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The Same Mold Story: What Toxic Mold Is Teaching Us about Causation in Toxic Tort Litigation

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INTRODUCTION

> She blinded me with science.
>
> —Thomas Dolby[1]

Pity the molds. For millennia, they have lived side-by-side with humans, efficiently tidying up our environment, providing us with nutrients, and even giving us penicillin, one of the most critical elements in the unending battle against infectious disease.[2] Without the molds, the human race would quickly succumb to disease and death.[3] And yet today the molds are maligned more than ever; blamed for ailments ranging from the benign to the life-threatening;

1. THOMAS DOLBY, *She Blinded Me with Science*, *on* GOLDEN AGE OF WIRELESS (Capitol Records 1982).

2. *See* CONSTANTINE JOHN ALEXOPOULOS ET AL., INTRODUCTORY MYCOLOGY 5–14 (4th ed. 1996).

3. NICHOLAS P. MONEY, CARPET MONSTERS AND KILLER SPORES: A NATURAL HISTORY OF TOXIC MOLD 21 (2004).

cursed for destroying lifetimes of wealth; faulted for ruining enormous man-made structures; cited as a threat to the viability of human industry. Today, "toxic mold" is affixed into the layperson's psyche alongside a cadre of other environmental concerns such as asbestos, arsenic, and mercury. Common household sales involve mold inspections—a rarity a decade ago.[4] Homeowners that file water damage claims typically face loss of insurance coverage and severe difficulty in obtaining alternate coverage.[5] Municipal buildings, including dozens if not hundreds of schools, have been shut down, and even destroyed, in response to mold concerns.[6] Our appreciation for the molds has evolved from one of curious respect to outright fear and hostility.

Predictably, this evolution in our appreciation of the molds has been accompanied by an onslaught of litigation.[7] Salacious

4. Susan Ware, *As Worries About Mold Grow, So Do Inspection Requests*, BOSTON GLOBE, Dec. 28, 2003, at NW2.

5. *See* Karen E. Klein, *Caught in a Storm*, L.A. TIMES, Nov. 24, 2002, at K1. Insurance coverage in virtually all states now specifically excludes mold-related claims or charges significant additional fees for mold coverage. Motoko Rich, *Nightmares on Mold Street*, N.Y. TIMES, Dec. 11, 2003, at D1; Ware, *supra* note 4. The rising cost of mold claims has been a significant financial burden for the insurance industry, causing it to reduce or eliminate coverage in some states. Christopher Oster, *Insurance Companies Just Say 'No' to Covering Mold*, WALL ST. J., Aug. 8, 2002, at D1. Industry officials have predicted that mold claims will also be financially crippling in North Carolina and have asked for significant leeway to raise homeowners' insurance rates. *See* WRAL.com, *Mold Problems May Cause Insurance Rates To Rise in N.C.*, *at* http://www.wral.com/news/2531272/detail.html (Oct. 23, 2003) (quoting representative of North Carolina Rate Bureau: "We pretty much assumed that what happened in Texas was likely to occur in North Carolina.") (on file with the North Carolina Law Review). The North Carolina Rate Bureau asked the state to allow homeowners' insurance rates to be raised fourteen percent. *Id.* The North Carolina Rate Bureau also asked the North Carolina Department of Insurance for a $5,000 cap on mold-related insurance claims. Allen Norwood, *Insurers Put Cap on Mold Payouts*, CHARLOTTE OBSERVER, July 27, 2002, at 1E. The cap was approved in December 2001 and went into effect on May 1, 2002. *Id.* The tumult in the insurance industry and the effect of mold on real estate transactions has been addressed extensively and will not be the primary focus of this piece. *See generally* Walter G. Wright Jr. & Stephanie M. Irby, *The Transactional Challenges Posed by Mold: Risk Management and Allocation Issues*, 56 ARK. L. REV. 295, 347–71 (2003) (discussing the effect of toxic mold on real property transactions); D. Chris Harkins, Comment, *The Writing Is on the Wall . . . and Inside It: The Recent Explosion of Toxic Mold Litigation and the Insurance Industry Response*, 33 TEX. TECH L. REV. 1101 (2002) (discussing the effect of mold litigation on the insurance industry); Sylvia Peña-Alfaro, Comment, *The Toxic Mold Terrifying Texas: Mold's Hold on the Insurance Industry*, 34 ST. MARY'S L.J. 541 (2003) (discussing the effect of mold litigation on the insurance industry).

6. *See, e.g.*, David Pierson, *Mold May Force Razing of Planned High School Site*, L.A. TIMES, Oct. 1, 2002, at B3; WRAL.com, *Mold Forces Closure of Durham Building*, *at* http://www.wral.com/news/2459430/detail.html (Sept. 5, 2003) (describing closure of community center in Durham, N.C.) (on file with the North Carolina Law Review).

7. *See* Rich, *supra* note 5.

allegations involving exotic species of molds that release toxins into the air have piqued the interest of the media and the public. Great fanfare has accompanied litigation involving famous celebrities pitted against insurance companies and homebuilders.[8] Eight-figure awards have been realized in cases involving homeowners and workers allegedly injured by toxic molds.[9]

Despite an absence of high-profile litigation, concern about the public health impact of molds has been a concern in North Carolina. Recent natural disasters such as Hurricane Floyd, which produced devastating floods in eastern North Carolina, created ideal moisture-rich conditions for mold proliferation.[10] North Carolina Central University in Durham experienced a mold infestation that led to the closing of several dormitories and a major university library;[11] remediation costs have been estimated as high as $26 million.[12] Similar conditions have arisen at UNC-Pembroke and Duke University.[13] Mold has also forced the closing of numerous county schools.[14] Even where the mold is of an innocuous variety, the heightened concern over children's exposures and liability has led

8. For example, Ed McMahon, of television fame, received a multimillion dollar settlement after he and his wife were driven from their California home by a mold infestation so dangerous it allegedly led to the death of their dog, Muffin. Jean Guccione, *Ed McMahon Settles Suit over Mold for $7.2 Million*, L.A. TIMES, May 9, 2003, at B1. Erin Brokovich was also involved in litigation against the builder of her home. Andrew LePage, *Activist's New Fight Hits Home*, SACRAMENTO BEE (California), Mar. 8, 2001, at D1.

9. *See* Centex-Rooney Constr. Co. v. Martin County, 706 So. 2d 20, 25 (Fla. Dist. Ct. App. 1997) (per curiam) (affirming award of $14 million to a county in a breach of contract lawsuit alleging that faulty design and construction of a county courthouse led to an infestation of mold that sickened county employees); Allison v. Fire Ins. Exch., 98 S.W.3d 227, 237 (Tex. App. 2002) (describing the now infamous $33 million jury award (much of which was reduced on appeal) won by Melinda Ballard and husband Ronald Allison, of Dripping Springs, Texas, in their lawsuit against the insurer of their mansion for failure to respond adequately to a toxic mold outbreak).

10. *See* Press Release, North Carolina Department of Health and Human Services, Public Health Officials Warn of Flooding's Delayed Dangers (Sept. 22, 1999), http://www.dhhs.state.nc.us/pressrel/9-22-99b.htm (on file with the North Carolina Law Review).

11. *See* Vicki Cheng, *Mold Plagues NCCU Library*, NEWS & OBSERVER (Raleigh, N.C.), Aug. 13, 2003, at 1A.

12. *See* Jane Stancill, *UNC Board Wants $29 Million for Mold*, NEWS & OBSERVER (Raleigh, N.C.), Nov. 14, 2003, at 3B.

13. *See id.*; *Mold on Books Closes Two Duke Library Floors*, NEWS & RECORD (Greensboro, N.C.), Nov. 2, 2002, High Point Edition, at B2.

14. *See* WRAL.com, *Cumberland County Health Building, School Affected by Mold, at* http://www.wral.com/news/2662743/detail.html (Nov. 25, 2003) (on file with the North Carolina Law Review); WRAL.com, *Mold Problems Show Up at Bunn High School, at* http://www.wral.com/news/2526887/detail.html (Oct. 2, 2003) (on file with the North Carolina Law Review).

school officials to take an expensive, but prudent, better-safe-than-sorry approach.[15]

This Comment focuses on the courts' treatment of mold personal injury claims that are a frequent component of mold lawsuits. Much speculation has occurred over the financial impact of these claims, with many drawing comparisons to the archetypal modern-day toxic tort action, the asbestos lawsuit.[16] The conclusion among practitioners and academics is that mold injury claims are inherently deficient because mold cannot be linked to specific adverse health outcomes at identifiable exposure levels.[17] And yet, a significant number of lawsuits alleging mold-related injuries have reached a successful conclusion for plaintiffs.

This Comment delves into the scientific merit of mold injury claims and focuses on the treatment of causation evidence in recent state and federal court decisions. To date, courts have exhibited widely divergent reactions to such evidence, due in part to varying tests of admissibility for scientific evidence, but also due to a growing divide between the courts on the relevance and admissibility of certain types of medical causation evidence, including differential diagnosis. Much of the unease is due to the generality of symptoms linked to non-toxigenic mold species and scientific uncertainty surrounding the health effects of toxigenic molds. These are valid concerns when determining whether a party to a toxic tort action has met the burden of proving causation. However, recent court opinions suggest that in their zeal to limit causation evidence, courts are overreaching by assigning relevance to the absence of mold exposure "standards."

Risk management exposure standards developed by governmental and regulatory agencies are not an appropriate proxy

15. *Mold Problems Show Up at Bunn High School*, *supra* note 14 (quoting local school official after discovery of potentially toxic mold: "Mold isn't something to play with.").

16. *See, e.g.*, Thelma Jarman-Felstiner, *Mold Is Gold: But, Will It Be the Next Asbestos?*, 30 PEPP. L. REV. 529, 551–52 (2003) (concluding that mold-related personal injury claims likely will never reach the same magnitude as that seen in asbestos litigation); Elizabeth L. Perry, Comment, *Why Fear the Fungus?: Why Toxic Mold Is and Is Not the Next Big Toxic Tort*, 52 BUFF. L. REV. 257, 260 (2004) ("Due to the extremely attenuated link between toxic mold growth and severe personal injury, it is unlikely that mold litigation will reach the same level as that of notorious toxic torts like asbestos and that of lead paint.").

17. *See, e.g.*, Jarman-Felstiner, *supra* note 16, at 552 ("Unless more conclusive scientific evidence is discovered to link toxic mold with specific, identifiable health problems, the personal injury component of all mold claims will probably never reach the level of asbestos litigation.").

for scientifically documented threshold health effect levels in the causation analysis. Risk management standards are not held to the same standard of proof as evidence of causation, and they incorporate additional considerations such as costs, technical feasibility, and legislatively mandated safety factors. Empirically-based threshold health effect values, in contrast, are derived directly from scientific data, and thus provide more defensible evidence of causation. As legislatures strive to develop the mold exposure "standards" clamored for by the public and legal commentators, there is a distinct risk that such guidelines will be misapplied by courts struggling to identify general or specific causation in individual cases. Recent decisions suggest that courts making complex evidentiary decisions may be on the verge of doing just that.

Part I of this Comment presents background information on molds, including toxigenic species such as *Stachybotrys chartarum*. Part II provides a brief summary of mold litigation in the United States, including an overview of a typical mold personal injury claim. Part III examines how courts historically have dealt with personal injury causation evidence in toxic tort cases generally. Part IV discusses how the courts have reacted to such evidence in mold-related litigation, and compares the courts' handling of mold-related toxic tort claims to decisions involving other toxicants, focusing on the courts' analyses of differential diagnosis evidence. Part IV also focuses on the recent interest expressed by commentators and the courts in "acceptable" exposure standards for mold, and highlights the risks presented by relying on such information in legal causation analysis.

I. MOLD AND HUMAN HEALTH

The molds are a ubiquitous and essential component of our planet's biosphere that contribute to the orderly transformation of dead and dying life forms into the simplest building blocks of life.[18] Molds are pervasive in both indoor and outdoor environments and can survive under an astonishing range of environmental conditions.[19]

18. *See* INST. OF MED., DAMP INDOOR SPACES AND HEALTH 4 (2004) [hereinafter IOM] (noting that molds consume dead and decaying organic matter), *available at* http://www.nap.edu/books/0309091934/html (on file with the North Carolina Law Review); Am. Coll. of Occupational & Envtl. Med., *Adverse Human Health Effects Associated with Molds in the Indoor Environment*, 45 J. OCCUPATIONAL & ENVTL. MED. 470, 470 (2004) [hereinafter ACOEM Statement].

