Diagnosis of Acute Transverse Myelitis*, Seminars In Neurology, vol. 28, no. 1 (2008).

This article discusses clinical presentations of myelophathies and diagnostic categories of acute myelopathy. The article expressly identifies Aspergillus as a fungal "infectious

agent" that causes acute myelopathies such as TM. Plaintiffs' Ex. 16, pp. 112-13 and Table 6.

8. Andrew W. Campbell at al., *Mold and Mycotoxins: Effects on the Neurological and Immune Systems in Humans*, Advances in Applied Microbiology, vol. 55 (2004).

This article summarizes the authors' findings concerning health and environmental histories of symptoms, pulmonary function, alterations in peripheral lymphocyte phenotypes, autoantibodies, and neurological abnormalities observed in patients. Plaintiffs' Ex. 17, pp. 377-78. The article identifies various toxigenic molds, "[t]he mycotoxins isolated from the molds and their general toxic effects" and associated "health concerns," including, among others: the relationship between Stachybotrys and proinflammatory cytokines; the relationship between various types of Aspergillus and CNS injury, immune damage by gliotoxin, and neuropathy; and the relationship between Penicillium and neuropathy and immune toxicity. *Id.* at 379, Table 1. In a section titled "Immune Complexes," the article states that "inflammation and autoimmune reactions may exist in mold-exposed patients." *Id.* at 392. With respect to "Neurological Abnormalities," the article states that "[m]ycotoxins, being relatively nonpolar, pass through the blood-brain barrier and thereby have access to synapses," and that "trichothecene mycotoxins from Stachybotrys cause neurological disorders by being neurotoxic." *Id.* at 393. "Patients affected [by trichothecene mycotoxicosis] develop seizures, central nervous system dysfunction, hypotension, and myelosuppression," and "[s]tudies have shown that exposure to molds can cause optic demyelinating neuritis and multifocal choroiditis." *Id.* at 393-394.

9. William J. Rea, *Effects of Toxic Exposure to Molds and Mycotoxins in Building-Related Illness*, Archives of Environmental Health, vol. 58, no. 7 (July 2003).

This article reports results of a study of 100 patients who had been exposed to toxic mold in their homes. The patients reported "symptoms of respiratory, neurological, and immune dysfunction. (Children were not included in th[e] study.)" Plaintiffs' Ex. 18, p. 399. "Signs and symptoms were recorded for each organ system by an examining physician." *Id.* at 400. "Immune abnormalities were confirmed by individual T- and B-cell counts, which were performed by flow cytometry," and "[i]mmune globulin was evaluated by the mold antibody assay." *Id.* The study found that "[o]ne mold (*Stachybotrys*) and 1 mycotoxin (trichothecene) seemed particularly virulent, damaging all 3 systems: immune, respiratory, and neurological." *Id.* at 404.

According to the study:

> "[p]arameters suggestive of more severe problems than just an average mold sensitivity included alterations in white blood cell count (in 19% of patients), alterations in T-lymphocyte subsets of helper cells (72%), changes in T_8 lymphocytes (33%), and changes in B lymphocytes (24%). These findings further substantiate that the immune system was significantly altered in these patients with massive mold/mycotoxin exposure. Cell-mediated immunity decreased in 71% of 83 patients measured, again pointing to abnormal T-cell function. It appears that the mycotoxin overload significantly disrupted the immune system of these severely exposed patients."

Id. at 404. The study concludes that "[s]igns and symptoms reported were related primarily to the respiratory tract and to the neurologic and immune systems," and that "tests for mold sensitivity demonstrated a high correlation with exposure and with alteration of the immune

system." *Id.* at 405. Moreover, "[o]bjective alteration of the neurologic system was found by autonomic nervous system testing," which "confirmed clinical neurological involvement . . . ." *Id.*

10. Jack D. Thrasher et al, *The biocontaminants and complexity of damp indoor spaces: more than what meets the eyes*, Toxicology and Industrial Health 25(9-10), 583-615 (2009).

This paper reviewed "indoor biocontaminants resulting from water intrusion and their associated toxicity to animals and humans," and "the peer-reviewed research that points to the impacts on human health, including neurological, respiratory and immune system and other organs, from exposure to damp indoor spaces." Plaintiffs' Ex. 19, p. 584. In a table identifying toxic molds, mycotoxins, and their associated health concerns, the study identifies, among others: various types of Aspergillus with the health concerns of immune toxicity, immune suppression, and neurotoxicity; and Stachybotrys with the health concerns of neural tube defects, protein synthesis inhibition, neurotoxicity, cytotoxicity, and immune toxicity. *Id.* at 593, Table 2. The paper concludes that "[d]iagnostic tests should be developed and recommended to determine the nature of building-related illness, e.g. allergy, hypersensitivity pneumonitis, encephalopathy, fungal infections, bacterial infection, etc." *Id.* at 601.

11. Ebere C. Anyanwu, *The validity of the environmental neurotoxic effects of toxigenic molds and mycotoxins*, The Internet Journal of Toxicology, vol. 5, no. 2 (2008).

