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Cognitive Impairment Associated With Toxigenic Fungal Exposure: A Replication and Extension of Previous Findings

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In this study, neuropsychological data and symptom reports from 31 individuals exposed to toxic mold were examined. Most participants were found to have reduced cognitive functioning in multiple domains, with memory and executive functions the most commonly affected areas. Rates of dysfunction were significantly greater than chance on more than half of the tests. Number of cognitive impairments was found to be related to depression, although few neuropsychological test scores were correlated with depression. Results also indicated that symptom report of the mold-exposed participants was not significantly different from that of matched groups of 65 persons with mild traumatic brain injury (TBI) and 26 individuals with moderate TBI. The mold-exposed participants reported significantly more symptoms than 47 people with no disability. This study adds to a growing body of literature (e.g., Baldo, Ahmad, & Ruff, 2002; Gordon, Johanning, & Haddad, 1999) relating exposure to mycotoxins to cognitive dysfunction.

Key words: toxic mold, brain injury, cognitive impairment, mycotoxins, neuropsychological

In 1999, Gordon, Johanning, and Haddad (1999) reported findings based on a battery of neuropsychological tests administered to a group of 20 individuals who were exposed to toxic mold (i.e., mycotoxins). They found that that the distribution of scores on some measures of cognitive function (i.e., integration of visual-spatial information, verbal learning, attention, and set-shifting) was negatively skewed toward the low end of the normal curve. This was surprising given that participants' mean IQ score fell in the high average range. Participants also reported many cognitive, physical, and behavioral symptoms. The data suggested that cognitive impairment was a common sequela of exposure to these molds. The negative health effects resulting from exposure to mycotoxins are well documented. These include immune suppression (with increased susceptibility to disease), upper respiratory symptoms (e.g., dyspnea, cough), gastrointestinal disturbances, fatigue, dermatological problems, and in-

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creased risk of cancer (Etzel, 2002; Johanning, 1999). Neurological symptoms such as memory loss, disorientation, and confusion have also been documented (e.g., Augerson, 2000; Etzel, 2002).

Gordon et al. (1999) were the first to describe an association between cognitive impairment and exposure to mycotoxins using results from neuropsychological testing. An earlier study by Hodgson et al. (1998) compared neuropsychological test performance in a sample of persons exposed to mold and in a matched sample of controls. The results did not "support the hypothesis of lower cognitive function among cases" (p. 246). Although this finding may, at first, appear to contradict those of Gordon et al. (1999), there are several factors that limit the conclusions that can be drawn from them. First, and perhaps most important, the authors do not provide key information about cognitive impairment in their participants. They do not state how many of the participants complained of cognitive impairments, the nature of the complaints of these individuals is not reported, and the number of individuals with cognitive complaints who were actually tested is not reported. This is critical issue because not all persons exposed to mold experience cognitive difficulties. Thus, the sample may not have been an appropriate group for the study of cognitive deficits associated with exposure to toxic mold. Second, the neuropsychological battery was not described (only two tests were mentioned). Thus it is impossible to determine whether the tests were appropriate to the (unspecified) "changes in mental status" in an unspecified number of participants. Finally, no descriptive or inferential statistics from the tests that were administered were reported.

Baldo, Ahmad, and Ruff (2002) offered evidence of cognitive impairments on neuropsychological tests in persons exposed to mycotoxins. In a sample of 10 persons exposed to mold, they documented impairments on several measures of cognitive functioning, including, in particular, visuospatial and verbal learning, visuospatial memory, and psychomotor speed. In addition, they reported: (a) the neuropsychological test scores of persons exposed to mold were similar to those of a group of individuals with traumatic brain injury (TBI) and (b) participants' scores on the Beck Depression Inventory–Second Edition (BDI–II) were significantly correlated with the number of impairments found on neuropsychological tests.