19. IOM, *supra* note 18, at 4; ACOEM Statement, *supra* note 18, at 470.

Molds flourish indoors given appropriate levels of moisture.[20] When building structures become waterlogged during construction or flooding, or experience chronic moisture problems as a result of improper ventilation or construction deficiencies, mold colonies will frequently develop in living or working quarters.[21] There are over 100,000 species of mold worldwide and over 1,000 different species have been identified in homes in the United States.[22] Indeed, the prevalence of indoor molds was succinctly described by the Institute of Medicine: "no indoor space is free of them."[23]

The benefits of molds are many,[24] but molds have also long been known to cause adverse health effects in humans ranging from allergic reactions[25] to infections[26] and overt toxicity, including

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20. *See* D.M. Kuhn & M.A. Ghannoum, *Indoor Mold, Toxigenic Fungi, and* Stachybotrys chartarum*: Infectious Disease Perspective*, 16 CLINICAL MICROBIOLOGY REV. 144, 145 (2003). Some species appear in virtually all damp indoor environments. *See id.* (tabulating results from several indoor air studies). *Penicillium* molds were found in ninety-six percent of damp buildings in one study of molds in domestic buildings. *Id.* at 145. Other species appear less frequently. *Id.* at 146 (reporting that in most studies the toxigenic species *Stachybotrys* was found in less than three percent of dwellings). Some commentators have suggested that the nutritional requirements for *Stachybotrys* growth are not met by some common culturing techniques, leading to underreporting of prevalence. *See id.* (theorizing that this phenomenon could explain underreporting in early mold studies). Molds also multiply quickly under less humid conditions if provided with warmer temperatures and suitable food sources. *Id.* at 145.

21. Occupational Safety and Health Admin., *A Brief Guide to Mold in the Workplace* [hereinafter OSHA] (explaining that with adequate ventilation and low moisture levels, the establishment and growth of mold colonies can easily be avoided), *at* http://www.osha.gov/dts/shib/shib101003.html (last visited Nov. 15, 2004) (on file with the North Carolina Law Review).

22. *See Mold: A Growing Problem: Joint Hearing Before the House Subcomm. on Oversight and Investigations and the Subcomm. on Hous. and Cmty. Opportunity of the Comm. on Fin. Servs.*, 107th Cong. 57 (2002) (statement of Stephen C. Redd, M.D., Chief, Air Pollution and Respiratory Health Branch, National Center for Environmental Health, Centers for Disease Control and Prevention) [hereinafter Redd] (Sup. Docs. No. Y4.F49/20:107-77), *available at* http://financialservices.house.gov/media/pdf/107-77.pdf (on file with the North Carolina Law Review); OSHA, *supra* note 21.

23. IOM, *supra* note 18, at 4.

24. *See, e.g.*, Nobelprize.org, *The Discovery of Penicillin*, *at* http://www.nobel.se/medicine/educational/penicillin/readmore.html (last visited Nov. 15, 2004) (describing Alexander Fleming's surreptitious discovery that *Penicillium notatum* mold secreted an antibacterial substance leading to the development of the antibiotic penicillin, which has saved millions of lives) (on file with the North Carolina Law Review).

25. *See* ACOEM Statement, *supra* note 18, at 470–72 (describing allergic and other hypersensitivity reactions to indoor molds).

26. Life-threatening fungal infections arising from mold exposure are observed in a limited number of circumstances. *See* IOM, *supra* note 18, at 235–37. These infections primarily affect people with weakened immune systems, such as cancer patients receiving chemotherapy and persons suffering from AIDS. *See* ACOEM Statement, *supra* note 18, at 472. More common infections include superficial fungal infections of the skin and nails,

cancer.[27] Allergic reactions to mold are the most commonly observed health effect. Approximately ten percent of the human population has allergic antibodies that will specifically recognize and react to the presence of mold proteins.[28] Exposure occurs when humans inhale airborne mold spores or hyphal fragments, which deposit on the surface of the airways, often triggering an allergic response.[29] Typically, allergic reactions are relatively benign and manifest as sinus irritation, hay fever, or asthma.[30] However, more serious allergic effects have been documented at elevated exposure levels[31] or in highly sensitized individuals exposed to moderate mold levels.[32] Molds are the most important biological contributor to "sick building syndrome," which describes the symptoms suffered by individuals in unhealthful indoor work or home environments.[33] A recent Mayo Clinic study concluded that the chronic sinus problems experienced by thirty-seven million Americans are largely the result of exposure to molds.[34]

There is an entire subset of health effects that arise from exposure to chemical toxins, known as mycotoxins, released by

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which are widely prevalent in the human population. *Id.* at 473. The CDC has estimated that up to nine percent of hospital-acquired infections are caused by fungi. *See* Redd, *supra* note 22, at 8.

27. Toxic endpoints, including respiratory tissue damage and nervous system impairment, have been observed in humans and animals exposed to toxigenic mold species. *See* IOM, *supra* note 18, at 125–82. Some mold toxins have even been linked to the development of human cancer. Redd, *supra* note 22, at 59 (referring to the carcinogenic mycotoxins aflatoxin (from peanut molds) and ochratoxin A (from species of *Penicillium* and *Aspergillus* found in cereal grains)); IOM, *supra* note 18, at 166–70.

28. *See* ACOEM Statement, *supra* note 18, at 471. It is further estimated that perhaps one-half of these persons will have experienced allergic symptoms arising from mold exposure. *Id.*

29. *Id.* at 471; IOM, *supra* note 18, at 92. Hyphal fragments are microscopic filaments comprised of the fungal cells that make up mold colonies. ACOEM Statement, *supra* note 18, at 470.

30. *See* ACOEM Statement, *supra* note 18, at 470–72.

31. *See id.* (describing "[a] rare, but much more serious immune-related condition, hypersensitivity pneumonitis" and other even more rare conditions including allergic bronchopulmonary aspergillosis and allergic fungal sinusitis).

32. *See id.* at 471 (explaining that an individual's "profile of allergic sensitivity" influences an individual's allergic airway disease).

33. *See* U.S. Envtl. Prot. Agency, *Children's Health Initiative: Toxic Mold*, *at* http://www.epa.gov/appcdwww/iemb/child.htm (last visited Nov. 15, 2004) (on file with the North Carolina Law Review). Sick building syndrome is an umbrella term that describes "a combination of nonspecific symptoms related to residence or work in a particular building." IOM, *supra* note 18, at 250. Symptoms most frequently include fatigue, headache, and irritation of the eyes, nose, or throat; however, studies describing sick building syndrome often vary in the requirements for the nature of symptoms. *Id.*

34. *See* Seema Mehta, *Mold Quickly Spreads as Health and Legal Issue*, L.A. TIMES, Feb. 4, 2001, at B3.

toxigenic species of mold.[35] These mycotoxins, which may have evolved as a defense mechanism to combat other microorganisms, insects, or animals,[36] rank among the most potent toxins known to man.[37] Human exposure occurs following ingestion of moldy foods, direct contact with the skin, or inhalation exposure to dust, mold spores, or hyphal fragments carrying mycotoxins.[38] A small number of occupational diseases have been linked to inhalation exposure to high levels of airborne toxigenic molds, primarily in industrial and agricultural occupations.[39]

Most of the recent research into mold and mycotoxin exposure has focused on inhalation exposures to mold in the indoor environment.[40] Numerous studies have suggested an association between home dampness and respiratory complaints,[41] although the causal relationship behind these associations remains unclear.[42] In recent years, a small number of controversial studies linked indoor

35. *See* Kuhn & Ghannoum, *supra* note 20, at 149–55. Mycotoxins are metabolites that are most frequently produced under "suboptimal growth conditions." *Id.* at 150.

36. *See* Ruth A. Etzel, *Mycotoxins*, 287 JAMA 425, 425 (2002).

37. For example, *aflatoxin*, produced by the peanut mold *Aspergillus flavus*, is one of only a handful of chemicals which has been definitively linked to human cancer. Populations with significant exposure to aflatoxin suffer from unusually high levels of hemangiosarcoma, a form of liver cancer. For a thorough account of the discovery and characterization of aflatoxin, see JOSEPH A. RODRICKS, CALCULATED RISKS: UNDERSTANDING THE TOXICITY AND HUMAN HEALTH RISKS OF CHEMICALS IN OUR ENVIRONMENT xvii–xxv (1994).

38. ACOEM Statement, *supra* note 18, at 473. Several notable disease outbreaks in human populations have been attributed to the consumption of foods contaminated by molds and mold toxins. *See* Kuhn & Ghannoum, *supra* note 20, at 150 (describing an outbreak of disease in the Middle Ages involving joint pain, hallucinations, and gangrene that arose from consumption of moldy rye and came to be known as "St. Anthony's Fire").

39. *See* Redd, *supra* note 22, at 59–60 (describing a variety of conditions including farmer's lung, malt worker's lung, and respiratory tract injuries caused by exposure to moldy hay and barley dust containing mold spores).

40. *See generally* IOM, *supra* note 18, at 90–110 (discussing methods for assessing exposure to indoor agents, including molds and mycotoxins).

41. *See* Bert Brunekreef et al., *Home Dampness and Respiratory Morbidity in Children*, 140 AM. REV. RESPIRATORY DISEASE 1363, 1363 (1989); Robert E. Dales et al., *Respiratory Health Effects of Home Dampness and Molds Among Canadian Children*, 134 AM. J. EPIDEMIOLOGY 196, 196 (1991); *see also* IOM, *supra* note 18, at 183–269 (assessing numerous studies purporting to show a link between damp indoor spaces and adverse health outcomes); Kuhn & Ghannoum, *supra* note 20, at 145 (summarizing several recent studies of indoor environments).

42. Kuhn & Ghannoum, *supra* note 20, at 145, 149 (explaining that concomitant exposure to volatile organic chemicals, bacteria, and other allergens, including dust mites, confounds the establishment of a clear causal connection and noting that most authors reporting ill effects related to *Stachybotrys* exposure are reporting "associations rather than proof of causation").

toxigenic molds, including *Stachybotrys chartarum*, to illnesses in workers[43] and pulmonary hemorrhage in infants.[44] These studies alarmed the public health community[45] and triggered investigations by several government agencies.[46] Generally, independent review bodies have backed away from the most troubling of the studies' conclusions, advising government agencies to adopt the position that the current science does not support a causal link between exposure to *Stachybotrys* and lung injury in infants.[47]

Nevertheless, public anxiety about *Stachybotrys* and other so-called "toxic molds" exploded in the years following release of the earliest toxic mold studies.[48] Suddenly, indoor molds became the cause of an increasingly terrifying laundry list of adverse health effects.[49] High-profile litigation, particularly involving celebrity plaintiffs, was covered widely in the popular press.[50] Several cases ended with enormous payouts to plaintiffs complaining of brain damage and serious respiratory damage stemming from residential toxic mold exposure.[51]

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43. *See* Eckardt Johanning et al., *Health and Immunology Study Following Exposure to Toxigenic Fungi (*Stachybotrys chartarum*) in a Water-Damaged Office Environment*, 68 INT'L ARCHIVES OCCUPATIONAL & ENVTL. HEALTH 207, 207–08 (1996).

44. Ctr. for Disease Control and Prevention, *Update: Pulmonary Hemorrhage/Hemosiderosis Among Infants—Cleveland, Ohio, 1993–1996*, 46 MMWR: MORBIDITY & MORTALITY WKLY. REP. 33–35 (1997).

45. *See* Frederick Fung et al., Stachybotrys*: A Mycotoxin-Producing Fungus of Increasing Toxicologic Importance*, 36 CLINICAL TOXICOLOGY 79, 84 (1998).

46. *See, e.g.*, Redd, *supra* note 22, at 8–9 (describing the CDC's efforts to understand the health effects of toxigenic molds); CDC WORKING GROUP ON PULMONARY HEMOSIDEROSIS, REPORT OF CDC WORKING GROUP ON PULMONARY HEMORRHAGE/HEMOSIDEROSIS (1999) [hereinafter CDC] (reviewing epidemiological studies and advising the CDC to address unresolved questions about the Chicago and Cleveland infant studies), *available at* http://www.cdc.gov/nceh/airpollution/mold/hemorrhage_report.pdf (on file with the North Carolina Law Review). The U.S. Environmental Protection Agency is also working towards defining the optimal growth conditions for molds in the indoor environment. *See* U.S. Envtl. Prot. Agency, *supra* note 33.