This paper reviewed peer-reviewed literature supporting "the validity of the environmental risks and adverse neurotoxic health effects of chronic exposures to toxigenic

molds and mycotoxins." Plaintiffs' Ex. 21, p. 1. It states that "[c]hronic indoor environmental exposure to toxigenic molds leads to various adverse neuroimmunologic and behavioral consequences," and that "[a]bnormalities in T and B cells, and subsets, were found in more than 80% of the patients." *Id.* at 7. The paper also states that "abnormal immunoglobulins (IgG, IgM, and IgA antibodies)" were found "in mold-exposed patients compared to the controls." *Id.*

12. Benjamin Greenberg, *Neuroimmunologic Disorders of the Central Nervous System: An Overview*, The Transverse Myelitis Association (2012).

This article provides an overview of TM and other immune-mediated disorders of the central nervous system. According to the article, "[t]he specific genetics in each of these disorders is not completely understood and environmental factors have not been clearly identified," but "[i]t is believed that the immune system response could be to a viral, bacterial or fungal infection, and in the case of TM, a significant number of people have flu-like symptoms," and "[t]his immune response might explain why the immune system was revved up." Plaintiffs' Ex. 22, p. 2. The article further explains that:

> "[t]he central nervous system is separated and protected from foreign agents by the blood brain barrier. For the immune system to attack anywhere in the central nervous system, cells from the immune system have to pass through this barrier. Thus, in the case of these disorders, not only does the immune system become confused, it also has to find a way to cross this protective barrier to get to the brain, the spinal cord and/or the optic nerves. These mechanisms are not very well understood."

Id. "Sometimes the inflammation has no clear cause and is referred to as Idiopathic TM. The majority of these cases are probably post infectious events, but this can be difficult to prove" (*id.*), and "[e]ach of these neuroimmunologic disorders remain a challenge to diagnose." *Id.* at 4. "The diagnostic criteria for the other disorders are neither entirely clear-cut nor universally accepted in medicine (i.e., there appear to be numerous exceptions to every rule.)" *Id.*

13. Michael R. Gray, *Mixed mold mycotoxicosis: immunological changes in humans following exposure in water-damaged buildings*, IEQ Review (November 21, 2006).

This article investigated "patients with chronic health complaints attributed to exposure to mixed colonies of indoor fungi and molds." Plaintiffs' Ex. 23, p. 1. The investigation included evaluation of "209 patients who presented with multiorgan system symptoms resulting from exposure to molds in their homes, schools, or workplaces." *Id.* The article states that "[p]otentially toxic and immunogenic byproducts of fungi and molds include mycotoxins," and that "[o]ccupants of affected structures can develop symptoms in multiple organ systems, including . . . central and peripheral nervous system . . . ." *Id.* In addition, "[h]uman illness can result from 1 or all of the following: mycotic infections or mycoses, immunoglobulin (Ig) E-mediated sensitivity and asthma; hypersensitivity pneumonitis and related inflammatory pulmonary diseases; cytotoxicity; immune suppression/modulation . . . ." *Id.*

The patient evaluations included physical examinations and routine diagnostic tests. *Id.* at 3. In these patients, "[s]erology tests for all viruses were negative for either active or

reactive infections." *Id.* The article states that "[o]verall, the symptom complex we observed was consistent with observations reported by others. The preponderance of symptoms involving the CNS and state of well-being are reflective of injury to the CNS," and "[t]he increased frequency of symptoms in females is consistent with their greater representation in several other clinical conditions (e.g., fibromyalgia and related disorders, autoimmune disease, and exposure to molds)." *Id.* at 5. The article explains that "mold-exposed patients demonstrated increased expression of various activation markers when compared with expected laboratory ranges," including "activated T cell[s]" and "nonallergic clinical conditions (e.g., autoimmune disorders . . . and inflammatory conditions)." *Id.* The article concludes that, "[c]ollectively, the increased presence of activated T cells, and increased 13 cells, implies a proinflammatory state," and "the increase in HLA-DR+ expression reflects the presence of increased autoimmunity. In the aggregate, this situation represents a proinflammatory, immune toxic state." *Id.*

According to this investigation:

> "[t]hese observations corroborate the immune changes reported herein and support the conclusion that exposure to mixed molds and their byproducts causes the expression of immune markers of activation, as well as at least 1 inflammatory cytokine – TNF-[alpha]. Furthermore, the decreases we observed in the percentage of peripheral blood NK cells and response to PHA further support the concept that immune dysregulation is occurring, and represents a 'promoter' state for the expression and development malignancies."

Id. at 6. The patients in this study "exhibited a high risk for producing autoantibodies to nuclei, smooth muscle, CNS and PNS myelin, and neurofilament," and the study states that

"individuals exposed to mixed molds produce several different autoantibodies. Work is in progress to determine the significance of these antibodies in conditions such as lupus erythematosus, autoimmune neuropathy, and a multiple-sclerosis-type syndrome." *Id.* The article concludes that "the presence of autoantibodies is commensurate with immune activation, and, finally, the antineuronal antigen(neurofilament)-specific antibodies are strongly associated with a wide array of degenerative neurological disorders of undetermined origin." *Id.* at 6-7.

14. M.C. Brouwer, *Vertebral Osteomyelitis Complicating Pneumococcal Meningitis*, American Academy of Neurology (August 19, 2008).

This article describes two cases of vertebral osteomyelitis complicating community-acquired bacterial meningitis due to *Streptococcus pneumoniae*." Plaintiffs' Ex. 24, p. 612. It states that "[b]acterial meningitis is a serious and life-threatening disease with a broad spectrum of complications," and that [v]ertebral osteomyelitis is a rare complication of bacterial meningitis and has only been described in patients with meningitis due to uncommon pathogens." *Id.*

15. M. Cimerman, *Femur Osteomyelitis Due to a Mixed Fungal Infection in a Previously Healthy Man*, Journal of Clinical Microbiology, vol. 37, no. 5, 1532-1535 (May 1999).

