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Neuropsychological exploration of alleged mold neurotoxicity

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Abstract

Cognitive and emotional correlates of toxic mold exposure and potential dose–response effects for both outcomes were investigated. Self-reported length of exposure, time since last exposure, and serum immunoglobulin (IgG) levels were assessed. Despite CNS complaints often seen with mold exposed individuals, overall results did not uncover concomitant cognitive deficits suggested in previous studies or a significant reduction in intellectual functioning. Fewer subjects were excluded as result of failing effort/motivation assessment than expected. Correlations of IgG and cognitive function are discussed. A dose-effect for self-reported length of exposure and cognitive outcome was not seen. The sample's overall Minnesota Multiphasic Personality Inventory II (MMPI-2) profile indicated elevations on scales 1, 2, 3, 7 and 8. MMPI-2 clinical scales 1 and 3 were significantly correlated with length of exposure. The MMPI-2 may be sensitive to increasing physical and emotional sequelae as length of exposure increases. A potential subgroup of cognitively impaired outliers within mold exposure litigants is explored. Limitations of self-reported and objective measurements for mold exposure and exploratory statistical methodology are discussed.

Keywords: Mold; Cognition; Neurotoxicity; Litigation; MMPI; Stachybotrys

The purpose of the present study is to explore potential cognitive and emotional correlates of toxic mold exposure as well as the potential dose–response effects for both outcomes. There are more than 1000 different kinds of molds that have been found in U.S. homes (CDC, 2002). *Stachybotrys chartarum* (*S. chartarum*) in particular has received an increasing amount of attention in the media and is often cited in an increasing number of personal litigation cases throughout the country. Despite increased attention by the public and among health and mental health professionals regarding potential mold neurotoxicity, there is a paucity of research in this area along with many unresolved scientific questions and issues. *S. chartarum* is one of many kinds of molds that can produce mycotoxins, which are chemicals that may be able to cause a multitude of symptoms and illnesses in humans and animals. Mycotoxins may become airborne, inhaled, and once deposited in the lungs may react with biomolecules and can be absorbed into the blood (IOM, 2004). The effects of *S. chartarum* were noted at least as far back as the 1930s and 1940s, when horses in Eastern Europe experienced immune suppression, infection and bleeding that was fatal after eating heavily *S. chartarum*-contaminated

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fodder (CADHS, 2000). Human health effects such as dermatological and respiratory syndromes were seen in the Russian farm workers that handled musty straw (Drobotko, 1945; Drobotko et al., 1946; Linnik, 1949, as cited in Kuhn & Ghannoum, 2003).

The casual relationships between mold exposure and subsequent inhalation of mycotoxins in the home or work/office environments and detrimental health effects remain unclear. However, clinical and epidemiological studies have suggested that complaints often include respiratory symptoms, ocular disturbances, skin irritations, headache, fever/chills, and CNS complaints (i.e. concentration and memory difficulties, fatigue, dizziness, etc.) (Gordon et al., 2004; Johanninh, Landsbergis, Gareis, Yang, & Olmstead, 1999). Despite the frequent list of CNS-related complaints reported by individuals who claim mold exposure it is presently unclear whether airborne mold or its byproducts found in the home or at the workplace are in fact "neurotoxic". Some initial neuropsychological studies that have been conducted suggested exposure may have a detrimental direct and/or indirect adverse impact on cognitive functioning (Baldo, Ahmed, & Ruff, 2002; Gordon, Johanning, & Haddad, 1999). However, it appears that methodological limitations of existing research limit the ability to make more clearly informed statements regarding the effects of mold on human CNS and neuropsychological functioning (Lees-Haley, 2003).

There are many problematic issues inherent in the scientific study of mold exposure in humans. One of the most difficult issues is quantifying an individual's level of exposure. Air samples deriving the number of colony forming units (CFU) per cubic meter of air (m^3) are somewhat misleading and the exact value of this information is not yet clear. It is possible that the minute particles in question are susceptible to varying environmental conditions as it is their airborne ability itself that allows spores to enter the lungs. It is likely that there can be significant variation in different specimens taken and drastic increases in airborne mold samples have been shown to be affected by the actual clean-up procedures themselves such as vacuuming or construction (Hunter, Grant, Flannigan, & Bravery, 1988). Another misleading issue related to mold exposure is that even if potentially toxogenic fungi are found in the home it does not necessarily imply the presence of mycotoxins, and even if a specific mycotoxin is found it does not necessarily mean that a particular species is currently present (Kuhn & Ghannoum, 2003; Tuomi et al., 2000). A recent study by Crago et al. (2003), which reported a dose–response relationship with neuropsychological functioning, utilized a sophisticated formula for measurement of exposure and included average hours per day in a building, days of exposure, air samples, and whether *S. chartarum* was present. However, their estimate of exposure is a combination of self-report and objective data, and as suggested from the above literature the accuracy of "objective measures" by sampling air particle units may be subject to significant variability. In addition to objective estimates, self-report data as it pertains to mold exposure and cognitive dysfunction will be explored in the present study and discussed in sections to follow.

Although many physicians use blood antibody tests as a biologic marker of mold exposure, this is also problematic. For instance, a *S. chartarum* antibody test is sensitive to cross-reaction and can thus be positive when an individual has been exposed to other types of molds altogether (CDC, 2002). *S. chartarum* species rarely exist in isolation and the mixed mold exposure model utilized by Crago et al. (2003) is likely more appropriate than merely isolating *S. chartarum* as a variable for study. While many patient's seen in a clinical neuropsychological setting often report positive blood antibody findings little is currently known about the quantitative relation between serum IgG and airborne exposure (IOM, 2004) and it is unclear whether the "objective data" currently utilized is significantly contributory toward an accurate estimate of an individual's overall level of exposure. Using a control group in comparison to a mold exposed group Gray et al. (2003) found significant group differences in levels of antibodies between groups, but these groups were not compared on objective neuropsychological and/or psychological outcome measures.

Despite these problematic issues, scientific investigation must continue to try and accurately uncover the potential neurocognitive and emotional correlates of mold exposure, including the presence of any dose–response effects for both outcomes. The findings of a recent study by Crago et al. (2003) that "a dose–response relationship between measures of mold exposure and abnormal neuropsychological test results and QEEG measures suggested that toxic mold causes significant problems in exposed individuals" (p. 452), are provocative. The present study sought to add to our understanding of possible mold neurotoxicity among individuals who report mold exposure and subsequent CNS, cognitive and behavioral effects.

Specifically, dose–response effects for mold exposure on both cognitive and emotional outcome measures were explored. Our hypotheses were: (1) the sample would show overall neurocognitive impairment in multiple domains and a decline from estimates of previous functioning, (2) objective estimates of exposure (IgG) will correlate with poor neuropsychological performance and increased emotional sequelae, (3) that increased length of time of self-reported mold exposure is associated with poorer neuropsychological performance and increased emotional sequelae,