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Commentary on Neuropsychological Performance
of Patients Following Mold Exposure

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Thank you to Dr. Dan Sudakin, Dr. Ron Gots, Dr. Bruce Kelman, and Dr. Don Millar.

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Recently Baldo, Ahmad and Ruff (2002) published a paper entitled, “Neuropsychological Performance of Patients Following Mold Exposure.” In this paper, they reported that they “investigated the effects of mold exposure (ME) on human cognition” by comparing neuropsychological data from ten toxic mold plaintiffs and ten mild traumatic brain injury plaintiffs (p. 193). The following paper reviews several issues raised by the study.

The context of the problem

Allegations of mold neurotoxicity have grown exponentially in the past decade in United States personal injury litigation. Mold health complaints have been made by celebrities such as Ed McMahon and Erin Brockovich, and the Ballard case in Dripping Springs, Texas caught the attention of attorneys, forensic neuropsychology experts, insurers, the media, and many others throughout the country when Ms. Ballard won a verdict of \$32 million dollars (later reduced but the case is still on appeal as of this writing).

Articles on toxic mold claims have appeared in most major newspapers and news magazines. For example, Time magazine’s Anita Hamilton, in her June 24, 2001 article “Beware: Toxic Mold” warned us 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). One indication of the continuing rise in the level of interest in mold claims is on the Internet. On March 15, 2003 the search term “toxic mold” on Google produced 120,000 hits, up from 63,400 only one year earlier (March 18, 2002). The content of the websites produced by this search is dominated by advocacy rather than health information. They routinely focus on the amounts of financial settlements and verdicts and many are operated by lawyers.

The article by Baldo et al. deserves attention because as the latest of only three papers reporting neuropsychological test findings, only two of which have appeared in peer-reviewed journals, it is likely to capture the attention of the toxic mold community. It merits critical review partly because of the level of impact it is likely to have and partly because there are problems with the study from a scientific perspective.

Examples of study limitations and unsupported conclusions

This cross-sectional sample is so small and diverse that this is 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.

A primary concern with this article is that 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 example, they make statements such as “The mitigating role of emotional factors, however, does not negate the seriousness or impact of ME [mold exposure]. That is, ME may have a detrimental impact on cognition indirectly via its impact on physical and emotional health” and “Although there is reason to believe that some of the observed effects on cognition were indirect, it is also possible that the ME itself had a direct impact on neurologic and thus cognitive functioning” (p. 200). In their conclusion, they state, “In summary, this study showed that (a) patients with ME are impaired on a number of measures of cognitive functioning, (b) the pattern of cognitive and psychological impairment following ME bears some similarity to that following [mild traumatic brain injury and (c) mitigating psychological factors play a role].” They go on to say, “Clinical evidence is growing to suggest that airborne mycotoxins have a direct, detrimental impact on the human brain (K. E. Gordon et al., 1993)” (p. 200) without mentioning the more numerous and more recent peer-reviewed publications that contradict this assertion (see below).

The sole study they cite as evidence of their claim that there is “growing” clinical evidence that airborne mycotoxins have this detrimental impact on the human brain is a case report of a young man exposed to moldy silage in an agricultural setting – a young man whose symptoms all resolved in one week (the young man’s father and brother recovered within 48 hours). In contrast, Baldo et al. concluded that their plaintiffs were still impaired after an average of 3.17 years from exposure onset to time of testing. Moreover, although the authors of the Gordon et al. case report indicated that the young man’s brief illness *may* have resulted from inhalation exposure to a mycotoxin, they also said that after they investigated the matter they did not reach a definitive diagnosis. Also, agricultural environments are notoriously well known to present different mold exposure levels than the offices and homes we observe in litigation, so the comparison of plaintiffs exposed in their homes and offices to the agricultural setting of a dusty silo chute is misleading if not irrelevant. Exposure to moldy silage dust near the discharge chute in a dairy farm silo is a dramatically different context than the office and residential environments in which the Baldo et al. plaintiffs were exposed. As Gordon et al. noted, in reference to the agricultural context of the exposure in their study, “Such dust can contain huge numbers of micro organisms and their metabolic by-products, including fungal elements and

mycotoxins” (p. 238). Agricultural settings commonly involve exposures exponentially greater than those seen in residential and office environments, e.g., 10,000 to 1,000,000,000 spores per cubic meter (Gots, March 3, 2003, personal communication; Lacey & Lacey, 1964; Malmberg, Rask-Andersen, & Rosenhall, 1993; Millar, March 1, 2003, personal communication).

