



# industry line

MANAGING THE HAZARDS OF ENVIRONMENTAL TOXIC AND TORT LITIGATION

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## Mold Caused Neuropsychological Injuries: Fact Or Fiction?

By Bruce A. Truex

Mold neurotoxicity is an increasingly common allegation in personal injury litigation. The current mold neurotoxicity controversy is, however, driven more by lawyers than scientific disagreement. These claims are variously referred to as brain damage, toxic encephalopathy, cognitive deficits, neurobehavioral deficits, and neuropsychological impairment (Lees-Haley, Ph.D., ADP, *Mold Neurotoxicity: Validity, Reliability & Baloney*) Others admit that science knows very little about how mycotoxins attack the brain, but are nonetheless certain that mycotoxins damage the brain. (Richard J. Kelly, MS, CIH, *Toxic Mold in Houses & Office Buildings*) Kelly goes on to claim that the “effect of poisoning the ‘mind’ has been examined on a few occasions” but provides the reader with no reference to any study that supports his assertion that inhaled mold spores have a physiological effect on brain function.

The primary problem with these allegations of neuropsychological impairment is that speculation has been substituted for scientific reasoning based on empirical data. There is presently no scientific basis for the allegations that breathing mold spores or mycotoxins in household or commercial offices causes neuropsychological impairment. Science simply does not know the neuropsychological effect, if any, of these exposures.

“The present alarm over harmful exposure to molds in the indoor environment derives from a belief that inhalation exposure to mycotoxins causes numerous and varied, but generally nonspecific, symptoms. Mycotoxins are “secondary metabolites” of fungi, which is to say that mycotoxins are not required for the growth and survival of the fungi species that are capable of producing them. The amount, if any, and type of mycotoxins produced is dependent on a complex and poorly understood interaction of conditions that probably include nutrition, growth substrate, moisture, temperature, maturity of fungal colony, and competition from other micro-organisms.”<sup>1,2,3,4,5</sup>

Additionally, even under the same conditions of growth, the profile and quantity of mycotoxins produced by toxigenic species can vary widely from one isolate to another.<sup>6,7,8,9</sup> Thus, it does not necessarily follow from the mere presence of a toxigenic species that mycotoxins are also present.<sup>10,11,12</sup> Mycotoxins are relatively large molecules that are not significantly volatile.<sup>13,14</sup> They do not evaporate or “off-gas” into the environment, nor do they migrate through walls or floors independent of a particle. In contrast, microbial volatile organic compounds (MVOC) are low molecular weight alcohols, aldehydes, and ketones.<sup>15</sup> Having very low odor thresholds, MVOCs are responsible for the musty, disagreeable odor associated with mold and mildew, and they may be responsible for the objectionable taste of spoiled foods.”<sup>16,17</sup> *Adverse Health Effects Associated With Mold & Indoor Environment*, American College of Occupational Environmental Medicine, Oct. 27, 2002.

The idea that mycotoxins cause toxic encephalopathy comes largely from a non-peer-reviewed presentation that was published in a book by Dr. Eckhardt Johanning. The study reported in Johanning’s book was performed by Wayne Gordon, Ph.D., and a neuropsychologist in the Department of Rehabilitation Medicine at Mt. Sinai School of Medicine. That study initially evaluated twenty patients (now increased to 38 patients) who were exposed to mycotoxins. Gordon examined the patients using a standard neuropsychological battery of tests and symptoms checklist. Using these tests, Gordon reported detecting subtle differences between the twenty patients and the control group, which had presumably not been exposed to the mycotoxins. Gordon nonetheless admitted that, “while other neurotoxins may have been present, the details were not provided.” Gordon then concluded that preliminary findings suggested exposure to toxigenic molds is associated with cognitive impairment, specifically defects in verbal memory, verbal learning, attention/concentration and set shifting. This “association” has formed the basis for numerous opinions asserting a causal connection between mold exposure and the neuropsychological injuries claimed by plaintiffs.

