

**Toxic Mold and Mycotoxins in Neurotoxicity Cases:
Stachybotrys, Fusarium, Trichoderma, Aspergillus, Penicillium,
Cladosporium, Alternaria, Trichothecenes**

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Summary

This paper presents the argument that psychologists and neuropsychologists have no scientific basis for rendering opinions about causation given the current state of the literature. The critical question is whether residential or office inhalation of mold spores or mold metabolites, including mycotoxins, causes neuropsychological impairment or mental and emotional disorders. There has not been sufficient research to support such conclusions. Nonetheless, in the context of litigation, speculative opinions are rendered in lieu of scientifically well-founded conclusions. Resources for recognizing and coping with pseudoscientific arguments are suggested.

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Introduction

Mold neurotoxicity is an increasingly common allegation in personal injury litigation, although conspicuously absent in customary clinical practice. Time magazine's Anita Hamilton, in her June 24, 2001 article "Beware: Toxic Mold" warned readers that "Like some sort of biblical plague, toxic mold has been creeping through homes, schools and other buildings across the U.S." She went on to say, "The biggest winners are the industries feeding off mold mania" (p. 54). The current toxic mold controversy appears to be driven more by lawyers, testers, and remediators than by scientific disagreements. Alleged central nervous system effects are variously referred to as brain damage, toxic encephalopathy, cognitive deficits, neurobehavioral deficits, neuropsychological impairment, and as facets of sick building syndrome or environmental illness. The symptoms we see in forensic cases include memory deficits, difficulty concentrating, problems with language and reasoning, mental fatigue, depression, anxiety, and numerous others (Baldo, Ahmad, & Ruff, 2002; Sudakin, 1998). In some cases the plaintiffs allege dozens of symptoms they attribute to mold exposure.

The primary problem with the allegations of psychological and neuropsychological impairment due to mold inhalation is that speculation has been substituted for empirically established scientific reasoning. Based on the absence of research in the neuropsychology and psychology literature, as of this writing, there is no scientific basis for the allegation that breathing mold spores or mycotoxins or other mold metabolites in household and commercial office settings causes neuropsychological impairment. The psychological and neuropsychological effects of these exposures have not been established. But experts are expressing opinions about causation in forensic cases, saying they cannot think of any reason that a person suing for millions of dollars might make subjective complaints other than because they inhaled mold, and that the scientific literature does not *disprove* their speculative opinions. These are not scientific arguments; they are ar-

guments based on false assumptions, e.g., (1) patient attributions can establish cause and (2) although causation has not been established scientifically, it can be proven in a specific case.

The complaints that plaintiffs have been making in toxic mold cases are variable and nonspecific. They do not constitute a syndrome or pattern of essentially identical complaints from one case to the next. Neither do their neuropsychological test scores fall into a consistent pattern. There is no fingerprint test profile or pattern of complaints generally recognized as hallmarks of mold neurotoxicity.

The psychological and neuropsychological literature is devoid of research on psychological and neuropsychological effects of inhalation of mold spores and mold metabolites such as mycotoxins. Searches of the PsycINFO database as recently as February 8, 2003 for mycotoxin, mycotoxins, mycotoxicity, mycotoxicosis, mold and metabol*, mold and cognit*, mold and neuropsych*, mold and memory, mold and concentration, mold and attention, mold and toxic*, mold and mental disorder, mold and encephal*, fungi and toxic*, fungi and neuropsych*, fungi and cogni*, fungi and memory, fungi and concentration, fungi and attention, fungi and encephal*, and fungi and mental disorder, revealed *not one* controlled study of humans despite the current wave of claims seen in forensic psychological and forensic neuropsychological settings. These searches included journal articles, books, reports, dissertations, and chapters from 1872 to 2003. Repeated searches in April and June 2003 did not produce new findings based on psychological or neuropsychological testing, with one exception. In the spring of 2003, a publication cited as a 2002 article by Baldo, Ahmad, and Ruff (discussed below) appeared on the databases. The psychological and neuropsychological effects of inhalation of mold spores and mold metabolites such as mycotoxins in residential and office settings simply have not been studied in the psychological and neuropsychological literature sufficiently to offer a scientific basis for opinions about their

hypothetical effects. Other than the article above and another brief exception mentioned below, similar searches in the medical literature (Medline/PubMed) that employs psychological and neuropsychological testing have revealed no such studies.

The paper most often cited in forensic cases as evidence of neuropsychological impairment due to mold neurotoxicity is not a scientific study (Gordon, Johanning and Haddad, 1999). It was not peer reviewed in any conventional sense and the methodology was fatally flawed. The paper reports having evaluated persons exposed to *Stachybotrys atra* but used no control group and did not include a standardized test battery administered to all the participants. Alternative toxic exposures were not investigated, not even other mold exposures. The neuropsychological test scores of the people studied were notable for being *normal*, not impaired. Due to these and other limitations the report is mostly uninterpretable and does not constitute scientific evidence of mold neurotoxicity. Notwithstanding these problems, lawyers and a small number of experts refer to these findings as evidence of mold neurotoxicity, e.g., in the famous Texas case of Melinda Ballard and Ronald Allison v Farmers Insurance (Allison v. Fire Insurance Exchange, 2002). In another relevant study involving objective testing, as distinct from subjective reports (a study that *was* published in a peer-reviewed journal), the briefly mentioned finding was that the persons exposed to mold performed *better* on cognitive testing than the controls (Hodgson, Morey, Leung, Morrow, Miller, Jarvis, Robbins, Halsey, & Storey, 1998).