The purpose of this study was to build on the findings of previous research (Baldo et al., 2002; Gordon et al., 1999) using data collected from a new sample of persons exposed to toxic mold. In particular, we were interested in determining (a) whether findings from previous studies could be replicated in terms of the nature and extent of cognitive impairments associated with mold exposure and (b) the similarities or differences in symptom report of individuals exposed to mycotoxins, persons with TBI, and persons without brain injury or mycotoxin exposure. The following questions were addressed:

1. Is the symptom report of persons exposed to toxic mold similar to that of individuals with TBI or to that of individuals without brain injury?
2. What is the nature of cognitive dysfunction found in persons exposed to toxic mold?
3. What is the relation between cognitive dysfunction in persons exposed to toxic mold and self-report of mood?
4. How extensive is cognitive dysfunction among persons exposed to toxic mold?
5. Are specific memory processes impaired in persons exposed to toxic mold?

Methods

Participants

Participants were 31 of 38 individuals evaluated at both the Eastern New York Occupational and Environmental Health Center by Eckardt Johanning and the Department of Rehabilitation Medicine at Mount Sinai School of Medicine between August, 1998 and July, 2001. Seven of the participants were excluded from the study because of a comorbid or premorbid condition that could potentially account for any cognitive impairments observed (e.g., psychiatric history, substance abuse, learning disability, neurological disorder). All data on the mold-exposed group were obtained through chart reviews approved by an institutional review board.

All participants reported cognitive difficulties as well as other health problems after exposure to toxic mold such as *stachybotrys atra, penicillium,* and *aspergillus*. Participants may have been exposed to other mycotoxins as well. Typical mold-related health complaints such as irritation and allergies, chronic rhino-sinusitis, lower airway problems, and immune system disorders were diagnosed in all participants. Apart from exposure to mycotoxins, participants reported no history of brain injury, neurological disease, substance abuse, significant psychiatric disorder, or other risk factors for cognitive impairment prior to their exposure to mold. Exposure to mold was either at home

or at work and was verified by review of environmental testing results. Duration of exposure varied between months and years and was often difficult to determine because exposure may have begun prior to onset of symptoms. Indeed, the onset of symptoms was often the stimulus for efforts to locate toxic mold in the person's environment. Therefore, it could not be determined how long the mold was present prior to its discovery. In all cases, exposure was terminated by the time testing was initiated, as participants were no longer living or working in the "contaminated" environment. Twenty nine of the participants were involved in litigation when tested. No evidence of malingering or symptom exaggeration was found in participants exposed to mold. Malingering and symptom exaggeration were assessed using several tests and methods, including the Test of Memory Malingering (Tombaugh, 1996), Memorization of 15 Items (Rey, 1964), and examination of data for response patterns consistent with malingering (e.g., very low scores on forced choice recognition tasks). Demographic data on the sample are provided in Table 1.

For purposes of comparing symptom report, three comparison groups were used: Group 1 was comprised of 65 individuals with mild TBI (less than 20 min of altered mental status); Group 2 was made up of 26 individuals with moderate TBI (20 min to 24 hr of altered mental status), and Group 3 was made up of 47 persons with no disability. The data for the individuals in the comparison groups were selected from a large database maintained by the Research and Training Center on Community Integration of Individuals with TBI (RTC) at Mount Sinai School of Medicine. All members of the TBI group met American Congress of Rehabilitation Medicine criteria for TBI. Mean length of time since injury was 3.89 years ($SD = 7.43$) for the mild TBI group and 2.68 years ($SD = 2.27$) for the moderate TBI group. This difference was not statistically significant. Demographic data on the comparison groups are provided in Table 1. Computation of analysis of variance (ANOVA) and chi-square tests indicated that there were no significant differences between the four groups in age, sex, or education. Informed consent was obtained from all participants in the nondisabled and TBI groups. The individuals in the TBI groups were participants in research projects on quality of life following TBI. No neuropsychological test data or information on litigation were available from these individuals.

