
DEFENSE CONSIDERATIONS IN MOLD LITIGATION:

INVESTIGATION, CAUSATION, MITIGATION & REMEDIATION

By: DALE O. THORNSJO and SHAMUS P. O'MEARA

JOHNSON & CONDON, P.A.



These materials are intended to provide a general overview of the topic. The views expressed in the following pages are not necessarily those of the co-authors, Johnson & Condon, P.A. or their clients.

This is the first of a two - part article dealing with mold litigation issues. Part I provides an overview of mold, and discusses issues involved in personal injury claims which seek to causally link mold's presence and the claimant's medical condition. Part II, which will appear in the next issue of *Minnesota Defense*, discusses property damage considerations when mold is present in a building. Part II will analyze building integrity issues as part of the indoor air quality environment, remediation of mold-related damages, use of appropriate professionals, measure of damages, and spoliation of evidence, among other items.

DALE O. THORNSJO and SHAMUS P. O'MEARA are partners at the suburban Minneapolis law firm of Johnson & Condon, P.A. Mr. Thornsjo focuses his practice on the defense and coverage issues raised by complex toxic tort, environmental and food contamination matters. Mr. O'Meara practices in the construction, recovery, public sector and indoor air quality areas. The authors are frequent lecturers at national and state forums on mold and indoor air quality, and have written extensively on the subject.

INTRODUCTION

Historically, concern about air quality and its effects on human biology focused primarily on outdoor air pollution. Through the 1970s, air quality debates focused on city smog and the effects of acid rain deforestation. However, numerous factors such as science's ability to detect smaller and more minute levels of materials in air and water, a more focused application of advanced medicine to public health, a greater acceptance of stricter building and construction standards and codes, and a more "toxic tort" litigation environment, have shifted air quality discussions from the atmosphere to the air inside the structures where the American public spends as much as 90 percent of its time.¹

This evolution has given rise to a wide divergence of opinion about how, if at all, indoor air quality impacts public health and property values. Certain advocates contend that people, particularly the young, the old, the chemically sensitive, or those who are suffering from asthma or immune deficiency problems, become ill from exposure to indoor air contaminants such as mold (fungi), bacteria, chemicals embedded in construction materials, tobacco, and office and cleaning products. Phrases such as "Sick Building Syndrome" or "Building Related Illness" have been coined to describe the phenomenon of illness due to questionable indoor air quality. Buildings with "lower" indoor air quality are alleged to cause occupant absences, a lack of productivity of the persons who otherwise use the structure, and increased health care costs.

Others stress that, while there may be merit to some of these observations when applied to certain isolated circumstances, any widespread effects are, at worst, temporary, and end when the persons involved leave the structure in question. Therefore, the epidemic claimed by some is likely exaggerated, overblown, and driven by incentives unrelated to health.

Mold is the latest substance of widespread concern in the indoor air quality debate. Mold is naturally occurring and ubiquitous. Mold can occur in buildings as a result of

¹U.S. Environmental Protection Agency, Office of Air and Radiation, *Report to Congress on Indoor Air Quality, Volume II: Assessment and Control of Indoor Air Pollution*, pp. 1, 4-14. EPA 400-1-89-001C, 1989. See, also, *Indoor Pollutants*, Report of Committee on Indoor Pollutants, Board on Toxicology and Environmental Health Hazards, Assembly of Life Sciences, National Research Council (1981) (on average, employed men spend 90% of day (21.7h) indoors, whereas married housewives spend 95% of the day (22.8h) indoors).

such common events as an increase in the level of humidity, premature sprinkler activation, poor construction practices which result in water infiltration, or poor heating, ventilation and air conditioning (HVAC) system maintenance. Media reports of "heightened" levels of certain types of molds present on the surfaces or in the air of energy efficient buildings have "sensitized" (but not necessarily medically) the public in a way not seen since asbestos or lead. The mere presence of mold in a building, while possibly being a sanitary issue, does not usually create a public

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health or property damage concern. Mold nonetheless does have the potential to alter a claim's character from one of simple water infiltration to one demanding as much in compensation as was expended to construct the building.

From a defense perspective, mold cases:

"ha[ve] become particularly troublesome because the defendants in these claims are not the chemical companies, who are experienced and well-equipped to deal with such personal injury suits. Rather, defendants include builders, subcontractors, architects, municipalities and homeowner insurers whose attorneys have rarely dealt with toxic injury allegations. Furthermore, the science of these matters is sufficiently convoluted and complex to confuse even the most experienced physicians, most of whom have never heard of mold toxins, and seasoned attorneys. These cases, moreover, are being championed by prolific authors whose scientific positions are often extreme, but whose extensive writings and lectures have engendered respect in this relatively new and unpopulated field."²

The following pages, as well as Part II of this article, address certain defense considerations raised by mold cases, and provide some suggestions on how to focus the mold analysis on insight, not innuendo, and science instead of speculation. In the end, from a personal injury perspective, the issues raised in defending the mold case are no different than those seen in other personal injury and toxic tort cases. The defenses should be pursued just as aggressively in mold cases as they are on other cases.

²Gots, R.E., *Mold and Mold Toxins: The Newest Toxic Tort*, 8 Journal of Controversial Medical Claims 1 (February 2001).