Sudakin (1998) found an increase in self-reported neurobehavioral symptoms in a case report of alleged mold-exposed individuals but cautioned readers that these individuals were aware of reports of adverse health effects of toxigenic fungi prior to relating their subjective complaints, which improved substantially after leaving the building. Sudakin (personal communication, 2002) reported that many of the people in this study were making claims for compensation, and described his paper as one

that discussed the hypothetical role of mycotoxins (not mold exposure) in the context of behavioral effects, and he concluded that this was a hypothesis with unknown applicability to humans. He also said his paper did not suggest that mold exposure may result in central nervous system changes.

The recent paper by Baldo, Ahmad and Ruff (2002) included neuropsychological testing but was based on a cross-sectional sample that was so small and diverse that this was a case study reporting anecdotal information, not a controlled study that might shed light on causation. The information in the article is so general that it is not clear what population is the focus of this study. The plaintiffs were not exposed to the same fungi, or exposed for a similar duration, or exposed in any measured amounts, and no data were presented to clarify which molds or mycotoxins are the independent variables in this study. Given that every human on earth is exposed to mold, and there are an estimated 100,000 fungi with varying properties (Terr, 2001), lack of further clarification of the independent variable was a critical omission. The neuropsychological test results were implausible. The Baldo et al. article has too many limitations to be used as a basis for opinions about effects of mold on human neuropsychological functioning. Unfortunately, despite qualifying their work as preliminary, the authors both imply and state conclusions that are not supported by their study or by the literature they cite (for further details, see Lees-Haley, in press).

Establishing Proof without Evidence

Because their methodologies lack objective evidence that mold or mycotoxin inhalation has caused brain injury, experts claiming to have found such injuries are relying heavily on subjective reports of symptoms and on tests that are affected by response biases associated with litigation and hazard perception (e.g., see Gots, 1993). This approach to forming causation conclusions is problematic for a number of reasons that will be discussed in more detail below. One key limitation, as noted above, is that there

is no known pattern that constitutes a neuropsychological or psychological syndrome or diagnosable mental disorder associated with mycotoxin inhalation or inhalation of mold spores.

Many experts in litigated cases are ignoring the growing body of research showing that patients in litigation behave differently from other persons in ways that directly affect assessment of the patient's condition. Artifacts associated with litigation appear to be affecting neuropsychological test results (Binder & Rohling, 1996; Feinstein, Ouchterlony, Somerville, & Jardine, 2001; Rohling, Binder, & Langhinrichsen-Rohling, 1995; Youngjohn, Burrows, & Erdal, 1995). Plaintiffs often report their pre-injury history in unusually benign terms, and discount alternative explanations for their complaints, such as important stressors in their lives (Lees-Haley, Williams, Zasler, Margulies, English, & Stevens, 1997). They respond to neuropsychological tests more frequently in an impaired range even when no one is claiming they have brain damage – not their lawyers, their doctors, or the plaintiffs themselves. For example, plaintiffs making mild brain injury claims often respond to psychological tests in a more impaired way than persons with documented severe brain injury – again, the test results appear to be contaminated by biases arising from litigation rather than being a true reflection of the extent of injury (Binder & Willis, 1991; Suhr, Tranel, Wefel, & Barrash, 1997; Youngjohn, Davis, & Wolf, 1997). There is growing evidence that attorneys and other advocates associated with litigated claims influence the psychological and neuropsychological evidence in ways that cause misleading and erroneous results (e.g., see Gervais, Green, Allen, & Iverson, 2001; Lees-Haley, 1997; Rosen, 1995; Wetter & Corrigan, 1995; Youngjohn, 1995). Finally, it is becoming increasingly clear that the amount of effort the patient makes during neuropsychological testing has a greater impact on the test results than the degree of injury (e.g., see Green, Rohling, Lees-Haley, & Allen, 2001). In summary, the influence of the claims context is such a powerful confounding force

that it must be considered in the process of differential diagnosis and ruled out.

Genuinely troubled people are particularly susceptible to these influences, at times to their detriment. For example, individuals with somatoform characteristics and histrionic personalities tend to be suggestible (APA, 1994, 2000) and therefore vulnerable to zealots and advocates who tell them they are brain-damaged and permanently injured by their toxic environment. Most of us more or less ignore, or notice and discount common “symptoms of life” such as transient aches and pains, fluctuating ability to concentrate, temporary fatigue, feeling stressed, or inability to recall various sorts of information such as a word or name or where we left something (e.g., see Angell, 1996). However, when an expert claims these are symptoms of mold neurotoxicity, a suggestible person may focus more attention on these experiences, become alarmed, and become involved in a vicious cycle of overinterpreting mild symptoms, becoming anxious, developing more symptoms caused by the anxiety, and becoming even more alarmed, sometimes to the point of virtually obsessing over the symptoms (Lees-Haley & Brown, 1992). Adding medication, which many plaintiffs take in connection with these symptoms, increases the likelihood that medication side effects will further complicate the clinical picture, e.g., by interfering with concentration, memory, energy level, et cetera (Maxmen & Ward, 2002; Schatzberg, Cole, & DeBattista, 2003). It is an easy next step for the patient to conclude that, because these feelings are more noticeable and more frequent lately, they must have been caused by mold exposure as has been suggested to them.