This paper reports "the only known occurrence of a primary mixed *Aspergillus* and *Chalara* osteomyelitis in one of the long bones in a healthy individual with no evidence of any other primary fungal infection." Plaintiffs' Ex. 25, p. 1532. The patient suffered a closed fracture of the femur and underwent a corrective operation, after which he developed

chronic osteomyelitis. *Id.* According to the paper, this was "the only known instance in which a long bone was affected in an immunocompetent individual, with no evidence of any systemic infection, by mixed population of two different *Aspergillus* spp. and the rare filamentous fungus *C. Ellisii.*" *Id.* The paper states that, "[i]n most cases of fungal osteomyelitis, gross defects in immunocompetence are present." *Id.* at 1534.

16. *Methods for Evaluation of Indoor Mold Growth*, New York Committee for Occupational Safety and Health (published at www.nycosh.org as of the date of this decision).

This pamphlet explains methods for evaluating mold growth and interpreting sampling results, relying upon the New York City Department of Health and Mental Hygiene, the United States Environmental Protection Agency, and the United States Occupational Safety and Health Administration. Plaintiffs' Ex. 28, pp. 1-2. It recommends visual inspection, and also explains and recommends interpretations for bioaerosol (air) sampling, spore trap (air) sampling, micro-vac sampling, surface wipe sampling, surface swab sampling, and tape lift sampling. *Id.* It defines "active mold growth" as greater than 1,000 colonies per cubic meter of air sampled in bioaerosol samples, with assessment "based on relative comparison of indoor and outdoor types and concentrations." *Id.* at 1. Surface wipe samples of 5,000 to 10,000 colony forming units per gram is considered an "elevated level of contamination." *Id.* at 2. Surface swab samples of greater than 10,000 colony forming units per square inch (or 1,500 per square centimeter) constitutes "probable contamination." *Id.* In the absence of standards, this pamphlet recommends interpreting mold sampling results by comparing outdoor versus indoor amounts, types, and microbial dominance. It states that "[t]he elevated

presence of certain pathenogenic (disease-causing) or toxin-producing (poisonous) micro-organisms is considered unacceptable," including: stachybotrys chartarum, aspergillus fumigatus and versicolor, and "various penicillium species." *Id.* at 2.

17. Douglas A. Kerr et al., *Immunopathogenesis of acute transverse myelitis* (2002).

This is the same article cited by The Bakery, discussed above, and, therefore, its contents are not summarized here.

18. Stanzani M, *Apergillus fumigatus suppresses the human cellular immune response via gliotoxin-mediated apoptosis of monocytes*, Blood (March 15, 2005).

This abstract states that "gliotoxin, the most abundant mycotoxin produced by AF [Aspergillus fumigatus], was able to suppress functional T-cell responses following [cytomegalovirus] or staphylococcal enterotoxin B (SEB) stimulation." Plaintiffs' Ex. 40. The abstract concludes that "[t]hese studies suggest that the production of gliotoxin by AF [Aspergillus fumigatus] may constitute an important immunoevasive mechanism that is mediated by direct effects on antigen-presenting cells and both direct and indirect effects on T cells." *Id.*

19. Animal Studies

Plaintiffs also submit several articles involving animal studies with molds and mycotoxins. *See e.g.* Plaintiffs' Exs. 2, 4-5, 8, 26, 32-33, 41. On the instant motion, the court need not rely upon these animal studies, as plaintiffs have submitted sufficient medical and scientific literature involving human exposure to various mold types and the mycotoxins they produce. While the court makes no determination as to the admissibility of these

articles, the court notes that other courts have questioned the relevance and admissibility of animal studies, reasoning that "laboratory animal studies * * * are generally viewed with more suspicion than epidemiological studies, because they require making the assumption that chemicals behave similarly in different species," and that "[a]nimal studies are aimed at discovering a dose-response relationship, while epidemiological studies show an association between exposure and disease." *In re "Agent Orange" Prod. Liab. Litig.*, 611 F.Supp. 1223, 1241 (E.D.N.Y. 1985) (internal quotation marks and citations omitted), *aff'd* 818 F.2d 187 (2d Cir. 1987), *cert. denied* 487 U.S. 1234 (1988).

D. Plaintiffs' Expert Affidavits and Reports

1. McGuinness

As discussed above, McGuinness is plaintiffs' mold expert, and the certified industrial hygienist who prepared the RK Report. McGuinness reviewed the results of the RK Report, the ECMS Report, and the JLC Report. McGuinness also reviewed several scientific papers submitted by plaintiffs. According to McGuinness, "[t]here are no standards for dose-response and there are no dose-response curves available regarding mold exposure. In other words, there is no established threshold of exposure to cause pathological responses in humans." McGuinness Aff. in Opp., p. 5. McGuinness states that, therefore, "it is generally accepted that the indoor bioaerosol levels should be less than those levels seen outdoors in mechanically ventilated facilities and the species and biodiversity of fungi identified should be similar indoors and outdoors." *Id.* McGuinness explains that results falling outside of these guidelines indicate "unusual fungal conditions or populations and are considered

unacceptable from an occupant health perspective and a building performance perspective." *Id.* (emphasis in original).