Materials and Procedures

All 31 participants exposed to toxic mold were administered a comprehensive neuropsychological battery. Although there was some variability in the tests administered, all participants were given the Wechsler Adult Intelligence Scale III (WAIS–III; Wechsler, 1997a), Wechsler Memory Scale–III (WMS–III; Wechsler, 1997b), and California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987). Most batteries also included the Continuous Performance Test (CPT; Conners and Multi-Health Systems Staff, 1995, $n = 30$), Trail Making Test A ($n = 27$) and B ($n = 28$; TMT A and B; Heaton, Grant, & Matthews, 1991), Short Category Test (CT; Wetzel & Boll, 1987, $n = 26$), Watson Glaser Critical Thinking Appraisal – Form B (Watson–Glaser; Watson & Glaser, 1980, $n = 25$) and subtests from the Woodcock Johnson Tests of Cognitive Abilities (WJTCA; Woodcock & Mather, 1989, $n = 29$). Thus, in addition to the WAIS–III, scores were used from 16 tests or subtests of memory and learning (WMS–III, CVLT), 3 tests of executive functions (TMT B, CT, Watson-Glaser), 4 tests of attentional abilities (CPT, TMT A), and 2 tests of processing speed (WJTCA). Other tests administered (e.g., Grooved Pegboard, Purdue Pegboard, Iowa Silent Reading Test–Level 3) are not included in the present analyses

Table 1. *Demographic Data*

Variable	Groups			
	Mold Exposure[a]	Mild TBI[b]	Moderate TBI[c]	No Disability[d]
M Age	44.10 ($SD = 9.91$)	44.74 ($SD = 11.09$)	44.91 ($SD = 10.52$)	44.38 ($SD = 14.48$)
Sex				
Male	19 (40.4%)	10 (32.3%)	22 (34%)	13 (50%)
Female	21 (67.7%)	43 (66%)	13 (50%)	28 (59.6%)
Years of Education[e]				
≤High school	10 (32.3%)	16 (24.6%)	7 (26.9%)	8 (17%)
Some post-secondary	2 (6.5%)	18 (27.7%)	11 (42.3%)	15 (31.9%)
≥Bachelors degree	17 (54.8%)	30 (46.2%)	8 (30.8%)	24 (51.1%)

Note: TBI = traumatic brain injury.
[a]$n = 31$. [b]$n = 65$. [c]$n = 26$. [d]$n = 47$. [e]In the Mild TBI group, one subject did not provide education information.

because they were administered to less than 80% of the sample.

In addition, 30 participants completed the Brain Injury Screening Questionnaire (BISQ). Those in the comparison groups were administered the symptom inventory portion of the BISQ as part of a larger structured interview. The BISQ (Research and Training Center on Community Integration of Individuals with Traumatic Brain Injury, 1997) was developed as a screening instrument for brain injury. The format for the BISQ is based on the "HELPS" instrument (Picard, Scarisbrick, & Paluck, 1991). Parts of the content of the BISQ were adapted from two brain injury symptom checklists developed by Lehmkuhl (1988) at TIRR and at the Medical College of Virginia (undated).

The BISQ documents: (a) events that lead to brain injury, (b) functional difficulties and symptoms associated with brain injury, and (c) events and conditions other than brain injury that might lead to symptoms similar to those seen in brain injury. The BISQ includes an inventory of 100 cognitive, physical, and emotional/behavioral symptoms commonly found after brain injury such as memory problems, thinking slowly, trouble following instructions, difficulty in dealing with people, feeling frustrated, dizziness, and headaches. Fifty seven of these symptoms were adapted from existing brain injury symptom checklists (Lehmkuhl, 1988; TBI Symptom Checklist, undated).

Results

BISQ Data

Means and standard deviations of physical, cognitive, behavioral, and total symptoms reported on the BISQ by the mold-exposure group and comparison groups are presented in Table 2. In a study that formed part of the basis for the development of the BISQ, Gordon, Haddad, Brown, Hibbard, and Sliwinski (2000) used the TIRR Symptom Checklist (Lehmkuhl, 1988) to examine the symptom report of more than 1,100 individuals with mild TBI, individuals with moderate/severe TBI, individuals with spinal cord injury, HIV-positive persons, postoperative liver transplant patients, and persons with no disability. They used logistic regression to identify symptoms that were both sensitive (reported by more than 33% of the TBI groups) and specific (reported by less than 10% of the non disabled group and less than 25% of the other groups) to TBI. Twenty five symptoms (23 cognitive, 1 behavioral, 1 physical) that were sensitive and specific (S&S) to mild TBI were identified. Five (all cognitive) of these 25 symptoms were also S&S to moderate/severe TBI. Means and standard deviations for these 25 S&S symptoms for the four groups in this study are also reported in Table 2.