47. *See* CDC, *supra* note 46, at 5 ("CDC should adopt the official position that the epidemiologic evidence does not provide strong support for the association of *S. atra* or other toxigenic fungi with [pulmonary hemorrhage and related conditions in infants]."). The Institute of Medicine concluded that the role of *Stachybotrys* in the Cleveland cluster is "controversial" and that available studies provide "inadequate or insufficient information" to determine whether an association exists between acute idiopathic pulmonary hemorrhage and *Stachybotrys* in the indoor environment. IOM, *supra* note 18, at 242–43.

48. *See* Anita Hamilton, *Beware: Toxic Mold*, TIME, July 2, 2001, at 54, 54–55.

49. *See id.*

50. *See, e.g.*, Guccione, *supra* note 8 (describing lawsuits involving Ed McMahon and Erin Brokovich).

51. *See id.* (describing multimillion dollar verdicts won in Texas and California mold

As mold spread across the legal landscape of the late 1990s and early 2000s, researchers have continued to pursue evidence of a causal relationship between mold exposure and adverse health outcomes. Recent studies have greatly advanced our knowledge of human health effects associated with exposure to indoor molds, routes of human exposure, sampling methods for molds, and the pervasiveness of the most suspect toxigenic molds, especially *Stachybotrys*.[52] Many scientists have distanced themselves from the most sensational claims regarding exposure to toxigenic molds.[53] Nevertheless, indoor molds are considered responsible for a wide array of adverse health outcomes, many of which are general in nature and difficult to trace back to a single causative agent.[54] The wide range of symptoms and uncertainty concerning ill effects from toxigenic molds present a daunting challenge for scientists and legal professionals striving to establish a causative link between exposure to toxigenic mold and human illness.

II. THE RECENT UPSURGE IN MOLD LITIGATION

As many as ten thousand mold-related lawsuits have been filed in the United States within the last decade,[55] a huge increase over the period prior to the mid-1990s when lawsuits involving mold appeared only sporadically.[56] Theories for the recent surge in lawsuits include a

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lawsuits). The Texas litigation included allegations that toxic mold exposure caused toxic encephalopathy, a brain disease. Allison v. Fire Ins. Exch., 98 S.W.3d 227, 240 (Tex. App. 2002).

52. *See* IOM, *supra* note 18, at 183–269 (reporting findings from numerous recent scientific publications); Kuhn & Ghannoum, *supra* note 20, at 149–50 (summarizing recent developments in health effects research for indoor toxigenic molds such as *Stachybotrys*).

53. *See, e.g.*, ACOEM Statement, *supra* note 18, at 476 ("Current scientific evidence does not support the proposition that human health has been adversely affected by inhaled mycotoxins . . . .").

54. *See supra* notes 28–34 and accompanying text.

55. Barbara Anderson, *Risks, Lawsuits Linked to Household Mold Are Growing*, FRESNO BEE (California), Jan. 3, 2004, http://www.modbee.com/local/story/7959687p-8833528c.html (on file with the North Carolina Law Review).

56. An electronic search of state and federal case law from prior to 1995 reveals a relatively small number of cases; most of these claims did not specifically allege injurious exposure to toxigenic molds. *See, e.g.*, Miller v. Lakeside Vill. Condo. Ass'n, 2 Cal. Rptr. 2d 796, 798 (Cal. Ct. App. 1991) (alleging that condominium association's negligent failure to repair and maintain plumbing system led to tenant's exposure to mold, causing asthma symptoms); Komatsu v. Bd. of Trs., 693 P.2d 405, 412 (Haw. 1984) (awarding disability benefits to employee suffering asthmatic bronchitis as a result of exposure to mold emitted from a defective air conditioning system); Washington Courte Condo. Ass'n v. Washington-Golf Corp., 501 N.E.2d 1290, 1293 (Ill. App. Ct. 1986) (alleging negligent installation of windows and exterior doors led to allergic reaction in child exposed to resulting mold infestation). One of the first appellate court decisions involving a toxigenic

greater understanding of the causal connection between molds and adverse health outcomes,[57] greater actual exposure resulting in part from new construction materials and standards,[58] greater attention being paid to the issue by the public through mass media consumption,[59] and opportunistic plaintiffs' lawyers.[60]

A. *Mold-related Causes of Action*

Although toxic mold-related lawsuits are a relatively new phenomenon, the claims being brought are generally founded on well-settled law.[61] Many of these lawsuits are filed by tenants, office workers, or homebuyers against landlords, construction companies, or

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mold species was *Centex-Rooney Construction Co. v. Martin County*, 706 So. 2d 20 (Fla. Dist. Ct. App. 1997) (per curiam). The court in *Centex-Rooney* affirmed the trial court's award to Martin County in a lawsuit for breach of contract regarding a courthouse that became infested with numerous mold species. *Id.* at 29.

57. Anderson, *supra* note 55 (noting the position of lawyers and advocates for homeowners that more lawsuits are being filed because people are better educated about mold risks).

58. *See id.* (suggesting that new home construction practices used today create optimal conditions for mold growth). The role of construction practices or energy efficient construction standards is much more controversial. Some have asserted that airtight houses prevent the ventilation that is key to reducing moisture buildup and mold growth. *See* Deborah K. Dietsch, *Exorcising a Mold Monster*, WASH. POST, Oct. 26, 2002, at H1 (contrasting a remediation company executive's view that energy-efficient homes are an "ideal environment for mold" with those of a homebuilders' association representative that "there is no clear evidence that newer homes have more mold than older homes"). Others have pointed to newer, inexpensive construction materials as a contributing factor. For example, cellulose insulation materials and plaster board used in newer homes retain moisture and may contribute to mold growth. Anderson, *supra* note 55; *see also* Kuhn & Ghannoum, *supra* note 20, at 146 (suggesting that *Stachybotrys*'s "fondness" for cellulose may explain its appearance in buildings). It has even been suggested that the residential building boom of the 1990s contributed to the problem as builders were hastily assembling houses with materials that became wet at the construction site. Rich, *supra* note 5. Still, assigning all the blame on energy efficient home construction standards is unsupportable, since these standards have been in place since the energy crisis of the 1970s, see Wright & Irby, *supra* note 5, at 309–10, while the exponential rise in lawsuits and insurance claims has been confined to the past five to ten years. *See* Rich, *supra* note 5 (noting that mold-related insurance payouts rose from near zero in 1999 to $3 billion by 2002).

59. *See* Mary Umberger, *The Star that Upstaged the Economy*, CHI. TRIB., Jan. 13, 2002, § 16, at 1 (describing how insurance industry representatives blame a "coordinated media campaign" for public concern about indoor molds).

60. *See* Anderson, *supra* note 55 ("[L]awyers for builders, subcontractors and landlords say modern construction isn't at fault. They refer to a 'mold is gold' phenomenon, claiming publicity about multimillion-dollar settlements is an incentive for people to sue."); *see also* Thomas Grillo, *After 8 Years, a Milestone in Battle over Mold*, BOSTON GLOBE, Nov. 25, 2003, at A1 ("[O]pponents of toxic mold litigation say the claims stem from greedy lawyers looking for the next cash cow as asbestos lawsuits diminish.").

61. Erin Masson Wirth, Annotation, *Toxic Mold in Residences and Other Buildings: Liability and Other Issues*, 114 A.L.R.5th 397, 408–09 (2003).

home sellers. Claims include breach of contract, breach of duty of good faith and fair dealing, failure to warn, negligence, unfair and deceptive trade practices, and breach of implied warranty of habitability.[62] A typical claim involves an allegation that negligent construction practices or improper maintenance of a rental unit resulted in a mold infestation that sickened residents.[63]

The largest number of mold-related lawsuits have been directed against homeowners' insurance companies for refusing to recognize mold-related insurance claims.[64] Many of these lawsuits incorporate claims of bad faith, which permit recovery of punitive damages.[65] Juries have handed down multimillion dollar awards in lawsuits against insurance companies,[66] attracting the attention of the

62. *Id. See, e.g.*, RCDI Constr., Inc. v. Space/Architecture Planning & Interiors, P.A., No. 01-1676, 2002 U.S. App. LEXIS 640, at *3–*6 (4th Cir. Jan. 15, 2002) (per curiam) (upholding dismissal of unfair trade practices claim filed against architectural firm that advised owners of mold-infested building to terminate their contractors); Roche v. Lincoln Prop. Co., 278 F. Supp. 2d 744, 746 (E.D. Va. 2003) (claiming landlord negligently failed to maintain apartment), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004); Caldwell v. Curioni, 125 S.W.3d 784, 788 (Tex. App. 2004) (reversing summary judgment for landlord on claim of breach of duty to inform tenants of mold infestation); Gifford v. Matejka, No. 25886-2-II, 2001 Wash. App. LEXIS 1560, at *3 (Wash. Ct. App. July 20, 2001) (involving allegation that landlord's agent failed to represent the true condition of the residence).

63. For example, in 1997 the Florida District Court of Appeal affirmed a $14 million judgment against a construction company for breach of contract in design and construction of a county courthouse. Centex-Rooney Constr. Co. v. Martin County, 706 So. 2d 20, 29 (Fla. Dist. Ct. App. 1997) (per curiam). Window and exterior wall leaks led to an infestation of toxigenic mold that sickened numerous court employees, including several judges. *Id.* at 24. The court found that the plaintiffs successfully "proved that Centex's construction defects caused moisture problems in the buildings, resulting in extensive mold growth." *Id.* at 25. The county was awarded over $11 million in damages. *Id.* at 28.

64. *See* Umberger, *supra* note 59 (citing insurance industry statistics estimating that approximately half of the recent lawsuits have been directed against insurance companies).

65. *See id.*; R.J. Maniloff, *Mold: The Hysteria Among Us: Exposure to Mold Causes Bad Faith Claims Against Insurers*, 16 MEALEY'S LITIG. REP.: INS. BAD FAITH, No. 13, Nov. 6, 2002, at 1. In the typical mold-related bad faith claim, a homeowner alleges that her homeowner's insurance company refused to repair a covered defect in the home that contributed to mold proliferation, or refused to repair mold-related damage to the home. The elements of a typical bad faith claim require that the insured prove that the insurer "acted in bad faith by refusing to settle or negotiate with the plaintiff and that the insurers' [sic] actions have been a misuse of power and authority tantamount to outrageous conduct reflecting a reckless and wanton disregard of the plaintiff's rights under the insurance policy." Johnson v. First Union Corp., 128 N.C. App. 450, 457, 496 S.E.2d 1, 6 (1998) (reciting the elements of a bad faith claim in North Carolina).

66. *See* Allison v. Fire Ins. Exch., 98 S.W.3d 227, 234–37 (Tex. App. 2002) (discussing the jury's award of over $33 million against an insurance company).

plaintiffs' bar and sending a shudder through the insurance industry.[67]

Perhaps the most infamous of the mold-related bad-faith claims (and arguably the most infamous mold-related case bar none) is *Allison v. Fire Insurance Exchange*.[68] In *Allison*, Melinda Ballard and husband Ronald Allison, of Dripping Springs, Texas, suffered a series of leaks that led to extensive water damage inside their home.[69] After filing several claims against their insurance provider, they eventually resorted to filing a lawsuit, alleging, *inter alia*, breach of contract and breach of duty of good faith.[70] The jury eventually awarded the couple over $33 million, including $12 million in punitive damages.[71] On review, the Texas Court of Appeals considered several of the alleged acts, including specific instances of misrepresentation,[72] unnecessary extensions of time to complete the claim investigation,[73] refusal of prompt payment, and fraudulent bids.[74] Citing evidence of the claim processor's lack of authority and experience and the extension of the claim investigation well beyond the determination of liability, the court held there was sufficient evidence to support the jury's finding of bad faith.[75] Notably, however, the court reversed the punitive damages award,[76] finding no basis for claims of fraud, unconscionable conduct or involvement in deceptive business practices.[77]

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67. *See* Oster, *supra* note 5.

68. 98 S.W.3d 227 (Tex. App. 2002).

69. *Id.* at 234–37 (describing the background of the case).

70. *Id.* at 236.

71. *Id.* at 237.

72. *See id.* at 245 (reviewing the allegation that insurance company representatives misrepresented that complete plumbing tests were performed).