Although most of us think of ourselves as not presently suffering the effects of brain injury due to mold or anything else, empirical investigations have repeatedly shown that surprisingly high rates of brain injury symptoms such as memory and concentration complaints and fatigue are normal in diverse populations such as adults in general, students and medical patients (e.g., see Dunn, Lees-Haley, Brown,

Williams, & English, 1995; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992; Gouvier, Uddo-Crane, & Brown, 1988; Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995a, 1995b; Lees-Haley, 1992; Lees-Haley & Brown, 1993). Likewise, epidemiological studies of prevalence of psychiatric symptoms show that rates of psychiatric illness are common and “greater than previously thought to be the case” (Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, Wittchen, & Kendler, 1994, p. 8; see also Kessler & Walters, 2002; Regier, Boyd, Burke, Rae, Myers, Kramer, Robins, George, Karno, & Locke, 1988; Robins, Helzer, Weissman, Orvaschel, Gruenberg, Burke, & Regier, 1984). Thus, when someone begins a search for “psychopathology” it is not hard to find. Administration of diagnostic tests to a random or non-complaining population detects false positives and actual pathology that was not apparent prior to the testing, so it is no surprise that testing a group of plaintiffs reveals apparent injury whether or not there is any (Feinstein, 1985, 1988). Normal people produce scores in the impaired range on several tests in a detailed neuropsychological test battery (e.g., see Heaton, Grant, & Matthews, 1991), but some experts ignore these base rates (“normal levels of abnormality”) and overinterpret a few low scores as indicative of toxic brain injury.

Teaching Eyewitnesses to See Invisible Phenomena

In ambiguous settings, psychological influences play an especially important role in human perception, so it is not surprising that they are important in controversial arguments over what is known about mold neurotoxicity. The information disseminated by media, politicians, activists, litigating parties, experts, and attorneys may cause important emotional reactions in susceptible people. A claim may be false but it can cause true alarm and genuine emotional distress if misleading allegations are taken seriously (Foster, Bernstein, & Huber, 1994; Guidotti & Jacobs, 1993; Harbison,

2003; Pratkanis & Aronson, 2001; Showalter, 1997).

It is critically important to use good science rather than advocacy in the evaluation of mold neurotoxicity. Lawyers and clinicians who cannot wait for facts (or actively reject them) are relying largely on propaganda and social influence techniques such as those described by Cialdini (1993) and Pratkanis and Aronson (2001). For example, they use social proof, repeated affirmations, appeals to authority, and vividness to persuade people that baseless views are true. *Social proof* is the tendency to believe what other people believe. If an advocate creates the impression that other people have concluded that mold caused them brain damage, there is a natural tendency to agree, and a subtle implication that one who disagrees lacks credibility. Identifying a few people who believe a proposition and encouraging them to go public (especially repeatedly) creates the impression that numerous people are discovering something real (Gilovich, 1993). For example, celebrities are called upon to join in public outcries against perceived toxic exposures. *Repeated affirmations* – literally just saying your claim over and over – creates the impression that the assertion is true. After all, as everyone knows, where there is smoke, there must be dry ice. *Appeals to authority* add weight to these persuasions; if one or more of the people affirming a belief is authoritative, e.g., a civic leader or expert, more people will be persuaded. Sometimes politicians are persuaded to join in unfounded but politically popular rhetoric, e.g., when the U. S. Congress condemned the American Psychological Association based upon the mistaken belief that research published by the APA was endorsing child abuse (Baird, 2002; Lilienfeld, 2002; McNally, 2003). If we like the source of an opinion, we are more likely to believe what is said. Thus, if a popular actor, media figure, politician or local hero joins the process, more of us will endorse the perceived reality. In mold litigation, household names such as Ed McMahon and Erin Brockovich have appeared in the national news related to the toxic mold contro-

versy. *Vivid examples* – especially dramatic, anecdotal case histories – often influence judgments more than dull but more accurate quantitative examples. Presenting a dramatic, close up picture of frightening black mold (*Stachybotrys*) may generate the feeling in the observer that “this is bad” and permit a scientifically unfounded emotional segue to the conclusion that anything that looks this bad must cause the alleged harm.

A closely related problem is recent research showing how false memories can be implanted or developed even when there is no reason to suspect the individual of any intent to deceive. For example, Elizabeth Loftus and her colleagues have dramatically demonstrated the potential for introducing erroneous memories in interviews (e.g., see Loftus, 1997; Mazzoni, Loftus, & Kirsch, 2001). Such false memories are not necessarily subtle shades of gray; some are dramatic and blatantly inaccurate recollections of events that never happened (Braun, Ellis, & Loftus, 2002; Thomas & Loftus, 2002).

Plaintiffs are Different from Patients without Claims

Patients pursuing litigation report more intense, frequent, and persistent symptoms than do non-litigating patients. For example, a number of prospective studies have found that non-litigating individuals with mild brain injury typically recover from their symptoms within a few months of injury (Barth, Alves, Ryan, Macciocchi, Rimel, Jane, & Nelson, 1989; Dikmen, Machamer, Winn, & Temkin, 1995; Dikmen, McLean, & Temkin, 1986; Gronwall & Wrightson, 1974; Hugenholtz, Stuss, Stethem, & Richard, 1988; Levin, Mattis, Ruff, Eisenberg, Marshall, Tabaddor, High, & Frankowski, 1987). However, recovery of patients in litigation often defies expectations. Their complaints, including memory loss, headache, dizziness, concentration difficulty, blurred vision, photophobia, ringing in the ears, irritability, fatigue, anxiety, and depression (World Health Organization, 1978) are reported to continue long after such symptoms normally resolve (Binder, Rohling, & Larrabee, 1997).