According to McGuinness, The Bakery incorrectly stated that ECMS's mold tests revealed higher counts of mold, including Aspergillus, outside plaintiffs' residence than inside. *Id.* at 13; Moretti Aff. in Supp., ¶ 16. Specifically, the Aspergillus/Penicillium present in Maya's room and the office was 798, while the outdoor amount was only 252. Moretti Aff. in Supp., Ex. 23, pp. 2-3. In addition, Stachybotrys was present in both the nursery and office, but none was detected outdoors. *Id.* McGuinness also points to ECMS's express statement that "[t]he presence of surface growth of Memnoniella & Stachybotrys is considered to be problematical especially when found indoors and a child or adult with a sickness or immune compromising sickness is present such that physiological conditions could be worsened by exposure." *Id.* at 3. He concludes that the sampling conducted at plaintiffs' residence contained "unusual fungal populations." *Id.* at 12.

McGuinness analyzed the discrepancy between the JLC Report and the ECMS Report. Although both reports identified molds in surface samples, the JLC Report indicated that air samples reflected outdoor conditions, as is argued by The Bakery. However, the JLC Report stated that the apartment "[w]indows had been open, but were closed at the onset of inspection." Moretti Aff. in Supp., Ex. 24, ¶ 2.0. According to McGuinness, leaving the windows "open for an undetermined period of time prior to JLC's site visit and sampling" left "ample time . . . for indoor and outdoor conditions to equilibrate . . . the conditions seen in the air samples collected on June 30, 2006 . . . ." McGuinness Aff. in Opp., p. 11.

McGuinness emphasizes the significant airborne levels of Stachybotrys and Aspergillus/Penicillium and surface levels of Stachybotrys and Memnoniella in plaintiffs' residence, and the close proximity in the timing of plaintiffs residing in the apartment, the mold test results, Keiko's return visits to the apartment after plaintiffs had vacated, and Keiko and Maya's illnesses. *Id.* at 12. McGuinness concludes that Keiko and Maya were exposed to toxigenic, mycotoxin-producing fungi sufficient to cause significant adverse health consequences. *Id.*

2. Dr. Grant

Dr. Irene Grant is a practicing physician specializing in internal medicine and infectious disease. Grant Aff. in Opp., pp. 1-2. Dr. Grant's affidavit is based upon and refers to the medical and scientific literature submitted by plaintiffs, the mold test results from plaintiffs' residence, and the medical records of Maya and Keiko. In her affidavit, Dr. Grant states that a significant portion of her practice is dedicated to treating patients with environmental mold exposure and mold-related illnesses. Dr. Grant states that the molds identified in plaintiffs' residence are toxigenic, and that "Aspergillus/Penicillium are opportunistic molds that cause infections in the immunocompromised." *Id.* at 3.

With respect to Maya, Dr. Grant states that no testing was performed to diagnose a fungal infection, but that Maya's blood cultures grew multiple bacteria, which is "a marker for a break in tissue barrier protection," whereas "[i]ntact tissues are part of immune protection." *Id.* at 4. Dr. Grant stated that, "[w]ith pneumococcal meningitis only pneumococcus would be expected to be in the blood," and that, "[h]aving 2 different

organisms in 2 out of 2 blood culture specimens occurs when relatively large fungal hyphae attempt to puncture vessels which allow multiple smaller bacteria to rapidly gain access to the blood stream." *Id.* In addition, Maya's

> "cerebrospinal fluid (CSF) showed a markedly decreased glucose <20, a markedly elevated CSF protein and WBC 550, 69% PMN, (markers for unusually severe bacterial infection) and red blood cell (RBC) 40. The presence of red blood cells in the CSF suggests a process damaging tissue blood vessels was occurring (molds especially Aspergillus/Penicillium are known to have [a] predilection for trying to invade blood vessels causing bleeding . . .). Thus bleeding is a key marker for fungal infection, especially Aspergillus."

Id. According to Dr. Grant, Maya's development of "a prolonged seizure coincident with multiple laboratory abnormalities" was "suggestive of a fungal infection and progressive inflammation: her C-Reactive Protein (CRP) (marker for inflammation and immune activation) rose to 3.0, WBC to 16.2K with markedly elevated absolute monocyte count . . . ." *Id.* In addition, Maya's "platelets (PLT) also rose to 763 [and] remained elevated until after her discharge (Thrombocytosis [is] a marker for deep tissue infection such as a persistent fluid collection or abscess)." *Id.* Dr. Grant maintains that Maya exhibited "abnormal [test] results" which "would occur in an uncontrolled ongoing fungal infection." *Id.* Maya's "glucose was elevated 131 mg/dL <99 (recognized risk for fungal infection), with further elevations in CRP, Complement total CH 50 with a markedly elevated IgM antibody titer," and "[a] low IgG subclass 4 was documented supporting the fact that her immune system was not yet developed." *Id.* According to Dr. Grant, Maya's symptoms persisted after being discharged from the hospital – fevers, congestion, watery eyes, left sided

otitis media, elevated monocyte count, platelets, and sedimentation rate – "are consistent with an unrecognized noninvasive fungal infection." *Id.* at 4-5.

