The Growing Hazard of Mold Litigation

Papers commissioned by the U.S. Chamber Institute for Legal Reform and the Center for Legal Policy at The Manhattan Institute

Released July 17, 2003

U.S. Chamber Institute for Legal Reform

The U.S. Chamber Institute for Legal Reform was founded in 1998 as a 501(c)(6) tax-exempt, separately incorporated affiliate of the U.S. Chamber of Commerce. The mission of ILR is simple: to make America's legal system simpler, fairer and faster for everyone. ILR's multi-faceted program seeks to promote civil justice reform through legislative, political, judicial and educational activities at the national, state and local levels.

Center for Legal Policy at the Manhattan Institute

The Center for Legal Policy at the Manhattan Institute is a leading voice for reform of America's civil justice system. The Center's mission is to communicate thoughtful ideas on civil justice reform to real decision-makers through books, publications, conferences and public or media appearances. Founded in 1986, hundreds of news reports have cited the Center's work, with The Washington Post going so far as to call Senior Fellows Peter Huber and Walter Olson the "intellectual gurus of tort reform."

EXECUTIVE SUMMARY

"A New Plague – Mold Litigation: How Junk Science and Hysteria Built an Industry"
and
"A Scientific View of the Health Effects of Mold"

The insurance industry has reported "toxic" mold claims in the billions of dollars. Insurance companies in Texas alone paid $1.2 billion in mold claims in 2001. Is mold the next asbestos? The U.S. Chamber Institute for Legal Reform, partnering with the Center for Legal Policy of the Manhattan Institute, commissioned two papers that take a close look at mold litigation and the science of mold. The first, by Cliff Hutchinson and Robert Powell, two experienced litigators with Hughes and Luce in Dallas and Austin, provides a legal perspective on mold claims. The second, written by a team of scientists led by Dr. Bryan Hardin, former Deputy Director of NIOSH and former Assistant Surgeon General in the Public Health Service, addresses the scientific evidence – or lack thereof – that forms the foundation of these claims.

In "A New Plague – Mold Litigation: How Junk Science and Hysteria Built an Industry," Hutchinson and Powell explain the phenomenon of mold litigation by opening with an overview of litigation over Alar and plastics, both based on a media-generated fear of alleged health hazards – fear without scientific support. It segues into a discussion of the 1980s media reports of an emerging illness – "sick building syndrome." Although this new health hazard resulted in buildings being shut down and in some cases abandoned, clear-eyed scientists have shown the threat to be highly exaggerated – more due to psycho-social factors than to any disease entity. Nonetheless, litigation over alleged health effects from indoor air quality has endured. Against this backdrop of public suspicion of indoor air and media generated fear of phantom toxics, mold claims emerged in the mid-1990s and quickly grew.

In November 1994, a Centers for Disease Control and Prevention (CDC) task force looked for possible causes of a rare bleeding lung disorder in eight babies in Cleveland. The CDC explored the possibility that molds could be at fault and concluded there could be a link. The Cleveland study generated a spate of publicity, so much publicity that the CDC convened a working group to reevaluate the findings. The second working group published a report in June 1999 contradicting the Cleveland study. It was about as negative as possible in rejecting the evidence of any association between mold and infant pulmonary hemorrhage. Despite a further CDC report in 2000 also refuting the Cleveland study, "the juggernaut of media frenzy, tort lawyers, and newly-coined [mold] remediators was rolling too fast to be slowed by mere science."

Hutchinson and Powell lay out the development of mold litigation, including some significant cases with large verdicts, and point out that the proliferation of "junk science" claims that form the foundation of mold litigation ironically occurred at the same time that the U.S. Supreme Court Daubert decision laid down new guidelines that tightened the standards for scientific testimony. The Supreme Court said that federal judges need to be gatekeepers – that they have an obligation to be vigilant against "expertise that is fausse and science that is junky." The authors examine mold litigation through the Daubert microscope and argue that the serious health claims that pervade mold litigation – brain damage, lung hemorrhage, and cancer – cannot withstand scrutiny under the "reliable science" standard of Daubert.

The scientific community has not been unresponsive to the spurious nature of mold claims. Probably the most complete examination of the scientific record was conducted by Cleveland microbiologists who published their findings in January 2003, concluding that there is no supportive evidence for serious illness from toxic mold in the contemporary environment. Other studies from the American Industrial Hygiene Association and the National Institute of Occupational Safety and Health (NIOSH) came to similar conclusions. The authors note that "science has confirmed common sense" since mold is not some rare, exotic toxic material but is everywhere, making

up 25 percent of the earth's biomass. If mold were extremely toxic, one could expect to see epidemics wherever people are exposed to the highest levels of mold – vacation spots and outdoor camps, for example.

Nonetheless, the opportunism of trial lawyers and the media's love for scary stories have kept the litigation "mushrooming." The pace of litigation is increasing. The Insurance Information Institute indicates that 10,000 mold-related suits are pending nationwide, a 300 percent increase since 1999. This number may be conservative. A California plaintiffs' lawyer asserts that he has "thousands" of claims himself, including one brought by Erin Brockovich.

Hardin and his team of scientists provide a detailed primer on mold in "A Scientific View of the Health Effects of Mold." Fungi, they point out, play an "essential role in the cycle of life as the principal decomposers of organic matter, converting dead organic material into simpler chemical forms that can in turn be used by plants for their growth and nutritional needs. Without fungi performing this essential function, plant and animal debris would simply accumulate." Mold is everywhere.

The paper examines in depth each type of health complaint associated with mold and offers an extensive survey of the scientific literature on the topic. It determines that mold can cause allergies for those who are "atopic" or prone to allergic reactions. And, "despite the fact that it can produce toxic substances under appropriate growth conditions, years of intensive study have failed to establish exposure to Stachybotrys ["toxic" mold] in home, school, or office environments as a cause of adverse health effects." The paper concludes that infections caused by mold are rare, except for those individuals who are "immune-compromised." Finally, it asserts that "there is no sound scientific evidence that mold causes 'toxicity' in doses found in home environments."

A New Plague – Mold Litigation: How Junk Science and Hysteria Built an Industry

By Cliff Hutchinson and Robert Powell
Partners, Hughes & Luce LLP

A New Plague – Mold Litigation: How Junk Science and Hysteria Built an Industry

By Cliff Hutchinson and Robert Powell Partners, Hughes & Luce LLP

By Cliff Hutchinson and Robert Powell

"Biblical plague" or mundane mildew? The answer depends on whether one consults the news media or peer-reviewed science. "Toxic mold" tortmania is sweeping the country, elevating a household fungus to a public enemy. But it is a frenzy brought on by misinformation, promulgated by the mass communication of television and the Internet. How this latest plague came to pass is an interesting and evolving story.

I. INTRODUCTION

Molds are members of the Fungi family, numbering over 100,000 species. Unlike plants, which use sunlight as a source of energy, molds and other fungi contain no chlorophyll and require organic matter for food. In their digestive process, molds produce secondary metabolites including enzymes, which can be allergenic and, depending on the presence of the proper growing conditions, substances called "mycotoxins," which can be toxic if ingested. The affinity for organic matter makes mold nature's vacuum cleaner for organic substances, and, indeed, molds are ubiquitous. They need only water, organic food and oxygen to thrive. As this is also the recipe for human habitation, mold has co-existed with man since time immemorial. Even the Old Testament contains instructions for dealing with mold.[1]

Since the mid-1990s our easy relationship with household mold has undergone a sea change. Mold is seldom mentioned in the media without the precursor "toxic," and the mere sight of mildew has homeowners calling authorities, or even evacuating and abandoning their homes and furnishings, with scenes of moon-suited remediators reminiscent of a major industrial toxic spill. A confluence of hurried – and inaccurate – science and good old American entrepreneurship has created a "toxic" cottage industry out of mundane fungi.

Not that some health concerns about mold are either new or wrong. Like ragweed pollen, animal dander and house dust, molds have long been associated with allergy

1 *Leviticus* 14: 33-57.

symptoms.[2] "The common health concerns from molds include hay-fever like allergic symptoms."[3] Allergy alone, though, doesn't sell newspapers or airtime. Media interest requires more, a new and graver threat. And what's new is the unfounded claim that "toxic" mold causes much more – brain damage, cancer, bleeding lungs, and inevitably death. Pseudo-scientists and legislators[4] have been quick to climb on the bandwagon, and the human interest of a new "household death threat" has ensured publicity. Love Canal has overflowed into America's living room.

This paper reviews the extraordinary development of public hysteria over household mold. We will consider the interplay of science[5] with the role of the media and of the mold "industry," as well as the inevitable train of legal filings seeking to capitalize on the situation. Finally, in the specific legal context, we will describe the evidentiary framework that has been developed to deal with claims allegedly supported by science and apply that framework to the current state of the toxicological science of human exposure to molds.

II. SETTING THE STAGE

Mold hysteria, and the industry that feeds it and feeds upon it, did not arise overnight or in a vacuum. Instead the mold crisis arose from the synergism of a number of social and historical factors that created a "Perfect Storm" of insurance claims and litigation.[6] This phenomenon is a paradigm of how opportunistic and over-zealous regulators, businesses and attorneys can manipulate public opinion. The American public is

2 See John E. Salvaggio, et al., *Emerging Concepts In Mold Allergy: What Is the Role of Immunotherapy?*, 92(2) J. ALLERGY & CLINICAL IMMUNOLOGY 217 (1993).

3 *Questions and Answers on* Stachybotrys chartarum *and other molds*, Centers for Disease Control and Prevention, National Center for Environmental Health (March 9, 2000).

4 California enacted the Toxic Mold Protection Act of 2001 to develop standards for permissible levels of mold and for mold remediation. Congressman John Conyers has proposed "The United States Toxic Mold Safety and Protection Act," asserting that the growth of "toxic mold" is becoming a problem of "monumental proportions." *See* http://www.house.gov/conyers/mold.htm.

5 The science of mold toxicology will be addressed in detail in the companion paper by Drs. Hardin, Kelman, Robbins, and Saxon.

6 *See* Keith T. Borman and Christopher M. McDonald, *The Perfect Storm*, FOR THE DEFENSE 59 (April 2003) (Discussing factors in publicity for mass tort litigation); Randy J. Maniloff, *Mold: The Hysteria Among Us*, 14 ENV. CLAIMS J. 1, 2 (2002) ("Mold is a litigation perfect storm.").

attuned to the notion that the modern world is filled with toxic substances, foisted upon them by business and industry. They are thus primed to accept any confirmation of their fears, especially if it comes with the authority of government.

A. THE TOXIC SOCIETY

Since the rise and radicalization of the environmental movement, Americans have been bombarded with pronouncements that man-made contaminants are increasingly harming public health. As early as 1975, a United States Senator stated that 90 percent of all cancers are caused by "contaminants placed in the environment by man."[7] As Supreme Court Justice Stephen Breyer pointed out in his important book on regulation, the public's evaluation of risk from toxic exposure is skewed and differs radically from the consensus of experts.[8] Part of this reaction is fear of the unknown or the uncommon; people react more strongly to the unusual.[9] For example, they may be more anxious about a passing nuclear-waste truck than a far more dangerous gasoline truck. And the media have tended to emphasize toxic scares and exposures.

The impetus behind this media bias to skew risk assessment is that the news value of the story increases when "risks" become "hazards" and when "hazards," ultimately, become "crises."[10] Rare hazards are more newsworthy than common ones, new hazards are more exciting than old ones, and dramatic new hazards (such as Legionnaire's Disease, radon, or "toxic mold") are best of all.[11] Researchers have found that television news, in particular, tends to focus on "dramatic visual opportunities, controversial health risk information, parties who can be blamed, and opportunities for political symbolism."[12]

7 Remarks by Senator John Tunney at an October 24, 1975, Senate Commerce Committee (Subcommittee on Environment) hearing on the Toxic Substances Control Act, quoted in ROBERT LICHTER & STANLEY ROTHMAN, ENVIRONMENTAL CANCER – A POLITICAL DISEASE 54 (Yale Univ. Press 1999).

8 STEPHEN BREYER, BREAKING THE VICIOUS CIRCLE – TOWARD EFFECTIVE RISK REGULATION 33 (Harvard Univ. Press 1993).

9 *See* William Baldwin, *Where's My Face Mask?* FORBES at 18 (April 28, 2003).

10 See William C. Adams, The Role of Media Relations in Risk Communication, 37 PUBLIC RELATIONS QUAR. 28 (Dec. 22, 1992).

11 See id.

12 Michael Greenberg and Daniel Wartenberg, *Risk Perception: Understanding Mass Media Coverage of Disease Clusters*, 132 AMERICAN J. EPIDEMIOLOGY S192, S192-93 (1990).

In recent years, scares prompted by news releases and articles have affected products as diverse as fruit and plastic wrap. In 1989 the television program "60 Minutes" aired a scientifically unfounded report on the pesticide Alar. The report claimed that Alar in apples was "the most potent cancer-causing agent in our food supply," and that as many as 5,300 pre-school children could contract cancer from eating apples.[13] One Oregon mother was reportedly so panic-stricken that she called 911 to dispatch police to stop the school bus taking her child to school because she had put an apple in the child's lunch box.[14] The facts, however, were that Alar posed virtually no risk; state regulators calculated it might cause 3.5 cancer cases per 1 trillion apple eaters. But the resulting sensationalism swamped the apple market and the pesticide producer, which took Alar off the market. The irresponsible report is estimated to have cost the apple industry $100 million in Washington State alone.[15] More recently, a Consumer Reports article on the alleged danger of pesticides set off a scare regarding consumption of fruits and vegetables.[16]

Time Magazine in its March 1, 1999, issue broached the suggestive headline: "Poisonous Plastics?" The magazine then proceeded to lambaste the plastics industry based on an unsubstantiated but "growing" body of evidence that "chemicals" migrate from plastics to foods. These migrating chemicals would make consumers "very sick indeed." Faced with an outcry from responsible scientists, Time issued a retraction three weeks later.[17] But the fear factor and presumably increased magazine sales were a fait accompli. Plastics have been riding a wave of fear since scientists asserted that the hydrocarbon chemicals used in the industry "disrupted" hormone activity.[18] A 1996 Tulane University

13 Dwight Lee, *Eco-Hype Working Against the Cause*, WASHINGTON TIMES at F3 (Nov. 27, 1992).

14 Reported in Thomas L. Kurt, *Multiple Chemical Sensitivities – A Syndrome of Pseudotoxicity Manifest as Exposure Perceived Symptoms*, 33 J. TOXICOLOGY: CLINICAL TOXICOLOGY 101 (March 1995).

15 Riley E. Dunlap and Curtis E. Beus, *Understanding Public Concerns About Pesticides: An Empirical Examination*, 26(2) J. CONSUMER AFFAIRS 418 (1992).

16 *See* Michael Fumento, *Fear of Fruit*, WALL STREET J. (Feb. 26, 1999).

17 *See* Michael Fumento, *Soft Plastics, Softer Science*, WALL STREET J. (April 2, 1999).

18 *See, e.g.*, Steve F. Arnold, et al., *Synergistic Activation of Estrogen Receptor with Combinations of Environmental Chemicals*, 272 SCIENCE 1489 (1996); THEO COLBORN, et al., OUR STOLEN FUTURE: ARE WE THREATENING OUR FERTILITY, INTELLIGENCE, AND SURVIVAL? – A SCIENTIFIC DETECTIVE STORY (1996).

study reported such effects and gained enormous publicity.[19] But numerous groups tried and failed to duplicate the results and, in an extraordinary move, the Tulane scientists formally withdrew their support.[20] Today, linking industrial chemicals to endocrine effects may be "more of a social phenomenon than a scientific one."[21] Nevertheless, environmental controversy dies hard, if at all, and only this spring USA Today trumpeted the headline "Out of the Frying Pan, Into a Fire,"[22] to promote a story claiming that plastic containers, the clothing material Gore-Tex, and the Teflon lining of cooking pans are poisoning Americans.

As a result of three decades of conditioning by such media hype, society is easily alarmed by "toxic" scares. Despite statistics showing that they live in the world's safest nation, Americans are fixated on risk, "thanks to research labs, tort law and media hype."[23] Americans suffer from a toxic paranoia, believing that business or industry is out to do them in with some chemical brew, and only environmental watchdogs keep the poisoners away. The idea of "toxic" or "killer" mold simply confirms these fears. Although mold is a naturally occurring agent, it allegedly results from the exposure of man-made cellulose construction materials to moisture. So the public imagination, thoroughly conditioned, is a fertile field for the mold scare.

B. ANTHROPOMORPHISM RUN AMUCK: WHEN BUILDINGS GET "SICK"

Another important factor in the mold scare is the public's growing concern with indoor air contamination. Given environmentalist alarms about potential toxic exposures in outdoor air and water, it was only a matter of time before fears about *indoor* air arose. Arguably, concerns about indoor air quality ("IAQ" to environmental cognoscenti) were

19 *See* Jocelyn Kaiser, *Synergy Paper Questioned at Toxicology Meeting*, 275 SCIENCE 1879 (1997) (quoting government scientist as saying, "I never saw a paper have such impact.").

20 John A. McLachlan, *Synergistic Effect Of Environmental Estrogens: Report Withdrawn*, 277 SCIENCE 459 (1997).

21 *Endocrine Disruptor Fever in Japan*, 280 SCIENCE 2053 (1998) (quoting Hideyuki Kobayashi of the Japan Environment Agency).

22 USA TODAY, 1D (April 24, 2003).

23 Jane Spencer and Cynthia Crossen, *Fear Factors: Why Do Americans Feel that Danger Lurks Everywhere?*, WALL STREET JOURNAL A1 (Apr. 24, 2003).

precipitated by energy conservation measures in the 1970s. Buildings were built "tighter," with fewer air exchanges with the outside atmosphere, and air conditioning was economized such that stale air could exist.

Stale indoor air could certainly affect sensitive individuals by increasing levels of such things as cleaning solvents, dust particles, and, in residences, animal dander. Additionally, an unrelated threat to the indoor environment was highlighted in the outbreak of Legionnaire's Disease in Philadelphia that resulted in the death of 29 conventioneers. The cause was traced to bacteria present in the ductwork of a hotel. Growing publicity thus spread the idea that buildings, primarily the workplace, could be easily contaminated by toxic chemicals or microorganisms.