Numerous empirical investigations have documented discrepancies between patients seeking compensation and patients not seeking compensation (see e.g., Berry, Wetter, Baer & Youngjohn, 1995; Gold & Frueh, 1999; Levin, et al., 1987; Youngjohn, Davis, & Wolf, 1997). Fee and Rutherford (1988) compared the frequency of reported symptoms among patients with mild brain injury who were in litigation with symptoms of others not in litigation. After normalizing for severity of initial injury, reporting rates were assessed for various symptoms including headache, anxiety, irritability, dizziness, depression, and insomnia. Litigating patients reported nearly twice as many symptoms as non-litigating patients.

The discrepant nature of self-reported symptoms in compensation seekers has been established with respect to psychological as well as neuropsychological injuries. Compensation-seeking patients have been found to report more persistent psychological symptoms than patients with similar injuries not seeking compensation. Frueh, Smith, and Barker (1996), for example, found that combat veterans seeking service-connected disability for posttraumatic stress disorder (PTSD) had significantly more pathological scores on a wide range of psychological inventories and on MMPI-2 validity indices than did combat veterans with equivalent PTSD diagnoses not seeking compensation (also see the review of this problem by Frueh, Hamner, Cahill, Gold, & Hamlin, 2000). Pope, Butcher, and Seelen (1993) noted that MMPI profiles of patients with pending disability evaluations exhibit more exaggeration and pathology. Their research also indicated that genuinely disabled people not awaiting a disability determination tend to produce MMPI profiles with normal scale scores. Compensation seeking has been cited as one of the most serious obstacles to successful treatment of PTSD within the VA system (Richman, Frueh, & Libert, 1994). Campbell and Tueth (1997) reported that the system of compensation payments creates a disincentive for recovery and noted that, “Rewarding individuals for pain and disability, particularly on a long-term basis, can

have numerous negative consequences, and ultimately may be a disservice to the patient” (p. 42). Frueh, Gold, and de Arellano (1997) similarly identified compensation as a confounder in cases of Posttraumatic Stress Disorder.

Health-care providers have noted fundamental differences between litigating and non-litigating patients, and “have become increasingly suspicious of the genuineness of symptoms exhibited by plaintiffs because of the large disparity often found between subjective complaints and objective findings” (Weissman, 1990, p.71). Compared with non-litigating patients, patients seeking financial compensation may find treatment withheld because plaintiffs are perceived as resistant and noncompliant with psychotherapeutic and rehabilitative services. When incentives such as settlements may outweigh incentives such as getting better, there may be noncompliance with helpful therapy and searches for claim-supporting tests and care.

Not only do litigating patients report current symptoms at higher rates and as persisting longer than do non-litigating patients but litigating patients tend to recall pre-injury psychological and neuropsychological functioning as superior to controls (Lees-Haley, Williams, & English, 1996; Lees-Haley, Williams, Zasler, Margulies, English, & Stevens, 1997). For example, Lees-Haley, et al. (1997) asked litigating and non-litigating patients to recall how problematic specific symptoms, behaviors, and aspects of life were in the past. Compared to non-litigants, patients seeking compensation recalled pre-injury functioning as less problematic. These recalled variables, all relevant to evaluations of “toxic mold” plaintiffs, included concentration, memory, fatigue, depression, anxiety, ability to attend school or work, irritability, headache, confusion, self-esteem, marriage, and relationships with children. Because the apparent severity of injury depends upon a comparison of pre- and post-injury functioning, this apparent worsening from pre- to post-injury bolsters the claim for compensation. Therefore, by failing to take into account the tendency of plaintiffs to overestimate their pre-

injury status, forensic examiners and triers of fact may overestimate the severity of the injury.

It is important for examiners to recognize that these tendencies are trends, not absolutes. They are group differences analogous to characteristics of a diagnostic group, e.g., persons diagnosed as suffering from depression. It is a mistake to presume that being a claimant or seeking compensation is automatically equivalent to being a malingerer. The majority of plaintiffs do not appear to be malingering. The problem of assessing claimants is analogous to problems with interpreting data from job applicants and persons examined by psychologists while seeking to win custody of their children. We know from years of experience that job applicants and individuals seeking custody of their children have a distinct tendency to present themselves in a favorable light during psychological evaluations (Bagby, Nicholson, Buis, Radovanovic, & Fidler, 1999; Corr, & Gray, 1995; Martin, Bowen, & Hunt, 2002). Similarly, claimants show a bias in the other direction, tending to emphasize ways of describing themselves that are consistent with their claims of injury or disability.

Exaggeration or Malingering

Malingering is defined as the intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives such as financial gain, seeking drugs, avoiding work, evading criminal prosecution, etc. (American Psychiatric Association, 1987, 1994). Contrary to what many of us used to believe based on our clinical treatment experience, empirical studies are finding that malingering is a common phenomenon in forensic matters. The courts perhaps were wiser than clinicians – they have long recognized this phenomenon. References to the problem of fraudulent claims and false testimony recur in published cases and legal commentary throughout the history of law. The oldest known code of laws, the Code of Hammurabi, addressed this issue (Hammurabi’s policy was to slay witnesses who testified falsely) (Johns, 2000). Indeed, the problem of false testimony is

the first issue addressed with every witness in every court in every case: “Do you swear to tell the truth, the whole truth, and nothing but the truth, so help you God?”