DOES A MOLD "PROBLEM" EXIST?

Mold growth in a corner of a room where moisture has infiltrated does not immediately translate into a toxic indoor air quality problem. However, there are occasions where a closer, and potentially extensive and thorough, investigation may be required to determine the scope and extent of a mold problem. Depending on the extent of the growth, and the type of claims being asserted, investigation, medical workup and/or remediation are best handled by professionals with specialized knowledge and experience in the implicated areas.

An accurate assessment of whether thousands of dollars in expert investigation, medical and remediation costs are required begins with a functional understanding of mold, how it occurs, and what its effects are on the body and the property involved.

MOLD 101

Mold is a type of fungus. Fungi are multi-cellular organisms that feed on and decompose organic material.³ Fungi are very similar to plants in that they are usually associated with soils. However, fungi are considered sufficiently unique to constitute their own kingdom. Approximately 70,000 species of fungi have been positively identified, and include mushrooms, mildew, mold, and puff balls. Fungi are important to the environment. They decompose dead organic materials, recycle nutrients back into the ecosystem, and help in the production of food, antibiotics, and other chemicals.

Molds themselves have been described as:

"... simple, microscopic organisms, found virtually everywhere, indoors and outdoors. Molds can be found on plants, foods, dry leaves, and other organic material. Molds are needed for breaking down dead material. Mold spores are very tiny and lightweight, and this allows them to travel through the air. Mold growths can often be seen in the form of discoloration, ranging from white to orange and from green to brown and black. When molds are present in large quantities, they can cause allergic symptoms similar to those caused by plant pollen."⁴

³Fungus is defined as any of a group of thallophytic plants (phylum *Thallophyta*) mainly characterized by an absence of chlorophyll. Van Nostrand's SCIENTIFIC ENCYCLOPEDIA (5th ed. 1976).

⁴Indoor Air Quality Info Sheet, *Mold in My Home: What Do I Do?*, California Department of Health Services. This publication may be found at www.dhs.ca.gov/ps/deode/ehlb/iaqs/molds.htm. See, also, *Molds in the Environment*, Center for Disease Control and Prevention (CDC) -- National Center for Environmental Health (NCEH) -- Factsheet (April 3, 1997).

Because mold is ubiquitous, it is found in all indoor and outdoor environments. The type and concentration of mold spores present varies due to different environmental conditions. Molds names are technical and difficult to pronounce. In addition to those described in the media such as *Stachybotrys*, *Aspergillus* and *Fusarium*, molds have names such as *Alternaria*, *Bipolaris*, *Chaetomium*, *Cladosporium* and *Penicillium*, among others.

Even though molds play a positive role in the ecosystem, in large doses they can potentially cause harm to plants, animals, and humans. Given the right situations, molds can grow on or in buildings and furnishings, which in turn creates the possibility that building occupants might be exposed to excessive levels of fungal spores, their bioeffluents and certain mycotoxins which exceed background outdoor levels.

Some mold strains produce large, complex molecules called mycotoxins. These metabolites have the potential, under certain circumstances, to produce a toxic reaction. While over 300 mycotoxins (including tricothecenes) are known, how they are produced is poorly understood. Production could be affected by fungal strain, genetic susceptibility of the host plant or commodity, moisture content, temperature, aeration, microbial population, and stress factors.⁵ Therefore, even if a potentially toxigenic mold is present, there is a serious question whether mycotoxins have been produced, or if they have been released into the air or into the breathing zone of an occupant.⁶

Penicillin is manufactured from a mycotoxin produced by *Penicillium*. *Aspergillus flavus* and *Aspergillus parasiticus* produce the mycotoxin aflatoxin B1. Aflatoxin B1 has been seen in foods, and therefore is found in the human diet. Many foods in several food groups contain some level of aflatoxin B1 (vegetables [peas and corn ears], starches and grains [bread, rice, grain, sorghum and wheat] and dairy [cheese and milk]). Aflatoxin has been reported as a cause of liver cancer. However, these mycotoxins do not seem to be of concern.

Stachybotrys appears to be the mold of greatest interest to public health officials. *Stachybotrys* is black and slimy in

texture, and requires an ongoing water source, or extremely high humidity, to grow. Due to its texture, *Stachybotrys* typically does not become airborne; however, if detected in the air it is possible that the strain has been present for some time. *Stachybotrototoxicosis* is the disease resulting from high exposures to this type of mold. Early *Stachybotrototoxicosis* was reported in the 1930s in Eastern Europe and Russia in association with hemorrhaging horses.⁷ Straw containing mold also reportedly caused skin rashes, bloody noses, and breathing problems for farm workers. However, recent analysis of the medical literature involving *Stachybotrys* (and other mycotoxin-producing molds) has shown that there is no scientifically established link between a building occupant's exposure and human toxic illness.⁸

In addition, some types of molds and fungi are capable of producing various volatile organic compounds (VOCs) such as alcohols, ketones, hydrocarbons and aromatics. These microbial VOCs, (MVOCs) can produce the distinctive musty "mold" odors smelled when large amounts of mold is present. However, mere odor does not mean the exposure is *per se* toxic.