ANOVA was used to compare means across groups. As shown in Table 2, significant differences were found across groups for all categories of symptoms (physical, cognitive, behavioral, total, S&S). Post hoc Bonferroni tests revealed that in all cases but one (the moderate TBI group had more physical symptoms than the mold group, $p < .04$), there were no significant differences in symptom report between individuals with TBI and persons exposed to mold. In all categories but one (behavioral symptoms), both individuals with TBI and persons exposed to mold reported significantly more symptoms than participants with no disability ($p < .001$). In the case of behavioral symptoms, there was no significant difference between the number reported by the mold-exposure group and the nondisabled group. These findings indicate that the symptom report of individuals exposed to mold is similar to that of those with known brain injury and different from that of persons without brain injury.

Table 2. *BISQ Symptom Report: Means and Standard Deviations and Results of ANOVAs Comparing Mean Numbers of BISQ Symptoms Between Groups*

	Groups										
	Mold[a]		Mild TBI[b]		Moderate TBI[c]		No Disability[d]				
Type of Symptoms	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*df*	*F*	*p*
Physical	6.07	3.73	7.11	4.88	9.12	4.78	1.87	2.67	3	21.74	<.001
Cognitive	18.67	11.06	21.25	14.59	25.65	14.34	2.66	5.47	3	29.48	<.001
Behavioral	8.13	6.82	11.89	8.20	13.00	7.98	4.19	5.76	3	12.84	<.001
All	32.87	19.37	40.25	26.17	47.77	24.65	8.72	12.37	3	25.78	<.001
25 S&S[e]	10.10	6.23	8.72	6.38	11.27	6.85	1.02	2.53	3	25.43	<.001

Note. BISQ = Brain Injury Screening Questionaire; ANOVA = Analyses of Variance; TBI = traumatic brain injury.
[a] $n = 30$. [b] $n = 65$. [c] $n = 26$. [d] $n = 47$. [e] S&S = symptoms sensitive and specific to TBI (Gordon et al., 2000).

Table 3. *Mean Test Scores, Standard Deviations, and Proportions of Individuals Exhibiting Reduced Function and Impairments on Neuropsychological Tests and Subtests*

Tests by Test Type	*M*	*SD*	Below 16th and 5th Percentiles
Attention			
TMT A	49[a]	28	.22/.03
CPT hits	58[a]	28	.00**/.00
CPT omissions	58[a]	28	.03*/.00
CPT commissions	44[a]	30	.17/.03
Processing speed			
WJTCA Visual Matching	62[a]	31	.10/.10
WJTCA Cross Out	52[a]	27	.17/.06
Executive functions			
TMT B	54[a]	27	.11/.00
Watson–Glaser	40[a]	32	.36*/.29**
Booklet category	45[a]	35	.31*/.16*
Memory			
CVLT Trials 1–5	38[b]	15	.48**/.26**
CVLT Trial 1	−.97[c]	1.01	.68**/.26**
CVLT Trial 5	−1.15[c]	1.77	.52**/.32**
CVLT List B	−.63[c]	1.03	.58**/.16*
CVLT Long delay cued recall	−1.07[c]	1.70	.52**/.26**
CVLT Short delay cued recall	−1.05[c]	1.66	.48**/.39**
CVLT Short delay free recall	−1.10[c]	1.53	.65**/.35**
CVLT Long delay free recall	−1.26[c]	1.63	.58**/.35**
WMS–III General memory	100[d]	19	.29*/.06
WMS–III Working memory	103[d]	14	.10/.03
WMS–III Auditory immediate	99[d]	16	.17/.06
WMS–III Visual immediate	97[d]	16	.33*/.06
WMS–III Immediate memory	98[d]	19	.23/.10
WMS–III Auditory delayed	101[d]	17	.17/.03
WMS–III Visual delayed	97[d]	18	.33*/.13
WMS–III Auditory recognition delayed	101[d]	19	.13/.06

Note. TMT = Trail Making Test; CPT = Continuous Performance Test; WJTCA = Woodcock Johnson Tests of Cognitive Abilities; CVLT = California Verbal Learning Test; WMS–III = Wechsler Memory Scale–Third Edition.
[a]Percentiles. [b]*T* scores. [c]*Z* scores. [d]Standard scores.
*Significantly different from proportion expected by chance ($p < .05$, chance = .16). **Significantly different from proportion expected by chance ($p < .01$, chance = .05 or .16).