Dr. Grant states that Maya's illness "was more probable than not at root a fungal infection, especially Aspergillus, of the upper airways and ears trying to extend into the CNS, and thereby promoting a bacteremia and bacterial meningitis," which "are difficult to recognize. Since Maya was immunosuppressed, the progression of her infection was dramatic and acute." *Id.* at 5. Dr. Grant states that "Maya was immunosuppressed from the get-go due to her immune immaturity. In addition she was documented with deficiencies in protein and IgG subset 4." *Id.* According to Dr. Grant:

> "[l]iving and sleeping in a mold contaminated environment as Maya did for months is consistent with a chronic heavy exposure. Since Stachybotrys' products instantaneously destroy tissue surfaces as well [as] suppress the innate immune response, it is also highly probable that the Stachybotrys exposure Maya experience inflamed and impaired her respiratory tract, facilitating colonization with biofilm establishment by Aspergillus/Penicillium in her sinuses and ears. Maya developed upper respiratory inflammation with sinusitis, otitis and mastoiditis due to her immune immaturity and was therefore immunosuppressed. The pathogenesis of pneumococcal meningitis is considered to result from seeding of the blood from another infected locus such as the nose, sinuses, pharynx[,] ears, mastoids, or lungs.
>
> Thus it is highly probable that exposure to the molds documented in the apartment caused (1) mucosal injury to her respiratory tract due to contact with Stachybotrys spores, metabolites and particulates debris, and (2) immunosuppression due to exposure to spores and toxic immunosuppressive products of known immunosuppressive molds (Stachybotrys and Aspergillus/Penicillium[)] and (3) establishment of a mold infection, most probably Aspergillus, in her sinuses and ears that spread and damaged (a) her inner ear, spread to her mastoids,

> with attempted extension into her meninges promoting bacterial blood brain barrier resulting in a secondary bacterial meningeal infection, and triggering a fluid loculation which developed while she was on antibacterial therapy that should have arrested a pneumococcal infection.
>
> . . .
>
> Stachybotrys spores, particulate debris and metabolites, including the trichothecene mycotoxins all inflame the respiratory tract. Many suppress the immune responses as well. This is relevant to both Maya['s] demise as well as [Keiko] Honda's.
>
> . . .
>
> Surgical drainage and resection has always been essential in the management of mold infections until recently with the advent of antifungal therapy. Therefore, Maya did get treated with antifungal therapy - her infected subdural loculation was surgically drained. Her immune system eventually controlled the fungal infection (again most likely Aspergillus). However the resultant damage to her ear and potential life-long risk of easy bacterial entry to her CNS resulted.
>
> In my experience treating and monitoring patients exposed to environmental Stachybotrys and Aspergillus/Penicillum, sinusitis is the most frequent infectious complication followed by otitis. Neurological symptoms occur in over 65%, including seizures."

Id. at 5-7.

Dr. Grant concedes that, unlike Maya, Keiko was not immunosuppressed. Dr. Grant acknowledged that Keiko had "mild idiopathic thrombocyptopenic purpura a few years ago, which self-resolved," and that "[t]here is no family history of autoimmune disease." *Id.* at 8. Dr. Grant states that Keiko's exposure to mold debris, mycotoxins and spores allowed mycotoxins to "gain[] access into her nervous system via the olfactory neurons," causing

"irreversible destructive lesions within the central nervous system and spinal cord resulting in ATM [acute transverse myelitis], quadriparesis, chronic neuropathic pain, spasticity and bowel & bladder dysfunction." *Id.* at 7. Dr. Grant cites to the medical and scientific literature submitted by plaintiffs, in support of her conclusion that exposure to Stachybotrys is "unequivocally a health hazard to anyone regardless of immune status," as "this toxigenic mold is known to produce immunosuppressing products." *Id.* at 12. Dr. Grant concludes that "[t]he highly elevated levels of Aspergillus and Stachybotrys" in plaintiffs' residence "is sufficient evidence that the amount of exposure to viable mold and toxic mold products caused the illnesses sustained by both [Keiko] Honda and Maya." *Id.* at 10.

3. Dr. Kerr

Dr. Kerr founded the only clinical center dedicated to the diagnosis and treatment of Transverse Myelitis in 1999, and he served as the director of this center until 2010. Kerr Aff. in Opp., p. 2. His November 20, 2012 affidavit is based upon and refers to his review of Keiko and Maya's medical and scientific literature, and the medical records and physical examinations of Keiko and Maya. *Id.* at 1. According to Dr. Kerr, Keiko's transverse myelitis "is not a part of a multifocal CNS disease like neuromyelitis optica . . . or multiple sclerosis," and, "[t]herefore, her TM fits into the category of idiopathic or parainfectious TM." *Id.* at 4.

Dr. Kerr found that Keiko had abnormal lab results, including "low Protein S function (35%) and free Protein S (36%), low C4BP (46%), elevated high thrombin time (96, nl is<30), Low IgA=40 and Ig=253." *Id.* at 3. According to Dr. Kerr, "[t]his means . . . that

two proteins that inhibit blood clotting, protein S and C4 Binding protein (C4BP) were less than half the level they should have been in [Keiko's] blood, and her clotting time was over 3 times higher than it should have been." *Id.* at 11. Together with the low immunoglobulin levels, Dr. Kerr concludes that these results suggest "a systemic hematologic/immunologic derangement, triggered by the mycotoxins that caused the TM." *Id.*

In his assessment of relevant exposures, Dr. Kerr noted that Keiko sought medical attention for knee discomfort and quadriceps tenderness on November 16, 2006, and she was diagnosed with quadriceps tendonitis. Dr. Kerr maintains that this incident may "suggest a developing immune response to the toxic mold to which she was exposed in the apartment." *Id.* at 5. Dr. Kerr also noted that Keiko returned to the apartment on December 5, 2006 for more than two hours, after which she developed flu-like symptoms, vertigo and vomiting, and the onset of TM on December 11, 2006.