How Mold Growth Occurs

In most cases, mold in buildings arises either from moisture intrusion, or from condensation in mechanical ventilation systems or building envelopes. The moisture then combines with a number of conditions that must exist for mold and fungal growth to occur on building materials. These conditions include an appropriate temperature and oxygen availability (in similar levels enjoyed by humans), and a nutrient source. These nutrient sources include wood, paper on sheetrock, other building materials produced from organic materials, or even skin flakes, soap residue, plants and food products. Since modern construction techniques have and will continue to use materials which in part are manufactured from mold's organic food sources, the most effective mold prevention technique is to eliminate moisture sources.

Once growth occurs, the mold spores can be disseminated into the indoor environment either by physical disturbance or through natural air currents. Because fungi can grow on substances with very low moisture content,

⁵ Ciegler, A., "Fungi that Produce Mycotoxins: Conditions and Occurrence." *Mycopathologia* 65:5011 (1978), cited in Page, E.H. and Trout, D.B., *The Role of Stachybotrys Mycotoxins in Building-Related Illness*. AIHAJ 62:644-648 (2001).

⁶ Gots, *supra* at note 2.

⁷ Forgacs, J. "Stachybotrototoxicosis. Microbial Toxins." In *Fungal Toxins*, Kadis, S.; Ciegler, A.; Ajl, S.J. eds. Academic Press, New York (1972), 95-128.

⁸ Page, E.H. and Trout, D.B., *The Role of Stachybotrys Mycotoxins in Building-Related Illness*. AIHAJ 62:644-648 (2001).

uncontrolled removal of fungal contamination can lead to additional growth and further bioamplification.

IS MOLD CAUSING PERSONAL INJURY?

Simply because mold is a “new” issue does not mean that traditional tests of medical reliability should be abandoned. While levels of mycotoxins and molds can be measured with great accuracy in many cases, the mere presence of mycotoxins or molds does not mean that any symptom is, by definition, caused by mold. To date, science has not been able to establish specific threshold levels (with one exception relating to eye and throat irritation) beyond which symptoms or illness will occur. However, it appears that, like many other substances, there is some dose-response relationship between exposure to molds (an amount over time) and the body’s reaction to the substance.

The Body’s Reaction to Mold

Adverse health effects from massive doses of mold are not new. Historic mold exposure health concerns were limited to specific occupations or exposure situations. A number of hypersensitivity diseases were named for exposure in specific occupations, such as Farmers Lung (*Aspergillus Umbrosus/Fumigatus*), Maize Grain Hypersensitivity (*Aspergillus Flavus*), and Woodworkers Lung (*Alternaria*).

Currently, there are three types of physiologic responses which, from an academic standpoint, relate to mold. The two seen most often in personal injury litigation are allergic/immunologic responses, and toxigenic responses. Less frequently seen in the current litigation are infectious/pathogenic responses.

It is claimed that common allergic conditions can be exacerbated by, and possibly caused by, exposure to the allergens contained in or on the mold spores. These conditions include allergic rhinitis, sinusitis, asthma, hypersensitivity pneumonitis and allergic skin diseases. Mold allergens can persist for years, whether the spore itself is alive or dead. It is not uncommon for allergens to persist, as seen most often with cat dander, housedust mites, and cockroach or rodent allergens. Whether mold allergens cause, as opposed to exacerbate, allergic reactions or sensitizations in building occupants is questionable, at least as to the general population.⁹

Infectious diseases such as Histoplasmosis and Aspergillosis have been recorded as a result of exposure to certain mold organisms, especially in certain highly susceptible populations. Typically, these infections will be limited to persons with suppressed immune systems, transplant patients, or young children or elderly who are frail. However, since the typical personal injury plaintiff does not fit within this profile, it is expected that few infectious claims would be a part of a typical building occupant mold claim.

Finally, it is theoretically possible to have a toxic reaction as a result of mycotoxin exposure. Many mycotoxins produced from mold have been identified as potentially harmful to humans and animals when inhaled, ingested, or coming into contact with skin. A wide variety of symptoms have been potentially associated to toxic effects of mold, including rashes, flu-like symptoms, headaches, fatigue, and central nervous symptom disorders. However, because health effects of mold exposure are dependent on various factors, the simple presence of mold cannot entitle a person to compensation for personal (or emotional) injuries.

Is the Plaintiff’s Environment the Cause of the Claimed Illness?

Indoor air quality is affected by climate outside of the building, the location of the building on the particular property, the building’s architecture and mechanical systems, how the building was constructed, and the materials and people in the building.¹⁰ The term “Sick Building Syndrome” has been defined by the Environmental Protection Agency (EPA) as describing “situations in which building occupants experience acute health and comfort effects that appear to be linked to time spent in a building, but no specific illness or cause can be identified.”¹¹ In contrast, the term “Building Related Illness” is defined as “symptoms of diagnosable illness which are identified and can be attributed directly to airborne building contaminants.”¹²

Sick building cases are characterized by the occupant alleging a variety of conditions including headaches, eye, nose, or throat irritation, dry cough, dry or itchy skin, dizzi-

⁹ See generally, Bardana, E.J., *Sick Building Syndrome – a Wolf in Sheep’s Clothing*. Ann. Allergy, Asthma & Immunol. 79:283-294 (1997).