In their review of the literature, Baldo et al. note, “To our knowledge, only one published study has conducted a detailed investigation of the neuropsychological effects of ME” (p. 194). The paper they are referring to (W. A. Gordon, et al., 1999) was not published in a peer-reviewed journal. Rather, it appeared in a conference proceedings without conventional peer review. To describe this paper as a “detailed investigation” and cite its conclusions without critical qualification is misleading. Rather than being a “detailed investigation”, the paper was quite brief and superficial and the methodology was fatally flawed. The paper reported 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. Results from only four tests were selected from the various batteries administered to support the conclusions, and a review of the entire set of test data shows that the neuropsychological test scores of the people studied were conspicuous for being *normal*, not impaired. Due to these limitations the report is mostly uninterpretable and provides no evidence for scientific purposes. In the only other 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 mold cases performed *better* on cognitive testing than the controls (Hodgson et al., 1998). This study was not mentioned in the Baldo et al. paper.

Baldo et al. cite Sudakin's 1998 study as evidence of mold causing central nervous system changes but did not mention that what Sudakin actually found was an increase in self-reported neurobehavioral symptoms in a case report, in circumstances that led him to caution readers that these individuals had been exposed to reports of adverse health effects of toxigenic fungi exposure prior to making their subjective complaints in hindsight after a delay. Baldo et al. also did not mention that Sudakin said the purportedly mold-related symptoms improved substantially after leaving the building, or that many of the people Sudakin studied were making claims for compensation. Upon review of the Baldo et al. paper, Dr. Sudakin's comment was “This article mischaracterizes my manuscript, which did not suggest that mold exposure may result in central nervous system changes. My manuscript discussed the hypothetical role of mycotoxins (not mold exposure) in the context of behavioral effects, and concluded that this was a hypothesis with unknown applicability to humans. The article by Baldo et al. does not test this hypothesis, but is rather an example of a pseudo-epidemiological study which does not provide any objective definition or quantification of exposure to mold or mycotoxins. While the authors' conclusions may be of value to plaintiff attorneys, they are not based upon sound epidemiological or toxicological principles.” (Sudakin, March 6, 2003, personal communication).

Another critical omission in the Baldo et al. article is the absence of a normal control group. Use of only plaintiffs involves an immediate selection bias. Of course plaintiffs complain of neuropsychological problems when sent to a neuropsychology expert in the course of their lawsuit – that is usually why lawyers send them to neuropsychologists. This sample selection is analogous to surveying patients in the waiting room at a physician's office and discovering that they report health symptoms. Although it occasionally happens, a plaintiff with no complaints is relatively rare. We already know that plaintiffs complain of a high rate of psychological and

neuropsychological symptoms even if neither they, their doctors, nor their lawyers are making neuropsychological claims (see e.g., Berry, Wetter, Baer & Youngjohn, 1995; Gold & Frueh, 1999; Lees-Haley, et al., 1997; Levin et al., 1987; Youngjohn, Davis, & Wolf, 1997). For a better perspective on the context of current neuropsychological evaluations, the reader should be aware that the number one referral source for neuropsychologists is lawyers, not doctors (Sweet, Moberg, & Suchy, 2000).

There were no reported controls for effects of common confounders such as medication or disease history. Apparently there was no attempt to rule out alternative toxic exposures, including other mold exposures. No objective evidence of brain injury was provided for any of the claimants, e.g., CT or MRI. There was apparently no blinding.

Although it is now well established that malingering needs to be ruled out in forensic cases (e.g., see APA, 1994, 2000), the authors performed only a superficial screening for this important problem (the Rey 15-Item Test and the Rey Dot Counting Test). To their credit, Baldo et al. acknowledge, "...future studies should include a more comprehensive assessment of motivation and malingering" (p. 200). However, based on these two Rey tests, they paradoxically make the misleading statement, "These data suggested adequate motivation, allowing the observed deficits to be interpreted with reasonable certainty" (p. 200). They make this comment despite conspicuous alarm signals such as the finding that one of their mold-exposed plaintiffs scored "in the impaired range on 10 out of 13 of the [WAIS-III] subtests given" (p. 195). To accept this finding as valid with no expression of skepticism or doubt is naïve. The average testable traumatic brain injury patient whose injury was so severe that the patient was in a coma for a month does not present with such dramatic losses on the WAIS-III one year post injury. It is common knowledge among neuropsychologists that the WAIS IQ tests are not sensitive to traumatic brain injury. Nonetheless, the authors stated that "We did not find any evidence of attempts to exaggerate..." and "All patients appeared to exert full effort..." These statements are especially puzzling given that they did not administer any substantial effort tests and they stated that future studies should be more thorough than they were in assessing motivation and malingering.

