Comparison of Work-Related Symptoms and Visual Contrast Sensitivity Between Employees at a Severely Water-damaged School and a School Without Significant Water Damage

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Background *The National Institute for Occupational Safety and Health (NIOSH) conducted a health hazard evaluation (HHE) of a water-damaged school in New Orleans (NO), Louisiana. Our aim in this evaluation was to document employee health effects related to exposure to the water-damaged school, and to determine if VCS testing could serve as a biomarker of effect for occupants who experienced adverse health effects in a water-damaged building.*

Methods *NIOSH physicians and staff administered a work history and medical questionnaire, conducted visual contrast sensitivity (VCS) testing, and collected sticky-tape, air, and dust samples at the school. Counting, culturing, and/or a DNA-based technology, called mold-specific quantitative PCR (MSQPCR), were also used to quantify the molds. A similar health and environmental evaluation was performed at a comparable school in Cincinnati, Ohio which was not water-damaged.*

Results *Extensive mold contamination was documented in the water-damaged school and employees (n = 95) had higher prevalences of work-related rashes and nasal, lower respiratory, and constitutional symptoms than those at the comparison school (n = 110). VCS values across all spatial frequencies were lower among employees at the water-damaged school.*

Conclusions *Employees exposed to an extensively water-damaged environment reported adverse health effects, including rashes and nasal, lower respiratory, and constitutional symptoms. VCS values were lower in the employees at the water-damaged school, but we do not recommend using it in evaluation of people exposed to mold.* Am. J. Ind. Med. 55:844–854, 2012. *Published 2012. This article is a U.S. Government work and is in the public domain in the USA.*

KEY WORDS: mold; school; visual contrast sensitivity; respiratory; asthma; health hazard evaluation

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INTRODUCTION

The National Institute for Occupational Safety and Health (NIOSH) conducted a health hazard evaluation (HHE) at a 1930s public high school in New Orleans (NO), LA. Employees requested the HHE because they were concerned about visible mold and flaking paint. Employees reported respiratory symptoms and difficulty concentrating, irritability, and trouble remembering things.

In 2009, the World Health Organization (WHO) published guidelines for protection of public health from mold and other exposures in damp buildings [WHO, 2009]. Based on its review of the scientific literature, the WHO concluded that there was sufficient epidemiologic evidence that occupants of damp buildings are at risk of developing upper and lower respiratory tract symptoms (e.g., cough, wheeze, and dyspnea), as well as respiratory infections, asthma, and asthma exacerbation. The WHO also concluded that limited evidence suggested associations between bronchitis and allergic rhinitis and damp buildings.

Visual contrast sensitivity testing (VCS) measures deficits in visual perception as a result of effects on the central nervous system even though visual acuity, which is a function of the eye itself, is normal [Regan, 1989]. VCS testing has been reported as useful in "diagnosing" and monitoring treatment of "biotoxin-related illness" among individuals working or living in water-damaged buildings [Shoemaker and House, 2005, 2006]. "Biotoxin-related illness" is not a generally accepted medical condition, and reportedly consists of multiorgan system symptoms, among which neurobehavioral symptoms are prominent. Interpretation of these studies is hampered by methodological limitations, including a nonrepresentative sample, medical conditions that often present with multiple system symptoms (fibromyalgia and chronic fatigue syndrome), the lack of a comparison group, and poor exposure characterization. These limitations could account for the lower VCS values in the participants of these studies, rather than illness from working or living in water-damaged buildings.

VCS is adversely affected by exposure to toxic substances that affect the central nervous system such as solvents [Frenette et al., 1991; Broadwell et al., 1995; Schreiber et al., 2002; Boeckelmann and Pfister, 2003; Gong et al., 2003; Hitchcock et al., 2003]. In addition to toxic chemical exposures, VCS test results can be affected by a number of factors/conditions, for example, hypertension, diabetes, head injury, alcohol consumption, attention deficit hyperactivity disorder, depression, and various eye conditions such as cataract, glaucoma, LASIK, and other eye surgery [Atkin et al., 1979; Sokol et al., 1985; Roquelaure et al., 1995; Pearson and Timney, 1998, 1999; Trick et al., 1988; Nomura et al., 2003; Hammond et al., 2004; Shamshinova et al., 2007; Bartgis et al., 2009; Bubl et al., 2009]. Therefore, any assessment of possible chemical or toxin exposures by VCS testing must take into consideration these other factors/conditions.

Our aim in this evaluation was to document employee health effects related to exposure to the water-damaged school, and to determine if VCS testing could serve as a biomarker of effect for occupants who experienced adverse health effects in a water-damaged building.

MATERIALS AND METHODS

This evaluation was conducted under a blanket institutional review board approval for the HHE program because HHEs are generally not considered research but workplace evaluations. In April 2005, NIOSH physicians and staff conducted a walk-through assessment of the water-damaged school and held confidential, open-ended interviews with school employees. In addition, preliminary environmental sampling (collection of tape samples for microscopic analysis for fungal spores, collection of bulk paint samples for lead analysis, and use of a moisture meter to qualitatively assess wall moisture levels) was performed. In May 2005, a work and health history questionnaire and VCS testing were performed at the water-damaged school. We also collected environmental samples for mold. We went to a comparison school (without water-damage) in Cincinnati, OH in February 2006 to do a similar environmental evaluation, a questionnaire survey, and VCS testing.