¹⁰ Indoor Air Quality Tools for Schools, IAQ Coordinator’s Guide at Environmental Protection Agency § 5 (Understanding IAQ Problems) (March 23, 1998).

¹¹ *Id.*

¹² Indoor Air Facts No. 4 (revised), Sick Building Syndrome, Office of Radiation and Indoor Air (6607J) (April 1991).

ness and nausea, difficulty in concentrating, fatigue, and sensitivity to odors. The cause of symptoms is claimed to be unknown, and most of complainants report relief soon after leaving the building.¹³ The usual difference between this description of sick building cases and mold cases is that mold is claimed to be the cause of the symptoms.

Admissibility of Medical General and Specific Causation Evidence: The *Daubert* Factors

As with all tort cases, a personal injury Plaintiff cannot recover unless the Plaintiff proves causation. "In tort law, 'causation' is the linchpin joining substance and disease."¹⁴ Numerous physical conditions are alleged to be "by" exposure to various mold strains. However, to prove causation, a Plaintiff must prove: (1) that mold *generally* causes personal injuries of the type claimed by the Plaintiff; *and* (2) that mold caused the *specific* injuries involved in the case.¹⁵ To meet these proofs, a Plaintiff must call an expert who will provide relevant and reliable testimony. "Without expert testimony supporting causation, the cause of action typically fails."¹⁶

Admissibility of novel or questionable medical causation evidence will turn in Federal Courts on whether the opinion satisfies the *Daubert* standard.¹⁷ In making this assessment, the Court acts as a "gatekeeper" to make sure that, not only is the evidence reaching the fact finder relevant, but reliable.¹⁸

In performing this function, the Court weighs several factors to ascertain whether the opinion was derived by the "scientific method." While not a conclusive list, it appears these factors will constitute the major considera-

tions the trial court will give to expert admissibility issues in order to determine whether the expert can account for the "how and why" the challenged opinion was reached,¹⁹ and applied the same "intellectual rigor" the expert would exert in their field of expertise.²⁰

The first factor considered by the Court is the extent to which an expert's theory has been, or can be, tested.²¹ For a scientific theory to be reliable, it must be capable of being tested, and must have actually been tested. Thus, a Trial Court should give priority to examining whether the substance of the expert's testimony has been tested, and whether the theoretical or logical form of the expert's scientific explanation makes that explanation amenable to empirical testing.²² In addition, the testing of the witnesses' entire discipline is justified; if the discipline or sub-discipline itself is unreliable, the testimony should be excluded.²³

In most cases, defense experts will easily meet this factor. Defense experts, unlike those for the Plaintiff, are not advocating their own theory or technique. Rather, the defense expert is simply opining with respect to the theory or technique advocated by the Plaintiff's expert.²⁴ "In short, the Plaintiff's expert has the burden of demonstrating causation, but the defendant need only present evidence tending to refute the Plaintiff's causation hypothesis. Accordingly, it is relatively easy for a defendant to argue that the testing factor can only apply to the Plaintiff's experts."²⁵

The second *Daubert* factor is "whether the theory or technique has been subjected to peer review and publication."²⁶ Peer review and publication are critical in determining the reliability of expert testimony because these hurdles increase the likelihood that substantive flaws in the advanced methodology will be detected.²⁷ Unlike peer

¹⁴ Perrone, Patrick J., et. al., *Excluding Expert Witness Testimony in Mold Litigation*, Mealy's Mold (June 2001).

¹⁵ *Id.*, citing *Merrell Dow Pharmaceuticals v. Havner*, 953 S.W.2d 706, 714-15 (Tex. 1997).

¹⁶ *Id.*, citing Smith, Frederick T., *Daubert and its Progeny: Scientific Evidence in Product Liability Litigation*, Washington Legal Foundation, Washington, D.C. (2000).

¹⁷ See *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993); *General Electric Co. v. Joiner*, 522 U.S. 136 (1997); *Kumho Tire Co. Ltd. v. Carmichael*, 526 U.S. 137 (1999). Some states have retained the pre-*Daubert* expert admissibility standard first enunciated in *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). See, e.g., *Goeb v. Tharaldson*, 615 N.W.2d 800 (Minn. 2000) (rejecting the *Daubert* standard in favor of the "Frey/Mack" standard previously adopted in Minnesota state court).

¹⁸ *Daubert*, 509 U.S. at 589.

¹⁹ *Joiner*, 509 U.S. at 144.

²⁰ *Kumho Tire*, 526 U.S. at 152.

²¹ *Daubert*, 509 U.S. at 593.

²² Elliston, Gary D., et. al., *Defending the Daubert Objection*, DRI Asbestos Medicine Seminar (November, 2001) citing *Frosty v. Textron, Inc.*, 891 F.Supp. 551, 554 (D. Or. 1995).

²³ See e.g., *Summers v. Missouri Pacific R.R. Sys.*, 132 F.3d 599, 603 (10th Cir. 1997).

²⁴ See *Id.*

²⁵ Elliston, *supra* at note 22.