Based on interest and research in Europe in the late 1970s, the World Health Organization addressed indoor air quality at a meeting in Geneva in 1983 and coined the term "sick building syndrome" for a new symptom complex – dry skin, mental fatigue, headaches, and airway infections.[24] The phrase was catchy, albeit misleading, and the idea was readily accepted by an environmentally sensitized public. The syndrome was not specifically linked to mold, though. An article published by the American Association for the Advancement of Science listed a host of indoor contaminants that could be troublesome, including combustion by-products, tobacco, smoke, radon, and organic chemicals.[25] Mold was only mentioned in the group of indoor allergens including pollen, dust mites, and animal dander.[26]

The new "illness" got wide play in the press, and its notoriety grew when a number of incidents were reported in which office workers claimed a galaxy of symptoms and walked out of their workplaces. Buildings were shut down and abandoned, even the

24 Emil J. Bardana, *Sick Building Syndrome – A Wolf in Sheep's Clothing*, 79 ANNALS OF ALLERGY & IMMUNOLOGY 283 (Sept. 1997).

25 John D. Spengler and Ken Sexton, *Indoor Air Pollution: A Public Health Perspective*, 221 SCIENCE 9 (July 1, 1983).

26 *Id.*

new EPA headquarters.[27] *Newsweek* reported that "study after study" has discovered that indoor air could be laden "with a witch's brew of chemicals, gases, smoke, bacteria and other pollutants."[28] An EPA scientist told the magazine: "We're all living in a chemical soup."[29] Government regulators actually encouraged the notion of "sick buildings." The Consumer Product Safety Commission's Sandra Eberle's advice to the public: "If you believe that the air is making you sick, it probably is."[30]

The American Bar Association was quick to recognize the tort possibilities of this new "disease," and the ABA Journal touted "SBS" as an "insidious" menace killing thousands and making millions ill every year.[31] "Indoor pollution presents attorneys and the legal system with a whole new field of law," said a New York attorney.[32]

In the 20 years since the WHO designation of the new syndrome, clear-eyed scientists have shown that the "threat" of SBS is wildly exaggerated. While some incidents of actual ill effects caused by indoor pollutants occur, they are rare and generally not serious, and many incidents are more somatic than real.[33] Toxicologists investigating a "sick building" outbreak at an aircraft manufacturing plant in California found that the workers as a group had no objective evidence of physical illness but had a somatic condition characterized by anxiety and depression.[34] At Washington National Airport, reports of an airborne toxic pollutant caused hundreds to report illness. The source was traced to rotting bananas in a trash facility. A recent study by British researchers of 44 offices with 4100 workers found that "psycho-social factors" were more important

27 *See Sick Buildings*; *Danger in Living*, ECONOMIST at 31 (May 26, 1989).

28 John Carey, et al., *Beware 'Sick Building Syndrome'*, NEWSWEEK at 58 (Jan. 7, 1985).

29 *Id.*

30 *Id.*

31 Mark Diamond, *Liability in the Air: The Threat of Indoor Pollution*, 73 A.B.A.J. 78 (Nov. 1, 1987); *see also* Anthony Borden, *Environmental Law Moves Indoors*, AMERICAN LAWYER at 23 (June, 1988); Charles-Edward Anderson, *Sick-Building Syndrome: Suits Increase for Indoor Pollution Despite Absence of Favorable Verdict*, 76 A.B.A.J. 17 (Dec. 1990).

32 Quoted in Diamond, *supra* note 31, at 78.

33 Sick building syndrome has been included with such ersatz medical conditions as "multiple chemical sensitivity" under the heading of functional somatic syndromes. *See* Arthur J. Barsky and Jonathan F. Borus, *Functional Somatic Syndromes*, 130 ANNALS INTERNAL MED. 910 (1999).

34 Patricia J. Sparks, et al., *An Outbreak of Illness Among Aerospace Workers*, 153 WESTERN J. MED. 28 (July 1990).

health concerns than indoor air in the workplace.[35] After numerous controlled studies could not confirm claims about the ailment, the American Medical Association's Council on Scientific Affairs concluded that evidence for the existence of "sick building syndrome" as a separate disease entity is weak.[36]

Nevertheless, "sick building" litigation continued in the 1990s. One attorney commented at an air quality seminar, "I feel a little like an undertaker in saying this, but ... business will be very good."[37] Typical legal fees to defend a sick building case have been estimated as "close to $1 million or more."[38] And, increasingly, the claims involved allegations that mold and fungi were making the buildings "sick."[39] Conditioned to suspect the air in the workplace, Americans could now point to a specific culprit.

C. THE TRIAL LAWYER/REMEDIATOR COMPLEX

Research and media reports tinged with sensationalism and an anxious public do not, alone, fuel an ongoing panic. That requires an entrepreneurial driving force. In the case of indoor mold, the entrepreneurs were already in place and ready for new business – the trial lawyers and asbestos removal companies that had developed the multi-billion dollar asbestos remediation industry.

Asbestos, a fire retardant building material commonly-used in the U.S. until the 1970s, helped spawn modern toxic tort litigation. Medical studies showed that high levels of exposure to asbestos could lead to fatal cancers, and asbestos producers were accused of covering up the dangers of asbestos exposure to construction workers.

35 *See Sick Buildings 'A Myth'*, BUILDING DESIGN at 32 (July 11, 1997); *see also* Ronald E. Gots, *'Sick Building Syndrome': Real or Imagined*, PRIORITIES at 18 (Summer 1992).

36 *See* Sally S. Hughes and Barbara A. Holt, *Is Sick Building Syndrome for Real?*, 59(4) J. PROPERTY MANAGEMENT 32 (July 1994).

37 Ed Bas, *Sick Buildings: Legal Grist for the 90s*, 186 AIR CONDITIONING, HEATING & REFRIGERATION NEWS 32 (May 25, 1992); *see* Ed Bas, *Better Indoor Air: Another Goal of the 90s*, 212 AIR CONDITIONING, HEATING & REFRIGERATION NEWS 112 (April 30, 2001) ("floodgates" of litigation opened after federal judge awarded compensation for building-related illnesses in 1993).

38 Martha Neil, *Sick-Building Syndrome Lawsuits Virulent to Defendants*, CHICAGO DAILY LAW BULLETIN 1 (Nov. 4, 1998).

39 *See Molds, Fungi Cause Sick Building Syndrome*, 65 OCCUPATIONAL HEALTH & SAFETY 13 (Jan. 1996).

The explosion of asbestos litigation that followed in the 1970s led to economic growth in two areas.

First, lawyers, for plaintiffs and defendants, began to focus on, and grow wealthy through, asbestos litigation. Fees brought in from asbestos cases provided substantial capital, allowing plaintiffs' lawyers aggressively to advertise widely and to develop the ability to efficiently file large numbers of cases.[40] Because asbestos has not been widely used for decades, and most high exposures for workers occurred during or before World War II, asbestos claims could be expected to decline. Nevertheless new cases continue to roll in; 90,000 new asbestos lawsuits were filed in 2001 alone.[41] A recent Rand study reports that more than 8,400 companies have been named as asbestos defendants.[42] Even so, asbestos lawyers are wary of the future.[43] To fill the expected void, trial lawyers are seeking the "next asbestos" – and many think that mold fills the bill.

Second, an asbestos remediation industry sprang up to remove asbestos from tens of thousands of existing buildings. The cost to the public for what is now seen as an overreaction in the remediation area has been enormous and is, unfortunately, prognostic of the potential cost of the mold scare. *USA Today* called asbestos removal "the biggest environmental cleanup project in U.S. history," costing an estimated $50 billion. "It has forced schools to lay off teachers, caused owners to abandon buildings and added considerably to the cost of remodeling."[44] Asbestos sealed in building materials poses little or no threat, but the removal business continues to percolate to the tune of $3 billion to $4 billion per year. Experts predict another $50 billion will be spent

40 *See In re Joint Eastern and Southern Districts Asbestos Litigation*, 237 F.Supp.2d 297, 305 (E. & S.D.N.Y. 2002).

41 *Dooley, Centrist House Democrats Unveil Asbestos Legislation*, Congressional Press Release (April 10, 2003).

42 Deborah Hensler, et al., *Facts & Figures About Asbestos Litigation: Highlights from the New RAND Study* (Rand Institute for Civil Justice, January 2003).

43 After years of pleas from the courts to resolve the logjam of asbestos lawsuits, Congress is considering legislation to provide a statutory framework to deal with asbestos claims *See* Jonathon Groner, *Plaintiffs' Bar On the Ropes in Court, On Hill*, 29 CONN. L. TRIBUNE 1 (Apr. 21, 2003).

44 Dennis Cauchon, *Risk of Cancer in USA is Barely Measurable*, USA TODAY, February 11, 1999, at 1A.

on remediation before the clean up winds down in 20 years.[45] But even though asbestos remediation continues, two factors have set the industry searching for new business. First, the pace of asbestos removal is slackening.[46] The public is becoming less fearful of asbestos when it is sealed into construction materials in older buildings, and health authorities agree that it poses little, if any, threat. Second, asbestos remediation is now heavily regulated. Removal companies must be licensed and their workers specially trained and equipped. Mold removal is not so regulated, and the work can be profitable indeed, as we'll discuss below.

III. THE CDC CLEVELAND STUDIES

By the mid 1990s, the stage was set for another environmental panic, and the public was already conditioned to suspect indoor air. The trigger was a study from a governmental agency typically associated with careful scientific work, the Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia. In November 1994, a pediatric pulmonologist in Cleveland, Ohio, alerted the CDC to a cluster of eight babies suffering from a rare disorder characterized by bleeding lungs. A CDC task force looked for possible causes and observed that the houses with sick babies had water damage. The CDC scientists took samples and concluded that molds, including *Stachybotrys chartarum*, could be at fault.

A. THE MEDIA DISCOVER *STACHYBOTRYS*

The initial CDC report was cautious, as was the limited media coverage. Judith Zimomra, director of public health for Cleveland, described the report as preliminary. "These are not hypotheses, but suspicions. ... There seems to be a certain combination of factors

45 The final bill for school asbestos removal alone has been estimated at $30 billion. Peter Cary, *The Asbestos Panic Attack*, U.S. NEWS & WORLD REPORT 61 (Feb. 20, 1995).

46 The asbestos removal business peaked in 1990 and has leveled off after a decline of some 25 percent. Jim McKay, *Survival Instincts; Hard Times Behind It, PDG Environmental Looks Around for Acquisitions*, PITTSBURGH POST-GAZETTE F-5 (Oct. 26, 1997).

contributing to [the illness], but nothing is definite."[47] By the spring of 1995, though, CDC scientists were having press conferences claiming they could link the disease to *Stachybotrys*.[48] And by the fall, researchers on the project began to ratchet up their rhetoric. "The public health impact of this investigation is immense. ... Natural environmental toxins, particularly those from fungi have received little attention in the United States. Now we have evidence these toxins can and do produce a serious, sometimes deadly, illness in vulnerable infants."[49] By September 14, 1995, the Cleveland Plain Dealer could boldly announce, *"The Fungus Did It."*[50]

Nevertheless, *Stachybotrys* was anything but a household term until 1997, when the CDC issued its second report on the Cleveland studies.[51] This report was also cautious, suggesting a link with mold exposure but concluding that "further efforts are needed to clarify the association. ..." Clarification wasn't needed for television news, though, and the heightened, and carelessly phrased, publicity can be traced to 1997. ABC's "Primetime Live" featured "toxic mold" in a report aired on April 9, 1997.[52] Sam Donaldson led off: "Doctors in Cleveland have been working on a frightening medical mystery that every parent should know about ..." With the sound of a siren as background, Chris Wallace replied, "Sam, it's a terrifying and potentially fatal disease – healthy newborns suddenly bleeding from their lungs." The presentation was plainly geared to heighten fears and sensationalism and left no doubt that a link between *Stachybotrys* and infant deaths had been established. The print media did not dampen the message with hyperbolic headlines about a "Baby-Killing Fungus."[53]

47 Quoted in Evelyn Theiss, *Fungus May Hold Key to Lung Disease in Babies*, CLEVELAND PLAIN DEALER at 1A (Jan. 13, 1995).

48 *Science Update*, DALLAS MORNING NEWS at 8D (April 3, 1995).

49 Dr. Dorr Dearborn, quoted in *Natural Toxins Apparent Cause of Bleeding Lungs in Babies*, PR NEWSWIRE (Sept. 11, 1995).

50 CLEVELAND PLAIN DEALER at 12B (Sept. 14, 1995) (researchers "99.999 percent certain they've pinpointed" fungus as cause).

51 Update: Pulmonary Hemorrhage/Hemosiderosis Among Infants – Cleveland, Ohio, 1993-1996, 46(2) MORBIDITY & MORTALITY WEEKLY REPORT 33 (Jan. 17, 1997). At about the same time, scientists supporting the mold theory released another study asserting that "toxic fungi" threatened office workers. See Fungi Posing Risk to Office Workers, Water-Damaged Buildings to Blame, 27 OCCUP. HEALTH & SAFETY LETTER (Feb. 18, 1997).

52 Air of Mystery, ABC PRIMETIME LIVE (Broadcast April 9, 1997).

53 See, e.g., Katherine Rizzo, US Backs Baby-Killing Fungus Study, ASSOCIATED PRESS ONLINE (July 31, 1997).

On November 5, 1997, CNN aired a story titled "A Look at a Deadly Mold Found in Homes."[54] CNN anchor Leon Harris sonorously introduced a story about "a mold that's more than a health hazard. This one is deadly."[55] CNN editorially increased the mortality in the CDC study, asserting that all 10 exposed infants had died.[56] In the CNN story, about a family purportedly suffering health effects from a flooded basement, CNN reporter Joan MacFarlane stated flatly, and without support, "The Griffins' symptoms [breathing problems, headache, feeling ill] are common when *Stachybotrys* is present." Television, more than the print media, probably stimulated public hysteria.[57] A Staten Island woman told the New York Times she was alerted to the cause of her family's fatigue and flu-like symptoms by watching a news magazine show on television during the summer of 1997.[58]

The next year the American Academy of Pediatrics contributed to the growing concern by issuing a policy statement citing the Cleveland studies for evidence that mold caused pulmonary hemorrhage.[59] Dr. Ruth Etzel, also of the CDC, one of the authors of the pediatric paper, told the press that pediatricians should consider "toxic mold" as a cause of bleeding lungs.[60] Interestingly, her colleague at the CDC, Dr. David Mannino, was more measured, advising that "we are just not sure molds cause illness very often. There needs to be more research."[61] Thoughtful journalists and scientists cautioned that the link between mold and serious toxic diseases was not proven.[62] Dr. Harriet Burge of Harvard, a leading researcher in the area, summed it up: "Reports

54 CNN THE WORLD TODAY (Nov. 5, 1997).

55 *Id*.

56 *Id*. In the 1994 study, Dr. Dearborn reported that 10 infants were in the exposed group, of which one died, and there were 30 controls.

57 *See* David Baltimore, *SAMS – Sever Acute Media Syndrome?*, WALL STREET J. A-12 (April 28, 2003) (discussing SARS media scare) ("People fear whatever scary new thing TV shoves under their noses.").

58 Lynette Holloway, *Families Plagued by a Home-Wrecking Mold*, New York Times at 39 (Nov. 9, 1997).

59 American Academy of Pediatrics, *Toxic Effects of Indoor Molds*, 101 PEDIATRICS 712 (April 1998). The report was sponsored by the Academy's Committee on Environmental Health, which was chaired by the CDC researcher who lead the 1994 Cleveland study.

60 *Mold May Be Cause of Bleeding Lungs*, Pediatricians Told, CHICAGO TRIBUNE at 6 (April 7, 1998).

61 Quoted in Philip J. Hilts, *The Mold Scare: Overblown or Not?* NEW YORK TIMES at F3 (Oct. 23, 1997).

62 *See, e.g., id*. at F10.

of disease resulting from exposure to fungal toxins in buildings are anecdotal and generally lack sufficient data to document a clear connection between exposure and disease."[63]

B. THE CDC RETRACTS ITS REPORTS

With so much publicity arising from the 1995 report, the CDC formed two expert panels to re-examine the initial findings. The CDC convened a panel of outside experts and an internal working group, including epidemiologists and an industrial hygienist, to evaluate the Cleveland study. The working group issued a report on June 17, 1999, which was dramatic in its scientific indictment of the initial study.[64] The Cleveland researchers apparently misdiagnosed the illness of the affected infants and made numerous statistical and survey errors.[65] The illness suffered by the infants, AIPH, did not appear compatible with illnesses caused by *Stachybotrys* and is not prevalent in the flood prone areas favored by *Stachybotrys*. Moreover, the plausibility of the association was diminished by the absence of concurrent illness or symptoms of household members in the homes studied. In the muted world of scientific criticism, this report was about as negative as possible in rejecting the proposed evidence of an association between *Stachybotrys* and infant pulmonary hemorrhage.

The CDC published a further report in 2000, concluding that an association between *Stachybotrys* and pulmonary hemorrhage in infants "was not proven."[66] "Serious shortcomings in the collection, analysis, and reporting of data resulted in inflated measures of association and restricted interpretation of the reports."[67] This update

63 Harriet A. Burge, *The Fungi: How They Grow and Their Effects on Human Health*, 69 HEATING, PIPING, AIR CONDITIONING 69 (July 1997).

64 *Report of the CDC Working Group on Pulmonary Hemorrhage/Hemosiderosis* (June 17, 1999).

65 *See also* Michael Hagmann, *A Mold's Toxic Legacy Revisited*, 287 SCIENCE 243 (April 14, 2000).

66 *Update: Pulmonary Hemorrhage/Hemosiderosis Among Infants – Cleveland, Ohio*, 1993-1996, 49(09) MORBIDITY & MORTALITY WEEKLY REPORT 180 (Mar. 10, 2000).

67 *CDC: Mold Not Cause of Mid-90s Ohio Infant Deaths*, HEALTH LETTER ON THE CDC (Mar. 20, 2000).

noted, with some apparent concern, that the earlier findings had been cited in health guidelines, congressional testimony,[68] and the popular media. Unfortunately, the CDC's retraction came too late. The juggernaut of media frenzy, tort lawyers, and newly coined remediators was rolling too fast to be slowed by mere science.