Patients seeking compensation have more incentive to produce false or exaggerated symptoms than patients seeking treatment. Over the years, estimates of the proportion of plaintiffs feigning psychological deficits ranged from a low of 1% (Keiser, 1968) to over 50% (Miller & Cartlidge, 1972). In personal injury cases, feigned cognitive deficits have been estimated at 64% (Heaton, Smith, Lehman, & Vogt, 1978) with 47% of workers' compensation possibly involving malingering (Youngjohn, 1991). Another study estimated the percentage of invalid memory deficits in patients claiming persisting postconcussive syndrome as being between 33% and 60% (Greiffenstein, Baker, & Gola, 1994). Research by Binder (1993) indicated that 33% of mild brain-injured subjects seeking compensation malingered deficits on psychometric testing. Such studies are relevant because the complaints made by persistent postconcussive plaintiffs are remarkably similar to those of many “toxic mold” plaintiffs. Both often appear to be exhibiting response biases related to litigation. Mittenberg, Patton, Canyock, & Condit (2002) studied 33,531 cases involved in personal injury, disability, criminal or medical matters and found that 29% of personal injury, 30% of disability, 19% of criminal, and 8% of medical cases involved probable malingering and symptom exaggeration. Mittenberg et al. found that 39% of mild head injury, 35% of fibromyalgia/chronic fatigue, 31% of chronic pain, 27% of neurotoxic, and 22% of electrical injury claims resulted in diagnostic impressions of probable malingering. In their study, diagnosis was supported by multiple sources of evidence. Forensic examiners should consider and rule out the possibility that symptom reports and test results are the product of inaccurate presentation by plaintiffs during interviews and exaggeration of symptoms through self-report inventories or tests. It has become the standard of care to consider the possibility of malingering when per-

forming a differential diagnosis in forensic cases (Slick, Sherman, & Iverson, 1999).

Some psychologists and psychiatrists claim that plaintiffs are unable to malingering mental disorders or neuropsychological deficits without detection by psychological experts. However, there is substantial contrary empirical support. Naïve individuals can fake psychological and neuropsychological symptoms successfully when provided minimal information about disorders (e.g., Albert, Fox, & Kahn, 1980; Faust, Hart, & Guilmette, 1988; Lamb, Berry, Wetter, & Baer, 1994; Rogers, Bagby, & Chakraborty, 1993; Rogers, Ornduff, & Sewell, 1993; Wetter, Baer, Berry, Robison, & Sumpster, 1993). In one investigation, participants asked to fake brain injury were provided detailed information on the type of validity scales on the MMPI-2 and were given information on how to avoid getting caught. This information enabled subjects to produce clinically elevated profiles without significantly elevated validity scales (Lamb, et al., 1994).

Uncoached individuals may report symptoms similar to those of genuine patients on certain kinds of inventories, such as symptom checklists (Lees-Haley, 1989a, 1989b). Research indicates that untrained individuals are able to endorse accurately symptoms and experiences of post-concussion syndrome (Mittenberg, DiGiulio, Perrin, & Bass, 1992) as well as major depression, generalized anxiety disorder, and PTSD (Lees-Haley & Dunn, 1994; also see Burges & McMillan, 2001). That naïve individuals can simulate psychopathology without raising suspicion of malingering is problematic for expert evaluators. Information about some psychological and neuropsychological disorders is readily accessible to plaintiffs motivated to deceive. Berry (1995) points out that “Fabricators may become familiar with psychiatric symptoms through personal acquaintances, perusal of volumes such as the DSM-IV (APA, 1994), textbooks in psychiatry, or even through exposure to lay sources such as magazine articles and movies about individuals with mental disorders” (p. 88). Bury and Bagby note that a claimant motivated to do so “can

easily learn what symptoms must be reported to qualify for the diagnosis” of Posttraumatic Stress Disorder (2002, p. 472). The Internet provides a wealth of information about mental disorders and neuropsychological tests.

The World Wide Web

Many plaintiffs perform Internet searches that produce claims and opinions of wildly varying reliability and validity. Although certain websites are a great aid to legitimate research, innocent but suggestible persons may accept misinformation, hyperbole and speculation as fact. Attorneys, clinicians and various other interested parties are posting a plethora of information on the web. On March 18, 2002 I entered “toxic mold” on Google.com and found 63,400 hits. At the top of all the 63,400 hits was the headline, “Learn about the side effects of toxic mold!” The text beneath this headline was not a referral to a scientist or physician or toxicologist. Rather, there was a hypertext referral to www.injurylawyershop.com, which led immediately to <http://www.toxicmoldinfocenter.com>, a site introduced with the line “*Toxic Mold info and access to attorneys who specialize in Mold litigation.*” On January 26, 2003 and June 16, 2003, I repeated the search for “toxic mold” and found 115,000 and 138,000 hits, respectively, on Google. A site laced with legal information, with no readily identifiable responsible party, continued to head the lists. The dramatic increase in web postings on toxic mold in is a sign of the remarkable growth of interest, information, and misinformation about the topic.

At <http://www.doctorfungus.org/> a variety of information was presented but at the top center of the web page there was news about someone suing for \$65 million. This is not unique. Scanning another website called “Toxic Mold and Tort News Online Safety, Prevention, and Information” at <http://www.toxic-mold-news.com/> we found the following introductory statement: “The Toxic Mold Website is a comprehensive guide to information regarding mold, toxic mold, safety, and prevention.

Our site also provides important legal rights and information for those who have been adversely affected by Toxic Mold in their home, workplace, and elsewhere.” After a couple of introductory paragraphs about “potentially fatal dangers” and legal rights there were four paragraphs of news, all four of which were about how much money people had collected in mold cases.

At The Mold Source at <http://www.themoldsource.com/starter.html> there was a list of medical-legal experts about whom the following claim was stated: “The following professionals have established themselves, through their dedication, commitment and their overwhelming concern for mankind, as the experts. They are the best the world has to offer “us”, the fungi contaminated. Collectively, they retain the majority of all known knowledge on fungi and fungal poisoning related illnesses...” My point is not to question the sincerity or integrity of the unidentified author of these views. It is to illustrate the intensity of apparent belief that we see in this issue and how this belief anoints “toxic mold” experts.