²⁶ *Daubert*, 509 U.S. at 593.

²⁷ See Elliston, *supra* at note 22.

review publication, the litigation process is not a valid test of the scientific propositions advocated by the expert. Lack of peer review or publication of an expert's test or theory is strong evidence that the concept is not within the "scientific knowledge."²⁸ "In short, expert witnesses should not be permitted to opine for the jury that which they have been unwilling to opine for their peers. The failure of a party's courtroom expert to subject opinions to peer review is a factor supporting exclusion of the testimony."²⁹

As with the first *Daubert* factor, defense experts normally need not meet this factor as these responding experts ordinarily are not espousing their own unique theory or technique. Rather, defense experts are evaluating the theory or technique given by the Plaintiff's experts, as compared with actual peer review and published matters.

The third factor is the known or potential rate of error of the expert's techniques.³⁰ "[T]he frequency with which a scientific technique leads to erroneous results bears heavily on its reliability."³¹ This factor almost never applies in a products liability context; rather, the factor primarily relates to issues arising in the criminal context.³²

The fourth factor from *Daubert* is whether the relevant scientific community has generally accepted the underlying theory or technique as valid.³³ Scientific knowledge is cumulative and progressive. A known technique or theory which has been able to attract only minimal support within the scientific community, or which is not consistent with accepted theories, is properly viewed with skepticism. "General acceptance remains a valid measure of an expert's reliability given that scientific theories are almost never generated in a vacuum."³⁴ Accordingly, if an expert presents theories that are not generally shared by other scientists, a court can exclude the opinion if the expert fails to identify and defend the reasons

why his conclusions are valid despite their atypical nature.³⁵

It is important to note that, even if some of the earlier-cited factors weigh in favor of admitting the expert evidence, it seems the Court can conclude that the evidence is inadmissible under this last factor simply because there is too great a gap between the underlying data and the expert's opinion.

General Causation

Causation is usually established in difficult or novel tort cases by satisfying both general, and specific, causation.³⁶ General causation in mold litigation focuses on whether mold is capable of causing a particular injury or condition in the general population.³⁷ This is generally addressed by analyzing the epidemiologic evidence available.

Epidemiology. "Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations." *Reference Manual on Scientific Evidence* (Second Ed.), p. 335 (Federal Judicial Center). Epidemiology is the best method of establishing general causation in the toxic tort context.³⁸ Courts consistently and universally agree that epidemiology is the most relevant evidence to determine if there is an association between the agent involved and the injury or disease claimed. Epidemiological studies can be interpreted to show that exposure to a particular toxin increases the risk of a particular injury.³⁹ It has been stated that an initial reliance on epidemiological studies may be proper if the study's mechanics (1) are "properly" designed, (2) are "properly" executed, (3) results in an increased risk, (4) are "unbiased" in design, and (5) results in a 95% confidence level.⁴⁰ It is also observed that, if an association is initially "found" between the agent and the disease, the study could still be invalid if there are alternative explanations for the association such as bias or other confounding factors.⁴¹

²⁸ *Id.*, citing *In re Air Crash Disaster at New Orleans, La.*, 795 F.2d 1230, 1234 (5th Cir. 1986) (finding that the admissibility of expert opinions should be carefully scrutinized where the experts "present studies and express opinions that they might not be willing to express in an article submitted to a referred journal of discipline or in other contexts subject to peer review").

²⁹ *Id.*

³⁰ *Daubert*, 509 U.S. at 594.

³¹ *DeLuca v. Merrell Dow Pharmaceuticals, Inc.*, 911 F.2d 941, 956 n.9 (3d Cir. 1990).

³² See Elliston, *supra* at note 22, at p. 25.

³³ *Daubert*, 509 U.S. at 594.

³⁴ See Elliston, *supra* at note 22, at p. 26.

³⁵ See *Lust v. Merrell Dow Pharmaceuticals, Inc.*, 89 F.3d 594, 598 (9th Cir. 1996).

³⁶ See Perrone, *supra* at note 14.

³⁷ *Id.*, citing *Merrell Dow Pharmaceuticals v. Havner*, 953 S.W.2d 706, 714-15 (Tex. 1997).

³⁸ See, e.g., *Brock v. Merrell Dow Pharmaceutical, Inc.*, 875 F.2d 307, 311 (5th Cir.), modified, 884 F.2d 166 (1989), cert. denied, 494 U.S. 1046 (1990).

³⁹ See Perrone, *supra* at note 14.

⁴⁰ *Id.*

⁴¹ See generally, *Reference Manual on Scientific Evidence* at pp. 354-373.