C. THE ONSET OF MOLD LITIGATION

At about the time of the initial Cleveland study, the first lawsuits involving mold began to trickle in. A number of the early cases dealt with mold in the context of generalized "sick building" claims. Ironically, several lawsuits dealt with courthouses. In 1991, DuPage County, Illinois, opened its new judicial center, touted as the "Taj Mahal" of courthouses, built at a cost of $53 million.[69] A year later the building was closed due to claims by more than 450 employees of various health problems, ranging from headaches to skin rashes.[70] County officials sued the builders and designers, but a jury found that the problems were caused by the county's own poor maintenance.[71] A subsequent lawsuit by 140 employees reportedly resulted in a settlement of millions of dollars.[72]

One of the most publicized cases involved the new courthouse in Martin County, Florida, which was completed in 1989 but abandoned in 1992. After complaints by workers and visitors, the courthouse was ultimately evacuated and virtually rebuilt. Martin County adopted a remediation plan that stripped the buildings to their concrete framings and completely removed the HVAC systems.[73] The county sued the contractor

68 Dr. Dearborn testified before the House Appropriations Subcommittee on Labor, Health & Human Services & Education on February 3, 1998, in support of a request for $5 million for further investigation, asserting that *Stachybotrys* was an urgent threat: "I urge you to help us attack this newly recognized environmental hazard that is killing young infants in our communities."

69 Mark Hansen, *Toxic Torthouse?: Ailing Employees Sue Builders of New Courthouse in Suburban Chicago*, 78 A.B.A.J. 26 (Dec. 1992).

70 *Id.*

71 Kenneth M. Block, *Sick Building Syndrome Returns to the Courthouse*, N.Y.L.J. 1 (Dec. 6, 1995).

72 Alice Fabbre, *More Students Suing St. Charles East High*, CHICAGO DAILY HERALD 3 (April 4, 2001).

73 One of the defense attorneys gives a thorough discussion of the case in Robert E. Geisler, *The Fungusamongus: Sick Building Survival Guide*, 8 ST. THOMAS L. REV. 511 (Spring 1996).

and others and won a $14 million jury verdict, which was upheld in large part on appeal.[74] In one bit of good news, the county's chemistry professor expert testified that mold in the building would kill lab rats,[75] so presumably the new building was rodent free. Just across the state, in Polk County, Florida, the new courthouse was described in mold damage litigation as "a ten story, 500,000 square foot petri dish," and the county obtained a $7 million settlement from the builder and construction professionals.[76]

These large "sick building" cases drew mostly localized media attention, and, because they involved large new or remodeled buildings, they were by definition an insufficient number to fuel another mass tort wave. That required more potential cases – fact patterns that consistently reappear – and that's what the mold controversy provided. The residential environment was perfect. With a well-oiled plaintiff's toxic tort bar idling and in search of the "next asbestos," the CDC study was a godsend. Mold had all the elements for a profitable mass tort industry: an ubiquitous toxicant invading the sanctity of the home; widespread and continuing publicity; unpopular defendants (insurers, lenders, and landlords); significant injury (according to medical activists); and, potentially, huge damages.

In 1999, one of the first, and largest, landlord-tenant mold lawsuits was filed in New York. Residents of a federally subsidized East Side housing development filed an $8 billion lawsuit claiming unrepaired plumbing leaks had led to mold infestation and a variety of health problems ranging from skin rashes to memory loss.[77] A number of single-family residential cases were brought nationwide, but it wasn't clear whether these would be simply low-dollar, mundane property loss cases or something more.

74 See Centex – *Rooney Constr. Co. v. Martin County*, 706 So.2d 20 (Fla. Ct. App. 1997).

75 Greg Saitz, *Professor: Courthouse Mold Fatal to Lab Rats*, THE STUART NEWS B2 (Mar, 27, 1996).

76 Geisler, *supra* note 73, at 512.

77 See Salvatore Arena, *Mold's Toxic Tenants Say in $8B Suit*, N. Y. DAILY NEWS (May 18, 1999). Ultimately, claims totaling $12 billion were filed by the complex's residents. *U.S. Attorneys Cash In On $12Bn Mould Claim*, INSURANCE DAY (April 30, 2002).

Then in January 2001, a Texas jury provided a thunderclap answer, a $32 million verdict in what has been called the Marbury v. Madison of mold cases.[78]

In 1998 Melinda Ballard noticed that the wooden floors in her 7,400 square foot Texas hill country home had begun to buckle. When a flooring contractor was unable to solve the problem, Mrs. Ballard decided to turn it over to her insurance company, but the floors remained wet during several months of inspections. In a chance encounter on a commercial airplane, the owner of an indoor air quality contracting firm told Mrs. Ballard she probably had mold in her home. His subsequent testing reportedly revealed airborne mold spores including *Stachybotrys* – although the indoor air company admitted the presence of *Stachybotrys* in the air was the result of "aggressive testing."

The Ballard family moved from the home in late April 1999 with their toothbrushes and the clothes on their backs, leaving everything in the home, including their young son's toys. Mrs. Ballard lodged a criminal complaint with the District Attorney against the insurance company for child endangerment and, with her husband, filed a lawsuit against the insurance company for property damages and personal injury.[79] In preliminary evidentiary rulings the trial court excluded expert testimony that mold exposure caused brain damage to Ron Allison, Melinda Ballard's husband. He had begun having increasing memory problems and difficulty concentrating in the period April through July 1999; and he was diagnosed with toxic encephalopathy. Thereafter, the trial court granted summary judgment on his personal injury claims based on lack of any evidence that mold exposure was a proximate cause.

Although the trial court excluded some expert testimony linking mold to Mr. Allison's medical problems, the court allowed testimony by various other witnesses of health concerns over the toxicity of mold because it was relevant to Mrs. Ballard's mental

78 Maniloff, *supra* note 6, at 15.

79 *Ballard v. Fire Insurance Exchange*, Cause No. 99-05252, 345th Judicial District Court, Travis County, Texas.

anguish claim – not for her own fear of getting ill but her concern for her family. The problem for defense counsel in this situation was that they could not properly rebut the claimants' expert testimony for mental anguish without running the risk of "opening the door" to the very junk science evidence the trial court just excluded.

On June 1, 2001, the jury awarded approximately $32,000,000, including approximately $2,500,000 to replace the home; $1,150,000 to remediate the home prior to replacing it; $2,000,000 to replace the contents of the home; $5,000,000 for mental anguish; and $12,000,000 in punitive damages. On appeal, the trial court's admission of the expert testimony of mold toxicity for mental anguish was sustained.[80]

Ballard was only one of a number of multi-million dollar verdicts nationwide, from Delaware to California. In fact, a California verdict in the same year came in at an eye-popping $18 million.[81] In late 2001, an Arizona jury awarded a homeowner more than $4 million against an insurance company for allegedly delaying the remediation of mold contamination.[82] But what was the role of mold toxicity in these cases? After all, *Ballard* was primarily a property damage case based on insurance bad faith in connection with the total loss of a very expensive ($3 million) home. The personal injury claims were actually precluded by the *Ballard* court.

The answer is that the alleged toxicity of mold is the key to these verdicts. Even when damages for health effects are excluded, as in *Ballard*, the jury is able to consider the need to evacuate, remediate, reconstruct, or even raze, the mold-infested property. And the specter of "killer mold" certainly amplifies the mental anguish allegations that often accompany construction defect claims. An important question then for evaluating these past verdicts, and in considering future claims, is whether the scientific basis

80 Allison v. Fire Ins. Exchange, 98 S.W.3d 227 (Tex. App. – Austin 2002, pet. filed). The appellate court found no evidence to support the jury's findings of fraud and unconscionability and no evidence that the insurance company knowingly violated its duty of good faith and fair dealing. It accordingly reversed the award of mental anguish damages and punitive damages, reducing the jury's award to approximately $4,000,000, and remanded the award of attorney's fees.

81 Anderson v. Allstate Ins. Co., No. CIV-00-97 (E.D.Cal. 2001), discussed in John T. Waldron III and Timothy P. Palmer, Insurance Coverage for Mold and Fungi Claims: The Next Battleground?, 38 TORT & INS. L. J. 49 (Fall 2002).

82 Hatley v. Century-National Insurance Co., No. CV 2000-006713 (Ariz. Super. Ct., Maricopa County).

for mold toxicity in these contexts provides admissible evidence. The answer is not a close one – under the standards of science or law, evidence for these new claims of heightened mold toxicity is lacking.

IV. SCIENTIFIC EVIDENCE

Just as good science is key to an understanding of the purported health threat of household mold, so, too, good science is essential to questions of legal liability in the judicial context. The question of causation arises both in claims of personal injury for the asserted health effects of mold exposure and claims for property damage for lost value due to the presence of mold and fear of personal injury. But the courtroom is not well designed for scientific debate, and complex scientific questions have long troubled courts dealing with differing opinions among witnesses on technical and scientific issues.

Fortunately, the United States Supreme Court, in a trilogy of decisions over the last decade,[83] set firm and workable guidelines for how federal courts should address questions of scientific evidence. With the now-famous *Daubert* test, the Supreme Court recognized that the scientific method is needed in judicial analysis just as in research outside the context of litigation. For the last ten years the *Daubert* test has helped curb the abuses of unscrupulous "junk science" advocates in the Courts. The *Daubert* framework is well suited for legal review of mold claims.

A. THE PROBLEM OF "JUNK SCIENCE" IN THE COURTS

Scientific questions are resolved in litigation through opinion testimony, not through the personal knowledge of fact witnesses, the traditional ammunition of Anglo-Saxon jurisprudence. This relatively recent innovation requires the use of "expert" witnesses,

83 Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993); General Elec. Co. v. Joiner, 522 U.S. 136 (1997); Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999).

who are empowered under the federal rules to offer opinions based upon "facts or data of a type reasonably relied upon by experts in the particular field."

FEDERAL RULES OF EVIDENCE 702 and 703 govern the admissibility of expert opinion evidence and provide that a witness "qualified as an expert by knowledge, skill, experience, training, or education" may testify in the form of an opinion if "scientific, technical, or other specialized testimony will assist the trier of fact." Rule 703 allows the expert to rely on facts or data that may not otherwise be admissible.

These rules as originally drafted provided limited guidance, and courts, for years, tended to interpret them as allowing virtually any opinion testimony from a witness who qualified as an expert. And because, unlike fact witnesses, experts could be paid for their testimony, there arose a thriving business of "hired gun" testifiers who were willing to testify to whatever opinion was needed.[84] The problem became popularly known as "junk science," pseudo-scientific theories with no true scientific basis, concocted solely to support a position in court.[85]

The controversy reached its judicial zenith with the *Bendectin* cases, claims arising from a drug made by Merrell Dow and taken by pregnant women to prevent "morning sickness." This drug allegedly caused birth defects. Bendectin, however, was one of the most widely studied drugs, and more than 30 epidemiological studies failed to find any statistically significant relationship between the drug and birth defects. That did not deter some thousands of lawsuits, culminating in Merrell Dow's removing the drug from the market. The litigation continued, though, culminating in an appeal to the Supreme Court, which held that scientific opinion testimony such as that relied

84 S*ee, e.g.*, Michael H. Graham, *Expert Witness Testimony and the Federal Rules of Evidence: insuring Adequate Assurance of Trustworthiness*, 1986 U. ILL. L. REV. 43, 45 ("Today practicing lawyers can locate quickly and easily an expert to advocate nearly anything the lawyers desire."); Jack B. Weinstein, *Improving Expert Testimony*, 20 U. RICH. L. REV. 473, 482 ("An expert can be found to testify to the truth of almost any factual theory, no matter how frivolous ...").

85 *See* PETER W. HUBER, GALILEO'S REVENGE: JUNK SCIENCE IN THE COURTROOM (New York: Basic Books, 1991). Huber's influential book was cited by the Ninth Circuit in its *Daubert* remand opinion. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995).

upon in the Bendectin cases must be shown to be reliable, that is, based upon the principles of the scientific method, in order to be admitted.

B. THE TEST OF DAUBERT AND ITS PROGENY

The Supreme Court rejected a simplistic single standard, in favor of an approach that inquires whether the expert's methodology or reasoning is grounded in the procedures of science. The Court derived its authority from Rule 702's direction that expert scientific testimony be based on "scientific knowledge."[86] The Court set out a nonexclusive list of factors that bear on the inquiry:

- The "falsifiability" of the theory or technique;
- The technique's known or potential error rate and the existence and maintenance of standards controlling its operation;
- The extent to which the theory or technique has been subjected to peer review and publication; and
- The extent to which the theory or technique has been "generally accepted" within the relevant scientific community.

In a footnote the opinion referred to numerous other indicia of scientific reliability that could be applied by a reviewing court.[87] The test, however, was not limited to a precise list of factors. It required that the validity of an expert opinion be measured by the standards of the particular field of knowledge at issue. The ultimate aim was to ensure that an expert be qualified and that his or her testimony be based on a reasonable foundation.

86 *Daubert*, 509 U.S. at 593-94.

87 *Id.* at 594n.12.

In two subsequent cases, the Supreme Court further refined the *Daubert* test. *General Electric Co. v. Joiner*, 522 U.S. 136 (1997), set the standard for review — abuse of discretion — for appeals of Daubert rulings. More importantly, *Joiner* emphasized the link between an expert's methodology and conclusions. An expert might employ a well-accepted and reliable methodology yet still make an unsupported leap from that methodology to unreasonable final opinions. But a district court is not required "to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert." *Id.* at 146. Experts must explain how and why they reach their conclusions. *Id.* at 144.

Another issue left open in *Daubert* was whether it applied to non-technical experts. The breadth of *Daubert* gatekeeping was clearly defined six years later in *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), which ruled that the test applies to all expert opinion testimony. The Court emphasized that gatekeeping is an obligation of the district court, which is required to be vigilant against "expertise that is *fausse* and science that is junky."[88] The Supreme Court's defining cases and the jurisprudence that has developed since 1993[89] have established a regimen for careful review of expert opinion. And in December 2000, the Federal Rules of Evidence were amended to incorporate the *Daubert* test.[90]

C. JUDICIAL GATEKEEPING AND TOXICOLOGY

The application of *Daubert/Joiner* gatekeeping to toxicological opinion is well established. Many of the scientific evidence cases analyzed under these admissibility standards deal with toxicology, including, of course, the *Daubert* case itself. Moreover, the gatekeeping factors mirror the analysis used by epidemiologists and toxicologists in applying their science.

88 526 U.S. at 159.

89 A review of case citations on LEXIS reveals the extent of Daubert's reach in recent litigation. The case was cited in almost 3000 state and federal decisions in the last five years.

90 See Catherine Brixen and Christine Meis, Codifying the 'Daubert Trilogy': The Amendment to Federal Rule of Evidence 702, 40 JURIMETRICS J. 527 (2000).

1. Dose Makes the Poison

The fundamental principle of toxicology is that "the dose makes the poison."[91] Virtually every substance, even pure drinking water or oxygen, is toxic above a certain level or *dose*.[92] While all substances can cause harm at some level of exposure,[93] the type of harm and the level of exposure required vary widely among toxic materials.[94] The causation analysis, then, must consider a dose/response relationship. At what dose, or level of exposure, does the substance have a toxic or harmful effect? "Evidence of exposure is essential in determining the effects of harmful substances."[95] The exposure required for the toxic effect is called the *threshold level*.

Toxicologists must also be concerned with the pathway of exposure – how does the substance enter the body and reach the site of injury. This analysis asks how the substance is absorbed (ingested, inhaled, absorbed through the skin) and how it is metabolized or biotransformed into other chemicals.[96] For mold, this means that the mere presence of a species of mold on material in a home is insufficient to demonstrate exposure. Rather, it must be shown that some byproduct of the mold has actually entered the ambient air in such a manner as to come in contact with people. Finally, toxicologists must consider the toxic endpoint or *target organ*: Where does the substance or its metabolite ultimately exert its harmful effect? Toxic substances produce

91 *See* Bruce N. Ames, *Six Common Errors Relating to Environmental Pollution*, 7 REGULATORY TOXICOLOGY & PHARMACOLOGY 379, 381 (1987).

92 Medical literature has reported cases of both acute and chronic intoxication, even fatalities, from excessive water intake. *See* M. ALICE OTTOBONI, THE DOSE MAKES THE POISON 43 (2d ed. 1991). Excessive amounts of common table salt can cause stomach cancer. Philip H. Abelson, *Testing for Carcinogens With Rodents*, 249 SCIENCE 1357 (1990).

93 *See* Ottoboni, *supra* note 92, at 31, 39.

94 *Cavallo v. Star Enterprise*, 892 F. Supp. 756, 764 (E.D. Va. 1995), *aff'd in part and rev'd in part and remanded*, 100 F.3d 1150 (4th Cir. 1996).

95 Bernard D. Goldstein and Mary Sue Henifin, *Reference Guide to Toxicology* at 424 in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed. Federal Judicial Center 2000) ("REFERENCE GUIDE").

96 *See* Karl K. Rozman and Curtis D. Klaassen, *Absorption, Distribution and Excretion of Toxicants* in CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 91-92 (Curtis D. Klaassen et al. eds., 5th ed. 1996). "Identification of the routes of exposure is essential in establishing the necessary proof of exposure, which needs to be consistent with the mechanisms of disease from a toxicologic standpoint and thereby provides an underlying basis for an opinion on medical causation." Thomas W. Henderson, *Legal Aspects of Disease Clusters, Toxic Tort Litigation: Medical and Scientific Principles in Causation*, 132 AMERICAN J. EPIDEMIOLOGY S69, S72 (1990).

characteristic injury to specific target organs or tissues.[97] Courts have recognized this principle and have required proof of causation between the suspect substance and the specific alleged malady.[98]

2. "General" Causation

In evaluating any particular toxicological exposure and its potential causal link to a disease, an expert must determine "general" and "specific" causation. "General" causation refers to whether a substance is capable of causing a certain physical effect.[99] The analysis of general causation relies upon well-developed principles of the field of epidemiology. Researchers in this discipline consider whether the scientific data show that an increase in the incidence of a disease would not have occurred had the affected persons not been exposed to the substance being studied.[100] "The central concept of epidemiology is the simple, commonsense proposition that if a factor causes a disease, then people exposed to the factor should be more likely to contract the disease than unexposed people."[101]

Researchers, and the courts, look at a number of factors to determine whether epidemiologic evidence is sufficient to establish causation.[102] In general, a study of an exposed population must demonstrate that the risk of harm is doubled (a relative risk of 2.0) before a link may be shown.[103] "[T]o demonstrate a causal relationship between an environmental toxin and its effects, several well designed epidemiologic

97 A central tenet of toxicology is that "each chemical agent tends to produce a specific pattern of biological effects that can be used to establish disease causation." REFERENCE GUIDE at 403.