The Toxic Mold Survivors Information and Support Group at <http://toxicmoldsurvivors.com/home.htm> provides a warning at the top of their home page about potential adverse health effects of molds (including *Stachybotrys*, *Aspergillus* & *Penicillium*, beneath the headline “Poisoned by Toxic Molds?”). This site tells us that potential adverse health effects include “neurotoxic:-toxic encephalopathy” [sic], memory and verbal problems, fatigue, malaise, vertigo, dizziness, and depression. Although ostensibly a website for a support group, litigation appears to be a major interest of these “survivors.” For example, the home page provides links to “Litigation” and “Next Asbestos?” (an article entitled “Toxic mold... The Next Asbestos?” by Sylvia Hsieh of [Lawyers Weekly USA](#)), plus links to the websites of lawyers and expert witnesses who testify in toxic tort litigation.

Medical Literature

Background information from the medical literature and relevant government agencies highlights the shaky foundation for psychological and neuropsychological experts testifying that mold or mycotoxin inhalation causes identifiable cognitive deficits. The following series of opinions and findings by medical experts should inspire some caution among psychological and neuropsychological experts.

Medical experts indicate that fungi can cause human disease in four ways: allergy, mycosis (fungal infection), irritation and mycotoxicosis (poisoning by ingestion of a mycotoxin) (Fung, et al., 1998a). Although it is known that many molds can cause toxicity if ingested at high levels, at present, there is no clear association of cause and effect between residential and office airborne (inhaled) mold exposure and illness. There have been no toxicologic and epidemiologic studies of humans with adequate scientific or statistical power to reach such conclusions (e.g., Fung, et al., 1998a; Mahmoudi & Gershwin, 2000). Most of the reported studies in the medical literature have been based on case studies or small samples, and most have flawed methodology. A number of authors have noted that we have not yet clearly determined what fungi or mycotoxin may produce illness or what dose is required (e.g., Mahmoudi & Gershwin, 2000). Many studies have used self-report measures of symptomatology and they have failed to rule out or control for alternative causation, including whether patients were involved in litigation (e.g., see Fung, et al., 1998a, 1998b). Fung, et al. (1998a, 1998b) point out that immunological laboratory studies are non-specific, which is ignored in some studies. The lack of symptom specificity is problematic, since complaints such as “chronic laryngitis, sinusitis, bronchitis, asthma, allergy, and toxic encephalopathy have numerous etiologies” (Fung, et al., 1998b, p. 633).

According to the American College of Occupational and Environmental Medicine (ACOEM) Evidence-based Statement, prepared

under the auspices of the Council on Scientific Affairs, peer-reviewed by the committee and council and approved by the ACOEM Board of Directors on October 27, 2002, “Current scientific evidence does not support the proposition that human health has been adversely affected by inhaled mycotoxins in home, school, or office environments” (Hardin, Kelman, & Saxon, 2002, p. 1).

In their review of the relevant literature, investigators from the National Institute for Occupational Safety and Health at the Centers for Disease Control and Prevention (CDC NIOSH) concluded that “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” (Page & Trout, 2001, p. 647). They also concluded, “To support hypotheses regarding potential adverse health consequences of mycotoxin exposure in the nonindustrial environment, objective measures of adverse health effects must be associated with some measure of mycotoxin exposure, and comparisons must be made with appropriate control populations; to date, such evidence has not been forthcoming” (p. 647).

In another review of the literature, Robbins, Swenson, Nealley, Gots, & Kelman (2000) concluded, “Health-based exposure standards for molds and mycotoxins do not yet exist. While there is general agreement that active mold growth in indoor environments is unsanitary and must be corrected, the point at which mold contamination becomes a threat to health is unknown” (p. 782). Robbins and her colleagues wrote that “...the current literature does not provide compelling evidence that exposure at levels expected in most mold-contaminated indoor environments is likely to result in measurable health effects” (p. 773). Robbins, et al. also noted, “Whether molds such as *Stachybotrys* should be treated differently than other molds, when considering cleanup or sampling and exposures issues, is also a controversial subject” (p. 782).

In his review of the literature concerning *Stachybotrys*, allergist Abba Terr concluded, “The current public concern for adverse health effects from inhalation of *Stachybotrys* spores in water-damaged buildings is not supported by published reports in the medical literature” (2001, p. 57).

According to Harriet Burge of the Harvard School of Public Health, “People have become concerned about the health effects of mycotoxins out of proportion to currently estimated risk” (2001, p. 52) and “The fact that a mold is growing in a home is not good evidence for exposure of any kind, and certainly not evidence of danger” (p. 55). Burge went on to say, “In general, then, one can reassure patients that the symptoms they are experiencing, although real, are probably not associated with mycotoxin exposure” (p. 56).

In May 2000 the American Industrial Hygiene Association convened a forum to summarize findings of a panel of scientists who had been assigned the task of evaluating the scientific literature suggesting causal associations between indoor exposure to mycotoxic fungi and adverse health effects. This review panel included experts in pediatric pulmonology, occupational health, epidemiology, microbiology, toxicology, and industrial hygiene. “Ultimately, the panel concluded that at 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” (Kirkland, 2001, p. 26).

Kuhn and Ghannoum (2003) conducted a detailed review of indoor mold and toxigenic fungi, emphasizing, but not limiting their discussion to, *Stachybotrys chartarum* (*atra*). They examined relevant case reports and studies and concluded, “In summary, despite many reported subjective complaints, there is no objective evidence for neurological compromise caused by indoor mold exposure, in particular from *S. chartarum*” (p. 160). They also concluded, “...we have not found supportive evidence for serious illness due to *Stachybotrys* exposure in the contemporary environment.