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In a recent deposition defended by Secrest Wardle, Dr. Gordon testified that his reported study is the only paper, as of the date of authorship, which addressed whether mold exposure caused cognitive impairment or brain injury. (Gordon Dep., p. 22) He admitted his paper indicated there was an association but not a causal link between mold exposure and cognitive impairment. (Gordon Dep., p. 23) Of equal importance, he admitted that the impairments he noted were those that could result from a number of conditions or exposures and that there was no control group. (Gordon Dep., pp. 40 and 83) Dr. Gordon's admissions make his study meaningless. Specifically, his admission that a causal link does not exist invites defendants to file *Daubert* motions seeking to strike any expert testimony alleging such a causal connection.

In the only other relevant study involving objective testing as distinguished from subjective reports (a study is published in a peer-reviewed journal), Hodgson, et al reported the results of a study of occupants of a courthouse in Florida that had previously had extensive mold growth in its walls.<sup>18</sup> The results of neuropsychological testing as reported in this study revealed that there was no difference in cognitive function between those exposed to mold and the control group. It was noted, however, that individuals with interstitial lung disease symptoms were more likely to endorse symptomology reflecting intense moods, anxiety, restlessness, irritability and other related symptoms.

As a part of ongoing research intended to support a preconceived causal connection between mycotoxins and neuropsychological injuries, additional studies are currently being conducted. K. Savolainen of the Finnish Institute of Occupational Health, after acknowledging that neuronal effects of the mycotoxin fumonisin B1 (FB1) are not well known, noted that FB1 inhibits normal growth of neuronal cells and disturbs myelin formation. The aim of the study was to investigate the mechanism whereby the mycotoxin FB1 affected neuronal and glial cells. The study concluded, among other things, that FB1 decreased the cell viability and that exposure leads to DNA fragmentation. Nonetheless, the study did not establish a causal link between neuropsychological symptoms and human inhalation exposure to mycotoxins or mold spores. Moreover, the study was critically flawed because it was an *in vitro* study that soaked the subject cells in purified toxins for six days without controls. There was no evidence presented that these mycotoxins could be absorbed in the bloodstream via inhalation and certainly not in the quantities used for the soaking of the cells. Nor was there any evidence that FB1 mycotoxins could cross the blood-brain barrier.

Based upon the limited scientific information available to date, a more logical explanation for a plaintiff's neuropsychological symptoms is litigation. Patients pursuing litigation report more intense, frequent and persistent symptoms than non-litigating patients do. 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, Imel, Jane & Nelson, 1989; Dikman, Ross, Machammer & Temkin, 1995; Dikman, 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 commonly does not conform to expectations, as complaints continue long after such symptoms normally resolve (Binder, Rohling & Larrabee, 1997). Numerous empirical investigations have documented discrepancies between patients in litigation and patients not seeking compensation. (*See e.g.*, Berry, Wetter & Youngjohn, 1995; Levin, et al 1987; Youngjohn, Davis & Wolf, 1997; Fee and Rutherford, 1988). Lees-Haley, Ph.D., ABPP (paul@lees-haley.com)

Patients seeking compensation have more incentives to produce false or exaggerated symptom reports than patients seeking treatment do. In personal injury cases, feigned cognitive deficits have been estimated at 64% (Heaton, Smith, Lehman & Vogt, 1978) and 47% of workers' compensation possibly involving malingering (Youngjohn, 1991). Another study estimated the percentage of manufactured memory deficits in patients claiming persistent post-concussive syndrome as being between 33-60% (Greiffenstein, Baker & Gola, 1998).

Despite these studies, some psychologists and psychiatrists claim that plaintiffs are unable to fake mental disorders or neuropsychological deficits without detection by psychological experts. There is, however, substantial empirical support for the fact that naïve individuals can fake psychological and neuropsychological symptoms successfully when provided minimal information about disorders (e.g., Albert, Fox & Kahn, 1980; Faust, Hart & Guilmetee, 1988; Lamb, Berry, Wetter & Baer, 1994; Rogers, Bagby & Chakraborty, 1993; Rogers, Orndeff & Sewell, 1993; Wetter, Baer, Berry, Robinson & Sumpter, 1993; Lamb, et al, 1994).

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