98 *See Porter v. Whitehall Labs., Inc.*, 9 F.3d 607, 614-15 (7th Cir. 1993, plaintiff's kidney disorder not associated with drug ibuprofen); *Hansen v. Mountain Fuel Supply Co.*, 858 P.2d 970, 973 (Utah 1993) (plaintiff's symptoms not related to exposure).

99 *Cavallo*, 892 F. Supp. at 771n. 34.

100 REFERENCE GUIDE at 374.

101 Bert Black, et al., *Guide to Epidemiology* 77 in EXPERT EVIDENCE: A PRACTITIONER'S GUIDE TO LAW, SCIENCE AND THE FJC MANUAL (Bert Black and Patrick W. Lee, eds. 1997).

102 These criteria, including the Austin Bradford Hill and Henle-Koch postulates, and numerous authorities on the topic of general causation are collected and discussed in *Merrell Dow Pharmaceuticals v. Havner*, 953 S.W.2d 706 (Tex. 1997). See also Bert Black and David E. Lilienfeld, *Epidemiologic Proof In Toxic Tort Litigation*, 52 FORDHAM L. REV. 732 (1984).

103 *Havner*, 953 S.W.2d at 716-20.

studies with sufficient power are necessary."[104]

An example of a general causation analysis in the judicial context is set out in *Grimes v. Hoffman-La Roche, Inc.*,[105] in which an ophthalmology patient sued her doctor and a pharmaceutical company, claiming her cataracts were caused by therapeutic doses of the drug Accutane. The district court applied a careful *Daubert* analysis to determine that the plaintiff's expert did not show that Accutane could, in general, cause cataracts.[106] There was thus no need for the court to go further and analyze the specific causation of the plaintiff's eye disorder.[107]

3. "Specific" Causation

"Specific" causation refers to whether the substance actually did cause the harm at issue.[108] For specific causation, it is essential that a claimant show that he was exposed to sufficient levels of a toxic substance for a sufficient duration to cause the injury sustained.[109] The importance of exposure evidence in litigation was emphasized in *Wright v. Willamette Industries, Inc.*,[110] which involved claims of injury from exposure to wood fibers and formaldehyde coming from Willamette's plant. The claimants had no evidence of any levels of exposure, however, so they could not establish whether they had encountered any hazardous amounts of the materials so as to satisfy specific causation.[111] Commentators have suggested that specific causation in a mold case should involve: (1) identification of the type of mold and mold byproduct allegedly

104 Frederick Fung, et al., Stachybotrys, *A Mycotoxin-Producing Fungus of Increasing Toxicologic Importance*, 36 J. OF TOXICOLOGY: CLINICAL TOXICOLOGY 79 (Jan. 12, 1998).

105 907 F. Supp. 33 (D.N.H. 1993). A good analysis of general causation using epidemiological principles is set forth in *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp.2d 584 (D.N.J. 2002) (rejecting general causation theory that dry cleaning fluid could cause specific form of leukemia).

106 907 F. Supp. at 38.

107 *Id.*

108 *See Amorgianos v. Nat'l Railroad Passenger Corp.*, 303 F.3d 256 (2d Cir. 2002) (claimant required to show "specific" causation that xylene actually did cause his specific alleged problems).

109 *See Abuan v. Gen'l Electric Co.*, 3 F.3d 329, 333 (9th Cir. 1993).

110 91 F.3d 1105 (8th Cir. 1996).

111 *Id.* at 1107-08.

causing injury; (2) development of specific information regarding exposure including proximity, duration and alleged exposure pathway; and (3) analysis of medical issues such as the onset or absence of symptoms relative to the alleged exposure.[112]

D. MOLD THROUGH THE *DAUBERT* MICROSCOPE

Daubert requires that the reliable scientific principles followed by epidemiologists and toxicologists in their professional practice also be applied in the courtroom. Thus in examining a mold injury claim, the trial judge must consider whether the type of mold at issue could cause, generally, the type of injury claimed. And, secondly, the court must consider whether that mold could have been inhaled in sufficient amounts to cause the specific injury to the claimant.

Mold as a cause of routine allergy symptoms among susceptible people is not disputed,[113] nor is that particularly newsworthy. Instead, the mold "scare" derives from three more serious toxicological claims – that mold in the home and workplace causes:

- Brain Damage;
- Lung Hemorrhage; and
- Cancer

These three claims have the cachet to get headlines and terrify the public. But none of them withstand scrutiny under the "reliable science" standards enforced by *Daubert.*

1. Need for Exposure Data

This first principle of toxicology applies to all the serious injury claims made in the

112 Stephen J. Henning and Daniel A. Berman, *Mold Contamination: Liability and Coverage Issues*, 8 HASTINGS W. – N.W.J. ENV. L. & POLICY 73, 91 (Fall 2001).

113 It is important to note, however, that dust mites may be a greater allergen problem in indoor air than mold. See Susan R. Tortolero, *Environmental Allergens and Irritants in Schools: A Focus on Asthma*, 72 J. SCHOOL HEALTH 33, 33 (Jan. 2002).

mold litigation. A claimant must establish that he or she has been exposed to mold spores or mycotoxins at a level and for a duration to cause the harm at issue. The initial problem is that science has established no standard or threshold level for toxicity of mold spores or mycotoxins. No toxicity level has been considered necessary because there is no credible basis to conclude that mold presents a significant public health risk.[114] And, even if such a level could be found, it would be virtually impossible to determine what amount is due to indoor mold growth versus ambient outdoor mold levels, because indoor ambient mold levels reflect a mixture of indoor and outdoor air. For example, a recent study of homes in Australia showed that 50% of the indoor mold concentrations were attributable to outdoor levels.[115]

Exposure levels cannot be extrapolated by reference to the quantity of mold growing on indoor surfaces. "There are no good guidelines for the amount of growth that is likely (or not) to result in exposure for any kind of fungus."[116] The amount of a mycotoxin produced by a mold depends on numerous variables, including the species and strain of mold, length of time the mold has been growing, the amount of available food and water, temperature, and the amount (and even wavelength) of available light.[117] The movement of mold spores also varies widely; some spores are readily airborne, but some, like *Stachybotrys*, require mechanical disturbance before entering the ambient air.[118] While many molds can produce mycotoxins as a byproduct, if the necessary conditions are present, these substances are not particularly volatile and would not be expected to be present in large quantities in the air.[119] Moreover, no procedure exists to test for the presence of mycotoxins in the air.

114 Testimony of Dr. Jack Snyder before the Texas Department of Insurance. ("I would emphasize that these standards have not been set primarily because clinical experience and the peer-reviewed literature do not support any credible objective basis for concluding that the presence of mold in homes, schools or offices presents a significant public health risk.").

115 Emil J. Bardana, *Indoor Pollution and Its Impact on Respiratory Health*, 87 ANNALS OF ALLERGY, ASTHMA & IMMUNOLOGY 33, 36 (Dec. 2001).

116 Harriet A. Burge, *Fungi: Toxic Killers or Unavoidable Nuisances?*, 87 ANNALS OF ALLERGY, ASTHMA & IMMUNOLOGY 52, 53 (Dec. 2001).

117 *Id. See* Jonathan S. Rutchick, *Evaluating Mold Exposure and Neurologic Complaints: No Easy Answers*, CLAIMS MAGAZINE 53 (March 2003).

118 Burge, *supra* note 116, at 53. See Abba I. Terr, Stachybotrys: *Relevance to Human Disease*, 87 ANNALS OF ALLERGY, ASTHMA & IMMUNOLOGY 57, 57 (Dec. 2001) (*Stachybotrys* is "difficult to find in undisturbed indoor air").

119 See Burge, *supra* note 116, at 52.

Nor can mold exposure be extrapolated from some type of physiologic testing of persons claiming the exposure. There is no "biomarker" to allow testing to determine if a person has been exposed to molds. The available blood tests have been shown to have no clinical application.[120] Even if some biomarker was identified, it could not be determined whether the effect resulted from outdoor or indoor exposure. "Ignoring these alternate sources is likely to invalidate any attempt at relating indoor fungal exposure and symptoms or disease."[121]

The notion that exposure can be postulated from symptoms, an argument made in *Ballard*, has no validity in science or law. Unless and until scientists can establish a threshold level for mold toxicity, they cannot make a reliable determination that a person has been exposed to a sufficient amount of mold to cause harm. But litigants are not likely ever to satisfy this requirement, because such a threshold level for a household mold exposure probably doesn't exist. Molds are everywhere and almost always at many times higher levels outdoors than indoors. If mold levels found in indoor air could seriously threaten health, the effect would long ago have reached epidemic proportions. As a preliminary matter, then, applying *Daubert* – and reliable toxicological science – no admissible evidentiary link can be shown between mold exposure and the types of serious illness and injury now being asserted by litigants.

2. Brain Damage

Loss of cognitive ability is a potent claim for litigants because it plays on the fears of juries and is based upon subjective measures that defy easy analysis. Claims about the neurotoxic effects of mold come from knowledge of "ergotism," a long recognized illness among animals and humans resulting from the eating of moldy grain products. Ergotism is characterized by symptoms including muscle spasms, seizures and

120 California Department of Health Services, Environmental Health Investigations Branch, Misinterpretation of Stachybotrys Serology. (Dec. 2000).

121 Bardana, *supra* note 115, at 36.

hallucinations.[122] The last major epidemic of ergotism was reported in India in 1975.[123] The leap from this rare disease caused by biologically moldy food to a claim that a similar neurotoxic effect results from inhalation of mold spores in ambient air is biologically implausible and has no scientific support. No peer-reviewed epidemiologic study has made such a connection. Indeed, the only study using valid objective testing found that the subjects claiming mold exposure performed better on neurologic tests than the control group.[124]

3. Lung Hemorrhage

After the 1994 scare that *Stachybotrys* caused pulmonary hemosiderosis, bleeding from the respiratory tract became a staple claim for litigants. But even assuming such a link to this rare disease of infants, a claim for a similar effect on adults is biologically implausible using the reasoning of the Cleveland researchers.

First, a look at the history leading to this theory. Russian scientists in the 1930s reported mucosal bleeding in horses that had eaten feed contaminated with *Stachybotrys*. The cause of the disease was isolated to a type of mycotoxin called trichothecene. But despite the widespread occurrence of *Stachybotrys*, there were few reports of human exposure to tricothecenes. In fact, one of the original Cleveland researchers pointed out in 1994 that "there is no clear association between inhalation and pulmonary hemorrhage noted in the literature."[125]

Concerns in the U.S. arose only after the Cleveland study and have been anecdotal. Moreover, Dr. Dearborn, one of the leaders of the Cleveland study, proposed that

122 *See* D.M. Kuhn and M. A Ghannoum, *Indoor Mold, Toxigenic Fungi*, and Stachybotrys chartarum: *Infectious Disease Perspective*, 16(1) CLINICAL MICROBIOLOGY REVIEWS 144, 159-60 (2003) ("There is no objective evidence for neurologic compromise caused by indoor mold exposure, in particular from *S. chartarum*").

123 *Id.* at 159.

124 M.J. Hodgson, et al., *Building-Associated Pulmonary Disease From Exposure to* Stachybotrys chartarum *and* Aspergillus versicolor, 40 J. OCCUP. ENVIRON. MED. 241, 246 (1998). The Hodgson paper studied occupants of the Martin County courthouse who were claiming injury, and was partially funded by the county. It concluded that the test results "do not support the hypothesis of lower cognitive function among cases."

125 Letter from Miriam K. Lonon to Ruth Etzel (Dec. 14, 1994), attached with the 1999 CDC Report.

Stachybotrys induced lung injury by affecting capillary fragility in rapidly growing infant lungs.[126] This mechanism could not apply to adults. In addition, a study of *Stachybotrys* in connection with a disease cluster in Chicago found a negative association between the mold and illness. The plausibility of this claim must also be tested against the physics of *Stachybotrys* exposure. At the outset, *Stachybotrys* is an unlikely lung toxicant because it produces spores "in a slimy mass that are unlikely to become airborne without dry conditions."[127] *Stachybotrys* spores are not easily disturbed, requiring "strong agitation or even direct abrasion."[128] And the spores that are finally airborne are 10 times too large to reach the lower respiratory tract in humans.[129]

The only conclusion that can be reached based on current research is that a general causal link between mold and serious lung damage is not proved sufficiently to satisfy a *Daubert* analysis. No valid epidemiologic studies make such a connection, and no reasoned proposal has been made for a biological mechanism for the alleged toxic effect.

4. Cancer

The cancer claims for mold are perhaps the most far-fetched, based on an extrapolation of the carcinogenic effects of ingested aflatoxins, which are produced in certain *Aspergillus*-contaminated food such as peanuts. But while there are over 100 toxic fungi and more than 300 mycotoxins, only two (aflatoxin and sterigmatrocystin) have been shown to cause tumors in monkeys.[130] There is only limited evidence regarding the carcenogenicity of inhaled aflatoxins.[131] The recent comprehensive medical review

126 *See* Dearborn, supra note 68.

127 Daniel L. Sudakin, Stachybotrys Chartarum: *Current Knowledge of Its Role in Diseases*, MEDSCAPE GENERAL MEDICINE (Feb. 29, 2000). "Measurements of the air in homes with extensive *Stachybotrys chartarum* growth have detected spores in the tens, not the tens of thousands per cubic meter." *The Truth About Mold*, HARVARD LAW LETTER (Jan. 2003).

128 *Clearing the Air: Asthma and Indoor Air Exposures* at 163, National Academy of Sciences, Institute of Medicine (2000).

129 Kuhn, *supra* note 122, at 159.

130 *Id.* at 161.

131 *Id.* at 162.

by Kuhn mentions three studies reporting possible associations between inhaled mycotoxins and cancer, but all were flawed. The state of the science, as reported in the Kuhn review, is that animal studies are conflicting regarding the carcinogenicity of mycotoxins generally, and no sound evidence links *Stachybotrys*–produced mycotoxins to human cancer or increased cancer risk. Given this lack of any sound toxicological or epidemiological evidence, expert opinion purporting to link indoor mold exposure to cancer must be excluded as unreliable science pursuant to *Daubert*.

V. THE MOLD CONTROVERSY POST-BALLARD

Since the "shock wave" of the $32 million verdict in *Ballard* in June 2001, legal pundits have posed the question: "Is mold still gold?"[132] The expert medical evidence for the *Ballard* plaintiff was excluded, sort of. The CDC has pulled back. Responsible scientists have spoken out on the lack of evidence for a link between mold and serious illness.[133] So what is the future for large personal injury claims? Apparently, the future is bright.

A. STATE OF THE SCIENCE

As with the Bendectin and breast implant mass tort controversies, mold lawsuits have kindled scientific interest in a thorough review of claims about mold toxicity. Probably the most complete examination of the scientific record was conducted by Cleveland microbiologists who published their findings, with some 465 references, in January 2003, concluding that there was no "supportive evidence for serious illness due to *Stachybotrys* exposure in the contemporary environment."[134] Other researchers reviewed all English language studies of indoor mold exposure from 1966 to 2002 and concluded, "specific toxicity due to inhaled fungal toxins has not been scientifically

132 *See* Stephen Pate, *Is Mold Still Gold? Personal-Injury Claims After Ballard*, 18 TEXAS LAWYER 36 (Feb. 24, 2003).

133 *See, e.g., Black Mold and Human Illness*, REPORT OF COUNCIL ON SCIENTIFIC AFFAIRS, TEXAS MEDICAL ASSOCIATION (O. Edwin McCluskey, chair, Sept. 2002) (proposition that mold can lead to illness other than infection or allergy is "an untested impression").

134 Kuhn, *supra* note 122, at 164.

established."[135] Numerous other reviews of mold toxicology concur:

- Texas Medical Association, Report of Council on Scientific Affairs – "Adverse health effects from inhalation of *Stachybotrys* spores in water-damaged buildings is not supported by available peer-reviewed reports in medical literature."[136]
- American College of Occupational and Environmental Medicine – "Current scientific evidence does not support the proposition that human health has been adversely affected by inhaled mycotoxins in home, school, or office environments."[137]
- Investigators from the National Institute for Occupational Safety and Health at the Centers for Disease Control (CDC NIOSH) – "This review of the literature indicates that there is inadequate evidence to support the conclusion that exposure to mycotoxins in the indoor (nonindustrial) environment is causally related to symptoms or illness among building occupants."[138]
- American Industrial Hygiene Association – "[A]t this time there is not enough evidence to support an association between mycotoxic fungi and a change in the spectrum of illness, the severity of illness or an increase in risk of illness."[139]

Regulatory authorities that have reviewed the issues do not warn of serious illness

135 F. Fung & W.G. Hughson, *Health Effects of Indoor Fungal Bioaerosol Exposure* 46, PROCEEDINGS: INDOOR AIR 2002, cited in Paul R. Lees-Haley, *Commentary on Neuropsychological Performance of Patients Following Mold Exposure*, THE SCIENTIFIC REVIEW OF MENTAL HEALTH PRACTICE (in press).

136 REPORT OF COUNCIL ON SCIENTIFIC AFFAIRS, *supra* note 132.

137 B. D. Hardin, et al., *Adverse Human Health Effects Associated with Molds in the Indoor Environment* (OCT. 27, 2002).

138 E. H. Page and D. B. Trout, *The Role of Stachybotrys Mycotoxins in Building Related Illness*, 62 AM. INDUSTRIAL HYGIENE ASS'N J. 644, 647 (Sept./Oct. 2001).