Our conclusion is supported by several other recent reports...” (p. 164).

In a review of current knowledge of *Stachybotrys Chartarum* (the most notorious mold in the litigation setting, which is where most mold complaints are found), Dan Sudakin, medical toxicologist, of the VA Medical Center in Portland Oregon stated, “Although the hazards associated with exposure to some mycotoxins have been well studied, the health risks from environmental exposure to *Stachybotrys* remain poorly defined” (2000, p. 1). In a related conclusion, Sudakin also stated, “Despite the far-reaching public health measures that have emerged as a result of recent publications, the health risks from environmental exposure to *Stachybotrys* remain poorly defined. The most current research is limited by indirect assessment of exposure, weak and inconsistent associations between exposure and disease, and inadequate assessment of known confounders. What is becoming clear is that *Stachybotrys* and other potentially toxigenic fungi are more common in the indoor environment than has been previously acknowledged” (p. 5).

Janet Weiss, medical toxicologist at the University of California, San Francisco, wrote, “Although several outbreaks of illness in humans have been attributed to respiratory exposure to [*Stachybotrys chartarum*], the causal link between fungal contamination in the indoor environment and adverse health consequences has yet to be established” (2001, p. 8). She further stated, “Data linking exposure with health effects are unavailable for spore concentrations found in typical indoor air environments” (p. 9-10). Regarding the emotional distress generated by clinicians who tell patients that such exposures may cause cancer, one should note that Weiss also stated, “There is no animal or epidemiological evidence to indicate that *S. chartarum* is a carcinogen, yet the popular press continues to raise this threat as if it were a proven fact” (p. 9).

Fung, et al. (1998a) summarized the results of published studies and reports regarding *Stachybotrys* mycotoxins related to human toxicology and concluded, “Recently, airborne

mycotoxins associated with water-damaged buildings have attracted the attention of health care professionals and the public. The controversy over airborne *Stachybotrys* mycotoxins originated from the extrapolation of data based on case series without specific medical diagnoses. Some reports imply a causal relationship between the presence of *Stachybotrys* and poisoning. However, to demonstrate a causal relationship between an environmental toxin and its effects, several well-designed epidemiological studies with sufficient statistical power are necessary” (p. 83). These studies have not yet been performed.

Based on their review of literature related to the microbiology of mycotoxin-producing molds and their potential role in human immunopathology in wet buildings, Assoulin-Dayana, Leong, Shoenfeld, and Gershwin concluded, “There has been increasing public attention to the potential health risks of mold exposure, particularly in wet buildings. A variety of molds has been isolated from both damaged homes and businesses, including agents that secrete toxigenic materials. One area that is attracting particular notice is the relative toxigenic potential of mycotoxins. Although exposure to molds can produce significant mucosal irritation, there are very few data to suggest long-term ill effects. More importantly, there is no evidence in humans that mold exposure leads to nonmucosal pathology. In fact, many of the data on toxigenic molds are derived from animal toxicity studies, and these are based primarily, on ingestion. Although every attempt should be made to improve the quality of indoor air, including avoidance of molds, the human illnesses attributed to fungal exposure are, with the exception of invasive infections and mold allergy, relatively rare” (2002, p. 191).

Based on their review of all English language studies on indoor mold exposure from 1966 to 2002, Fung and Hughson concluded, “specific toxicity due to inhaled fungal toxins has not been scientifically established” (p. 46). They also concluded, “Specific toxicity due to

inhaled mycotoxins is not well documented, and remains controversial” (2002, p. 50).

In another review of the medical literature related to clinical implications of mycotoxins and *stachybotrys*, Kaplan, Palmer and Revankar (2003) wrote, “There has been considerable interest and concern in recent years regarding the potential health effects of mycotoxins in the indoor environment. Although the existence of mycotoxins has been known for several decades, relatively little is known about their effects in humans. What is known comes almost exclusively from studies of ingestion as the route of exposure. This review summarizes what is known regarding health effects of mycotoxins in general and specifically examines the evidence for the role of indoor exposure to the fungi of the genus *Stachybotrys* as a cause of disease in humans. Much work remains to be done in the area of mycotoxin research. The risk of health effects from ingestion seems much more widespread than from indoor airborne exposure, although the latter has received considerably more media attention. Rigorously controlled studies are needed to clarify these issues.”

The Texas Medical Association issued a report on “Black Mold and Human Illness” that addressed *Stachybotrys chartarum* (McClusky, 2002). They first noted that public attention to *Stachybotrys* has increased, stating, “In Texas, this attention has been manifest not in scientific or medical publications, but rather in the lay press and in an increasing number of insurance claims filed for mold remediation of homes and workplaces” (p. 1). The Texas Medical Association’s Council on Scientific Affairs was asked to update the “state of the medical science” concerning *Stachybotrys*. After performing their review, they reported their method and conclusions: “To study this issue, the council conducted a search of medical and scientific literature and contacted Texas and national experts/specialists. After reviewing available data, the council has concluded that public concern for adverse health effects from inhalation of *Stachybotrys* spores in water-damaged build-

ings is generally not supported by published reports in medical literature” (p. 1).

Toxicologist Ronald Gots in his conference paper entitled “Correcting Mold Misinformation” wrote, “Mold toxins at indoor levels have never been shown scientifically to cause any illness. Physicians generally do not accept that there is any causal connection between them” and “Indoor exposure to mold or mold toxins has never been proven to cause brain damage.” He also added, “It is highly unlikely that there is a home in the world without some *Stachybotrys* spores in it” (Gots, 2002, p. 2).