Neuropsychological Test Findings

Did the sample group exhibit significant patterns of impairments on neuropsychological tests? As presented in Table 3, mean scores for the sample on all neuropsychological tests, except the CVLT, fell within the average range. The sample's IQ scores were normally distributed ($M = 108$, $SD = 16$, skewness = −.237, kurtosis = .003) and slightly above the population mean. Reduced functioning was operationalized as scores at or below the 16th percentile and impairment was operationalized as scores at or below the 5th percentile. Table 3 presents proportions of participants in the mold-exposure group who met criteria for reduced functioning or impairment on each neuropsychological test. All participants but one (97%) demonstrated reduced functioning on at least one of 25 possible measures (maximum = 17, $M = 6.97$, $SD = 5.09$) and over 70% exhibited impairments on at least one measure. The mean number of impairments was 3.61 ($SD = 4.08$).

Were the proportions of persons with reduced function or impairment found in the sample significantly greater than chance? To determine whether proportions of impaired participants on each test were significantly different from those that would be expected by chance, binomial tests were computed. Each of these tests compared the observed proportion of individuals with reduced function or impairments to that expected by chance (.16 and .05, respectively). Significant proportions of individuals with reduced function and impairment were found for some tests of executive functions (Watson–Glaser, reduced function $p = .012$/impairment $p < .001$; Booklet Category Test, reduced function $p = .045$/impairment $p < .018$) and

learning and memory (all CVLT subtests used $p < .001$, except CVLT List B, $p = .018$ for impairment). For some tests of memory, significantly more participants than would be expected by chance had reduced function (WMS–III General Memory, $p = .049$; WMS–III Visual Immediate Memory, $p = .015$; WMS–III Visual Delayed Memory, $p = .015$), but the proportion with impairments was not statistically significant. It should be noted that on the two CPT measures used, there were significantly *fewer* persons with reduced function than would be expected by chance.

Did the sample group exhibit reduced function and impairments in multiple cognitive domains? As shown in Table 4, the majority of the sample (65%) had reduced functioning in multiple domains and 35% had multidomain impairments. Table 5 indicates that memory dysfunction was the most common area of difficulty, followed by executive function disturbances. For attention, reduced function was more common than impairment and for processing speed the reverse held true. Impairments on tests of attention and processing speed were not found in the absence of memory problems.

Were participants' scores on memory tests significantly lower than their scores on IQ tests? Because the WMS–III was standardized on a group who were also given the WAIS–III, computation of comparisons between memory scores (WMS–III index scores) and overall ability (WAIS–III IQ scores) is possible (Psychological Corporation, 1997). These com-

Table 4. *Frequencies and Percentages of Reduced Functioning (RF) and Impairments in Individuals Exposed to Mold by Cognitive Domains and Number of Domains Affected*

Reduced Function/ Impairment by Domains	Frequencies and Percentages	
	16th Percentile	5th Percentile
No RF[a] or Impairment	1 (3%)	9 (29%)
RF/Impairment in one domain	10 (32%)	8 (26%)
Memory only	8 (26%)	8 (26%)
Executive functions only	2 (7%)	0 (0%)
Processing speed only	0 (0%)	0 (0%)
Attention only	0 (0%)	0 (0%)
RF/Impairment in two domains	12 (39%)	9 (29%)
Memory and Executive functions	6 (20%)	6 (20%)
Memory and Processing speed	0 (0%)	2 (6%)
Memory and Attention	6 (20%)	1 (3%)
Executive functions and Processing speed	0 (0%)	0 (0%)
Executive Functions and Attention	0 (0%)	0 (0%)
Attention and Processing speed	0 (0%)	0 (0%)
RF/Impairment in Three domains	7 (23%)	2 (6%)
Memory and Executive functions and Processing speed	2 (7%)	1 (3%)
Memory and Executive functions and Attention	3 (10%)	1 (3%)
Executive functions and Attention and Processing speed	0 (0%)	0 (0%)
Memory and Attention and Processing speed	2 (7%)	0 (0%)
RF/Impairment in All Four Domains	1 (3%)	0 (0%)
Memory and Executive functions and Processing speed and Attention	1 (3%)	0 (0%)
Total	31 (100%)	