73. *Id.*

74. *Id.*

75. *Id.* at 250.

76. *Id.* at 258.

77. *Id.* at 257–58. Insurance companies have enacted measures to limit their potential liability from mold-related claims, such as refusing to issue policies on homes that have suffered recent water damage and dropping policy holders after a single water damage claim. Oster, *supra* note 5. In some states, the insurance lobby has successfully pushed for legislative limits on mold-related damages. *See Mold Problems May Cause Insurance Rates To Rise in N.C.*, *supra* note 5 (quoting North Carolina Rate Bureau officials and citing a $5,000 cap for homeowners' insurance claims related to mold). Still, payouts are expected to continue into the foreseeable future as insurance companies, builders, and property owners battle over who should take responsibility for mold-related property damage. *See* Rich, *supra* note 5. The above cases only scratch the surface of the realm of mold-related property law claims. For recent comprehensive reviews on the matter of liability between insurance companies, builders, and homeowners, see generally Wright & Irby, *supra* note 5 (discussing the effect of mold on real estate transactions and potential means for addressing those challenges); Mike Bischoff, Comment, *Theories of Toxic Mold Liability Facing Arizona Homebuilders*, 34 ARIZ. ST. L.J. 681 (2002).

B. *Mold Personal Injury Claims*

Although Melinda Ballard prevailed on several claims against her insurance carrier, her husband, Ronald Allison, ultimately failed to convince the court on the merit of his personal injury claims.[78] His claims were certainly extraordinary. Allison contended that his exposure to *Stachybotrys* caused a host of ailments, including toxic encephalopathy—a degenerative disease of the brain.[79] However, prior to trial the district court granted the defendant's motion to exclude expert testimony that linked Allison's condition to *Stachybotrys* exposure,[80] and subsequently granted partial summary judgment to the defendants on these claims for lack of evidence.[81] These decisions were ultimately upheld on appeal.[82]

Allison illustrates a frequently encountered outcome in mold-related litigation: success on claims involving contract or property law principles, and a lack of success on personal injury claims.[83] Mold plaintiffs face substantial challenges in bringing forth personal injury claims. Courts insist on specificity and reliability of evidence linking mold exposure to health effects, and yet very few agents give rise to a greater range of health effects than the molds. The claims reflect this diversity: Ed McMahon complained of chronic coughing, sneezing, and congestion;[84] Erin Brokovich's lawsuit against the builder and former owner of her home claimed exposure to mold caused respiratory ailments, facial rashes, sinus infections, and frequent headaches.[85] Many other cases involve complaints of allergic reactions, including aggravation of asthma, hypersensitivity to odors, and difficulty breathing.[86] Invariably, additional and more general

78. *Allison*, 98 S.W.3d at 240.

79. *See id.* at 239.

80. *Id.* at 237 (describing the district court's exclusion of testimony on the grounds that "[Allison's] expert witnesses did not have reliable epidemiological studies about the health effects of exposure to mold").

81. *Id.* at 240. The evidence that Allison submitted as proof of causation as to his conditions is discussed in Part IV.A., *infra*.

82. *Id.* at 239–40.

83. *See* Grillo, *supra* note 60 (quoting the editor of *Massachusetts Lawyer Weekly*: "It's easy to prove that mold messed up your home but a lot harder to prove it messed up your body.").

84. Guccione, *supra* note 8 (describing the events leading up to the McMahon's lawsuit against their homeowner's insurance provider).

85. LePage, *supra* note 8.

86. *See, e.g.*, Roche v. Lincoln Prop. Co., 278 F. Supp. 2d 744, 746 (E.D. Va. 2003) (listing hypersensitivity to smells and shortness of breath among their symptoms), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004); Miller v. Lakeside Vill. Condo. Ass'n, 2 Cal. Rptr. 2d 796, 798 (Cal. Ct. App. 1991) (alleging severe allergic

symptoms such as fatigue, headaches, and body pain appear.[87] In one case, a plaintiff even claimed that exposure to mold was a "promoter and substantial contributing factor" to the development of cancer following concomitant exposure to asbestos.[88]

More serious health effects have been attributed to the toxigenic molds. Allison complained that exposure to *Stachybotrys* caused neurological impairment including memory loss, inability to concentrate, and toxic encephalopathy.[89] Similar symptoms were described by the plaintiffs in *Roche v. Lincoln Property, Co.*,[90] whose apartment was found to contain several molds, including *Stachybotrys*.[91] In *Caldwell v. Curioni*,[92] the plaintiffs experienced headaches, fever, diarrhea, severe nausea, and vomiting within a week of moving into a house contaminated with *Stachybotrys*.[93]

The personal injury claims are an important part of the plaintiff's case because they can give rise to large damage awards.[94] But thus far, plaintiffs have had mixed results with mold-related personal injury claims. The divergent outcomes observed in these cases reflect the complexity of the allegations, the effect of evolving scientific evidence, and the trepidation of courts making difficult determinations regarding evidentiary admissibility.

III. TOXIC TORTS AND THE ADMISSIBILITY OF CAUSATION EVIDENCE

Toxic mold personal injury claims are simply a unique form of

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reactions and asthma attacks).

87. *See, e.g.*, New Haverford P'ship v. Stroot, 772 A.2d 792, 796 (Del. Super. Ct. 2001) (noting claims of these symptoms in addition to asthma attacks and other allergic reactions including sinus problems).

88. *See* Watters v. Dep't of Soc. Servs., 849 So. 2d 724, 731–33 (La. Ct. App. 2003) (finding genuine issue of material fact in the plaintiff's expert testimony that mold exposure compromised his immune system, weakening his defenses against malignancy).

89. Allison v. Fire Ins. Exch., 98 S.W.3d 227, 239 (Tex. App. 2002).

90. 278 F. Supp. 2d 744, 746 (E.D. Va. 2003), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004).

91. *Id.* at 755.

92. 125 S.W.3d 784, 788 (Tex. App. 2004).

93. *Id.* at 788.

94. For example, in *New Haven Partnership v. Stroot*, 772 A.2d 792 (Del. Super. Ct. 2001), a jury awarded the plaintiffs over $1 million for medical expenses, permanent impairment, and pain and suffering. *Id.* at 801. More recently, a condominium owner was awarded $285,000 for flu-like symptoms arising from a mold infestation caused by her landlord's negligent failure to repair a leak in the basement of her unit. Grillo, *supra* note 60 (reading the condominium owner's case as "send[ing] a message that it's possible to link" mold exposure to adverse health effects).

toxic tort action. In a typical toxic tort action, the plaintiff alleges that the defendant caused or threatened to cause the plaintiff to be exposed to a toxic agent, resulting in a compensable injury.[95] The burden of proving causation is a weighty one for the plaintiff. The plaintiff must show that the toxicant in question is capable of causing the injury complained of (general causation) and must further prove that the toxicant in fact did cause that injury in the present case (specific causation).[96] As we will see, there are often pointed disagreements over issues such as whether and to what extent the plaintiff was actually exposed, and what evidence exists to support the determination that the offending substance was in fact "toxic" at the plaintiff's level of exposure.[97] In addition, the plaintiff must also rule out other potential causes of the same injury.[98] Every link in the causal chain is susceptible to attack by the opposing party.[99]

A. Evidentiary Hurdles for the Toxic Tort Plaintiff

For environmental agents such as the molds, the plaintiff's burden of proving injurious exposure is often profoundly difficult. Movement of a toxicant from a source to a human receptor is nearly impossible to accurately predict given the overwhelming number of environmental variables to consider.[100] In addition, many toxic agents are metabolized and excreted quickly by the exposed individual, precluding any opportunity to provide definitive evidence of exposure through medical examinations.[101] Finally, the appearance of symptoms such as cancer and chronic organ toxicity may be delayed until many years following exposure.[102] This delay introduces numerous challenges for the plaintiff, including the possibility of intervening exposures and the need to reconstruct exposure from decades-old data of questionable quality.[103] Because exposures are

95. *See* JEAN MACCHIAROLI EGGEN, TOXIC TORTS IN A NUTSHELL 2 (2d ed. 2000).

96. In re Breast Implant Litig., 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998); *see* Stephen J. Henning & Daniel A. Berman, *A Mold Claims Primer: Microbial Contamination Issues*, 33 BRIEF 22, 31–32 (2003).

97. *See* Henning & Berman, *supra* note 96, at 31–32.

98. *See* SHEILA JASANOFF, SCIENCE AT THE BAR: LAW, SCIENCE, AND TECHNOLOGY IN AMERICA 119 (1995).

99. *See id.* (explaining that most attacks occur where there are "disagreements among relevant communities of experts").

100. *See* RODRICKS, *supra* note 37, at 12–24 (describing "a process of staggering complexity and beauty").

101. *See id.* at 25–37 (providing an overview of the process of absorption, distribution, metabolism, and excretion of chemicals by the human body).

102. *See* EGGEN, *supra* note 95, at 5–6.

103. *See id.* at 6–7.

frequently fleeting and unpredictable events, accurate quantification of risk is nearly impossible.[104] Exposure evidence is therefore highly susceptible to accusations of unreliability.

Proving the particular level of exposure at which an agent like mold would be expected to give rise to adverse health effects is also difficult. Most substances cannot be characterized as toxic per se because they only manifest toxicity above a certain threshold level of exposure.[105] Determining the relevant threshold health effect level is one of the most heated issues in toxic tort litigation. Disagreements may arise over the quality of data supporting the determination, the use of the data to extrapolate to "safe" or no-effect exposure levels, and even the relevance of a particular health outcome.[106] Toxicity assessment[107] commonly involves relying on toxicological data from animal studies.[108] Interpreting how such results should apply to the assessment of human health risk involves making several inferential leaps—all of which are subject to debate.[109] Epidemiological studies

104. *See* Elaine M. Faustman & Gilbert S. Omenn, *Risk Assessment*, *in* CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 83, 97 (Curtis D. Klaassen ed., 6th ed. 2001) (explaining that exposure assessment is the most uncertain parameter in risk assessment).

105. Paracelsus (1493–1591) is often credited with issuing the mantra of toxicology: "What is there that is not poison? All things are poison and nothing [is] without poison. Solely the dose determines that a thing is not a poison." David L. Eaton & Curtis D. Klaassen, *Principles of Toxicology*, *in* CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 11, 13 (Curtis D. Klaassen ed., 6th ed. 2001).

106. For example, not all experts will agree as to whether certain health outcomes can be characterized as evidence of toxicity. Some ailments, such as headaches, body pain, or nausea, incorporate elements of subjectiveness; toxicological endpoints generally consist of quantifiable endpoints such as tumor incidence or observable tissue damage.

107. Toxicity assessment is the component of the traditional risk assessment process which incorporates hazard identification (involving "a description of the specific forms of toxicity . . . that can be caused by a chemical and an evaluation of the conditions under which these forms of toxicity might appear in exposed humans") and dose-response assessment (an examination of the expected quantitative relationship between dose of exposure and toxicity in exposed humans). COMM. ON RISK ASSESSMENT OF HAZARDOUS AIR POLLUTANTS, NAT'L RESEARCH COUNCIL, SCIENCE AND JUDGMENT IN RISK ASSESSMENT 56 (1994). Combined with an exposure assessment, this information is used to characterize risks in the human population. *Id.* at 68.

108. *See generally* Faustman & Omenn, *supra* note 104, at 88–90 (describing how animal bioassay data is used in the hazard identification process).

109. *See id.* (discussing the limited utility of high-dose animal bioassays for determining risks at lower exposure levels); *see also* Henning & Berman, *supra* note 96, at 31–32 (explaining that animal studies are of questionable value in proving causation in court cases). *See generally* Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, *in* FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 401, 406–09 (2d ed. 2000) (describing the assumptions in extrapolating data from animal studies to assess human health risks), *available at* http://www.fjc.gov (on file with the North Carolina Law Review); Michael D. Green et al., *Reference Guide on Epidemiology*,

are also frequently utilized to determine potential risks to human health.[110] But while these studies are preferable in that they involve human exposures,[111] they are also subject to confounding exposures and bias in study design.[112] In addition, while epidemiology is powerful for identifying association, it is substantially weaker at establishing modes of toxic action or dose-response characteristics.[113] Plaintiffs that rely solely on epidemiologic studies are relegated to arguing their claims in probabilistic language leading to mixed results.[114]

Frequently, the plaintiff will introduce a medical expert to discuss scientific evidence supporting a particular theory of causation. In addition to discussing relevant toxicological and epidemiological studies, the medical expert may present the results of a differential diagnosis, "a standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated."[115] A differential diagnosis typically involves a physical examination, review of the patient's medical history, and consideration of results from various clinical tests conducted on the patient.[116] The medical professional will often discuss clinical evidence of disease and any relevant toxicological or epidemiological information that rules in the suspected agent as the cause of the plaintiff's injury. The expert may also discuss evidence that rules out other potential causes of the plaintiff's injury—an especially important consideration when the suspected agent is not associated with a signature disease.[117] By ruling out other potential causal factors, differential diagnosis has the potential to establish specific causation.