139 K. H. Kirkland, *Health Hazards From Exposure to Mycotoxic Fungi in Indoor Environments* 26, THE SYNERGIST (April 2001), quoted in Paul R. Lees-Haley, *Commentary on Neuropsychological Performance of Patients Following Mold Exposure*, THE SCIENTIFIC REVIEW OF MENTAL HEALTH PRACTICE (in press).

from mold. For example, the EPA in its mold guide states that: "Symptoms other than the allergic and irritant types are not commonly reported as a result of inhaling mold."[140]

Thus science has confirmed common sense. Mold is not some rare, exotic toxic material. Instead, it is everywhere and makes up an estimated 25% of the Earth's biomass. If mold were extremely toxic, one could expect to see epidemics at summer camps and outdoor vacation spots where people are exposed to the highest levels of mold. But no outdoor pandemic has been reported, nor does valid science support the claims for an indoor mold plague. Even *Consumer Reports*, usually given to environmental hyperbole, had to admit that, "there's currently no convincing evidence that [mold] causes such serious conditions as internal bleeding, memory loss, and chronic lethargy."[141]

B. MORE MEDIA MISINFORMATION

Scientists have been attempting to get this balanced and accurate information to the public. "We're trying to calm down this mold frenzy," says Dr. Harriet Burge. "I've been in this business for 40 years and molds have always been there, and none of this hysteria was going on."[142]

Nevertheless, the scientific repudiation of the mold scare has been singularly ineffective. It's just too good a story for the media to let go. A recent search on LEXIS/NEXIS found over 3,500 news stories relating to toxic mold since the start of 2000. Typical is the front page of *USA Today's Weekend* edition, which featured a full-page colorized microphotograph of *Stachybotrys* with its cover story, *"When Mold Takes Hold."* As one commentator pointed out, the USA Weekend story largely ignored the scientific

140 Environmental Protection Agency, A BRIEF GUIDE TO MOLD, MOISTURE AND YOUR HOME 3 (EPA 402-K-02-003).

141 *Health Care Savvy: Is Mold Making You Sick?*, 15 CONSUMER REPORTS ON HEALTH 10 (May 2003).

142 Quoted in Thomas Grillo, *Mold: It's Drawing Attention As a Potential Hazard, But Some Health Experts Remain Skeptical About the Risks*, BOSTON GLOBE H1 (Sept. 29, 2002).

research that questioned claims of mold toxicity.[143] The magazine's distribution, in 580 newspapers to 48 million readers, helped ensure that the sensationalized report of mold health effects was the story received by most Americans.[144] In July 2001, *Time Magazine's* headline article was *"Beware: Toxic Mold,"* warning that, "Like some sort of biblical plague, toxic mold has been creeping through houses, schools and other buildings across the U.S."[145] Mold stories also have been highlighted on the major television networks ABC (*Toxic Intruder: Black Mold Panic Has Families Fleeing Their Homes*, Nov. 29, 2002), CBS (*Silent Killers: Toxic Mold*, Sept. 28, 2000), and NBC (April 17, 2002).[146] When the misinformation in these reports gets repeated or reprinted often enough, it becomes a "fact" to be adopted without equivocation even in a scholarly publication such as a law review. For example, a recent article published in the *Arizona State Law Review* states: "Toxic molds like *Stachybotrys*, on the other hand, release noxious spores into the air until a home becomes too toxic to inhabit."[147]

More problematic is the ready availability of unfiltered and inaccurate information on the Internet. "The Internet, e-mail, and satellite-enabled saturation media coverage have put public fear on steroids."[148] A recent search on Google for "toxic mold" sources got over 50,000 hits, leading off with mold remediator advertising and including numerous mold "information" sites and mold activist and survivor groups – invariably with links to mold lawyers, mold testers, and mold remediators. The lack of balance is particularly exaggerated on Internet sites targeted to potential claimants.[149] The intent of these sites clearly is not to inform but to generate more "sporaphobia."

143 Maniloff, *supra* note 6, at 6-7.

144 *Id.* at 6.

145 Anita Hamilton, *Beware: Toxic Mold*, TIME MAGAZINE 54 (July 2, 2001).

146 *See* Shawn Adams, *Identification and Treatment of Toxic Molds*, 71 OCCUPATIONAL HEALTH & SAFETY 38 (Oct. 2002). See discussion of "must-scare TV" news reporting in Gail Pennington, *Warning: Sweeps Month May Be Bad for Your Health*, ST. LOUIS-POST DISPATCH (May 2, 2002).

147 Mike Bischoff, *Comment: Theories of Toxic Mold Liability Facing Arizona Homebuilders*, 34 ARIZ. ST. L. J. 681, 683 (2002).

148 Baltimore, *supra* note 57, at A-12.

149 See Henning, *supra* note 112, at 73-74.

C. MOLD LITIGATION MUSHROOMS

The media frenzy, despite the lack of a scientific basis, continues to fuel litigation, which touches a wide range of potential defendants. The primary targets, and the defendants with the largest verdicts thus far, are insurers. The claims, typified by those in *Ballard*, are that the carrier failed to promptly deal with a covered water damage claim, allowing mold to grow in unrepaired areas, leading to property loss and personal injury. A homeowner can also sue the builder for construction defects[150] or any of the contractors or subcontractors involved in the construction, a prior owner, the developer,[151] or the homeowner's association for improper maintenance. Presumably, real estate agents, appraisers, and home inspectors could also be brought into the fray.[152] Other, potentially lucrative targets for litigants are product suppliers and manufacturers.[153] For example, Plaintiff's lawyers have filed class action lawsuits across the country against the manufacturers of synthetic stucco, known as an Exterior Insulation Finishing System (EIFS), which allegedly allows water to seep into the wood behind the exterior finish causing structural damage. EIFS lawyers are now beginning to assert health claims for mold associated with the water damage. A "second wave" of claims is now being asserted against the companies trying to repair the damage from the first wave of water/mold loss. In *America Mutual Insurance Co. v. Henderson*,[154] the homeowner sued the companies brought in to repair the initial water damage claim, including the remediator company, the plumbing contractor, and the environmental testing company.

Claimants can also sue for mold exposure in a wide variety of public buildings –

150 *See, e.g., Pulte Home Corp. v. Smith*, 823 So.2d 305 (2002) (class action alleging construction defects leading to "toxic mold").

151 *See, e.g., Johnson-Brown v. 2200 M Street LLC*, 2003 U.S. Dist. LEXIS 5947 (D. D.C. April 8, 2003).

152 *Mold Spores: Bad Science or Bad Dream?*, 24 NAT'L L.J. B13 (Feb. 18, 2002).

153 *See, e.g., Booker v. Real Homes, Inc.*, 2003 Tex. App. LEXIS 254 (San Antonio Jan. 15, 2003) (suit against builder and manufacturer of windows). A Louisiana case illustrates how these theories are frequently being alleged concurrently. Haskal and Carol Herzog claimed that their home had been damaged by toxic mold and sued their insurer seeking coverage, the roofing contractor, a contractor that installed a vapor barrier, and the manufacturer of allegedly contaminated insulation. *See Herzog v. Johns Manville Products Corp.*, 2002 U.S. Dist. LEXIS 22187 (Nov. 15, 2002) (affirming order allowing amendment).

154 2003 U.S. Dist. LEXIS 322 (N.D. Ill. Jan. 10, 2003).

schools are a popular target, as are courthouses and other government offices. Mold is even creeping into employment law. A school employee filed a class action against the Plano, Texas, school district alleging that the high school was defectively constructed, allowing water leakage and the formation of "toxic mold."[155] The employee claimed the school district violated due process rights of school employees and students by allowing her work place to become contaminated. In a recent California case, an employee sued her employer for injuries, including headaches, skin rashes and fatigue, under a state statute, alleging that the employer concealed the presence of "toxic mold" in the laboratory where she worked.[156]

The pace of these lawsuits is apparently increasing. The Insurance Information Institute reported that 10,000 mold-related lawsuits are pending nationwide, a 300 percent increase since 1999.[157] This number may be conservative. A California plaintiffs' lawyer claims that he has "thousands" of cases himself, including one for Erin Brockovich, and asserts that mold will surpass asbestos in case volume and value.[158] Asbestos isn't being used any more, but mold is "naturally occurring, and the supply is endless."[159]

Most of the cases are being filed in states with warm, humid climates, such as Texas, but claims are increasing in northern states as well, with 400 cases reportedly pending in New York[160] and numerous cases in Pennsylvania.[161] One report claims that 70% of the cases nationwide are pending in Texas state courts, and the pace in Texas is

155 See Greene v. Plano I.S.D., 227 F. Supp. 2d 615 (E.D. Tex. 2002) (refusing to apply "state created danger" theory to mold claim).

156 See Jensen v. Amgen, Inc., 105 Cal. App. 4th 1322, 129 Cal. Rptr.2d 899 (2003).

157 Deborah Sachs Felt, Toxic Mold Litigation: The Frenzy Continues, 228 NEW YORK L.J. 4 (Dec. 6, 2002); see Lori Lichtman, Mold in Suits Springing Up in Pa., 25 PENN. L. WEEKLY 1 (Sept. 2, 2002) ("There is a fungus among us, and it's heading to court room near you.").

158 Stephanie Francis Cahill, For Some Lawyers, Mold is Gold: Toxic Troubles Translate Into Millions of Dollars for a Practice That's Bound to Grow, 87 A.B.A.J. 22 (Dec. 2001).

159 Id.

160 Felt, supra note 157, at 4.

161 Lichtman, supra note 157 (reporting one practitioner alone with 25 to 30 cases).

rapidly accelerating.[162] Mold claims have gone up over 500% since 2001; Farmers Insurance says it is getting 30 to 40 new lawsuits per week in Texas.[163] Opportunistic insureds have even begun to "cook" mold in their homes to generate claims. Last year in Houston, police charged seven people with filing fraudulent insurance claims after they deliberately flooded homes to encourage runaway mold growth.[164]

D. RISE OF THE MOLD REMEDIATORS

"Environmental concerns lead to opportunities,"[165] touts a franchise industry trade journal. As the *USA Today* story on asbestos removal observed, environmental remediation has become a multi-billion dollar industry. When ordinary construction – building, remodeling, repair – is slow, contractors know that "remediation" is steady work. And steady work with an emotional "hook." Businesses and homeowners may put off some construction work because of economics or for a variety of other reasons. But if a tenant or homeowner is terrified of some alleged toxic substance, removal must come immediately, regardless of cost. "The biggest winners are the industries feeding off mold mania."[166]

Mold, as the "next asbestos," has clearly caught the eye of remediators. Moreover, while asbestos remediation has now become standardized by regulations and asbestos remediators must be certified, no standards apply to so-called "mold remediators," or, indeed, to other indoor air "experts." "Right now, anyone with a can of disinfectant and a vacuum cleaner can claim to be an IAQ expert, ..."[167] Any contractor or repairman can simply change the logo on the side of his truck, and he's in the

162 Sue Reisinger, *Toxic Mold is Good as Gold for Plaintiffs' Lawyers*, CORPORATE LEGAL TIMES 52 (Sept. 2002).

163 *Id.*

164 *The Truth About Mold*, HARVARD LAW LETTER (Jan. 2003).

165 Todd Sloane, *Franchisees Find It's So Easy Being, Earning Green*, 3 FRANCHISE TIMES 4 (Sept. 1997).

166 Hamilton, *supra* note 145, at 55.

167 Maya Bell, *Indoor Air Gives Life to Industry; As "Sick-Building Syndrome" Spawns Complaints and Lawsuits, Those Who Seek Its Causes and Cures Multiply Quickly, Profitably – and Sometimes Improperly*, ORLANDO SENTINEL H1 (May 21, 1995).

mold removal business. The lack of standards raises the potential for fraud and significantly inflated claims for repair and remediation.[168] Mainstream industry publications now push mold remediation as "black gold ... your next profit center."[169] It is, indeed, as one commentator decried, "a gold rush of entrepreneurialism" for indoor air "solutions" involving duct cleaners, air testers, makers of cleaning devices, remodellers, in a "motley army of providers" in search of a problem.[170] Disreputable remediators are not reluctant to sensationalize their sales pitch. A Houston "mold abatement" company recently sent a broadcast facsimile to area residents with the enlarged-type admonition: "Health Warning! Mold can kill you! There is a chance that killer mold is in your home right now."[171]

The new breed of mold remediators can turn a simple plumbing problem into a Superfund site. Instead of a routine repair, workers now don masks or moonsuits; contractors do massive reconstruction, or in some cases, raze the entire structure. In Texas, routine water damage claims that were handled at a cost of $3,000 now cost "upwards of $90,000."[172] One insurer reported that almost one-third of all water claims involved an allegation of mold contamination.[173] Such inflated remediation costs contributed to a 200% increase in the total cost of water damage claims in Texas from 1999 to 2001.

Competent and reputable contractors also have problems because of the lack of statutory or regulatory standards. Contractors making a good faith effort to address claims of mold damage thus have no firm guidelines and may face liability of their own from disgruntled property owners and residents no matter what remediation

168 Testimony of Jim Oliver, Texas Windstorm Insurance Association, before the Texas Department of Insurance (June 26, 2001).

169 John R. Hall, *"Is Mold Black Gold? IAQ testing, mold remediation could be your next profit center,"* 216 AIR COND., HEATING & REFRIGERATION NEWS 1 (July 8, 2002).

170 *See* Ronald E. Gots, *"Sickbuilding Syndrome": A Diagnosis in Search Of a Disease*, 10 PRIORITIES 8 (Mar. 31, 1998).

171 Advertisement on file with the Texas Department of Insurance (filed Oct. 29, 2001).

172 Testimony of Rick Gentry, Executive Director, Insurance Council of Texas, before the Texas Department of Insurance (Sept. 13, 2001).

173 Testimony of Denise Ruggiero, State Farm Insurance Company, before the Texas Department of Insurance (Sept. 13, 2001).

methodology they employ.[174] As a result, some remediation contractors may delay work until they have a plan from an environmental engineer, with the accompanying increase in repair costs.[175]

VI. CONCLUSION

The progress of mold litigation highlights the triumph of publicity and scare tactics over fact. High profile lawsuits, like the claim by entertainer Ed McMahon that recently settled for $7 million,[176] continue to make headlines. Activists continue to hit the hustings touting the "deadly" threat of household mold, and the Internet continues to spread misinformation. The economic cost alone is enormous. The insurance industry has reported claims in the billions of dollars – insurance companies in Texas alone paid $1.2 billion in mold claims in 2001,[177] and the losses to other target defendants in the 10,000-plus lawsuits pending nationwide are incalculable. Not to be overlooked, though, is the emotional cost to credulous Americans who are encouraged to believe that they have suffered property losses and serious physical injury from a mundane and ubiquitous organism.

The mold scare looks eerily familiar to the breast implant litigation explosion that forced companies into bankruptcy and generated tens of thousands of lawsuits.[178] It took almost 10 years before impartial scientists were able to calm public fears and defuse that controversy. Query how many negative scientific reports will be necessary

174 *See, e.g.,Amica Mutual Insurance Co. v. Henderson*, No. 02 C 5193, 2003 WL 21196261 (N.D. Ill. May 15, 2003)(refusing to dismiss homeowner claim against insurer, contractor, and environmental inspector), discussed in 21 TOXIC CHEM. LIT. REP. 12 (June 12, 2003). "Qualified contractors are faced with a tremendous dilemma. As a result of the current boondoggle of mold claims, contractors now have a more difficult time servicing customers with legitimate claims. The misinformation about mold has caused an explosion in claims and lawsuits. Qualified contractors should be part of the solution instead of scapegoats in this rush to litigate." Comments of Kirk Pickerel, president of Associated Builders and Contractors.

175 Testimony of Jim Oliver, supra note 168.

176 Jean Guccione, *Ed McMahon Settles Suit Over Mold for $7.2 million*, LOS ANGELES TIMES Part 2 at 1 (May 9, 2003).

177 Sue Reisinger, *Toxic Mold is Good as Gold for Plaintiffs' Lawyers*, CORPORATE LEGAL TIMES at 52 (Sept. 2002). Thanks to the new "mold rush," Texas policyholders saw a 34.5 percent increase in premiums.

178 *Mold Spores: Bad Science or Bad Dream?*, 24 NAT'L L. J. B13 (Feb. 18, 2002). Predictably, perhaps, the media has now hyped a combination of the two claims. See Lisa Collier Cool, *Breast Implant Horror, Leaking, Scarring, Black with Mold, Why Women Are Taking Them Out*, GLAMOUR (Nov. 2000).

before the mold panic subsides. There is a lesson here, one that we have been taught before, but we seem to have difficulty absorbing. It is that in a "wired" interconnected world, communication has extraordinary and immediate power, and those with the imprimatur of scientific credentials and media access have an increasing responsibility to get the facts straight. Bad science and worse journalism are to blame for the mold fiasco. Americans deserve better.

About The Authors

Clifton T. Hutchinson
Partner
Hughes & Luce LLP

Mr. Hutchinson is a partner in the law firm of Hughes & Luce LLP, in Dallas, Texas. His practice focuses on the trial and appeal of cases involving complex technical and scientific issues, and he is a frequent speaker and author on expert evidence topics. Since 1980, he has participated in the litigation of the various "waves" of mass toxic tort cases, including asbestos, benzene, formaldehyde, and mold claims.

Mr. Hutchinson received a Bachelor of Industrial Engineering degree from the Georgia Institute of Technology in 1969 and his law degree, cum laude, from Southern Methodist University in 1980. He is admitted to practice before the United States Patent and Trademark Office and is a member of the Dallas, Texas, and American Bar Associations.

H. Robert Powell
Partner
Hughes & Luce LLP

Mr. Powell is a partner in the Austin, Texas, office of Hughes & Luce LLP. Mr. Powell earned his undergraduate degree from Baylor University in 1964 and his law degree, cum laude, from Baylor Law School in 1966. While in law school, he was Editor-in-Chief of the Baylor Law Review. From 1966 through 1969 he served in the U.S. Navy. He began his law practice in Dallas in 1970 and joined Hughes & Luce in 1975. In 1983, he moved from the Hughes & Luce Dallas office to its Austin office.

Mr. Powell has more than 30 years of experience before state and federal, trial and appellate, courts. His litigation practice has centered upon complex civil litigation and has included products liability and insurance litigation concerning alleged indoor air pollution involving formaldehyde and mold claims.