Table 5. *Frequencies and Percentages of Reduced Functioning (RF) and Impairments in Individuals Exposed to Mold by Cognitive Domains*

Reduced Function/Impairment by Domains	Frequencies and Percentages	
	16th Percentile (RF)	5th Percentile (Impairment)
No RF or Impairment	1 (3%)	9 (29%)
RF/Impairment in Memory	28 (90%)	19 (62%)
RF/Impairment in Executive functions	14 (45%)	11 (36%)
RF/Impairment in Attention	12 (39%)	2 (7%)
RF/Impairment in Processing speed	5 (17%)	3 (10%)

parisons provide a means for examining relative deficits within individuals and, thus, provide a more sensitive measure of impairment than differences from group norms. Proportions of individuals with significant discrepancies between WAIS–III IQ scores and WMS–III Index scores are provided in Table 6. In all cases where significant discrepancies occurred, WMS–III scores fell *below* corresponding IQ scores. On all WMS–III tests, about a quarter or more of the sample had significant discrepancies between intelligence and memory. Auditory memory was the area of least discrepancy.

To determine whether the proportions of discrepant scores were greater than would be expected by chance, binomial tests were used to compare observed proportions to those found in the WAIS–III/WMS–III normative sample. These "expected" proportions are presented in Table 6. Significant differences were found for Full Scale IQ (FSIQ) versus Immediate Memory ($p = .004$), FSIQ versus General Memory ($p = .034$), FSIQ versus Immediate Memory ($p = .004$), FSIQ versus Working Memory ($p = .013$), Performance IQ (PIQ) versus Visual Immediate ($p < .001$), PIQ versus Visual Delayed ($p < .001$), and Verbal IQ (VIQ) versus Auditory Recognition Delayed ($p < .001$). The differences for VIQ versus Auditory Immediate and Auditory Delayed were not significant. Therefore, for all tests except two indexes of auditory memory, significantly more discrepancies between memory and IQ were found than would be expected by chance. These differences were not trivial, as the observed proportions for those measures where significant differences were found were two to four times greater than those expected on the basis of chance.

Table 6. *Proportions of Individuals Exposed to Mold with Significant Discrepancies Between IQ and WMS-III Scores and Expected Proportions*

Discrepancy	Observed Proportion	Expected Proportion
FSIQ vs. WMS–III General Memory	.29*	.15
FSIQ vs. WMS–III Immediate Memory	.35**	.15
FSIQ vs. WMS–III Working Memory	.40*	.20
PIQ vs. WMS–III Visual Immediate	.55**	.15
PIQ vs. WMS–III Visual Delayed	.40**	.10
VIQ vs. WMS–III Auditory Immediate	.20	.20
VIQ vs. WMS–III Auditory Delayed	.20	.20
VIQ vs. WMS–III Auditory Recognition Delayed	.60**	.25

Note. WMS–III = Wechsler Memory Scale–Third Edition; WAIS–III = Wechsler Adult Intelligence Scale–Third Edition; FSIQ = WAIS–III Full Scale IQ; PIQ = WAIS–III Performance IQ; VIQ = WAIS–III Verbal IQ. *Denotes observed proportion significantly greater than expected proportion derived from normative data ($p > .05$). **Denotes observed proportion significantly greater than expected proportion derived from normative data ($p > .01$).

Were specific patterns of memory dysfunction detectable in the sample? Because memory dysfunction was the most common type of cognitive deficit, we attempted to determine whether specific patterns of memory deficits were present in the sample. Curtiss, Vanderploeg, Spencer, and Salazar (2001) examined patterns of verbal learning in two samples of individuals with TBI. They calculated seven indexes of memory, six of which were based on Baddeley's (1976) model of memory. Indexes were computed using items from the CVLT and WMS–R Digit Span subtest. The indexes measured *Span* (holding items in working memory), *Central Executive* ("ability to manipulate information in working memory, regardless of span capacity," p. 576), *Consolidation* ("consistent ... and effective learning over five learning trials," p. 576), *Encoding* ("ability to impose and use an effective semantic strategy to encode information," p. 576), *Retention* (ability to remember information for a period of 20 to 30 min), and *Retrieval* (being able to retrieve newly learned information from long-term memory). *Control* (ability to avoid intrusions and perseverations), the seventh index, was not based on Baddeley's model but on previous findings concerning difficulties often experienced by individuals with TBI on the CVLT. Using cluster analysis, Curtiss et al. found five patterns of memory disturbances that corresponded to Baddeley's a theoretical model of memory.