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in FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 345–47 (2d ed. 2000) (discussing the disadvantages of animal toxicity studies for assessing human health risks), *available at* http://www.fjc.gov (on file with the North Carolina Law Review).

110. *See* Faustman & Omenn, *supra* note 104, at 88.

111. *See id.*; *see also* Henning & Berman, *supra* note 96, at 31 (asserting that epidemiological research is the "preferred method" for establishing a link between toxic substances and human diseases).

112. *See* JASANOFF, *supra* note 98, at 120–21; Lora E. Fleming & Judy E. Bean, *Epidemiologic Issues in Occupational and Environmental Health*, *in* PRINCIPLES OF TOXICOLOGY: ENVIRONMENTAL AND INDUSTRIAL APPLICATIONS 511, 519–20 (Phillip L. Williams et al. eds., 2000).

113. *See* Fleming & Bean, *supra* note 112, at 511–21.

114. *See* EGGEN, *supra* note 95, at 8.

115. Westberry v. Gislaved Gummi AB, 178 F.3d 257, 262 (4th Cir. 1999).

116. *Id.*

117. Perhaps the most obvious example of a signature disease is mesothelioma, a rare form of lung cancer that is almost exclusively caused by exposure to asbestos.

B. *Admissibility of Causation Evidence in Toxic Tort Actions*

Because of the need for expert medical testimony, nearly all toxic tort cases require the court to make admissibility determinations on causation evidence.[118] The courts are ultimately responsible for determining the admissibility of complex medical and scientific evidence.[119] Today, there are two primary standards for admissibility of expert testimony utilized by the courts: the *Frye* and *Daubert* tests. The *Frye* test[120] requires that a scientific theory or methodology be generally accepted in the relevant scientific community.[121] *Frye* was widely adopted by both federal and state courts[122] and is still utilized in several states.[123] However, in the 1993 decision *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,[124] the Supreme Court established that judges should be the ultimate arbiters of the relevance and reliability of scientific testimony under the Federal Rules of Evidence.[125] Thus, the admissibility determination in federal courts was shifted from acceptance by the relevant scientific community squarely back onto the shoulders of the judge.[126] Although *Daubert* is not binding on the

118. *See* EGGEN, *supra* note 95, at 10.

119. FED. R. EVID. 104(a); FED. R. EVID. 702; *see* Henning & Berman, *supra* note 96, at 31.

120. Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).

121. *Id.* at 1014.

122. *See* EGGEN, *supra* note 95, at 271.

123. *See* Alice B. Lustre, Annotation, *Post-Daubert Standards for Admissibility of Scientific and Other Expert Evidence in State Courts*, 90 A.L.R.5th 453, 520–37 (2004) (noting fifteen states and the District of Columbia follow the *Frye* test).

124. 509 U.S. 579 (1993).

125. *Id.* at 589 ("[T]he trial judge must ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable.").

126. Under *Daubert*, the judge must ensure that the "reasoning and methodology underlying the testimony is scientifically valid and . . . properly can be applied to the facts at issue." *Id.* at 592–93. Acceptance in the scientific community was retained as a factor to consider, but lack of such acceptance was no longer a fatal defect in the proponent's argument. Instead, a multitude of factors could be considered, including: (1) whether the theory or technique has been tested; (2) whether the theory or technique has been subjected to peer-review; (3) the known or potential rate of error for the theory or technique; and finally, (4) the degree of acceptance of the theory or technique in the scientific community. *Id.* at 593–94. The list is not exclusive, and consistent with guidance from the Supreme Court, see Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999), federal courts often consider other factors, including "(1) whether the expert has unjustifiably extrapolated from an accepted premise to an unfounded conclusion and (2) whether an expert has accounted for obvious alternative explanations." Roche v. Lincoln Prop. Co., 278 F. Supp. 2d 744, 749 (E.D. Va. 2003) (citing language from Supreme Court and other federal court decisions), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004).

states, many state courts have adopted it.[127]

As the causation element is a key component of the plaintiff's case, defendants frequently launch pre-trial evidentiary challenges.[128] Often, the precise etiology of the disease at issue is subject to very real debate,[129] resulting in completely contradictory expert testimony. Thus, evidentiary decisions made by the courts are profoundly challenging and have the potential to affect the outcome of a particular case.

Not all courts have insisted on the same degree of certainty when it comes to establishing causation in toxic tort cases. This variability is especially evident in the courts' treatment of differential diagnosis testimony. For example, while the prototypical claim involves providing evidence of actual exposure levels, an absence of precise knowledge on this point is not always fatal to the plaintiff's case. In *Westberry v. Gislaved Gummi AB*,[130] the Fourth Circuit recognized that it is often "difficult, if not impossible, to quantify the amount of exposure" in cases involving exposure to toxic agents.[131] Similarly, precise information about the threshold level of toxicity may be absent. The *Westberry* court, permitting a worker's claim that he was injured by exposure to airborne talc in the workplace, addressed the absence of known hazardous exposure levels for the compound:

> [W]hile precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff's exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial exposure and need not invariably provide the basis for an expert's opinion on causation.[132]

Accordingly, the Fourth Circuit reasoned that in the absence of precise data on exposure, the finder of fact may rely on a reliable differential diagnosis that includes evidence of substantial exposure, temporality of symptoms, and general notions of toxicity to determine whether the plaintiff was exposed to unhealthy levels of a toxic

127. North Carolina courts recently declined to adopt the *Daubert* standard. Howerton v. Arai Helmet Ltd., 358 N.C. 440, 469, 597 S.E.2d 674, 693 (2004).

128. EGGEN, *supra* note 95, at 270–71.

129. *Id.* at 7.

130. 178 F.3d 257 (4th Cir. 1999).

131. *Id.* at 264.

132. *Id. See generally* Goldstein & Henifin, *supra* note 109, at 404–05 (discussing causation issues and use of toxicological evidence in toxic tort cases).

agent.[133]

Other courts have scrutinized differential diagnosis evidence far more strictly. For example, in *Moore v. Ashland Chemical*,[134] the Fifth Circuit rejected testimony from a plaintiff's physician that argued for a link between exposure to toluene and occupational lung injuries.[135] The court refused to allow the plaintiff to rely solely on evidence of the temporality of exposure and subsequent health outcome, insisting on the need for scientific studies defining relevant threshold levels of exposure.[136] Other circuits have applied a similar logic, insisting that the plaintiff show "the levels of exposure that are hazardous to human beings generally."[137]

Disparate standards of admissibility can have a profound effect on the plaintiff's claim in a toxic tort lawsuit. If the toxicity of the agent at issue is acknowledged, but the dose-response characteristics are poorly defined, the fate of a plaintiff's personal injury claim may be in question. Where, as with mold, the scientific evidence supporting adverse health impacts is still developing and consists primarily of clinical studies and small-scale epidemiological findings, even compelling case-specific medical evidence may not be accepted by the court as reliable evidence of specific causation. The outcome may well depend significantly on that court's insistence on defining the levels of exposure that are hazardous to human health.

IV. HOW THE COURTS HAVE DEALT WITH THE CAUSATION QUESTION IN MOLD INJURY CLAIMS

The defense bar correctly considers the causation component the weakest link in the mold plaintiff's personal injury claim.[138] In addition to facing the standard litany of challenges inherent in any toxic tort personal injury claim, the mold plaintiff faces several uniquely difficult realities: mold is a ubiquitous, naturally occurring organism, it exists in an astounding variety of species, and

133. *See Westberry*, 178 F.3d at 262.

134. 151 F.3d 269 (5th Cir. 1998).

135. *Id.* at 279.

136. *Id.* at 278.

137. *See* Mitchell v. Gencorp, Inc., 165 F.3d 778, 781 (10th Cir. 1999) (quoting Wright v. Willamette Indus., Inc., 91 F.3d 1105, 1106 (8th Cir. 1996)); *accord* Allen v. Pennsylvania Eng'g Corp., 102 F.3d 194, 199 (5th Cir. 1996) ("Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case.").

138. *See* Julie S. Elmer, *A Fungus Among Us: The New Epidemic of Mold Claims*, 64 ALA. LAW. 109, 112 (2003) ("Causation is the Achilles heel of a mold claim.").

environmental sampling methods are evolving rapidly, making results from such surveys difficult to translate to actual human exposure.[139] In addition, surprisingly little is known about the impact of molds and mycotoxins on human health, and even the well-characterized symptoms of exposure, while real, are subjective and general in nature.[140] Despite decades of study, particularly intensive during the past several years, the scientific community has not reached any consensus on the expected range of adverse health effects[141] or the levels at which these adverse health effects might be observed.[142] Concurrent exposure to other indoor air pollutants such as bacteria, cigarette smoke, volatile organic chemicals, and dust mites can frustrate attempts to link specific symptoms to mold exposure.[143] Any of the above factors can be mined by the defense to cause irreparable damage to the plaintiff's case, particularly on the issue of specific causation.[144] In many respects, it is hard to conceive of a more challenging scenario for the toxic tort plaintiff.

A. Existing Case Law

Despite the significant evidentiary challenges, plaintiffs have prevailed in several mold-related personal injury lawsuits. *Centex-Rooney Construction Co. v. Martin County*[145] presaged many of the issues that would be raised in future toxic mold lawsuits. Martin County, Florida sued the construction management company in charge of building the county courthouse, alleging that negligent oversight of the project permitted window and exterior wall leaks to

139. *See* Kuhn & Ghannoum, *supra* note 20, at 146–47 (explaining the difficulties in measuring toxigenic molds and the mycotoxins that are believed responsible for adverse health effects).

140. *See supra* Part I.

141. *See* IOM, *supra* note 18, at 253–54 (finding sufficient evidence of an association between the presence of mold and several upper respiratory tract symptoms, but inadequate or insufficient evidence to determine whether an association exists for other health outcomes including asthma development, pulmonary hemorrhage in infants, skin symptoms, and fatigue); Redd, *supra* note 22, at 60–61.

142. *See* IOM, *supra* note 18, at 92–93, 170 (emphasizing the poor understanding of mold exposure patterns and concluding that the dose of mycotoxins required to cause adverse human health effects has not been determined).

143. Kuhn & Ghannoum, *supra* note 20, at 145. *See also* IOM, *supra* note 18, at 90 ("[T]he specific roles of infectious and noninfectious microorganisms and their components in diseases related to indoor environments are poorly understood.").

144. *See* Stephen J. Henning & Daniel A. Berman, *Mold Contamination: Liability and Coverage Issues*, 8 HASTINGS W.-NW. J. ENVTL. L. & POL'Y 73 (2001) (distinguishing general from specific causation in mold claims and defining causation as the weakest link in the plaintiff's case).

145. 706 So. 2d 20 (Fla. Dist. Ct. App. 1997).

create an infestation of toxigenic mold.[146] The resulting "sick building" conditions led to evacuation of the building.[147] Appealing the $14 million judgment against it, the construction company cited several allegedly erroneous evidentiary determinations—namely, the lower court's decision to exclude an environmental report showing that fungal and bacterial levels inside the courthouse were two to ten times lower than outside levels and to admit expert testimony suggesting the existence of a health hazard in the building.[148]

The Florida Court of Appeals agreed that denial of the environmental report was an abuse of discretion, but found the error to be harmless.[149] Significantly, the court affirmed the admission of the health hazard testimony, finding sufficient foundation for the trial court's conclusion, made following a *Frye* hearing, that the testimony was admissible.[150] The court noted that both experts cited "numerous publications" recognizing the adverse health effects of the toxigenic molds, thereby providing adequate proof "that the basic underlying principles of scientific evidence were sufficiently tested and accepted by the relevant scientific community."[151] Thus, *Centex-Rooney* represents an early evidentiary victory for proponents of toxic mold injury claims in a *Frye* jurisdiction, which is surprising given subsequent statements by scientists and courts emphasizing the lack of scientific consensus on toxic mold-related health effects.[152] Notably, no mention is made of the absence of known hazardous exposure levels.