Where the epidemiology fails to reliably demonstrate an association between the toxin and the injury, courts generally rule the medical causation evidence is neither relevant nor admissible. Currently, there are no reliable epidemiological studies that demonstrate within a reasonable degree of scientific certainty that a causal connection exists between mold in buildings and the illnesses typically involved in mold litigation; nor are there any relevant significant peer reviewed articles which irrefutably establish such a connection.⁴²

This is not to say that the literature has not propounded some type of association between mold in buildings and some medical conditions. However, associations do not automatically equate to causation in a legal sense.⁴³ “. . . [E]pidemiology cannot objectively prove causation; rather, causation is a judgment for epidemiologists and others interpreting the epidemiologic data.”⁴⁴ Therefore, there must be some assessment of the epidemiology to determine the reliability and appropriateness of the research and findings so that the association found in the study can be extended to find a general cause-effect relationship.⁴⁵

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Bradford Hill Criteria. In addition to epidemiological studies themselves, courts focus on additional “reliability” considerations, such as the “Bradford Hill” criteria,⁴⁶ to

⁴² See Perrone, *supra* at note 14, quoting Gots, *supra* at note 2 (“There are few epidemiological investigations of inhaled mycotoxins and disease in indoor air settings. Although some purport to show an association between inhaled mycotoxins and health effects, none has had sufficient data or experimental design to support this claim.”). See also, Page, *supra* at note 8.

⁴³ *Reference Manual on Scientific Evidence* at p. 336.

⁴⁴ *Id.* at p. 374.

⁴⁵ *Id.*

⁴⁶ See Perrone, *supra* at note 14, citing Austin Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Soc’y Med. 295, 299 (1965). See generally, *Reference Manual on Scientific Evidence* at pp. 375-79.

assist in the gatekeeping function. The Bradford Hill criteria has been described as “part of sound methodology generally accepted by the current scientific community” for establishing disease causation.⁴⁷ As expected, Plaintiff experts often refuse to accept the validity of the Bradford Hill factors in determining disease causation; alternatively, these experts dismiss some or all of the factors given the particular fact pattern involved in the current cause of action.⁴⁸ The *Reference Manual on Scientific Evidence* supports the proposition that not all factors need be present to determine causation.⁴⁹ However, attempts to distinguish or explain away the applicability of the Bradford Hill factors as a whole should not be persuasive as these factors were not created by defense counsel or a trade association; rather, the factors were created by the credible, objective scientific community of which Plaintiff experts supposedly are a part.⁵⁰

The Bradford Hill criteria factors are: 1) temporal relationship, 2) strength of association, 3) dose-response relationship, 4) replication of findings, 5) biological plausibility, 6) consideration of alternative explanations, 7) cessation of exposure 8) specificity of association, and 9) consistency with other knowledge.⁵¹

Temporal Relationship. This factor asks whether the injury started before or after the alleged exposure.⁵² If the injury occurred before the alleged exposure, it is obviously implausible that the particular exposure caused the injury. This sometimes overlooked factor can yield compelling evidence when it is shown that the Plaintiff has suffered the same symptoms as alleged from mold exposures consistently before the time of building occupation, or at time when the Plaintiff was not in contact with the air inside the building.

Strength of Association. Strength of association asks how strong the alleged association is between the exposure and the adverse health effect as compared to the health effect in

⁴⁷ Perry, et. al., *Sound Methodology in Evaluating Causation: The Bradford Hill Criteria in Toxic Tort Litigation*, For the Defense, p. 67, 68, 81, 87 (August 2001), quoting *Merrell Dow Pharmaceuticals, Inc., v. Havner*, 953 S.W.2d 706, 719 (Tex. 1997).

⁴⁸ *Id.*

⁴⁹ *Reference Manual on Scientific Evidence* at p. 375.

⁵⁰ See Perry, *supra* at note 47, citing *Amorgianos v. National Railroad Passenger Corp.*, 137 F.Supp.2d 147 (E.D.N.Y. 2001).

⁵¹ *Id.*; see also *Reference Manual on Scientific Evidence* at p. 375.

⁵² See Perry, *supra* at note 47.

the general population.⁵³ The ratio is quantified under the terms Relative Risk or Observed Risk.

A mechanical application of the Strength of Association arms the defense attorney with a powerful argument to exclude Plaintiff's experts when the Relative Risk of persons exposed to the product do not have a two-fold risk of developing the condition in question.⁵⁴ It can also be used to defuse sympathy toward the Plaintiff by showing that her or his ailment is just as common in the general population as in those who are/have been exposed. Conversely, the higher the Relative Risk, the less chance exists that some confounding factor or bias is the source of the heightened association.⁵⁵ If the factor is strong, there can also be an argument that this strength also plays an important role in determining specific causation (see discussion, below).

Dose-Response Relationship. The dose-response factor joins toxicology and epidemiology to assess disease causation. This criterion asks whether evidence indicates that, as the exposure increases, a given health effect also increases. The lack of a dose-response relationship casts serious doubt on the validity of a study because it is unlikely that the least exposed individuals have a greater risk for the adverse health effect than those with higher exposures.