According to Dr. Kerr, there are two mechanisms of injury to the spinal cord in TM: indirect neural injury, caused by alteration to the immune system and resulting in an autoimmune attack of the spinal cord; and exposure to agents that are directly neurotoxic to the spinal cord. Citing the above-referenced medical and scientific literature, Dr. Kerr concludes that "toxins from chronic mold exposure result in immune alterations and direct neurotoxic injury." *Id.* at 6. Dr. Kerr does not claim that Keiko and Maya's illnesses were caused by a "direct fungal invasion," but rather, that "the mycotoxins from the mold created an immune derangement," which would not be revealed in the results of Keiko and Maya's spinal taps. *Id.* at 11.

Dr. Kerr claims that it is unlikely for Keiko and Maya to "develop extremely rare immune-mediated disorders within a 6 months period of each other with no underlying trigger." *Id.* at 10. He states that TM, bacterial meningitis, and osteomyelitis are all associated with immune system suppression, which has been shown to be caused by the mycotoxins produced by Stachybotrys and Aspergillus. *Id.* According to Dr. Kerr, the chances of all three illnesses to occur within a single family in a six-month period is approximately one in 25 trillion. *Id.* Dr. Kerr states that, without exposure to mycotoxins, Maya may have still contracted pneumococcal meningitis, but she likely would not have developed osteomyelitis.

III. Analysis

The Bakery and its experts claim that there is insufficient scientific evidence to support plaintiffs' theory that mold exposure, in otherwise healthy people, could cause the illnesses sustained by Keiko and Maya, or that mold exposure could suppress the immune system causing an otherwise healthy individual to contract an infection. The Bakery argues that neither Keiko nor Maya had a suppressed immune system, and that Keiko's serology testing revealed no evidence of an infection. According to The Bakery, "there is no general acceptance in the medical community that mold exposure causes TM," and "[t]here are no peer-reviewed articles linking Maya's injuries, meningitis and osteomyelitis, to mold exposure." Moretti Aff. in Supp., ¶¶ 62-63. In addition, they argue, Dr. Kerr failed to mention that the mold tests revealed higher counts of mold, including Aspergillus, outside plaintiffs' residence than inside.

"An expert opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (i.e., general causation), and that the plaintiff was exposed to levels of the toxin sufficient to cause illness (i.e., specific causation)." *Nonnon v. City of New York*, 88 A.D.3d 384, 394 (1st Dep't 2011), *citing Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434, 448 (2006). "[T]he *Frye* test asks whether the accepted techniques, when properly performed, generate results accepted as reliable within the scientific community generally." *Parker*, 7 N.Y.3d at 446 (internal quotation marks and citations omitted). As stated in *Parker*, "*Frye* holds 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." *Id.* at 446-447 (internal quotation marks and citations omitted). "General acceptance does not necessarily mean that a majority of the scientists involved subscribe to the conclusion, but that those espousing the theory or opinion have followed generally accepted scientific principles and methodology in reaching their conclusions." *Nonnon*, 88 A.D.3d at 394. Stated differently, "[t]he focus of the *Frye* inquiry 'should not be upon how widespread [a] theory's acceptance is, but should instead consider whether a reasonable quantum of legitimate support exists in the

literature for [an] expert's views.'" *Cornell v. 360 W. 51st St. Realty, LLC*, 95 A.D.3d 50, 52 (1st Dep't 2012), citing *Marsh v. Smyth*, 12 A.D.3d 307, 312 (1st Dep't 2004, Saxe, J., concurring).

In *Cornell*, the First Department held that, "[s]ince plaintiff's expert's opinions relating plaintiff's condition to the mold infestation find 'some support in existing data, studies [and] literature' (*Marsh*, 12 A.D.3d at 313), namely, studies that have found a statistically significant relationship between mold and various respiratory maladies, the *Frye* standard is satisfied." *Id.* at 53.

> "[I]t is not necessary that the underlying support for the theory . . . consist of cases or studies considering circumstances exactly parallel to those under consideration in the litigation. It is sufficient if a synthesis of various studies or cases reasonably permits the conclusion reached by the . . . expert. The fact that there [is] no textual authority directly on point to support the [expert's] opinion is relevant only to the weight to be given the testimony, but does not preclude its admissibility."

LaRose v. Corrao, 105 A.D.3d 1009, 1009 (2d Dep't 2013) (internal quotation marks and citations omitted).

Thus, a *Frye* objection will be rejected where the medical literature, "when considered in the aggregate" and "[s]ynthesized . . . , provide[s] an objective basis for [the experts'] opinion[s]," even if none of the articles in the medical literature, "read in isolation, provides conclusive support for the theory of causation espoused by the plaintiffs' experts." *Lugo v. New York City Health & Hosps. Corp.*, 89 A.D.3d 42, 61 (2d Dep't 2011). A "[d]efendant's

factual disagreement with plaintiff's medical causation theory [does] not warrant a [*Frye*] hearing . . . [where] no scientific technique or novel application of science [is] at issue," and the objection will be rejected where "there [are] relevant examples and data accompanying the experts' opinions." *Gayle v. Port Auth. of N.Y. & N.J.*, 6 A.D.3d 183, 184 (1st Dep't 2004). Once the court makes the limited determination that "the experts' deductions are based on principles that are sufficiently established to have gained general acceptance as reliable," the court may not "intrude[] upon the jury's realm of weighing the evidence." *Marsh*, 12 A.D.3d at 308.