In *New Haverford Partnership v. Stroot*,[153] two apartment tenants sued their landlord to recover for mold-related health problems allegedly caused by the landlord's negligent failure to repair a leaky bathroom fixture.[154] The landlord challenged the admission of expert

146. *See id.* at 23–25. The County filed suit against the construction managers, masonry supply company, the project architect, and sureties involved in the project; allegations included breach of contract and negligent design and construction. *Id.* at 24.

147. *Id.* at 24.

148. *Id.* at 25–26.

149. *Id.* (noting that the trial court permitted the defendants to present several of the report's critical findings).

150. *Id.* at 26.

151. *Id.*

152. *See supra* notes 141–42 and accompanying text. Indeed, commentators discussing defense strategies for mold litigants in *Frye* jurisdictions emphasize the lack of scientific consensus on the causal relationship between mold exposure and adverse health outcomes. *See, e.g.*, Elmer, *supra* note 138, at 115 (discussing strategies for challenging causation in mold claims brought in Alabama, a *Frye* jurisdiction).

153. 772 A.2d 792 (Del. Super. Ct. 2001).

154. *See id.* at 795–96 (describing the facts of the case). The facts in this case are remarkable: a shower leak in the upstairs bathroom caused the plaintiff's ceiling to

testimony relating to mold exposure and causation of plaintiff's injuries, asserting, among other things, that one physician failed to rule out other possible causes of plaintiff's cognitive deficits and that none of the experts had a proper foundation to show causation.[155] On the first argument the court upheld the decision to admit the physician's conclusions with little discussion.[156] On the issue of causation, the court said the scarcity of monitoring data went to the weight of the evidence but did not justify excluding the testing data, while the medical testimony was properly based on the "scientifically accepted" procedure of utilizing a medical history, blood test results, and monitoring data to rule out other possible causes of the plaintiff's injuries.[157] Thus, *Stroot* illustrates an instance of a court accepting a mold personal injury claim on the basis of a differential diagnosis analysis that did not include introduction of dose-response data, consistent with the relaxed standard for differential diagnosis employed by the Fourth Circuit in *Westberry*.[158]

Similarly, in *Caldwell v. Curioni*,[159] renters of a home that was infested with mold including *Stachybotrys* prevailed on appeal of a ruling granting summary judgment to their landlord.[160] At trial, the plaintiffs' personal injury claims were rejected, despite expert testimony concerning their health problems and the presence of toxigenic mold in the residence.[161] The trial court may have been swayed by one expert's admission that "there are no established standards for permissible airborne fungal concentrations."[162]

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collapse, exposing debris "covered with black, green, orange and white mold." *Id.* at 796. Despite a strong, nauseating odor, plaintiff remained in the apartment that night, but by morning she could not breathe and was rushed to a hospital on doctor's orders. *Id.* It is not clear from the court opinion whether the plaintiff alleged an adverse reaction to toxigenic molds specifically. The opinion refers to "atypical" molds and the plaintiff's symptoms included allergy-related respiratory symptoms and cognitive deficits. *Id.*

155. *See id.* at 798. The plaintiffs introduced testimony from at least four experts, including a mycologist/microbiologist, two physicians and an architect. The physicians both testified that the plaintiff's physical and mental ailments were caused by exposure to "atypical" molds. *See id.* at 796–97.

156. *Id.* at 799 ("[The physician] took a history from [plaintiff] and relied on [another physician's] review of her medical records. Since his approach is accepted in the scientific community, we conclude that his opinion evidence was properly admitted.").

157. *Id.* at 800. Although the court referred to "acceptance in the scientific community" as supporting admittance of the physician's testimony, its decision was apparently based on the court's assessment of reliability. Delaware is a *Daubert* state. M.G. Bancorporation v. Le Beau, 737 A.2d 513, 522 (Del. 1999).

158. *See supra* notes 130–33 and accompanying text.

159. Caldwell v. Curioni, 125 S.W.3d 784, 788–89 (Tex. App. 2004).

160. *Id.* at 788–89.

161. *Id.* at 787–88.

162. *Id.* at 789.

However, the absence of standards was treated summarily by the Texas Court of Appeals, which declared that "the lack of any established standards does not confirm that the levels of mold present were not dangerous."[163] The court found plaintiffs' evidence of mold exposure and testimony from treating physicians compelling enough to overturn the lower court's summary judgment ruling.[164]

Not all personal injury claims have concluded favorably for mold plaintiffs, however. For example, Melinda Ballard's windfall judgment of $32 million against her homeowner's insurance company was tempered by a dismissal of her husband, Ronald Allison's personal injury claims—a decision later upheld by the Texas Court of Appeals.[165] Allison had offered evidence that toxigenic molds including *Stachybotrys* were present in the couple's home, and provided testimony from several medical professionals asserting that exposure to these molds caused his illnesses.[166] Allison's experts relied on an epidemiological study they had conducted in which twenty people experienced a range of health effects after being exposed to toxic mold in a building.[167] But relying on that study to show general causation proved fatal to Allison's claim. The district court found that the study met the *Daubert* standard of admissibility,[168] but fell short of Texas's *Havner*[169] standard of reliability for epidemiological evidence.[170] Without sufficient proof of general causation, Allison was precluded from demonstrating specific causation.

Allison demonstrates a willingness of courts to keep a tight leash on evidence supporting a mold plaintiff's injury claim. The court concluded that testimony from the plaintiff's experts was based on the results of an unreliable study, and as such, the testimony itself was

163. *Id.* at 793.

164. *Id.* at 794.

165. Allison v. Fire Ins. Exch., 98 S.W.3d 227, 237–40 (Tex. App. 2002). Mrs. Ballard's victory was also tempered by the court's decision to reduce her damages to $4 million. *Id.* at 264.

166. *See id.* at 239.

167. *Id.*

168. *See id.* The evidence was adjudged reliable under *Daubert* and the corresponding state court case which adopted the reliability standard in Texas. *See id.* at 237–38 (discussing application of *E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549 (Tex. 1995)).

169. Merrell Dow Pharms., Inc. v. Havner, 953 S.W.2d 706 (Tex. 1997).

170. *See Allison*, 98 S.W.3d at 239–40 (explaining that to be declared reliable, *Havner* requires that an epidemiological study exhibit "a ninety-five percent confidence interval, [or] . . . show that exposure to the substance more than doubles the risk of injury" (quoting *Havner*, 953 S.W.3d at 717–18, 722–23)).

unreliable.[171] Thus, reliance on a single epidemiological study of questionable reliability fatally undermined the plaintiff's claim.

In *Roche v. Lincoln Property Co.*,[172] two tenants allegedly suffering from exposure to toxic mold sued their landlord and apartment manager for negligent maintenance of their apartment.[173] In support of their claim, they offered the testimony of Dr. Bernstein, an allergist that had treated them.[174] Dr. Bernstein concluded, based on medical observations, a review of the scientific literature, knowledge of mold measurements taken from the plaintiffs' apartment, and his previous experience with patients suffering from sick-building syndrome,[175] that the plaintiffs' symptoms were caused by allergenic and mycotoxic effects of *Stachybotrys* and other indoor molds.[176] The defendants moved to exclude Dr. Bernstein's testimony as insufficient to meet the *Daubert* standard of admissibility on the issue of specific causation.[177]

The court ultimately concluded that Dr. Bernstein's testimony was inadmissible under *Daubert* because he did not establish, "through any acceptable methodology, that the plaintiffs were injured by the mold allegedly contained within their apartment."[178] Specifically, the court was unimpressed by Dr. Bernstein's "vague and generalistic" conclusion that mold exposure caused the plaintiffs' injuries,[179] his over-reliance on a temporal association,[180] and his reference to "conflicting facts."[181] In fact, the court spelled out no less than ten reasons why Dr. Bernstein's differential diagnosis testimony was deficient,[182] attacking everything from his lack of credentials as a toxicologist to his failure to rule out the plaintiffs' "significant

171. *Id.* at 240.

172. 278 F. Supp. 2d 744 (E.D. Va. 2003), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004).

173. *Id.* at 746.

174. *See id.* at 746 (reporting that symptoms included memory loss, sinus problems, mild hypersensitivity to smells, chronic nasal stuffiness, chronic upper respiratory sinus symptoms, chest congestion, and shortness of breath).

175. *Id.* at 749–50, 754 (noting that Dr. Bernstein reviewed blood tests, CAT scans, MRIs, X-rays, and skin sensitivity tests on the Roches).

176. *Id.* at 746.

177. *Id.* at 747.

178. *Id.* at 750.

179. *Id.* (finding that Dr. Bernstein failed to distinguish between reactions to mold and other common allergens).

180. *Id.* at 752.

181. *Id.* at 750 (criticizing Dr. Bernstein's reliance on equivocal scientific studies).

182. *See id.* at 751–52.

allergies to cats."[183] Dr. Bernstein, the court concluded, failed to "rule in the suspected molds and rule out other allergens," making his differential diagnosis insufficient as reliable evidence of specific causation.[184] Other recent decisions suggesting similar misgivings about causation evidence in mold litigation are increasingly prevalent.[185]

B. *Should Differential Diagnosis Pass Muster as Proof of Causation in Toxic Mold Litigation?*

The above cases expose a remarkable range of attitudes concerning the viability of differential diagnosis as proof of causation in toxic tort litigation. Courts will rightly insist on a differential diagnosis when a plaintiff's injury can be explained by more than one cause.[186] Defining what constitutes a valid differential diagnosis, however, is subject to significant dispute.[187] And to date, courts have exhibited widely divergent views on the matter.[188] The sheer volume of information included in a complex toxic tort differential diagnosis provides ample opportunity to question the relevance or reliability of proffered evidence. Similarly, the flexibility introduced by *Daubert* permits significant leeway to courts that choose to insist on more or higher quality data.[189]

The *Roche* opinion provides an excellent point of comparison to earlier toxic tort cases evaluating differential diagnosis testimony and a possible window on how such evidence will fare in mold-related lawsuits in federal courts. In many ways, *Roche* stands in stark contrast to the Fourth Circuit's handling of the plaintiff's injury claims in *Westberry*.[190] The plaintiff in *Westberry* failed to bring forth

183. *Id.* at 752.

184. *Id.* at 753.

185. *See, e.g.*, Graham v. Lautrec, Ltd., No. 01 031717 CE, 2003 WL 23512133 (Mich. Cir. Ct. July 24, 2003) (granting a motion to exclude expert testimony on causation in a mold personal injury claim).

186. *See* Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS., 107, 120 (Autumn 2001).

187. *See* Edward J. Imwinkelried, *The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology): Of Under- and Over-Estimations*, 56 BAYLOR L. REV. 391, 395 (2004). *See generally* Sanders & Machal-Fulks, *supra* note 186 (discussing differential diagnosis options).

188. *See* Imwinkelried, *supra* note 187, at 395; Sanders & Machal-Fulks, *supra* note 186, at 120–29 (discussing numerous recent cases evaluating differential diagnoses).

189. Sanders & Machal-Fulks, *supra* note 186, at 137 (concluding that overall, *Daubert* courts are less likely to admit differential diagnosis testimony).

190. The courts' differing attitudes become stark in their description of the standard of review under FED. R. EVID. 702. The *Roche* court emphasized the court's duty to keep

any epidemiological studies, animal studies, laboratory data, or any peer-reviewed published studies to support the medical conclusion that his sinus disease was caused by occupational exposure to talc dust.[191] In addition, his expert witness did not provide any tissue samples indicating evidence of exposure, nor any evidence to show that adverse effects would be experienced above the plaintiff's level of exposure.[192] Yet the *Westberry* court upheld the decision to admit the causation testimony, primarily on evidence of a temporal relationship between exposure and effect.[193] In contrast, the Roches and Dr. Bernstein offered numerous exhibits in support of their claim, including tissue samples and an extensive literature review.[194] But the district court systematically eviscerated each of the plaintiffs' exhibits, even finding occasion to attack Dr. Bernstein's status as an allergist[195] and refusing to analyze his testimony as a treating physician.[196]

Left with little more than a temporal association between exposure and symptoms, the plaintiffs' evidence was deemed insufficient proof of causation.[197] The court made the unnecessarily broad statement that "[a medical] opinion based primarily, if not solely, on temporal proximity does not meet *Daubert* standards."[198] To distinguish *Westberry*, the court pointed to that case's testimony from a treating physician, the prolonged period over which the plaintiff's symptoms were monitored, and exposure to "severe" levels of talc.[199] This explanation is deficient coming on the heels of the court's broad statement concerning temporal proximity of exposure and symptoms, especially given the awkward characterization of the quality of the *Roche* plaintiffs' exposure evidence.[200]

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out conjecture and unsupported speculation, 278 F. Supp. 2d at 748, while the *Westberry* court spoke of the "flexible" inquiry to be conducted by the district court under the "liberalize[d]" standard, which must focus on "principles and methodology" and not the conclusions reached. *See* Westberry v. Gislaved Gummi AB, 178 F.3d 257, 261 (4th Cir. 1999).