Given their finding that there was no consistent pattern of neuropsychological test performance in the mold plaintiffs, and that there was conspicuous variability between the mold plaintiffs in the number of scores in the impaired range and normal range, it is unclear how one can possibly argue that this study sheds light on cause-effect relations between mold exposure and neuropsychological deficits. The argument seems to be that some sort of undefined mold exposure causes something, but causes what? Concepts relevant to a causation analysis, e.g., strength of association, consistency of association, specificity, et cetera are conspicuously missing (e.g., see Hill, 1966). A more powerful explanatory variable for these findings is the context of litigation. Research in the last dozen years has revealed that compensation seeking has such a potent influence on psychological and neuropsychological findings that for all practical purposes it has the powerful level of impact of a diagnosable mental disorder or an important demographic variable (e.g., see Greiffenstein, Baker, & Gola, 1994; Lees-Haley & Brown, 1993; Rohling, Binder & Langhinrichsen-Rohling, 1995; Suhr, Tranel, Wefel, & Barrash, 1997).

The ten mild traumatic brain injury plaintiffs studied by Baldo et al. were defined as suffering traumatic brain injury by using the criteria proposed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Special Interest Group) (Mild Traumatic Brain Injury Committee, 1993). The Special Interest Group's criteria are so broad and nonspecific that

numerous persons report these complaints following experiences that scientists do not associate with traumatic brain injury, e.g., verbal insults, wrongful termination, various forms of discrimination, sexual harassment, and exposure to frightening events (e.g., see Lees-Haley, Fox, & Courtney, 2001).

Concluding that similarity between alleged impairments of mild traumatic brain injury plaintiffs 1.7 years post injury and mold plaintiffs implies genuine impairment related to mold is a mistake because the Baldo et al. mild traumatic brain injury group should not be testing as neuropsychologically impaired 1.7 years post-injury (Binder, Rohling, & Larrabee, 1997; Dikmen, Machamer, Winn, & Temkin, 1995). Despite the implausibility of such a finding, the authors uncritically concluded, "Some individuals in each group were quite impaired..." (Baldo et al., p. 196).

Baldo et al.'s use of a base rate >85 for the cutoff for scoring "in the clinically significant range" (p. 197) on the Millon Clinical Multiaxial Inventory – III (MCMI-III) may have led to an underestimate of pre-existing psychopathology associated with personality disorder traits and an underestimate of comorbid psychopathology. In the manual for the MCMI-III, Millon, Davis, & Millon (1997) note that for the 14 personality disorder scales BR scores of 75 suggest the presence of a trait, and BR scores of 85 indicate the presence of a disorder. For Scales A through PP, they suggest that BR 75 indicates the presence of a syndrome, and BR 85 is associated with the prominence of a syndrome. Despite their use of a higher than standard cutoff for evidence of psychopathology, 40% of the Baldo et al. mold plaintiffs scored in a range associated with at least one personality disorder, i.e., evidence of pre-existing psychopathology that could have affected the test results.

Finally, it is not clear why the time between testing and onset of exposure was used instead of time since termination of exposure, given the authors' assumption that they were evaluating the impact of a toxic exposure, and the normal expectation of improvement following removal of the toxin in most cases.

Relevant current medical views

To return to Baldo et al.'s assertion that there is a growing body of evidence that airborne mycotoxins have a direct, detrimental impact on the human brain: Following are a few of many examples of relevant and more up-to-date experts' views related to recent alarmist claims about so-called "toxic mold."

According to the American College of Occupational and Environmental Medicine (ACOEM) Evidence-based Statement, prepared under the auspices of the Council on Scientific Affairs and 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" (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 et al. (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).

According to Dr. 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). Dr. 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 that suggests 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).

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), Dr. 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, Dr. 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.

Janet Weiss, MD, 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).

In their review of literature related to the microbiology of mycotoxin-producing molds and their potential role in human immunopathology in wet buildings, Assouline-Dayana, Leong, Shoenfeld, & Gershwin concluded, “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... 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).

Kelman et al. created an inhalation exposure model to study exposure to mycotoxins in office and residential environments. They developed their model from peer-reviewed literature among other sources. Kelman et al. concluded, “It appears that even when using extremely conservative estimates, it is nearly impossible to inhale sufficient mycotoxin in office and residential environments to produce meaningful toxic effects” (Kelman, Robbins, & Swenson, in press).

Toxicologist Dr. Ron Gots in his 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).

Conclusion

In conclusion, 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. This article is likely to appeal to advocates who support the belief that vaguely defined “mold exposure” causes “brain damage” but it does not provide findings of value to the scientific community.

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