The dose-response factor is one of the primary cornerstones for the defense. It often exposes the subjective nature of the Plaintiff's claim in the face of remote exposures and the alleged harm.⁵⁶

Replication of Findings. Replication of findings asks whether the results of the studies, positive or negative, have been repeated by different investigators looking at different populations. This factor can require more than one positive study before it can be said that disease causation is established. Inconsistencies in various studies' findings can raise questions as to the causal link. "The Plaintiff's expert's tendency to cite a few positive studies while failing to address more recent and relevant studies is the hallmark of poor science and unreliable expert testimony."⁵⁷

Biological Plausibility. Biological plausibility requires an assessment of whether the causal link is possible based on

presently known biology. If the mechanics of how the condition develops is inconsistent with biological knowledge, there should be attempts at confirming the study's findings before it is extensively or exclusively relied upon in a legal setting. This factor is an important tool to challenge Plaintiff's experts' opinions by forcing the experts into a corner that shows their opinions are subjective, non-scientific speculation that contradicts the recognized science for both the exposure and the illness.⁵⁸

Alternative Explanations. This criterion simply asks whether other known causes of the Plaintiff's adverse health condition exist. Alternative explanations many times are the confounding factors or biases raised earlier.⁵⁹ Many courts use Plaintiff's experts' failure to address alternative explanations as one of the bases for excluding the experts' testimony.⁶⁰

Cessation of Exposure. The "cessation of exposure" factor tips in favor of a causal link if it can be shown that ending the exposure lowers the risk of the ailment. This can be confused with the dose-response factor discussed above, but should be given independent significance. Plaintiffs can use this factor to claim causation in reactive airway claims when the Plaintiff's condition almost immediately ends when they exit the building, but then immediately reappears when they return to the building.

Specificity of Association. This factor is based on an assumption that an agent will cause one disease, or one type of disease, and not a plethora of conditions or effects.⁶¹ It is generally contrary to accepted scientific understanding that a particular agent will cause a wide variety of unrelated health problems. However, if the exposure at issue involves numerous agents, the specificity requirement is weakened as the various agents might be the cause of the various agents. Whether the various mycotoxins produced by multiple possible molds present in a building constitutes such a variety of agents to reduce importance of this factor is yet to be seen.

Consistency With Other Knowledge. Consistency with other knowledge asks whether a causal determination makes sense in light of other information available which may reflect contrary exposure trends. For example, if a

⁵³ *Id.*

⁵⁵ Reference Manual on Scientific Evidence at p. 376.

⁵⁶ See Perry, *supra* at note 47, citing *Castellow v. Chevron USA*, 97 F.Supp.2d 780, 796-98 (S.D. Tex. 2000).

⁵⁷ See *Id.*, citing generally *Mitchell v. Gencorp.*, 165 F.3d 778, 782-84 (10th Cir. 1999).

⁵⁸ *Id.*

⁵⁹ Reference Manual on Scientific Evidence at pp. 363-73.

⁶⁰ See Perry, *supra* at note 47.

⁶¹ Reference Manual on Scientific Evidence at p. 379.

building's mold levels are decreased, but an individual's symptoms increase, or remain the same, the causal link may be questionable, especially if other confounding or bias factors are present. In essence, this factor can be considered a "common sense" check.

Specific Causation

The general causation discussion does not complete the Plaintiff expert's causation proof. The Plaintiff's expert must still prove that the agent at issue caused this Plaintiff's condition. "... [E]mploying the results of group-based studies of risk to make a causal determination for an individual Plaintiff is beyond the limits of epidemiology."⁶² Therefore, there must be a consideration of the particular individual's exposure, response, and injury in order to ascertain whether specific causation exists in a particular case.

Despite the understanding that epidemiology cannot be the determiner of specific causation, studies where the Relative Risk findings are greater than 2.0 times the baseline risk are said to meet the "more likely than not" specific causation analysis seen in civil cases. In fact, according to some of the Federal Circuits, if the epidemiological evidence does not show that exposure to an alleged toxin *doubles* the risk of a known disease, the Plaintiff cannot show causation.⁶³ In those cases, the courts have held that Plaintiffs must establish not only that the toxin increased somewhat the likelihood of harm, but that the toxin more than doubled the risk; only then it be determined that the toxin is more likely than not a source of the injury. In spite of this analysis, there is still a need to equate the specific Plaintiff's dose and response with those involved in the study to provide reinforcement to the bald causation statement.⁶⁴

Commentators have proposed that, in order for the Plaintiff expert to establish specific causation in a mold case, the expert must demonstrate: (1) that mycotoxins were present in the building; (2) the Plaintiff was exposed to the mycotoxins; (3) the dose and duration of exposure to mycotoxins was sufficient to cause Plaintiff's injury; and (4) the Plaintiff was injured.⁶⁵ However, many times the Plaintiff expert will mistakenly rely on a number of assumptions to

⁶² *Id.* at p. 337.

⁶³ See, e.g., *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1320-21 (9th Cir.), cert. denied, 516 U.S. 869 (1995); *Hall v. Baxter Healthcare*, 947 F.Supp. 1387, 1403 (D. Or. 1996).

⁶⁴ *Reference Manual on Scientific Evidence* at pp. 385-86.