191. *Westberry*, 178 F.3d at 262.

192. *Id.*

193. *Id.* at 265 (explaining that "depending on the circumstances, a temporal relationship between exposure . . . and . . . symptoms can provide compelling evidence of causation").

194. *Roche*, 278 F. Supp. 2d at 753–56.

195. *Id.* at 749.

196. *Id.* at 750.

197. *Id.* at 764–65.

198. *Id.* at 764.

199. *Id.* at 765.

200. *See id.* at 765 (dismissing expert testimony that the plaintiffs were exposed to severe mold concentrations because "not all mold spores are allergens and not all molds

Concededly, the plaintiffs' evidence in *Roche* was weak in several respects. First, only one of the plaintiffs showed an allergic reaction to mold when tested, yet both plaintiffs complained of suffering from mold exposure.[201] Second, medical tests showed the plaintiffs were allergic to numerous allergens, and Dr. Bernstein's testimony failed to rule out those allergens as potential intervening causes.[202] And finally, as the district court willingly emphasized, the published studies relied on by Dr. Bernstein contained language suggesting a lack of understanding about the precise toxic mold exposure levels associated with adverse health effects.[203]

Understandably, all of the above factors augured against a favorable outcome for the plaintiffs in their quest to demonstrate specific causation. But the most legitimate flaw in the plaintiffs' case was the failure to rule out confounding exposures. This failure is the factor that most clearly distinguishes *Westberry*'s facts from those in *Roche* and will determine the outcome of evidentiary challenges in future indoor mold personal injury claims. Although the plaintiff in *Westberry* suffered from symptoms of a general nature and provided little toxicological evidence to support his claim, he prevailed in part because his treating physician took steps to rule out other potential causes of the plaintiff's sinus condition, including a cold he had suffered and a summer's worth of waterskiing.[204] The *Westberry* court's discussion on this point was thin, which is remarkable considering the generality of the sinus symptoms experienced by the plaintiff.[205] Perhaps it was the nature of the occupational exposure, occurring in a more controlled environment than what might be expected in the home, which swayed the *Westberry* court and convinced it to overlook the minimal supporting toxicological evidence. Regardless, because the plaintiffs in *Roche* exhibited multiple allergic sensitivities, and perhaps more importantly, because their allegedly mold-related symptoms were consistent with general reactions to other airborne allergens, the failure to rule out intervening causes for the plaintiff's ailments doomed their case. This outcome was presaged by many commentators who follow mold litigation,[206] and it strongly suggests that the "ruling out" component

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produce mycotoxins").

201. *Id.* at 753.

202. *Id.* at 762–63.

203. *Id.* at 756.

204. Westberry v. Gislaved Gummi AB, 178 F.3d 257, 266 (4th Cir. 1999).

205. *See id.*

206. *See* Jarman-Felstiner, *supra* note 16, at 552; Perry, *supra* note 16, at 272–73.

of a differential diagnosis represents the most significant challenge to the mold plaintiff.[207]

C. *The Curious Role of "Standards" in the Causation Calculus*

As the preceding paragraphs suggest, courts considering differential diagnosis evidence in mold personal injury lawsuits will have considerable opportunity to question the reliability of the plaintiff's evidence introduced to rule out alternative causes of the disease. Similarly, the evolving state of scientific consensus regarding adverse health effects related to exposure of toxigenic mold will further undermine proof of general and specific causation. Both of these considerations were invoked by the *Roche* court in the course of its evidentiary analysis and would have formed a defensible basis for rejecting the plaintiff's causation evidence.

Rather than rein in its discussion to these basic, critical considerations, however, the *Roche* court chose to engage in a protracted discussion of additional, less relevant factors. It was here that the court made its most questionable determinations. One example was the *Roche* court's seeming obsession with "definable standards" for molds.[208] The *Roche* court's logic was particularly tortured on the issue of these mold "standards." The court repeatedly cited medical and scientific literature describing the generality of symptoms arising from mold exposure and the nearly indefinable range of exposures that would be expected in the population,[209] seemingly bolstering an argument that a single "bright line" health standard for mold would be impossible to craft. But then the court cited the absence of such a standard as a critical, fatal factor in the plaintiffs' proof of specific causation.[210]

The *Roche* court is not the lone voice calling for such health-based standards; numerous authors reviewing the landscape of mold-

207. Of course, the plaintiffs in *Stroot* prevailed on essentially the same evidence, but in state court. *See* New Haverford P'ship v. Stroot, 772 A.2d 792 (Del. Super. Ct. 2001). Similarly, the state court in *Centex-Rooney* did not exhibit as much skepticism over the quality of the plaintiff's causation data. *See supra* notes 147–52 and accompanying text; *see also* Caldwell v. Curioni, 125 S.W.3d 784, 794 (Tex. App. 2004) (reversing summary judgment for mold defendant where plaintiff offered some evidence of mold exposure and testimony from treating doctors).

208. *Roche*, 278 F. Supp. 2d at 756. The *Roche* Court discussed the need for exposure guidelines or standards repeatedly over eight pages of the opinion. *Id.* at 754–61.

209. The court embarked on a lengthy review of the scientific and medical literature pertaining to the health effects of mold exposure, quoting from articles offered by the plaintiffs and the defendants. *Id.* at 755–61.

210. *See id.* at 761.

related litigation have concluded that standards for mold exposure are a key factor in the viability of such claims.[211] The typical argument is that until such standards are articulated, plaintiffs will not be able to successfully establish specific causation because they will not be able to show that their exposure exceeded some "safe" threshold exposure level.[212] However, the courts do not speak with a unified voice on the need for such exposure standards in toxic tort cases.[213] Numerous federal courts have insisted that the plaintiff prove his actual exposure exceeded some quantifiable hazardous level of exposure as part of the process of ruling in the suspected causative agent in a differential diagnosis.[214] But other courts have been considerably less troubled by the absence of such "standards."[215]

The *Roche* court first broached the issue of "quantitative standards or guidelines"[216] for mold exposure in a section of its opinion entitled "A Toxicologist's Methodology."[217] Toxicologists, the court explained, must first establish the nature and extent of the actual exposure.[218] They must then perform an evaluation, based on the published scientific literature, of the exposure levels associated with adverse health outcomes.[219] The final step of this risk assessment

211. *See, e.g.*, Jarman-Felstiner, *supra* note 16, at 549 (asserting that "[u]ntil permissible mold exposure limits" are established by governmental agencies, mold litigation will be substantially hindered); Peña-Alfaro, *supra* note 5, at 575–76 ("Until air quality standards are implemented, much . . . uncertainty and conjecture will continue."); Perry, *supra* note 16, at 297 ("[S]tate and federal guidelines for acceptable levels of mold within the home are needed to help the scientific community, homeowners, insurers, and courts alike reach definitive conclusions about the true dangers of mold exposure."); *see also* Wright & Irby, *supra* note 5, at 323–25 (suggesting that an absence of permissible exposure limits and remediation standards for mold presents a problem for environmental issues in the transactional process).

212. *See Roche*, 278 F. Supp. 2d at 758; *see also* Graham v. Lautrec, Ltd., No. 01 031717 CE, 2003 WL 23512133, at *5 (Mich. Cir. Ct. July 24, 2003) (noting in dictum the conflict in the scientific community over the ability to establish a "standard related to mold exposure").

213. *See supra* notes 130–37 and accompanying text.

214. *See supra* notes 136–37 and accompanying text.

215. *See, e.g.*, Westberry v. Gislaved Gummi AB, 178 F.3d 257, 264 (4th Cir. 1999) (allowing differential diagnosis despite a lack of "precise information concerning the exposure necessary to cause specific harm"); Caldwell v. Curioni, 125 S.W.3d 784, 793 (Tex. App. 2004) (stating that lack of established standards for mold does not mean that the mold present was not dangerous).

216. *Roche*, 278 F. Supp. 2d at 754.

217. *See id.* at 754–55. The court apparently felt compelled to embark on this pedagogical exercise despite the fact that neither party raised the issue in multiple briefs. *Id.* at 754.

218. *Id.* at 754 (quoting Cavallo v. Star Enter., 892 F. Supp. 756, 764 (E.D. Va. 1995), *rev'd on other grounds*, 100 F.3d 1150 (4th Cir. 1996)).

219. *Id.*

is to compare the two values to determine the relevance of the subject's exposure.[220] Because the studies reviewed by the court revealed that there were no "standards or guidelines" for mold, it reasoned that Dr. Bernstein's anecdotal conclusions were unsupported by "the methods of science."[221] According to the court's view, in the absence of an exposure guideline, the mold plaintiff will almost certainly succumb to summary judgment for failure to provide evidence of specific causation.

The court's enunciation of the principles of toxicology and its subsequent dissection of scientific studies on the record are admirable, but the court's analysis also exposes the danger of permitting courts to march intrepidly into the scientific realm. The description of the steps taken by toxicologists to assess risks from exposure to toxic agents more or less accurately portrays the risk characterization process described above.[222] But the court risks making a serious misstep when it delves into the "standards or guidelines" supposedly used by toxicologists to assess risks. At one point, the court explained that toxicologists use "exposure levels associated with adverse health outcomes" as a barometer for assessing risks.[223] This definition is consistent with the toxicity assessment process described above.[224] However, the court's repeated reference to the absence of mold "standards or guidelines" risks focusing on regulatory values rather than scientifically established threshold exposure levels. To wit, the court discusses, at great length, several articles offered by the defense which emphasize the absence of definable standards for mold, none of which appears in the peer-reviewed scientific literature.[225] It appears from the language in these articles and others discussed by the court[226] that some of these authors

220. *Id.*

221. *See id.* at 754–55.

222. *See supra* notes 107–14.

223. *See Roche*, 278 F. Supp. 2d at 755.

224. *See supra* notes 107–09 and accompanying text.

225. *Roche*, 278 F. Supp. 2d at 755–58. Ironically, after the court explained that the toxicologist methodology involves relying on published scientific articles, the court dismissed as unhelpful peer-reviewed articles relied on by Dr. Bernstein and subsequently focused the bulk of its discussion on articles that were not published in the peer-reviewed scientific literature, including several government publications. *See id.* at 755–59.

226. The court also discussed an article from the New York City Department of Health and Mental Hygiene as further support for its argument that no established standards exist for mold exposure. The article, which is not published in the peer-reviewed scientific literature, was cited by the plaintiffs but not relied upon by Dr. Bernstein. *Id.* at 758. The court also referenced Internet publications and other state agency documents. *See id.* at 759.

are referring to the absence of *regulatory* standards.[227]

The relevance of the distinction between regulatory standards and scientifically determined threshold effect levels cannot be emphasized enough. Scientifically determined threshold effect levels are based on empirical observations, often derived from closely controlled animal experiments or peer-reviewed epidemiological studies;[228] they are considered critical components of risk assessment.[229] But regulatory standards are the result of risk management processes, which incorporate significant policy considerations such as the notion of "acceptable" exposure levels.[230] Regulatory standards are crafted to conform to statutory guidance rather than causation principles.[231] The regulatory values are typically far lower than documented effect levels, often by orders of magnitude, because their role is to be protective of health.[232] In

227. *See id.* at 754 (emphasizing that many of the cited articles discuss the absence of "quantitative standards or guidelines for assessing whether the mold contamination in an area is *acceptable* or not") (emphasis added); *see also* Graham v. Lautrec, Ltd., No. 01 031717 CE, 2003 WL 23512133, at *5 (Mich. Cir. Ct. July 24, 2003) (quoting Redd, *supra* note 22, on the lack of information concerning "acceptable" levels of mold). Acceptability is a common term used to define a regulatory aim. *Cf. Roche*, 278 F. Supp. 2d, at 758–59 (quoting Redd, *supra* note 22, at 9–10 (referring to "setting standards and guidelines for indoor mold exposure levels")).