A Scientific View Of The Health Effects Of Mold

By Bryan D. Hardin, Ph.D., Andrew Saxon, M.D.,
Coreen Robbins, Ph.D., CIH, and Bruce J. Kelman, Ph.D., DABT

A Scientific View Of The Health Effects of Mold

By Bryan D. Hardin, Ph.D., Andrew Saxon, M.D., Coreen Robbins, Ph.D., CIH, and Bruce J. Kelman, Ph.D., DABT

By Bryan D. Hardin, Ph.D, Andrew Saxon, M.D., Correen Robbins, Ph.D., CIH,
and Bruce J. Kelman, Ph.D., DABT

Judging by what appears in breathless television reports and bold newspaper headlines, the nation's health is under insidious attack by a silent killer: “toxic mold.” In this paper we will discuss what is real and what is imagined in those reports.

I. INTRODUCTION

Mold is familiar to all of us in the form of growths on our bathroom tile or as fuzzy patches in food containers lost in the backs of our refrigerators. In more general terms, molds are fungi. Like plants and animals, fungi comprise one of the “Kingdoms” of living organisms on Earth. Mushrooms in our garden salad are another familiar fungus. The fungi play an essential role in the cycle of life as the principal decomposers of organic matter, converting dead organic material into simpler chemical forms that can in turn be used by plants for their growth and nutritional needs. Without fungi performing this essential function, plant and animal debris would simply accumulate. Although we tend to notice fungi only rarely, as when mushrooms sprout in our lawn or mold appears unwanted on stale bread, fungi are always with us.

When mold is visible to our eye, most of what we see are the reproductive structures of the mold colony. Mold grows as tiny, hair like filaments called “hyphae” that are not individually visible to the naked eye. An intertwining mass of hyphae, called the “mycelium,” comprises the bulk of a fungal colony but may not be visible if it is growing beneath the surface. At an appropriate stage of maturity and when environmental conditions are right, the mycelium bears specialized reproductive structures that rise above the surface to produce and release spores. Like hyphae, individual spores are not visible to the naked eye. Air currents and other disturbances carry the spores away from the colony and they eventually settle onto a surface. If that surface provides conditions suitable for growth, the spore can give rise to a new mold colony. Even in the cleanest environments, the air we breathe inevitably contains mold spores.

Molds affect human health in three ways: allergic reactions, infections and toxic, or poisonous, effects. Each of these involves fundamentally different biological

processes but some symptoms and health complaints may be similar for allergic, infectious and toxic processes.

Therefore, when making a diagnosis the medical professional must first take care to establish that mold, rather than some other factor, plays a role in the patient's condition. If mold is involved, then it must be determined whether the patient's symptoms are allergic, infectious, or toxic in origin.

This paper examines in some depth each type of health complaint by surveying the extensive scientific literature. It determines that mold can be an allergen for atopic individuals; that infections caused by mold are rare, save for immune-compromised individuals; and that there is no sound scientific evidence that mold causes “toxicity” in doses found in indoor home environments.

II. ALLERGIC AND OTHER HYPERSENSITIVITY REACTIONS

Our immune system allows us to resist infection and disease caused by organisms such as viruses, bacteria and fungi. There are two major functional divisions of the immune system: “innate” and “acquired” immunity. The first of these, innate immunity, is our bodies’ normal response to “recognizing and reject” anything foreign, including molds where there are common non-human structures.

The second division of the immune system, acquired immunity, cannot react immediately upon initial exposure but must “learn” to recognize an unfamiliar foreign material. Once stimulated by one or several exposures, the acquired immune response is highly specific to a particular challenge agent (called an “antigen”), and both the speed and the intensity of the acquired immune response may increase if exposures are repeated. It is the acquired immune system that prevents a second mumps infection and that is deliberately activated when we receive an immunization. Specific antibodies are formed that recognize a particular antigen, and a response can be triggered when only a tiny amount of that specific antigen is present.

By Bryan D. Hardin, Ph.D, Andrew Saxon, M.D., Correen Robbins, Ph.D., CIH, and Bruce J. Kelman, Ph.D., DABT

A. INDIVIDUALS AND COMMON ALLERGIES

Unfortunately, in some individuals the acquired immune system is genetically predisposed to develop exaggerated responses to common, harmless environmental exposures. These people, said to be "atopic," generate a class of specific antibodies (so called IgE antibodies) in response to these common environmental factors. These immune responses are called "immediate hypersensitivity reactions," "Type I allergic reactions," or – most commonly – "allergies."[1]

Common inhaled environmental triggers for allergic reactions include animal dander, feathers, insect proteins from body parts, grasses, pollens and molds. Foods, medications, and insect stings may be additional triggers. Atopic individuals tend to have allergic reactions not just to one environmental factor but generally have allergic responses to a number of them. For these people, reactions to inhaled allergens (antigens) can vary from relatively mild irritation of the eyes and nose to severe congestion and breathing difficulties including allergic asthma. In the extreme, an immediate hypersensitivity reaction can be life-threatening.

Of the molds that commonly grow indoors, *Penicillium* and *Aspergillus* species are the most important as allergens. However, outdoor molds such as *Cladosporium* and *Alternaria* are generally more abundant and more important than indoor molds in causing allergic airway disease. When mold spores and plant pollens are abundant in outdoor air, they may also be found at high levels in the indoor air.[2]

Depending on methods used, estimates vary as to how common fungal allergies are. At least 30% of the population may be atopic, and 20% is affected by allergic diseases

1 Burns, L.A., et al., *Toxic responses of the immune system* 355-402 in CASARETT AND DOULL'S TOXICOLOGY – THE BASIC SCIENCE OF POISONS (C.D. Klaassen et al., eds.) (McGraw-Hill, New York 1995); Evans, R., III *Epidemiology and natural history of asthma, allergic rhinitis, and atopic dermatitis* 1109-1136 in ALLERGY -- PRINCIPLES AND PRACTICE, Vol. II (E. Middleton, Jr. et al., eds.) (Mosby – Year Book, Inc., St. Louis, MO 1993).

2 *See* Horner, W.E. et al., *Fungal allergens*, CLIN. MICROBIOL. REV. 8(2):161-179 (1995); Solomon, W.R. and Platts-Mills, T.A.E., *Aerobiology and Inhalant Allergens* 367-403 in ALLERGY : PRINCIPLES AND PRACTICE (E. Middleton, Jr. et al., eds.) (Mosby Co., St. Louis 1998).

such as asthma and rhinitis (runny nose), with 10% of these individuals having severe allergic disease. Skin prick tests and in vitro tests for allergic antibodies provide accurate information as to the presence of allergic antibodies to fungal and other allergens. The prevalence of fungal allergies as determined by these methods is highly variable with reactions occurring in 3% to 91% of the population, depending on the exact population studied and the source of the challenge material used.[3]

B. UNCOMMON ALLERGIC SYNDROMES

1. Allergic Bronchopulmonary Mycosis and Allergic Fungal Sinusitis

Antigen-antibody interactions are also involved in the uncommon allergic syndromes "allergic bronchopulmonary aspergillosis"[4] and "allergic fungal sinusitis."[5,6] In these conditions, the fungi actually grow within the patient's airways (either the lungs or the sinuses). These individuals generally have airway damage from previous illnesses or other conditions that impair normal drainage. That poor drainage provides a site at which fungi can grow within the body without actually invading adjacent tissues. Such fungal colonization is without adverse health consequence unless the subject is also allergic to the specific fungus that has taken up residence. In that case there can be ongoing allergic reactions to fungal substances being released directly into the body.[7]

---

3 *See* Evans, *supra* note 1; Horner et al., *supra* note 2.

4 Fungi other than *Aspergillus* are now recognized to cause this condition in the lungs, so the term "allergic bronchopulmonary mycosis" has been suggested to replace "allergic bronchopulmonary aspergillosis."

5 The number of fungal organisms recognized as being involved in allergic fungal sinusitis is increasing, but *Aspergillus* and *Curvularia* are the most common genera.

6 Specific diagnostic criteria have been established for both allergic bronchopulmonary mycosis and allergic fungal sinusitis.

7 *See, e.g.*, Cockrill, B.A. and Hales, C.A., *Allergic bronchopulmonary aspergillosis*, ANN. REV. MED. 50:303-316 (1999); deShazo, R D. and Swain, R.E., *Diagnostic criteria for allergic fungal sinusitis*, J. ALLERGY CLIN. IMMUNOL. 96(1):24-35 (1995); Greenberger, P.A. *Allergic bronchopulmonary aspergillosis, allergic fungal sinusitis, and hypersensitivity pneumonitis*, CLIN. ALLERGY IMMUNOL. 16:449-468 (2002); Greenberger, P.A. and Patterson, R., *Diagnosis and management of allergic bronchopulmonary aspergillosis*, ANN ALLERGY 56(6):444-448 (1986); Katzenstein, A. L. et al., *Allergic Aspergillus sinusitis: a newly recognized form of sinusitis*, J. ALLERGY CLIN. IMMUNOL. 72(1):89-93 (1983); Schubert, M.S., *Fungal rhinosinusitis: diagnosis and therapy*, CURR. ALLERGY ASTHMA REP. 1(3):268-276 (2001); Schubert, M.S. and Goetz, D.W., *Evaluation and treatment of allergic fungal sinusitis, I. Demographics and diagnosis*, J. ALLERGY CLIN. IMMUNOL. 102(3):387-394 (1998); Slavin, R.G., *Allergic bronchopulmonary aspergillosis*, CLIN. REV. ALLERGY 3(2):167-182 (1985); Zhaoming, W. and Lockey, R. F., *A review of allergic bronchopulmonary aspergillosis*, J. INVESTIG. ALLERGOL. CLIN. IMMUNOL. 6(3):144-151 (1996).

2. Hypersensitivity Pneumonitis

Unrelated to immediate hypersensitivity (allergic) reactions, hypersensitivity pneumonitis or "extrinsic allergic alveolitis" is a much more severe lung condition that results from an exaggerated immune response involving a different class of immunoglobulins. The scientific evidence suggests that this immune response is limited to intensive occupational exposures or rarely home animal protein (birds particularly) or bacterial (not mold) exposures.

Development of hypersensitivity pneumonitis requires both very high blood levels of specific immunoglobulin proteins and inhalation exposure to very large quantities of foreign antigens. The inhaled antigens and their specific immunoglobulin antibodies interact to produce an intense local immune reaction.

If the exposures continue, hypersensitivity pneumonitis can progress to a disabling fibrotic lung disease. Most cases of hypersensitivity pneumonitis result from occupational exposures to high levels of organic dust that includes plant debris, fungi and bacteria; but it has also been attributed to pet birds, humidifiers and heating, ventilation, and air conditioning (HVAC) systems. In the latter two exposures, the predominant organisms responsible for hypersensitivity pneumonitis are thermophilic Actinomyces, which are not molds but rather are filamentous bacteria that grow at high temperatures.[8]

C. CHANGES IN SCIENTIFIC TESTING

Immunoglobulin antibodies classically were detected in tests that involved diffusion in flat sheets of agar gel into which holes, or "wells," have been punched. Concentrated extracts from sources suspected of causing allergies (e.g., animal dander, bird droppings,

8 Fink, J. and Zacharisen, M.C. *Hypersensitivity Pneumonitis* 994-1004 in ALLERGY: PRINCIPLES AND PRACTICE (E. Middleton, Jr. et al., eds.) (Mosby Co., St. Louis 1998); Greenberger, *supra* note 7; Lacey, J. and Crook, B., *Fungal and actinomycete spores as pollutants of the workplace and occupational allergens*, ANN. OCCUP. HYG. 32(4):515-533 (1988).

mold samples) were placed in a row of wells from which antigens can diffuse through the gel. Antibodies could have been incorporated into the gel itself, or blood serum from the patient would be placed in a row of wells parallel to those containing suspected antigen sources. If a specific antigen encountered its corresponding specific antibody in the gel, a reaction took place that was visible as a "precipitin" band in the gel. These traditional tests were highly specific but relatively insensitive; i.e., they did not detect small immunoglobulin levels. (The traditional tests do, however, remain useful in diagnosing hypersensitivity pneumonitis where high levels of immunoglobulin antibody are found.)[9]

New immunoglobulin tests called "solid phase immunoassays" are faster, easier to perform and more quantitative than the older gel diffusion tests. The new tests are also more sensitive and can detect low immunoglobulin levels, much lower than can be detected in gel diffusion tests.

Elevated immunoglobulin levels in the new solid phase immunoassay tests have less diagnostic value than do elevated levels in traditional gel diffusion tests because many people may have elevated levels of a specific immunoglobulin such that the more precise newer tests give "false-positive" results, *i.e.*, such results indicate some previous exposure to the corresponding specific antigen but do not indicate hypersensitivity pneumonitis or other disease. Because an estimated 5% of the normal population has immunoglobulin levels above reference values for any one tested material, a panel of tests against a series of mold or other antigens has a high probability of producing a false-positive result. Thus, solid phase immunoassay tests should not be used to screen for mold exposure unless there is a pre-existing clinical suspicion for hypersensitivity pneumonitis.[10]

9 *See* Homburger, H.A. and Katzmann, J.A., *Methods in laboratory immunology –Principles and interpretation of laboratory tests for allergy* 554-72 in ALLERGY –PRINCIPLES AND PRACTICE, VOL. I (E. Middleton, Jr. et al., eds.) (Mosby – Year Book, Inc., St. Louis, MO 1993).

10 *See id.; see also* California DHS, *Misinterpretation of Stachybotrys serology* (2000), available at http://www.dhs.ca.gov/ps/deodc/ehib/ehib2/topics/serologyf2.htm (May 5, 2002); Fink and Zacharisen, supra note 8; Flaherty, D.K., et al., *Multilaboratory comparison of three immunodiffusion methods used for the detection of precipitating antibodies in hypersensitivity pneumonitis*, J. LAB. CLIN. MED. 84(2):298-306 (1974).

D. THE (POTENTIAL) LINK TO MOLD

Scientific studies have shown increases in the occurrence of asthma, wheezing, coughing and phlegm, particularly in children who live in damp or water-damaged homes. Dampness may favor the growth of mold, bacteria and dust mites, each of which is a potential contributor to the respiratory illnesses associated with damp homes.[11] Notably, however, even these studies showing increases in respiratory allergies in damp homes have only established that mold and other fungi exacerbate *existing* asthmatic conditions; one cannot conclude from the evidence that mold actually *causes* asthma in previously undiagnosed individuals.[12]

Although not relevant to indoor mold exposure, it is worth mentioning that some health practitioners and members of the public hold the belief that there is a vague relationship between mold colonization, molds in foods and a "generalized mold hypersensitivity state." Adherents claim that individuals are "colonized" with the mold(s) to which they are sensitized and that they react to these endogenous molds as well as to exposures in foods and other materials that contain mold products. The proposed hypersensitivity is determined by the presence of any of a host of non-specific symptoms plus an elevated (or even normal) level of immunoglobulins to any of a host of molds.

The claim of mold colonization is generally not supported with *any* evidence, *e.g.*, cultures or biopsies, to demonstrate the *actual* presence of fungi in or on the subject. Instead, proponents often claim colonization or infection based on the presence of a

11 *See, e.g.*, Billings, C.G. and Howard, P., *Damp housing and asthma*, MONALDI ARCH. CHEST DIS. 53(1):43-49 (1998); Burr, M.L., *Health effects of indoor molds*, REV. ENVIRON. HEALTH 16(2):97-103 (2001); Flannigan, B. and Miller, J.D., *Health implications of fungi in indoor environments – an overview* 3-28 in HEALTH IMPLICATIONS OF FUNGI IN INDOOR ENVIRONMENTS (R.A. Samson et al., eds.), VOL. AIR QUALITY MONOGRAPHS, VOL 2, (Elsevier, Amsterdam 1994).

12 The Institute of Medicine's Committee on the Assessment of Asthma and Indoor Air concluded there was "sufficient" evidence that exposure to house dust mites is causally related both to the development of asthma and to the exacerbation of asthma in sensitive individuals. The Committee judged the evidence "inadequate or insufficient" that exposures to bacterial endotoxins, fungi or molds may cause the development of asthma. Evidence that exposure to fungi and molds is associated with an exacerbation of asthma in sensitive individuals was judged "sufficient." *See* IOM, *Executive Summary* 1-18 in CLEARING THE AIR – ASTHMA AND INDOOR AIR EXPOSURES (National Academy Press, Washington, D.C. 2000).

wide variety of nonspecific symptoms and antibodies detected in serologic tests that represent no more than past exposure to normal environmental fungi. The existence of this supposed disorder is not supported by reliable scientific data.[13]

III. INFECTION

Unlike "acquired immunity," the misfiring of which underlies allergic responses, the second major functional division of our immune system, "innate immunity," does not have to "learn" to recognize invading foreign organisms. Instead, it immediately recognizes certain repetitive patterns of foreign organisms as a first line of defense to ward off potential infection before it can be established. Repeated exposure to the same challenge does not produce an increasingly strong innate immune response to that challenge.[14] Following after the innate response, the adaptive (learned) immune response then develops. Both are important in protecting us from infection with molds.

A. FUNGI AND INFECTION GENERALLY

Fungi are important causes of plant diseases but rarely cause serious diseases in animals, including humans, because the innate immune system recognizes fungi as "foreign" with high efficiency and clears them from the body before most can establish an infection. Many species of fungi normally live in or on the surface of the human body without producing any ill effect, particularly so long as the normal balance of microorganisms is maintained. *Candida albicans*, for example, may be cultured from more than half of the population that has no evidence of infection.[15]

13 Blonz, E.R., *Is there an epidemic of chronic candidiasis in our midst?* JAMA 256(22):3138-3139 (1986).

14 *See* Burns, L.A. et al., *supra* note 1; Tramont, E.C., *General or nonspecific host defense mechanisms*, 33-41 in PRINCIPLES AND PRACTICE OF INFECTIOUS DISEASE (G.L. Mandell et al., eds.) (Churchill Livingstone, Inc., New York 1990).

15 *See* Tramont, *supra* note 14.