Sudakin (2003) reviewed literature on trichothecenes as related to human health. He concluded, “Toxigenic fungi capable of producing trichothecenes can be found in indoor and outdoor environments throughout the world... While the effects of trichothecenes have been extensively studied in animals the toxicology of these important mycotoxins remains largely unexplored in humans... More recent reports suggesting human health risks from non-dietary routes of exposure are difficult to objectively interpret, as the assessment of hazard and exposure in these epidemiological studies has primarily focused on toxigenic fungi, not trichothecenes” (p. 104).

The Committee on Environmental Health of the American Academy of Pediatrics wrote, “Pediatricians should be aware that there is currently no method to test humans for toxigenic molds such as *Stachybotrys* or mycotoxins” (Committee on Environmental Health, 1998, p. 714).

The Centers for Disease Control and Prevention (CDC) posts mold-related information for the public on their website in the National Environmental Health section. In their posting entitled “Questions and Answers on *Stachybotrys chartarum* and other molds” they pose a series of questions and answers (Available at <http://www.cdc.gov/nceh/airpollution/mold/stachy.htm>). Following are two of those questions and the CDC answers:

Question: “I heard about toxic molds that grow in homes and other buildings.

Should I be concerned about a serious health risk to me and my family?”

Answer: “The hazards presented by molds that may contain mycotoxins should be considered the same as other common molds which can grow in your house. There is always a little mold everywhere - in the air and on many surfaces. There are very few case reports that toxic molds (those containing certain mycotoxins) inside homes can cause unique or rare health conditions such as pulmonary hemorrhage or memory loss. These case reports are rare, and a causal link between the presence of the toxic mold and these conditions has not been proven. A common-sense approach should be used for any mold contamination existing inside buildings and homes. The common health concerns from molds include hay fever-like allergic symptoms. Certain individuals with chronic respiratory disease (chronic obstructive pulmonary disorder, asthma) may experience difficulty breathing. Individuals with immune suppression may be at increased risk for infection from molds. If you or your family members have these conditions, a qualified medical clinician should be consulted for diagnosis and treatment. For the most part, one should take routine measures to prevent mold growth in the home.”

Question: “What are the potential health effects of mold in buildings and homes?”

Answer: “Mold exposure does not always present a health problem indoors. However some people are sensitive to molds. These people may experience symptoms such as nasal stuffiness, eye irritation, or wheezing when exposed to molds. Some people may have more severe reactions to molds. Severe reactions may occur among workers exposed to large amounts of molds in occupational settings, such as farmers

working around moldy hay. Severe reactions may include fever and shortness of breath. People with chronic illnesses, such as obstructive lung disease, may develop mold infections in their lungs.”

Conclusion

The mold neurotoxicity debate is not simply about health care and science – money and litigation are salient in the concerns of those claiming neuropsychological injuries are caused by toxic mold. The campaign being waged to convince people of the dangers of “toxic mold” deserves attention and efforts to understand the underlying concerns. But bypassing scientific evidence in favor of wholesale dissemination of “toxic mold” rhetoric is not a neutral act. If it turns out that these exposures are neuropsychologically harmless, the strong claims and loud alarms sounded by lawyers, doctors and others will nonetheless have harmed numerous victims. Who will be responsible for their pain and suffering or emotional distress at being told they have “chemical AIDS” or “significantly elevated risk of cancer” or “permanent brain damage”? On the other hand if we discover evidence of causation of neuropsychological deficits in this area, the findings need to be presented widely in a forum most helpful to affected patients, not through sensationalized hyperbole. The cure for misinformation is good information, but the treatment is not easy. A substantial number of advocates are ignoring science and making vigorous efforts to make mold neurotoxicity *appear* real *before* we know the truth. But sensationalism, unfounded conjecture and manipulation of perception are no substitute for facts. The way to determine the effects of inhalation of mycotoxins and mold spores is through high quality, well-controlled scientific studies, not speculation in adversarial settings.

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It is helpful to examine the flourishing concerns about mold neurotoxicity through the lenses of texts and Internet resources that explore mistaken arguments and ineffective science. Fascinating and instructive source books for this purpose include Angell, 1996; Fumento, 1993; Gilovich, 1993; Huber, 1993; Lilienfeld, Lynn, & Lohr, 2003; Milloy, 2001; Murray, Schwartz, & Lichter, 2001; Whelan, 1993; Wildavsky, 1995. On the Internet, see Quackwatch available at <http://www.quackwatch.org/>, the American Council on Science and Health available at <http://www.acsh.org/>, and Junk Science.com at <http://www.junkscience.com/>.

As of this writing in 2003, the answer to the question posed to psychological and neuropsychological experts, whether inhalation of mycotoxins or mold spores causes neuropsychological impairment or mental disorders, is “We don’t know.” However, healthy skepticism is in order. The potential field of investigation is so vast that clear answers will not be established soon. Experts have estimated that there are close to 100,000 recognized species of fungi (e.g., Terr, 2001). Given the number of possible exposures under different environmental circumstances to persons in dramatically different mental and physical condition, one can speculate endlessly. But speculation is not evidence.

At present there is no scientific basis for claiming that individuals have suffered mental and emotional injuries by inhalation of mold, mold spores or mold metabolites, including mycotoxins in residential or office environments. To the extent that experts are expressing conclusions that mold inhalation in residences or offices caused mental or emotional injuries or brain injury, their opinions are speculation, possibilities and guesses.

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