228. Goldstein & Henifin, *supra* note 109, at 406–09 (describing various measures of toxicological effect in animal studies, including the no-observable effect level ("NOEL") and the maximum tolerated dose ("MTD")).

229. *See generally* THE PRESIDENTIAL/CONG. COMM'N ON RISK ASSESSMENT AND RISK MGMT., 2 RISK ASSESSMENT AND RISK MANAGEMENT IN REGULATORY DECISION-MAKING (1997) [hereinafter PCRARM] (distinguishing between the risk assessment and risk management steps in the development of regulatory standards and guidelines), *available at* http://www.riskworld.com/Nreports/1997/risk-rpt/volume2/pdf/v2epa.PDF (on file with the North Carolina Law Review); *see also* Douglas Crawford-Brown, *Scientific Models of Human Health Risk Analysis in Legal and Policy Decisions*, 64 LAW & CONTEMP. PROBS., 63, 66 (Autumn 2001) (describing the series of steps involved in risk assessment).

230. *See* PCRARM, *supra* note 229, at 55, 84–85.

231. Sutera v. Perrier Group of Am., Inc., 986 F. Supp. 655, 664 (D. Mass. 1997).

232. *See* Crawford-Brown, *supra* note 229, at 78; *see also* STEPHEN BREYER, BREAKING THE VICIOUS CIRCLE: TOWARD EFFECTIVE RISK REGULATION 42–50 (1993) (discussing regulators' use of conservative dose-response and exposure models in justifying risk-based regulatory action). The EPA's reference doses ("RfD") and reference concentrations ("RfC") illustrate the effect of safety or uncertainty factors on final risk-based exposure guidelines. Both the RfC and RfD represent "[a]n estimate (with uncertainty spanning perhaps an order of magnitude) of a[n] . . . exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." U.S. Envtl. Prot. Agency, *Integrated Risk Information System: Glossary of IRIS Terms, at* http://www.epa.gov/iris/gloss8.htm (last visited Nov. 17, 2004) (on file with the North Carolina Law Review). The recently derived RfC for chronic inhalation exposure to benzene (0.03 mg/m^3) was derived by dividing the lowest human exposure level associated with adverse effects (8.2

addition, in developing exposure standards regulatory agencies are not held to the same standard of proof as the plaintiff seeking to show causation in a toxic tort action.[233] The *Roche* court risks overstating the relevance of exposure standards when it emphasizes the absence of an indicator of acceptable levels of mold as a factor in the plaintiff's failure to rule in *Stachybotrys* as a cause of his disease.[234]

Regulatory standards that purport to define acceptable levels of exposure should play no more than a token role in the toxic tort causation analysis. The Texas Court of Appeals was correct in *Caldwell* when it stated that "the lack of any established standards does not confirm that the levels of mold present [are] not dangerous."[235] A party may well suffer injury from exposure to an agent that has no regulatory exposure standard;[236] indeed, less than one percent of chemicals in commercial use have been fully evaluated for safety.[237] Similarly, an individual may experience an exposure exceeding some acceptable regulatory level and suffer no ill effects, due to the large safety and uncertainty factors incorporated into health-protective regulatory standards.[238] Attaching significance to

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mg/m^3) by a composite uncertainty factor of 300. U.S. Envtl. Prot. Agency, *Integrated Risk Information System: Benzene (CASRN 71-43-2)*, *at* http://www.epa.gov/iris/subst/0276.htm (last visited Nov. 17, 2004) (on file with the North Carolina Law Review). This uncertainty factor was compiled from separate uncertainty factors accounting for extrapolation from an effect level to a no-effect level (3), variability in human sensitivity to benzene (10), the use of a subchronic exposure study to derive a chronic exposure guideline (3), and finally, an uncertainty factor to account for a lack of reproductive toxicity studies (3). *Id.*

233. *Sutera*, 986 F. Supp. at 664; *accord* Nat'l Bank of Commerce v. Assoc. Milk Producers, 22 F. Supp. 2d 942, 961 (E.D. Ark. 1998) ("[R]egulatory agencies employ a different perspective in setting 'action levels' than do the courts in imposing tort liability.").

234. *See, e.g.*, Roche v. Lincoln Prop. Co., 278 F. Supp. 2d 744, 758–59 (E.D. Va. 2003) (claiming landlord negligently failed to maintain apartment), *summary judgment granted*, No. 02-1390-A, 2003 U.S. Dist. LEXIS 23353 (E.D. Va. July 25, 2003), *vacated on other grounds*, 373 F.3d 610 (4th Cir. 2004). The court cited Dr. Redd of the CDC for the proposition that because it is not known what indoor levels of mold are acceptable, setting standards for mold exposure would be problematic. *Id.*

235. Caldwell v. Curioni, 125 S.W.3d 784, 793 (Tex. App. 2004).

236. Data on actual injurious exposures often forms the scientific basis for exposure standards developed to protect against future exposures. *See* Indus. Union Dep't, AFL-CIO v. Am. Petroleum Inst., 448 U.S. 607, 617 (1980) (discussing the development of state and federal workplace exposure limits for benzene following evidence of blood disorders in workers exposed to benzene).

237. Goldstein & Henifin, *supra* note 109, at 412.

238. The aforementioned benzene RfC provides a capable example. *See supra* note 232. A person could conceivably be exposed to benzene at an exposure concentration one hundred times the RfC value and yet still be well below the concentration associated with adverse human health outcomes because of safety and uncertainty factors used to calculate the RfC value.

the presence or absence of acceptable mold exposure standards in the causation analysis introduces far too great a risk for misstating the merits of the plaintiff's evidence.

Although the absence of quantifiable threshold effect levels ultimately plays little more than a pedantic role in the *Roche* court's ultimate holding, its lax use of language to describe the toxicologist's methodology risks putting far too much weight on risk management-based regulatory standards where empirically-based values are preferred.[239] This risk is particularly acute because several states[240] and the federal government[241] have expressed a recent interest in defining mold exposure guidelines.[242] Proposed federal language describing exposure standards does not hint at whether a risk assessment outcome or a risk management-based exposure guideline is being sought;[243] however, the language in California's Toxic Mold Protection Act of 2001[244] is far more suggestive of a risk management goal. The Act charges the California Department of Health Services to "adopt practical standards to assess the health threat posed by the presence of mold ... in an indoor environment."[245] These "assessment standards" are to protect the public health and are to be

239. Commentators, too, have succumbed to this temptation, suggesting that definitive conclusions regarding health risks cannot be reached until exposure guidelines are generated. *See* Perry, *supra* note 16, at 297.

240. For example, California recently enacted the Toxic Mold Protection Act of 2001, 2001 Cal. Adv. Legis. Serv. 584 (Deering), on January 1, 2002.

241. Rep. John Conyers of Michigan is currently sponsoring a bill to address toxic mold. *See* H.R. 1268, 108th Cong. (2003), http://thomas.loc.gov (on file with the North Carolina Law Review).

242. There are currently no state or federal health-based standards for airborne exposure to toxic molds, a fact cheered by some. *See* Ins. Journal, *Mold Legislation Slows to a Crawl in Statehouses Alliance Says*, *at* http://www.insurancejournal.com/news/newswire/national/2003/09/08/32047.htm (Sept. 8, 2003) (quoting Kirk Hansen, director of Alliance of American Insurers: "We are happy to report that attempts to establish exposure or air-quality standards at the state level have failed.") (on file with the North Carolina Law Review).

243. The Conyers Bill directs the EPA to work with the CDC and NIH to jointly establish "minimum levels of exposure at which indoor mold growth is harmful to human health." H.R. 1268 § 102(a).

244. CAL. HEALTH & SAFETY CODE §§ 26100–26157 (Deering 2004).

245. *Id.* § 26105(a). Completion of these tasks will depend on establishment of the task force and receipt of specific funding from the public. DHS has anticipated that if sufficient funding is received, development of mold standards will take at least 2 years from the point at which the task force is convened. *See* California Dept. of Health Servs., *SB 732 (Toxic Mold Protection Act of 2001) Implementation Update*, *at* http://www.dhs.ca.gov/iaq/Mold/SB732update.htm (last visited Nov. 15, 2004) (on file with the North Carolina Law Review). The Act also required the DHS to develop several additional mold-related standards for mold sampling and remediation. *See* CAL. HEALTH & SAFETY CODE § 26105 (Deering 2004).

generated utilizing the latest scientific data, but these standards must also consider technological and economic feasibility.[246] Additionally, the standards are to consider "the adverse health effects of exposure to molds on the general population, including specific effects on members of subgroups that comprise a meaningful portion of the general population."[247] This directive thus seeks to incorporate factors outside of the realm of toxicology, including considerations of cost and technical feasibility. Furthermore, consideration of population subgroups of heightened sensitivity almost invariably leads to the application of large safety or uncertainty factors,[248] which drives the exposure standard even further from the observed adverse effect or no-effect level. Insisting that the plaintiff compare his actual exposure to such a "standard" to prove specific causation would be a dubious request at best.

CONCLUSION

Mold lawsuits continue to churn their way through the civil justice system. Although the initial panic has subsided, great public concern still exists over the impact of indoor molds on human health. Several years have elapsed since the first toxic mold lawsuits entered the litigation radar screen, and early results are mixed. Plaintiffs have scored some substantial victories in personal injury lawsuits, primarily at the state court level. Federal courts, to date, have been more

246. CAL. HEALTH & SAFETY CODE § 26105(b) (Deering 2004).

247. *See id.* § 26105(c) (specifying that subgroups comprising a meaningful portion of the general population include "infants, children age 6 years and under, pregnant women, the elderly, asthmatics, allergic individuals, immune compromised individuals, or other subgroups that are identifiable as being at greater risk of adverse health effects than the general population when exposed to molds.").

248. A recent example of a legislatively mandated safety factor can be found in the "additional tenfold margin of safety" provision for protection of children from pesticide residues, contained in the Food Quality Protection Act of 1996, Pub. L. No. 104-170, 110 Stat. 1489 (codified as amended in scattered sections of 21 U.S.C.). The Act specifically provides that:

> The Secretary of Health and Human Services and the Secretary of Agriculture, in consultation with the Administrator, shall conduct surveys to document dietary exposure to pesticides among infants and children. In the case of threshold effects . . . an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and the toxicity to infants and children.

21 U.S.C. § 346a(b)(2)(C)(ii) (2000). Even without such a specific legislative mandate, regulatory agencies regularly include safety factors for sensitive populations. *See supra* note 232 (describing the inclusion of an uncertainty factor of 10 to account for variability in human sensitivity to benzene).

exacting in their treatment of causation evidence, refusing to apply some of the more relaxed standards seen in earlier toxic tort cases. This outcome is due in part to the generality of symptoms attributed to mold exposure, which are difficult to distinguish from effects from other common allergens. In some cases, the failure of federal courts to allow causation evidence is also due to a lack of consensus about the adverse effects from exposure to mycotoxins released by toxigenic mold. But varying reactions to causation evidence are also due to disparate views regarding the amount and quality of information needed to constitute a reliable differential diagnosis. The prevalence of mold, the generality of exposure symptoms, and uncertainties regarding the ill effects of exposure to toxigenic molds will undoubtedly continue to expose jurisdictional differences in the treatment of differential diagnosis testimony.

As scientific understanding about the health effects of toxigenic molds advances, the public is clamoring for exposure and remediation standards for molds that will aid in defining the relevance of actual exposures. The courts, too, have suggested that such standards are necessary elements of the causation calculus. But great risks are presented when decisionmakers rely too heavily on such standards. Just as the absence of regulatory standards should not necessary signal a fatal defect in the mold plaintiff's claim, so the existence of standards will not necessarily be an appropriate guiding light for courts grappling with issues of general and specific causation. There are currently proposals at both the state and federal government level that involve the development of mold exposure standards. However, even if the science of mold toxicology has advanced to the point where a foundation for such a risk assessment might exist, these standards are likely to incorporate risk management concepts such as technical feasibility and default use of safety factors that will give rise to an end product that is quantitatively far removed from actual health effect levels. Accordingly, the courts that so zealously pine for such standards need to exercise extreme caution interpreting their relevance in the context of causation. In the legal arena, the mold "standard" may not be the panacea everyone seeks.

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