Superficial fungal infections of the skin or mucosal surfaces are common among people with normal immune systems. These superficial infections include infections of the feet (athlete's foot), nails, groin (jock itch), dry body skin (ringworm) and the oral or vaginal mucosa. Another fungal infection of the superficial layers of the skin is a condition known as "*Pityriasis (Tinea) versicolor*," in which there are patches of skin with variable pigmentation. Some of the organisms involved in superficial infections, for example *Trychophyton rubrum*, can be found growing as an indoor mold, while others, such as *Microsporum canis* and *Trychophyton mentagrophytes*, can be found on pets that live indoors. As previously mentioned, *Candida albicans* is commonly found on human mucosal surfaces, but it causes problems only if other normally resident microorganisms are eliminated or if the individual has a severely impaired immune system. Local factors such as moisture in shoes or boots and in body creases and the loss of skin integrity are important factors in development of superficial fungal infections.[16]

Blastomyces, Coccidioides, Cryptococcus, and *Histoplasma* are types of fungi that infect persons with normally functioning immune systems and they may cause fatal illnesses. However, these fungi do not grow indoors unless special conditions (*e.g.*, accumulated bird or bat droppings in an attic) provide the environment they require.[17]

B. FUNGAL INFECTIONS IN IMMUNE-COMPROMISED INDIVIDUALS

Apart from the exceptions noted above, fungal infections involving deep tissue invasion nearly always occur in persons who have severely impaired immune systems. Individuals at risk include those with blood disorders such as acute leukemia, cancer patients who are receiving intense chemotherapy, persons who are taking

16 Hay, R.J., *Dermatophytosis and other superficial mycoses*, 2017-28 in PRINCIPLES AND PRACTICE OF INFECTIOUS DISEASE (G.L. Mandell et al., eds.) (Churchill Livingstone, Inc., New York 1990); Walker, T.S., *Fungi that cause superficial, cutaneous, and subcutaneous mycoses*, 298-305 in MICROBIOLOGY (T.S. Walker, ed.) (W.B. Saunders Company, Philadelphia 1998).

17 Lenhart, S.W., et al., HISTOPLASMOSIS – PROTECTING WORKERS AT RISK, DHHS CDC National Institute for Occupational Safety and Health (NIOSH) and National Center for Infectious Diseases (NCID), NIOSH Publication No. 97-146.

immunosuppressive drugs to prevent rejection of organ transplants, people with uncontrolled diabetes and AIDS patients.

For all of these individuals, concern is greatest when they are hospitalized, at which time their immune systems may be the most severely compromised. In the hospital, intense measures are taken to avoid fungal, bacterial, and viral infections, but outside that restricted environment fungi are so ubiquitous that few protective actions can be taken beyond avoiding recognizable sources of intense fungal exposure.[18]

In sum, apart from clearly immune-compromised individuals, there is no sound scientific basis for believing that mold or other fungi in indoor environments cause infection beyond the common varieties mentioned above. Immune-compromised individuals should take special care, especially in hospitalized settings, but most individuals have little to fear infection-wise from indoor molds that over-the-counter remedies cannot cure.

IV. TOXICITY

Finally, we turn to "toxicity," the most serious poisonous effects attributed by the trial bar and common press to "toxic molds." We explore in this section the various chemical compounds molds produce, most specifically mycotoxins, which are alleged to cause numerous serious health effects in individuals exposed to indoor mold. Our survey of the extensive scientific literature on the subject leads us to conclude that there is no evidence that mold can be "toxic" at indoor exposure levels.

18 Hawkins, C. and Armstrong, D., *Fungal infections in the immunocompromised host*, CLIN. HAEMATOL. 13(3):599-630 (1984); Munoz, P. et al., *Environmental surveillance and other control measures in the prevention of nosocomial fungal infections*, CLIN. MICROBIOL. INFECT, 7 Suppl. 2:38-45 (2001); Singh, N., *Trends in the epidemiology of opportunistic fungal infections: predisposing factors and the impact of antimicrobial use practices*, CLIN. INFECT. DIS. 33(10):1692-1696 (2001); Walsh, T.J. and Dixon, D.M., *Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis and treatment*, EUR. J. EPIDEMIOL. 5(2):131-142 (1989).

A. MICROBIAL VOLATILE ORGANIC COMPOUNDS

A growing colony of mold produces many chemical substances. Some of these easily evaporate into the air and are called "microbial volatile organic compounds." These chemicals are responsible for the musty, disagreeable odor associated with mold growth. Microbial volatile organic compounds become toxic at concentrations *far* higher than are found in moldy homes, offices, or schools, but their odor is both apparent and disagreeable at very low concentrations. Microbial volatile organic compounds soon dissipate when mold growth ceases.[19]

B. MYCOTOXINS: BASIC EVIDENCE

1. Mycotoxins Generally

Many, but not all, molds are capable of producing another category of substances that are blamed for a multitude of problems and thus are the source of much of the alarm associated with molds. Called "mycotoxins," those substances are not required in the complete life cycle of the mold and are produced unpredictably even by those species ("toxigenic species") that are capable of producing them. A variety of environmental factors, including age of the colony, available nutrition and water, and competition with other species, influence whether mycotoxins are produced at all and, if produced, what kinds and to what extent. The wide variability on the part of toxigenic species to produce, or fail to produce, mycotoxins has been shown both in laboratory studies and by testing naturally growing environmental samples. Thus, even if a species of mold that is capable of producing mycotoxins grows in an indoor environment, it does not necessarily follow that mycotoxins will be produced.[20]

19 *See* Flannigan and Miller, *supra* note 11; Kaminski, E. et al., *Volatile flavor compounds produced by molds of* Aspergillus, Penicillium, *and* Fungi imperfecti, APPL. MICROBIOL. 27(6): 1001-1004 (1974).

20 Andersen, B. et al., *Characterization of Stachybotrys from water-damaged buildings based on morphology, growth, and metabolic production*, MYCOLOGIA 94(3):392-403 (2002); Burge, H.A., *Fungi: toxic killers or unavoidable nuisances*? ANN. ALLERGY ASTHMA IMMUNOL. 87(6 Suppl 3):52-56 (2001); Ciegler, A. et al. *Mycotoxins: occurrence in the environment*, 1-50 in MYCOTOXINS AND N-NITROSO COMPOUNDS: ENVIRONMENTAL RISKS, VOL. I, (R.C. Shank, ed.) (CRC Press, Inc., Boca Raton, FL 1981); Hendry, K.M. and

Unlike microbial volatile organic compounds, mycotoxins do not evaporate into the air but remain in the mold colony where they are produced and in the substrate the colony grows upon. Also unlike microbial volatile organic compounds, mycotoxins do not dissipate when active mold growth ceases. If mycotoxins are produced, they remain in place even after the mold is inactive or dead. These facts mean that when mycotoxins are produced, human exposure requires either direct skin contact with or ingestion (eating) of the moldy material, or inhalation (breathing in) of particles in the air that carry mycotoxins with them. In homes, offices and schools, only the inhalation route of exposure has potential to be meaningful.[21]

Most of what is known about the health effects of mycotoxins comes from veterinary and human illnesses caused by eating moldy foods. Episodes of acute toxicity are infrequent in developed nations, but experience demonstrates that mycotoxins consumed in the diet can cause severe, even fatal, health effects. The precise dose (amount) of mycotoxin required to produce these health effects is not known, but it seems clear that the dose in these episodes has been very high.[22]

2. Carcinogenic Mycotoxins

Several mycotoxins can induce cancer when administered to animals in laboratory

Cole, E.C., *A review of mycotoxins in indoor air*, J. TOXICOL. ENVIRON. HEALTH 38(2):183-198 (1993); Jarvis, B.B. et al., *Study of toxin production by isolates of Stachybotrys chartarum and Memnoniella echinata isolated during a study of pulmonary hemosiderosis in infants*, APPL. ENVIRON. MICROBIOL. 64(10): 3620-3625 (1998); Nikulin, M., et al., Stachybotrys atra *growth and toxin production in some building materials and fodder under different relative humidities*, APPL. ENVIRON. MICROBIOL. 60(9):3421-3424 (1994); NRC, PROTECTION AGAINST TRICHOTHECENE MYCOTOXINS, National Research Council (NRC) (National Academy Press, Washington, D.C. 1983); Rao, C.Y., *Toxigenic fungi in the indoor environment* 46-1 to 46-17 in INDOOR AIR QUALITY HANDBOOK (J.D. Spengler et al., eds.) (McGraw Hill, New York, NY 2001); Smith, J.E. et al., *Cytotoxic fungal spores in the indoor atmosphere of the damp domestic environment*, FEMS MICROBIOL. LETT. 79(1-3):337-343 (1992); Tobin, R.S. et al., *Significance of fungi in indoor air: report of a working group*, CAN. J. PUBLIC HEALTH 78(Suppl 2):S1-S14 (1987); Tuomi, T. et al., *Mycotoxins in crude building materials from water-damaged buildings*, APPL. ENVIRON. MICROBIOL. 66(5):1899-1904 (2000); Vesper, S.J. et al., *Hemolysis, toxicity, and randomly amplified polymorphic DNA analysis of Stachybotrys chartarum strains*, APPL. ENVIRON. MICROBIOL. 65(7):3175-3181 (1999).

21 *See* Hendry and Cole, *supra* note 20; Pasanen, A.-L. et al., *Laboratory experiments on membrane filter sampling of airborne mycotoxins produced by* Stachybotrys atra *Corda*, ATMOSPHERIC ENVIRONMENT 27A(1):9-13 (1993); Schiefer, H.B. Mycotoxins in indoor air: a critical toxicological viewpoint, INDOOR AIR 90:167-172 (1990); WHO, *Selected mycotoxins: ochratoxins, trichothecenes, ergot*, ENVIRONMENTAL HEALTH CRITERIA 105 (World Health Organization (WHO), Geneva, Switzerland 1990), at 30, 77, 169.

22 *See* Ciegler, A. and Bennett, J.W., *Mycotoxins and mycotoxicoses*, BIOSCIENCE 30(8):512-515 (1980); Ciegler, A. et al., supra note 20; Drobotko, V.G. *Stachybotryotoxicosis: a new disease of horses and humans*, AM. REV. SOVIET MED. 2(3):238-242 (1945); Forgacs, J. and Carll, W.T. *Mycotoxicoses*, ADV. VET. SCI. 7:273-382 (1962); Pohland, A.E. *Mycotoxins in review*, FOOD ADDIT. CONTAM. 10(1):17-28 (1993).

experiments. For example, aflatoxin B_1 produces liver cancers and ochratoxin A produces kidney tumors when fed to rats. These and other mycotoxins that are known or suspected of causing cancer are primarily associated with fungal contamination of grains and nuts; the suspected human cancer risk is associated with dietary exposure. In parts of the world where the food supply is consistently contaminated with aflatoxin or ochratoxin, there is evidence that the local human population suffers increased rates of liver or kidney cancer. However, these mycotoxins have rarely been identified as being produced by molds growing indoors.

Even if cancer-causing mycotoxins were being produced by molds growing indoors, the exposure levels and therefore any risk of cancer would be very low, and certainly lower than that encountered in dusty agricultural and grain-handling environments. Epidemiologic studies of workers with occupational inhalation exposure to dusts containing aflatoxin have suggested an increased cancer risk,[23] but these studies have had weak predictive power and were not in agreement on the specific kinds of cancer they associated with exposure. The International Agency for Research on Cancer (IARC) has evaluated the evidence pertaining to numerous mycotoxins but only the aflatoxins have been classified as human carcinogens, while others were either "possibly" carcinogenic to humans or could not be classified as to cancer risk.[24] Similarly, the U.S. National Toxicology Program classifies aflatoxin as "known" to cause human cancer and ochratoxin A as "reasonably anticipated" to cause human cancer.[25]

3. Organic Dust Toxic Syndrome

Agricultural workers who inhale high concentrations of dusts generated from silage and stored grains sometimes experience the acute illnesses "pulmonary mycotoxicosis,"

23 *See* Hayes, R.B. et al., *Aflatoxin exposures in the industrial setting: an epidemiological study of mortality*, FOOD CHEM. TOXICOL. 22(1):39-43 (1984); Olsen, J.H. et al., *Cancer risk and occupational exposure to aflatoxins in Denmark*, BR. J. CANCER 58(3):392-396 (1988).

24 IARC, SOME TRADITIONAL HERBAL MEDICINES, SOME MYCOTOXINS, NAPHTHALENE AND STYRENE 82 (2002); IARC, SOME NATURALLY OCCURRING SUBSTANCES: FOOD ITEMS AND CONSTITUENTS, HETEROCYCLIC AROMATIC AMINES AND MYCOTOXINS 56 (1993).

25 *See* REPORT ON CARCINOGENS, U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program (2002).

"grain fever," "silo unloader's lung," or "organic dust toxic syndrome." Workers experience these illnesses, a high fever with breathing difficulty, following hours of heavy dust exposure. The exact cause of these illnesses has not been established, but they appear not to be allergic in nature. The mixed dusts the farm workers are exposed to contain the organic debris from the stored agricultural products along with fungi and bacteria and their associated mycotoxins, endotoxins and glucans. Dust exposures associated with these illnesses are high enough to obscure clear vision, and spore counts have been measured in the range of 1 billion to 10 billion spores per cubic meter of air, more than 1000 times higher than the most extreme conditions encountered in a moldy indoor environment.[26]

C. "SICK BUILDING SYNDROME"

"Sick building syndrome" and "non-specific building-related illness" are terms applied when a variety of symptoms are alleged to be associated with occupancy in a building. Symptoms are reported to be reduced outside of the building. No specific illness can be diagnosed and symptoms reported often are sensory, such as dry or itching eyes, nose, or throat, disagreeable odors, or tightness of the chest and other breathing difficulties.

Careful investigation often finds no specific cause for the complaints, but mold growth is blamed if it is found.[27] Numerous recent reviews of the scientific literature on sick building syndrome have concluded that levels of microorganisms in the

26 *See* Brinton, W.T. et al. *An outbreak of organic dust toxic syndrome in a college fraternity*, JAMA 258(9):1210-1212 (1987); Di Paolo, N. et al., *Inhaled mycotoxins lead to acute renal failure*, NEPHROL DIAL TRANSPLANT 9 Suppl 4:116-120 (1994); Emanuel, D.A. et al., *Pulmonary mycotoxicosis*, CHEST 67(3):293-297 (1975); Lacey and Crook, supra note 8; Malmberg, P. et al., *Exposure to microorganisms associated with allergic alveolitis and febrile reactions to mold dust in farmers*, CHEST 103(4):1202-1209 (1993); May, J.J. et al., *A study of silo unloading the work environment and its physiologic effects*, AM. J. IND. MED. 10:318 (1986); *Preventing organic dust toxic syndrome*, NIOSH Alert National Institute for Occupational Safety and Health (NIOSH), NIOSH Publication No. 94-102 (1994); Pratt, D.S. and May, J. J. *Feed-associated respiratory illness in farmers*, ARCH. ENVIRON. HEALTH 39(1):43-48 (1984).

27 See Croft, W.A. et al., *Airborne outbreak of trichothecene toxicosis*, ATMOSPHERIC ENVIRONMENT 20(3):549-552 (1986); Hodgson, M.J. et al., *Building-associated pulmonary disease from exposure to* Stachybotrys chartarum *and* Aspergillus versicolor, J. OCCUP. ENVIRON. MED. 40(3):241-249 (1998); Jarvis, B.B., *Mycotoxins and indoor air quality* 201-14 in BIOLOGICAL CONTAMINANTS IN INDOOR ENVIRONMENTS (P.R. Morey et al., eds.) (ASTM, Philadelphia 1990); Johanning, E. et al., *Health and immunology study following exposure to toxigenic fungi* (Stachybotrys chartarum) *in a water-damaged office environment*, INT. ARCH. OCCUP. ENVIRON. HEALTH 68(4):207-218 (1996); Johanning, E. et al., *Clinical-epidemiological investigation of health effects caused by Stachybotrys atra building contamination*, PROCEEDINGS OF INDOOR AIR '93 - HEALTH EFFECTS 1:225-230 (1993).

indoor air are only weakly correlated with symptoms.[28]

D. *STACHYBOTRYS* AND THE CENTERS FOR DISEASE CONTROL REPORTS

Much of the present alarm over indoor mold exposure can be traced to a 1993-1994 series of cases of pulmonary hemorrhage among infants in Cleveland, Ohio. Those cases were investigated by the Centers for Disease Control and Prevention (CDC) and the initial reports did not suggest any cause for the cases studied. Later, these investigators suggested that the cause had been the presence of a particular mold, Stachybotrys chartarum, in the infants' homes. Because no illness was seen in adults and other children living in the same homes, they also suggested that very young infants might be unusually vulnerable.[29] In the immediate aftermath, despite the intense national attention to these cases in Cleveland, no new clusters and only a few additional isolated case reports have been published by independent investigators,[30] although the original investigators continue to add to their case series.[31] Nationwide surveillance by the CDC has failed to identify either clusters or isolated cases that meet the CDC's definition of "acute idiopathic pulmonary hemorrhage in infants" (AIPHI).[32]

28 *See* Burge, *supra* note 20; Fung, F. et al., Stachybotrys, *a mycotoxin-producing fungus of increasing toxicologic importance*, J. TOXICOL. CLIN. TOXICOL. 36(1-2):79-86 (1998); Kuhn, D.M. and Ghannoum, M.A., *Indoor mold, toxigenic fungi,* and Stachybotrys chartarum*: infectious disease perspective*, CLIN. MICROBIOL. REV. 16(1):144-172 (2003); Menzies, D. and Bourbeau, J., *Building-related illnesses*, N. ENGL. J. MED. 337(21):1524-1531 (1997); Page, E.H. and Trout, D.B., *The role of Stachybotrys mycotoxins in buildings related illness*, AM. IND. HYG. ASSOC. J. 62(5):644-48 (2001) ; Robbins, C.A. et al., *Health effects of mycotoxins in indoor air: a critical review*, APPL. OCCUP. ENVIRON. HYG. 15(10):773-784 (2000); Terr, A.I., Stachybotrys: *relevance to human disease*, ANN. ALLERGY ASTHMA IMMUNOL. 87(6 Suppl 3):57-63 (2001); Tobin et al., supra note 20.