⁶⁵ See Perrone, *supra* at note 14, citing Gots, *supra* at note 2.

prove these points. As discussed above, simply because mold spores are found in a building does not mean that:

- the spores contain mycotoxins;
- if mycotoxins were present, it is not necessarily a valid assumption that the mycotoxins became airborne;
- simply because mycotoxins were airborne does not mean the mycotoxins were in the occupant's breathing zone;
- if the mycotoxins were in the breathing zone, that they entered the occupant's body; and
- if the mycotoxins entered the occupant's body, there is no guarantee that mycotoxins inhaled were sufficient to cause injury⁶⁶

These are points which simply can not be presumed. The Plaintiff expert should be held to the strict standard of actual proof, and not be the beneficiary of presumptive evidence that the Plaintiff was specifically exposed to such levels.⁶⁷

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It has been said that there is no "biological marker" which links conditions to ingested, inhaled or absorbed mycotoxins from various mold spores.⁶⁸ Therefore, in the absence of other evidence of the occupant's exposure to mold spores from a building, it can be said that an examination of the occupant herself or himself will not determine whether they were exposed to mycotoxins coming from the structure.⁶⁹ Under this scenario, the specific causation factor should fail, and the expert should not be able to testify on this point.⁷⁰

⁶⁶ *Id.*

⁶⁷ See, e.g., *Austin v. Kerr-McGee Refining Corp.*, 25 S.W.3d 280, 292 (Tex. App. 2000); *Curtis v. M&S Petroleum, Inc.*, 174 F.3d 661, 670 (5th Cir. 1999).

⁶⁸ See Perrone, *supra* at note 14.

⁶⁹ Plaintiff experts claim that serology tests for antibodies (IgE and IgG for example) establish exposure to mold in the building. This is only partially true. The test can indicate whether the Plaintiff has been exposed to mold. However, the test is not time sensitive; a positive finding merely shows the Plaintiff had some exposure during the Plaintiff's life, which is nearly always a significantly larger time period than the time spent in the structure in question. Since mold is ubiquitous, the test results in and of themselves are of little use.

For a regulatory view on serology testing, please see the California Department of Health Services web page at <http://www.dhs.ca.gov/ps/deodc/ehib/ehib2/topics/Serologyf2.htm>.

⁷⁰ See Perrone, *supra* at note 14.

How Will the Occupants Prove Their Case?

Because science has not been able to establish specific threshold levels beyond which mold-related symptoms or illness will occur, occupants imply from case and animal studies that they were exposed to mold in sufficient levels to cause sickness. However, these studies are just as suspect in their application to mold cases as they are in other areas of litigation science and medicine. Mycotoxin animal inhalation studies measure acute effects at high exposure levels, and do not therefore logically equate to human low-level exposures. In fact, according to one medical expert, the data from these studies actually show that physiologic mechanisms may be able to address the toxicity if the specific dose involved is administered over a longer length of time than when the same massive dose is administered in a single instant.⁷¹ Therefore, these studies actually support the proposition that there is a threshold level for molds which must be exceeded before mold can cause symptoms.

There is, however, the continual temptation to be swayed by the “epidemiological study” created by the litigation itself if numerous individuals complain that a common building in which each spends a significant amount of time is causing all of the occupants to have the same symptomology.

There is, however, the continual temptation to be swayed by the “epidemiological study” created by the litigation itself if numerous individuals complain that a common building in which each spends a significant amount of time is causing all of the occupants to have the same symptomology. Plaintiffs can claim that the building occupants themselves create their own epidemiological cohort from which it can be definitively proven that all are suffering similar illnesses from the same mold. In these cases, it is difficult (but not impossible) to claim that all of the claimants are part of a mass hysteria, or motivated by matters other than illness. The subtle peer pressures placed on co-workers in these types of situations can be a source of a sympathetic response, even if unintended, and result in a

⁷¹ Gots, *supra* at note 2, p. 3.

skewed study.⁷² In addition, the level of scientifically confirming data the Plaintiffs’ expert needs to establish the reliability and relevancy of the “study” is typically not generated. As exemplified in the recent paper by Drs. Page and Trout, past studies attempting to show causation between mold and disease are deficient in their proofs, and therefore represent inadequate evidence to support causation.⁷³

Establishing the Plaintiff’s Actual Pre- and Post- Exposure Medical Condition

Traditionally, the defense counsel seeks to determine the Plaintiff’s pre-existing conditions as part of the medical defense. This approach is no different, and in fact is more significant, when mold is the alleged agent. Prompt collection of relevant documents, including school, healthcare and employment records may allow counsel to develop a pre-exposure baseline, and then compare that baseline to the Plaintiff’s current alleged conditions or complaints.

Even more so than in the more traditional injury case, a careful analysis of where, how and under what circumstances the Plaintiff lives will greatly aid in determining whether the Plaintiff’s alleged condition is exposure-related, or associated with some other activity such as a recent hobby, a new pet, a new location where the Plaintiff frequents, etc. However, if counsel considers testing alternative locations where the Plaintiff might possibly be exposed to other toxic substances, counsel should be ready to accept the negative inference that the particular site at issue might be a cause of the Plaintiff’s condition if the testing at the alternative site fails to yield elevated mold exposures.

CONCLUSION

Despite the newness of mold personal injury litigation, the issues involved in the cases are really no different than those seen in past personal injury and toxic tort cases. The medical defense must be aggressively developed with an eye towards eliminating or limiting the impact the Plaintiff’s medical causation expert has on the case. This is done by understanding and employing the time-tested strategies utilized in other toxic tort and personal injury cases. ▲

⁷² O’Neal, et. al., *Sick Building Claims*, The Construction Lawyer (January, 2000).

⁷³ Page, *supra* at note 8.