The burden of proof concerning general acceptance rests with plaintiffs, as the proponents of the scientific theory at issue, and the showing of "generally accepted reliability" may be based upon "'judicial opinions, scientific or legal writings, or expert opinion other than that of the proffered expert.'" *Selig v. Pfizer, Inc.*, 185 Misc 2d 600, 605 (Sup. Ct., N.Y. Co. 2000) (citation omitted), *aff'd* 290 A.D.2d 319 (1st Dep't 2002).

The expert testimony proffered by plaintiffs does not introduce a novel scientific technique. Rather, the *Frye* standard is implicated because The Bakery challenges whether plaintiffs' expert theory is supported by scientific data, and whether that theory raises a novel conclusion. Here, the medical and scientific literature submitted by defendants suggests that clear conclusions cannot be drawn concerning non-respiratory health problems associated with mold exposures, and the existence of a causal relationship between mold exposure and immune and neurological diseases. The Bakery also relies upon Dr. Kerr's conclusion that Keiko's illness "fits into the category of idiopathic [from unknown cause] or parainfectious

TM." Dr. Kerr aff, at 4. In essence, The Bakery relies upon the dearth of medical and scientific evidence establishing a direct link between mold exposure and meningitis, osteomyelitis, and transverse myelitis, the illnesses suffered by Keiko and Maya.

As a preliminary matter, the article *An Approach to the Diagnosis of Acute Transverse Myelitis*, submitted by plaintiffs, expressly identifies Aspergillus as a fungal "infectious agent" that causes acute myelopathies such as Keiko's transverse myelitis. Plaintiffs' Ex. 16, at 112-13 and Table 6. Virtually all of the evidence submitted by the parties, including The Bakery's medical and scientific literature, supports the conclusion that indoor mold is a common cause of life-threatening infections in immunosuppressed patients, and Drs. Grant and Kerr opine that Maya was immunosuppressed and predisposed due to her age and immature immune system. In any event, while much of the medical and scientific articles and position papers submitted by plaintiffs may not "exactly parallel" the facts of plaintiffs' case, "a synthesis" of this literature "reasonably permits the conclusion" that Keiko and Maya were exposed to high levels of mold that is known to produce harmful mycotoxins, and that mycotoxins damage immune and neurological systems, both of which are implicated by Keiko and Maya's illnesses. *LaRose*, 105 A.D.3d at 1009. Specifically, the mold sampling test results from plaintiffs' apartment, and the accompanying affidavit of plaintiffs' mold expert, show that the concentration of Aspergillus/Penicillium in the nursery and office of plaintiffs' apartment were more than three times higher than the exterior. This evidence also showed that the interior of the apartment contained medium to high levels of Stachybotrys, another toxic mold, while the outdoor samples did not show Stachybotrys. According to

plaintiffs' experts' affidavits and the medical and scientific literature that they rely upon, these levels could cause significant health problems, including neurological and immune system dysfunction that lead to Keiko and Maya's illnesses.

Plaintiffs' evidence provides a "'scientific expression' of plaintiffs' exposure levels" to several species of toxigenic fungi – that is, fungi that produce mycotoxins – in quantities evidencing high levels of contamination. *Nonnon*, 88 A.D.3d at 396. Thus, plaintiffs "have laid an adequate foundation for their opinions on specific causation." *Id.* The literature upon which plaintiffs' experts rely reasonably permits the conclusion that there is a significant relationship between the mold found in plaintiffs' apartment and immune and nervous system injury and dysfunction, thereby increasing Keiko and Maya's susceptibility to their illnesses. At a minimum, plaintiffs have presented "a reasonable quantum of legitimate support . . . in the literature for [their] expert's views," concerning the causal link between Keiko and Maya's illnesses and mold exposure. *Cornell*, 95 A.D.3d at 52 (internal quotation marks and citation omitted).

As discussed above, the lack of "textual authority directly on point to support the [expert's] opinion is relevant only to the weight to be given the testimony, but does not preclude its admissibility." *LaRose*, 105 A.D.3d at 1009. In essence, The Bakery's experts disagree with plaintiffs' theory of medical causation, but nothing contained in The Bakery's papers disagrees with the specific scientific methodology or techniques employed by the doctors and scientists who authored the literature relied upon by plaintiffs. Plaintiffs' theory is accompanied by scientific data and expert opinions, and the experts' disagreement about

plaintiffs' theory of causation neither warrants preclusion nor a *Frye* hearing. *Gayle*, 6 A.D.3d at 184.