29 *See* CDC, *Update: pulmonary hemorrhage/hemosiderosis among infants – Cleveland, Ohio, 1993-1996*, MMWR MORB. MORTAL WKLY. REP. 49(9):180-184 (2000); CDC, *Update: pulmonary hemorrhage/hemosiderosis among infants--Cleveland, Ohio, 1993-1996*, MMWR MORB. MORTAL WKLY. REP. 46(2):33-35 (1997); CDC, *Acute pulmonary hemorrhage/hemosiderosis among infants – Cleveland, January 1993-November 1994*, MMWR MORB. MORTAL WKLY. REP. 43(48):881-883 (1994); Etzel, R.A. et al., *Acute pulmonary hemorrhage in infants associated with exposure to* Stachybotrys atra *and other fungi*, ARCH. PEDIATR. ADOLESC. MED. 152(8):757-62 (1998); Montana, E. et al., *Environmental risk factors associated with pediatric idiopathic pulmonary hemorrhage and hemosiderosis in a Cleveland community*, PEDIATRICS 99(1):1-8 (1997).

30 *See* Elidemir, O. et al., *Isolation of Stachybotrys from the lung of a child with pulmonary hemosiderosis*, PEDIATRICS 104(4 Pt 1):964-966 (1999); Flappan, S.M. et al., *Infant pulmonary hemorrhage in a suburban home with water damage and mold* (Stachybotrys atra), ENVIRON. HEALTH PERSPECT. 107(11):927-930 (1999); Novotny, W.E. and Dixit, A., *Pulmonary hemorrhage in an infant following 2 weeks of fungal exposure*, ARCH. PEDIATR. ADOLESC. MED. 154(3):271-75 (2000); Tripi, P.A. et al., *Acute pulmonary haemorrhage in an infant during induction of general anaesthesia*, PAEDIATR. ANAESTH. 10(1):92-94 (2000).

31 *See* Dearborn, D.G. et al., *Clinical profile of 30 infants with acute pulmonary hemorrhage in Cleveland*, PEDIATRICS 110(3):627-637 (2002).

32 *See* Brown, C.M., Medical Epidemiologist, Air Pollution and Respiratory Health Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, E-mail message to Hardin, B.D., Subject: AIPHI (May 15, 2003); CDC, *Availability of case definition for acute idiopathic pulmonary hemorrhage in infants*, MMWR MORB. MORTAL. WKLY. REP. 50(23):494-495 (2001).

When questions arose regarding the validity of the suggestion that exposures in the home to *Stachybotrys* caused the Cleveland cluster of AIPHI cases, the CDC conducted a detailed internal reevaluation of the original data and also convened a panel of outside experts to evaluate the original study and its conclusions. These re-evaluations led the CDC to publish a statement that Stachybotrys had not been shown to be the cause of the series of pulmonary hemorrhages in Cleveland,[33] and other reviewers have also concluded that the available scientific evidence does not establish Stachybotrys (or other indoor mold) as the cause of AIPHI.[34]

E. MYCOTOXINS INDOORS AND HEALTH EFFECTS: EMPIRICAL RESULTS

Stachybotrys is only one of a number of molds that can grow indoors and that are capable of producing mycotoxins, but as previously described neither Stachybotrys nor any other toxigenic species always produces mycotoxins. Mycotoxins clearly can be produced by molds growing indoors on building materials, but little has been published regarding the amount of mycotoxins produced or the environmental factors that regulate their production under those conditions.

Most of the published information on mycotoxin production by molds was developed under the artificial conditions of mold growing on defined or semi-defined media in the laboratory, and the mold strains used often were standard laboratory strains known to be good producers of mycotoxins. Only rarely were strains brought to the laboratory from an indoor environment. When toxigenic molds have been grown on building materials (for example, wood, gypsum board, insulation, etc.) the amount of mycotoxin produced has been undetectable or low relative to amounts produced on defined or semi-defined media. These facts reinforce the notion that the mere presence

33 *See* CDC (2000), *supra* note 29.

34 *See* Hardin, B.D. et al., *Adverse human health effects associated with molds in the indoor environment*, J. OCCUP. ENVIRON. MED. 45(5):470-78 (2003); Kuhn and Ghannoum, supra note 28; Page and Trout, *supra* note 28; Sudakin, D.L., Stachybotrys chartarum: *current knowledge of its role in disease*, MEDGENMED :E11 (2000).

of a toxigenic mold species is not evidence that mycotoxins are present.[35]

1. The Basic Requirements for Toxicity in Fungal Exposure

If mycotoxins are to have human health effects in the indoor environment, three conditions must all be met. First, there must be an actual presence of mycotoxins. As we have just seen, that cannot be assumed on the basis of a toxigenic species being present. Second, there must be a pathway of exposure from the source to the susceptible person. As we have also seen, mycotoxins do not evaporate into the air. Exposure requires generation of airborne particles to carry the mycotoxin from the source into the breathing air of people in the area. Third, the amount of mycotoxin inhaled must be sufficient to cause toxicity. In other words, a toxic dose must be delivered.

2. An Assessment of the Scientific Literature with an Emphasis on Dose

"Dose" is a fundamental concept in the science of toxicology, but we all have a basic understanding of the concept from our daily lives. We all understand that too much of anything can be harmful. A medication taken as prescribed, *e.g.*, one pill three times daily, is beneficial, whereas all of the pills consumed at once could be fatal. We also understand from our daily lives that it generally takes more to affect a large person than a small person. A single alcoholic beverage might make a small person lightheaded, whereas it might require two or three drinks to have the same effect on a larger person. Toxicologists acknowledge that familiar concept by expressing dose as the amount taken in divided by body weight (mg/kg). The question is, if mold growing indoors produces mycotoxins and if spores or other particles containing mycotoxins are present in the breathing air, can a toxic dose of mycotoxins be inhaled?

35 *See* Jarvis, *supra* note 27; Nikulin et al., supra note 20; Ren, P. et al., *Comparative study of* Aspergillus *mycotoxin produced on enriched media and construction material*, J. INDUSTRIAL MICROBIOL. BIOTECHNOL. :210-213 (1999); Tobin et al., *supra* note 20.

That question has not been addressed directly in scientific publications, but it is possible to make calculations that suggest the minimal numbers of particles in the air that would be required. Single doses of *Stachybotrys* spores, known to contain mycotoxins, have been directly introduced into the noses of mice[36] and lungs of rats.[37] A variety of doses were used with the rats, and their condition was monitored using sensitive laboratory methods.

Severe effects were produced when the higher doses were placed directly into rat lungs, but there were no effects at the lowest dose used, which was 3 million spores per kilogram of body weight (3,000,000 spores/kg). Those spores were placed all at once directly into the lungs, but we can calculate how many spores would have to be in the air in order to inhale the same dose of spores over a 24-hour period. Since it has been hypothesized that small infants are especially vulnerable we can use standard reference values for the body weight and breathing rate of infants: according to the EPA, 95% of all one-month-old infants weigh more than 3.16 kg (7 pounds) and infants under one year of age breathe approximately 4.5 cubic meters (m^3) of air per day.[38] In order to inhale 3,000,000 spores/kg over a 24-hour period of continuous exposure, such an infant would have to be exposed to over 2 million spores per cubic meter of air (2,000,000 spores/m^3). Still higher spore concentrations would be required for the average school-aged boy (over 6 1/2 million spores per cubic meter) and for the average adult man (over 15 million spores per cubic meter). Thus, absorbing even the dose that caused no ill effects in rats would require numbers of airborne spores that vastly exceed the numbers actually seen even in heavily mold-contaminated homes, offices, or schools.

36 *See* Nikulin, M. et al. *Experimental lung mycotoxicosis in mice induced by* Stachybotrys atra, INT. J. EXP. PATHOL. 77(5):213-218 (1996).

37 *See* Rao, C. Y. et al. *Reduction of pulmonary toxicity of* Stachybotrys chartarum *spores by methanol extraction of mycotoxins*, APPL. ENVIRON. MICROBIOL. 66(7):2817-2821 (2000); Rao, C. Y. et al. *The time course of responses to intratracheally instilled toxic Stachybotrys chartarum spores in rats*, MYCOPATHOLOGIA 149(1):27-34 (2000).

38 *See* EPA, EXPOSURE FACTORS HANDBOOK, Update of May 1989 Office of Research and Development, US Environmental Protection Agency (EPA), Washington, DC 20460, EPA/600/P-95/002Fa, Washington, DC (1997).

The comparison is even more dramatic if we remember that the spores were introduced instantaneously and directly into the lungs of the rats. A sudden, direct application of that kind overwhelms the normal processes that protect the lungs by removing foreign materials, either by physically carrying them away or by changing them chemically. We can think of the rat experiments as representing a dose rate of 3 million spores per kilogram of body weight per minute (even though the spores were administered in much less than one minute). To make a more direct comparison not just with the total dose but with the dose rate that had no ill effects on rats, we can calculate how many spores would have to be in the breathing air to deliver 3 million spores per kilogram body weight in a one-minute exposure. For the small, one-month-old infant, that concentration would have to be 3 billion spores per cubic meter of air. The average school-aged child would have to be exposed for one minute to over 9 billion spores per cubic meter of air and the average adult to 22 billion spores per cubic meter of air.

Other studies have been done in which mycotoxin-containing *Stachybotrys* spores were introduced directly into the nasal passages of mice two times a week for three weeks.[39] Only two doses were used, and effects were seen with both. The higher dose caused severe inflammation and bleeding in the lungs visible at the end of three weeks. In contrast, the lower dose, which was 46,000 spores per kilogram body weight at each treatment, produced mild inflammation and no bleeding in the lungs of the treated mice. Combining all six treatments, the lower dose totaled 280,000 spores per kilogram body weight. We can calculate how many spores would have to be in the breathing air to deliver that dose over a three-week period of continuous, 24-hour per day exposure. For the 3.16-kilogram one-month-old infant, that concentration would be 9,400 spores per cubic meter of air. The average school-aged child would have to be exposed continuously for three weeks to over 29,000 spores per cubic meter of air, and the average adult to 68,000 spores per cubic meter of air.

39 *See* Nikulin, M. et al., *Effects of intranasal exposure to spores of* Stachybotrys atra *in mice*, FUNDAM. APPL. TOXICOL. 35(2):182-188 (1997).

Even though this calculation ignores the more severe impact expected from repeated instantaneous dosing (as compared to slow, continuous intake by natural breathing), it still produces a concentration of *Stachybotrys* spores in air that would not be seen in real home, school, or office settings. For example, in data from 9,619 indoor air samples from 1,717 buildings, *Stachybotrys* was detected in the indoor air of 6% of the buildings. In those 103 buildings, the median ("middle," meaning half of values were lower and half were higher) airborne concentration was 12 colony-forming units (viable spores) per cubic meter of air.[40]

The calculations above suggest minimum airborne concentrations of *Stachybotrys* spores (with all of them actually containing mycotoxins) that are required to achieve doses in humans that would be equal to doses that had essentially no effect in artificially exposed animals. These calculations do not tell us where to draw a line between "safe" and "unsafe" conditions, but they do make it clear that it would be difficult to deliver a toxic dose of mold toxins by inhaling spores in the indoor air. We can be confident that it is nearly impossible for anyone to inhale a harmful dose of mold toxins in homes, offices, or schools because even the most heavily contaminated of them have total spore concentrations that are far lower than the values calculated.

V. CONCLUSION

Molds are common and important allergens. During their lifetime, about 5% of individuals can be expected to have some allergic breathing reactions caused by molds. However, molds are not the most important allergens that sensitive people encounter in their lives, and for these allergic individuals outdoor molds are more important than indoor molds. For almost all allergic people, reactions will be limited to itchy, watery eyes and nose or asthma. To avoid making mold allergies worse, or

40 *See* Shelton, B.G. et al., *Profiles of airborne fungi in buildings and outdoor environments in the United States*, APPL. ENVIRON. MICROBIOL. 68(4):1743-1753 (2002).

developing them in the first place, it makes sense not to allow the unchecked growth of mold indoors.

Infections by molds and other fungi are rarely significant causes of disease in humans. Superficial fungal infections of the skin and nails are relatively common in normal individuals, but those infections can be treated effectively and they generally resolved without complications. The fungi which cause superficial infections are not the type which grow on building materials. Only a small number of fungi, *Blastomyces, Coccidioides, Cryptococcus,* and *Histoplasma*, cause serious disease in individuals who have normally functioning immune systems. Fortunately, those fungi do not grow in indoor environments, but their spores may come indoors with outdoor air if there is a source near open windows or building air intakes. Individuals who have severely impaired immune systems are at significantly increased risk of serious fungal infections. However, fungi are so common and ever-present that avoidance of fungi is very difficult even in the confines of hospital isolation units.

Some of the molds that can grow indoors are capable of producing toxic substances, mycotoxins, under appropriate conditions of growth. Adverse health effects caused by molds and mycotoxins have been recognized for centuries when contaminated foods have been eaten. Occupational diseases are also well known in association with inhalation of high levels of fungi, bacteria, and other organic matter in industrial and agricultural settings. However, despite a great number of studies spanning two decades, there is no proven association between indoor mold exposure and various nonspecific health complaints.

The mold *Stachybotrys chartarum* has come to cause particular fear when it is found indoors. However, the studies that stimulated that fearful response have been disavowed by the CDC, and no similar studies have been published by others. Despite the fact that it can produce toxic substances under appropriate growth conditions, years of intensive study have failed to establish exposure to *Stachybotrys* in home, school, or

office environments as a cause of adverse human health effects. Actual levels of exposure in indoor environments, dose-response data in animals, and dose-rate considerations all make it highly unlikely that a toxic dose of mycotoxins can be delivered by inhalation of indoor air.

Mold spores are present in all indoor environments and cannot be eliminated from them. Normal building materials and furnishings can support the growth of many species of molds, but only if there is an adequate supply of moisture. Where mold grows indoors there is an inappropriate source of water. Mold growth should be reduced to the extent practicable in the home, school or office, since it physically destroys the building materials on which it grows; it is unsightly and may produce offensive odors; and it is likely to sensitize and produce allergic responses in those who are susceptible.

Nevertheless, except for persons with severely impaired immune systems, indoor mold is not a source of fungal infections, and current scientific evidence does not support the idea that human health has been adversely affected by inhaled mold toxins in home, school, or office environments. Thus, the notion that "toxic mold" is an insidious, secret "killer," as so many media reports and trial lawyers would claim, is "junk science" unsupported by actual scientific study.

About The Authors

Dr. Bryan D Hardin
GLOBALTOX

Bryan D. Hardin, Ph.D., holds positions as a senior consultant with GlobalTox and Adjunct Assistant Professor at the Rollins School of Public Health, Emory University. He was commissioned into the US Public Health Service and began his public health career with the National Institute for Occupational Safety and Health (NIOSH) in 1972, where he served in research, policy, and management roles, culminating as Deputy Director of NIOSH and Assistant Surgeon General in the Public Health Service.

Dr. Hardin holds a Ph.D. in Environment Health Sciences from the University of Cincinnati. Dr. Hardin is a full member of the American Association for the Advancement of Science, the American Industrial Hygiene Association, the American Public Health Association, and the Teratology Society. He has served on working groups of the World Health Organization, the International Labor Office, and the International Agency for Research on Cancer.

Coreen A. Robbins, Ph.D., C.I.H.
GLOBALTOX

Coreen A. Robbins, M.H.S., Ph.D., CIH, holds a position with GlobalTox, Inc. as a consulting Industrial Hygienist for projects in field investigations and in litigation support activity. She has approximately 13 years of experience in industrial hygiene and has served as a consultant in many investigations throughout the U.S.

Dr. Robbins holds a master's degree in Occupational Safety and Health (1989), and a Ph.D. (1995) in Environmental Science from the Johns Hopkins University. Dr. Robbins is also a Certified Industrial Hygienist (CIH). Dr. Robbins has extensive practical experience in conducting industrial hygiene surveys in areas including indoor air quality, mold, asbestos and man-made mineral fibers, chemical exposure assessment and industrial noise exposure. Dr. Robbins is a full member of the American Academy of Industrial Hygiene and the American Industrial Hygiene Association (AIHA), and an affiliate member of the American Conference of Governmental Industrial Hygienists. She is currently serving on the AIHA's Task Force on Microbial Growth as the representative for the AIHA Toxicology Committee.

Andrew Saxon
Chief, Division of Clinical Immunology and Allergy
UCLA School of Medicine

Andrew Saxon, MD, is a professor and Chief of the Division of Clinical Immunology and Allergy at the UCLA School of Medicine. Dr. Saxon has over 25 years of experience in immunology, he has published approximately 165 peer-reviewed research articles, and he has three patents in the immunology field. Since 1999, Dr. Saxon has served as editor-in-chief of the journal Clinical Immunology.

Dr. Saxon received his MD from Harvard Medical School. He is board-certified in Internal Medicine, Allergy and Immunology, and Diagnostic Laboratory Immunology. He is a member of the American Academy of Allergy and Immunology, where he serves on the Research Awards Committee, the Nominating Committee, the Primary Immunodeficiency Disease Committee and the Clinical and Diagnostic Immunology Committee; and where has served in the past as Chairman of the Basic and Clinical Immunology Section.

Dr. Bruce J. Kelman
GLOBALTOX

Bruce J. Kelman, Ph.D., D.A.B.T., holds positions as Principal and President of GlobalTox, Inc. Dr. Kelman has approximately 25 years experience in toxicology and has served as a consultant and expert in numerous investigations across North America. He has evaluated numerous claims of personal injury and health impacts from many chemicals and drugs, and has presented a variety of health risk concepts to policy makers, government regulators, citizen groups, and individuals involved in all aspects of the legal process.

Dr. Kelman holds a Ph.D. from the University of Illinois (1975) and is certified in toxicology by the American Board of Toxicology (original certification in 1980 with recertifications in 1985, 1990, 1995 and 2000).Dr. Kelman is a member of the Society of Toxicology, American College of Occupational and Environmental Medicine, American College of Toxicology, American Society for Experimental Pharmacology and Therapeutics, Society for Experimental Biology and Medicine, and Teratology Society.

U.S. Chamber Institute for Legal Reform
1615 H Street, N.W.
Washington, D.C. 20062-2000
ph. 202-463-5724 fax: 202-463-5302

www.LegalReformNow.com