In this study, five of the seven indexes were computed using CVLT data. To compute index scores, Curtiss et al. (2001) derived z scores using data from their sample. Because the sample in this study was not large enough to derive z scores, index scores were calculated using normative CVLT data. The Central Exec-

utive and Span indexes were not calculated because normative data for the Forward and Backward subscores of the WMS–R Digit Span subtest have not been published and were not available from Psychological Corporation.

Mean levels of performance were worst on the Encoding index ($M = -.52$, $SD = 1.28$), suggesting that many participants had significant impairment in this area. Consolidation index scores ($M = -.35$, $SD = .89$) were somewhat greater and Control ($M = -.02$, $SD = .71$), Retention ($M = -.14$, $SD = .31$), and Retrieval ($M = .03$, $SD = .24$) scores varied very little from the mean. Inspection of the standard deviations for each mean index score shows high variability in the samples. This variability suggests a wide range of scores on different measures and may indicate that, rather than a single overall pattern of memory dysfunction, there were different patterns of memory impairments within the sample (as in Curtiss et al., 2000) or simply strengths and weaknesses idiosyncratic to individuals.

Is there a relation between severity of self-reported symptoms of depression and reduced function and impairments on neuropsychological tests? Baldo et al. (2002) found a significant association between scores on the BDI–II and the number of test scores in the impaired range (below the 10th percentile). Although the mean BDI–II score for the sample in this study was elevated ($M = 20.18$, $SD = 10.37$), there was no significant correlation between BDI–II scores and the number of tests on which there was reduced function. However, the number of impairments (scores at or below the 5th percentile) *was* correlated with BDI–II scores ($r = .41$, $p = .032$). Although it would appear that more severe disruption of cognitive functions was related to depression, BDI–II scores were significantly correlated with scores on only four of the neuropsychological tests and subtests. Three of the correlations were with scores on tests of memory and learning (CVLT Trials 1–5, $r = -.40$, $p = .034$; CVLT Trial 1, $r = -.40$, $p = .038$; CVLT List B, $r = -.56$, $p = .002$) and one with scores on a test of attention (TMT A, $r = -.52$, $p = .006$). Thus, the relation between depression and cognitive impairment appears to be task specific rather than generalized (i.e., correlated with the majority of measures).

Discussion

The current findings of cognitive impairment are consistent with those of Gordon et al. (1999) and Baldo et al. (2002). In addition, persons exposed to mycotoxins reported significantly more cognitive and physical symptoms than nondisabled individuals. This finding takes on added salience because there was virtually no significant difference in self-report of cognitive, physical, and emotional symptoms between the mold-exposure group and individuals with known brain injury. Furthermore, participants exposed to mold and persons with TBI reported similar numbers of symptoms found to be S&S to TBI (Gordon et al., 2000). These findings suggest that the experience of disruptions to cognition, somatic functioning, and emotional well-being in persons with mold exposure is similar in severity and quality to that of persons with brain injury and different from that of individuals without disabilities. Thus, it appears that, as indicated in Baldo et al.'s study, the self-report of individuals exposed to mycotoxins is consistent with that of persons with known brain injury.