The Bakery relies upon *Cubas v. Clifton & Classon Apt. Corp.*, where the plaintiff's expert affidavits "merely asserted, in conclusory fashion, that the injured plaintiff and the decedent became sick as a result of their exposure to toxic mold in the cooperative apartment building where they resided, which was owned by the defendant." 82 A.D.3d 695, 696 (2d Dep't 2011). The Second Department granted summary judgment dismissal, holding that the plaintiff's expert affidavits "failed to utilize objective standards to show that the toxic mold to which the plaintiff and the decedent were allegedly exposed was capable of causing their injuries, or that their exposure to the toxic mold was the actual cause of their illnesses and symptoms." *Id.* Here, in contrast, plaintiffs' expert affidavits are based upon dozens of peer-reviewed articles and position papers that substantiate their causation theory, thereby "rais[ing] a triable issue of fact as to whether any action or omission on the part of the defendants caused the alleged injuries." *Id.* Therefore, *Cubas* is distinguishable on its facts.

The Bakery also relies upon *Fraser v. 301-52 Townhouse Corp.*, where the First Department affirmed the trial court's dismissal of the plaintiff's personal injury claim, because the "plaintiffs . . . failed to meet their burden of establishing general acceptance of the theory on which the specific claims at issue [were] based." 57 A.D.3d 416, 418 (1st Dep't 2008). However, the Court expressly limited its holding to the evidence presented by the plaintiffs, stating: "[w]e 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." *Id.* Thus, the First Department "never disavowed the underlying theory that exposure to mold may, under certain circumstances, give rise to respiratory and other ailments." *Cornell*, 95 A.D.3d at 52. *Fraser* did not involve TM, meningitis, or osteomyelitis. The parties in that action did not rely upon the same medical and scientific literature as plaintiffs herein, and *Fraser* did not involve allegations involving an immuno-compromised child. Accordingly, *Fraser* is distinguishable on its facts.

The court notes that the parties challenge the credibility of certain experts and the authors of certain medical and scientific papers. For instance, Dr. Andrew Campbell, who authored the above-referenced article relied upon by plaintiffs, *Mold and Mycotoxins: Effects on the Neurological and Immune Systems in Humans*, surrendered his medical license in the State of Texas, by voluntary surrender order dated November 4, 2011. Moretti Reply Aff., Ex. 53. Even if the court were to make a credibility ruling with respect to Dr. Campbell, his article was co-authored by three additional individuals, who The Bakery criticizes but fails to discredit. The Bakery also criticizes plaintiffs' reliance upon medical and scientific literature authored or co-authored by Drs. Kaye Kilburn, Jack Thrasher, Eckhardt Johanning, Aristo Vojdani, Allan Lieberman, William Rea, Michael Gray, and Douglas Kerr. However, The Bakery fails to explain how or why the specific articles and position papers submitted by plaintiffs should be discredited. Moreover, many of these individuals' articles and position papers were co-authored by at least some individuals not challenged by The Bakery. A significant portion of the articles and position papers relied upon by plaintiffs were

authored by individuals that remain entirely unchallenged by The Bakery. In any event, "[i]t [is] the jury's prerogative to resolve the conflicting testimony from the medical causation experts" (*Gayle*, 6 A.D.3d at 184), and the court may not "intrude[] upon the jury's realm of weighing the evidence." *Marsh*, 12 A.D.3d at 308.

The court also notes that The Bakery submits several articles and position papers with its reply papers. These documents cannot "form a basis for their expert[] opinion[s] because [they were] only submitted in [The Bakery's] reply papers." *Mitrovic v. Silverman*, 104 A.D.3d 430, 431 (1st Dep't 2013). To the extent that The Bakery relies upon these documents to refute the medical and scientific literature submitted by plaintiffs, as The Bakery argued when oral arguments were heard on this motion (5/13/13 Tr., 44:18-21), they merely express disagreement with plaintiffs' theory of causation. Therefore, "such literature only affects the weight given to [the] expert[] opinion[s] and does not dictate an outcome as a matter of law." *Id.* Nor is the court persuaded by The Bakery's arguments, raised for the first time in its reply papers, that no tests were performed to determine the presence of known mycotoxins and that Dr. Kerr should be discredited because he engaged in research misconduct, as plaintiffs have had no opportunity to respond to these arguments and the evidence submitted in support thereof. *Id.*; *see also Schultz v. 400 Coop. Corp.*, 292 A.D.2d 16, 21 (1st Dep't 2002) ("[t]he consideration of arguments advanced at a time when the opposing party has no opportunity to respond is a procedure that this Court condemned").

For the foregoing reasons, The Bakery's evidence fails to eliminate material factual issues from the case. *Winegrad v. New York Univ. Med. Ctr.*, 64 N.Y.2d 851, 853 (1985).

Accordingly, The Bakery's motion for summary judgment is denied. As plaintiffs have substantiated their causation theory by showing general and specific causation, The Bakery's motion to preclude plaintiffs' expert witnesses from providing opinion evidence is denied. Moreover, a *Frye* hearing is unwarranted, as plaintiffs' causation theory is substantiated and supported by "relevant examples and data accompanying the experts' opinions." *Gayle*, 6 A.D.3d at 184. Moreover, as the parties' submissions make clear that they have "totally exhausted [their] arguments and authorities" on this motion, a hearing is unwarranted, as "the court cannot see how a *Frye* hearing can shed any more light on these issues." *Selig*, 185 Misc. 2d at 607, *aff'd* 290 A.D.2d 319.

Accordingly, it is hereby

ORDERED that the motion for summary judgment of defendant The Bakery, Inc. is denied; and it is further

ORDERED that counsel for the parties shall contact the Judge to whom this case is reassigned to schedule a conference.

This constitutes the decision and order of this Court.

Dated: New York, New York
April 28, 2014

ENTER:

Saliann Scarpulla, J.S.C