In addition to symptom report, dysfunction on cognitive testing was found in a variety of domains, including processing speed, attention, executive functions, and learning, as well as working, short-term, and long-term verbal and visual memory. Again, these findings are consistent with those of Gordon et al. (1999) and Baldo et al. (2002). The most common type of dysfunction was in the area of memory, particularly in the areas of verbal learning and visual memory. On measures of verbal learning, significant numbers of participants showed reduced function and impairment. Although the proportion of participants with significant impairment (scores below the 5th percentile) on measures of visual memory was not significantly above chance levels, the proportion with reduced function (scores below the 16th percentile) was. Most participants had reduced function in two or more cognitive domains, and over one third of the sample had significant impairments in two or more domains, suggesting that the cognitive impact of exposure to mycotoxins is often extensive. Indeed, Baldo, Ahmad, and Ruff also found significant impairments in other cognitive domains that were not examined in this study (visuospatial learning and psychomotor speed), indicating that impairments may be even more wide-ranging than this study suggests.

Impairment on most tests was established by comparing test performance to normative data. In the case of the WMS–III tests, however, it was possible to make relative comparisons between performance on tests of memory and ability (IQ). Examination of relative differences between WMS–III memory index scores and participants' intellectual ability revealed more numer-

ous significant discrepancies than comparison to normative data. Thus, significant differences between IQ scores and memory scores were more likely to be found in our sample of persons exposed to mycotoxins than in the sample of more than 1,250 individuals on whom the WMS–III was standardized.

To determine whether specific memory processes are impaired in persons exposed to toxic mold, analyses modeled on those of Curtiss et al. (2001) were conducted. Findings indicate that, in the group as a whole, deficits in encoding ("ability to impose and use an effective semantic strategy to encode information during learning," p. 576) were most pronounced. However, this was also the index on which participants' scores exhibited the most variability, suggesting that performance in this area varied widely in the group. Further studies with larger numbers of participants will be necessary to determine whether different patterns of impairment in specific, theoretically defined memory processes can be distinguished in persons with mold-related cognitive impairment as they have been in individuals with TBI (Curtiss et al., 2001).

Baldo et al. (2002) found increased levels of Axis I and Axis II disorders in their sample and a significant relation between number of impairments and BDI–II scores. In this study, elevated depression scores were also present. Consistent with the findings of Baldo et al., a relation was found between number of impairments (scores at or below the 5th percentile) and BDI–II scores, although this relation ceased to be statistically significant when a 16th percentile cutoff was used. Thus, depression appeared to be related to the severity of cognitive impairment, although when correlations between specific tests and depression scores were examined, this relation was shown to be limited to a small subset of the measures. It is not clear why depression should be specifically related to these tasks.

Although it might be argued that depression and other psychiatric disorders could account for the impairments found on neuropsychological testing in this study, the limited number of correlations between depression and measures of neuropsychological functioning does not give support to this hypothesis. A large, albeit conflicting literature exists on depression and performance on cognitive testing (see, e.g., Lezak, 1995). Some studies report a relation between the two (e.g., Cassens, Wolfe, & Zola, 1990), whereas others do not (e.g., Niederehe, 1986). The patterns of impairment found in this study are not consistent with an interpretation that reduced performance was a consequence of depression. Impairment related to depression should be associated with overall reductions in performance, obscuring of cognitive strengths (Lezak, 1995) and reduced attention (Cohen, 1993). In our sample, none of these issues apply, because performance was reduced on some memory tasks, but not on others, and correlations between depression and memory scores were found for some tests but not others. IQ scores were generally high relative to memory scores, providing a further indication that overall functioning was not reduced. Finally, the proportions of persons with impairments on tests of attention were not significant. Indeed the sample performed significantly *better* than would be expected by chance on two tests of attention, rather than worse as one would expect if they were impacted by depression. Thus, although depression and altered cognitive function were correlated, this study does not provide evidence for a causal relation between these two factors.

A limitation of this study is the absence of a matched cohort of control participants for neuropsychological testing data. A further limitation is the apparent under-representation of tests of domains of cognitive functioning other than memory. Of the 25 scores used from different tests, 16 were scores from memory tests. Further studies with more tests of other cognitive functions might help to provide a broader perspective on the nature and extent of cognitive impairments in individuals exposed to toxic mold. Finally, the relations among cognitive impairment and type, duration, or extent of mold exposure were not addressed in this study. Future studies should address this issue.

This study adds to a growing body of evidence (Baldo et al., 2002; Gordon et al., 1999) that suggests exposure to mycotoxins can result in significant and measurable cognitive deficits in memory, learning, attention, processing speed, and executive functions.

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