Medical Questionnaire

All employees at both schools were invited to participate in the evaluation. Each participant gave full informed consent and filled out a questionnaire about his or her age, medical history, work history, exposure to mold or moisture in the home, and symptoms experienced during the last month while working in the school. These included upper respiratory and mucus membrane, lower respiratory, constitutional, neurobehavioral, and dermal symptoms. If the participant reported having the symptom during the previous month while in the school and that the symptom improved or went away on days not at school, the symptom was considered "work-related." The presence of work-related wheezing or whistling in the chest, or two of the following three symptoms: cough, chest tightness, or unusual shortness of breath defines symptoms consistent with work-related asthma.

Visual Contrast Sensitivity

VCS testing [Ginsburg, 1993] was conducted with the Functional Acuity Contrast Test (F.A.C.T.^{TM}) handheld chart by NIOSH physicians. The F.A.C.T. sine-wave grating chart tests five spatial frequencies (1.5, 3, 6, 12, 18 cycles per degree) and nine levels of contrast. Cycles per degree refers to the number of alternating bands within one degree of visual angle and contrast refers to the difference in intensity between the light and dark bands. Because high levels of visual sensitivity for spatial form are associated with low contrast thresholds, a reciprocal measure (1/threshold), termed the contrast sensitivity score, is computed. Measures of VCS, rather than measures of refractory visual acuity, have been presented as better appraisals of visual dysfunction resulting from chemical exposures. However, if visual acuity is poor, then performance on the F.A.C.T. will also be poor. Therefore, we also measured visual acuity with a handheld Snellen chart. Results from persons with visual acuity of 20/50 or less were removed from further analysis.

The tests were conducted monocularly and binocularly under standard daylight illumination in the library of each school (68 and 239 candelas per square meter, as specified by the Ginsburg method). Participants who used corrective lenses were asked to wear them during testing. The test results place the VCS ability of the employee on the scale of average performance for 90% of the general population [Ginsburg, 1993] (to learn about the VCS test and to see how it is performed, go to: http://www.youtube.com/watch?v=lpvCoPqWb0Q).

Environmental Sampling

Environmental analyses during the first site visit at the water-damaged school included moisture measurements using a TRAMEX Moisture Encounter meter (Tramex Ltd., Littleton, CO). Also, 14 sticky tape samples (SKC Inc., Eighty-Four, PA) were collected in multiple rooms for microscopic analysis of mold spores and other indications of mold growth. In addition, five vacuum dust samples were collected after class, two from fourth floor and three from third floor classrooms, using a filter sock with an average pore size of 6.7 μm (Midwest Filtration Company, Fairfield, OH) attached to a high-efficiency particulate air vacuum, 497A JIC (3M, St. Paul, MN) for approximately 5 min each (time depends on filling of the sock). These five dust samples were analyzed via a DNA-based method called mold-specific quantitative polymerase chain reaction (MSQPCR) [Haugland et al., 2002, 2004; Brinkman et al., 2003].

During the second site visit to the NO school, additional environmental samples were collected. In four classrooms and one outdoor location, triplicate air samples were collected at a flow rate of 28.3 liters per minute (lpm) using Andersen N-6 samplers (Thermo Electron Corporation, Waltham, MA) in conjunction with malt extract agar (MEA) plates and triplicate spore trap samples were also collected with Air-o-Cell® samplers (Zefon International, Inc., Ocala, FL) at a flow rate of 15 lpm. All samples were collected for 5 min each. The vacuum sampling pumps were pre- and post-calibrated with a DryCal (Bios International Corporation, Butler, NJ).

At the comparison school in Cincinnati, one outside and four inside air samples were collected (during school) at a flow rate of 3.5 lpm for approximately 5 hr with SKC Inhalable Button samplers (SKC Inc.) in conjunction with a 2.0-μm pore size polycarbonate filters. These samples were analyzed using MSQPCR. Eight sticky tape samples were collected for microscopic analysis. In addition, triplicate samples were collected by both Anderson samplers and Air-o-Cell® samplers (as described above for the NO school) at one outside location (just outside the school entrance) and in four rooms, for 5 min each.

Lead concentrations in the water-damaged school paint were assessed by collecting flaking paint samples in seven rooms and analyzing them for lead content with NIOSH Method 7300, that is, inductively coupled plasma atomic emission spectrometry [NIOSH, 2012]. At the comparison school, no peeling paint was found and, therefore, no samples were collected for lead analysis.

Statistical Analysis

Statistical analysis was performed with SAS Version 9.1.3 software (SAS Institute, Cary, NC) and StatXact Version 6 software (Cytel Software Corporation, Cambridge, MA). Results with P-values ≤ 0.05 were considered statistically significant. Chi square or Fisher's exact tests were used to compare the prevalence of symptoms, certain demographic characteristics, and percent of abnormal VCS scores between schools. To examine the relationship between work-related symptoms and VCS test results, the symptoms were combined into the following symptom complexes: upper respiratory (sinus problems, dry or irritated eyes, nosebleeds, sore or dry throat, frequent sneezing, stuffy nose, or runny nose); and lower respiratory (cough, wheezing or whistling in the chest, chest tightness, or unusual shortness of breath).

The Wilcoxon two-sample test was used to compare alcohol consumption and VCS values between employees at each school and to examine the relationship between symptom complexes (i.e., upper or lower respiratory symptoms) and VCS values. MSQPCR mold concentration data having a minimum detection limit of 1 cell/mg dust were treated as left-censored data with appropriate statistical methods applied [Helsel, 2005]. Procedurally, nondetects were set at half the minimum detection limit, and given equal and lowest rank for nonparametric rank-based analyses [Helsel, 2005].

RESULTS

In the water-damaged school, condensation was observed in classrooms near the top of walls and in the adjacent hallways. Rusty spots on the light fixtures and stained or missing ceiling tiles were observed in fourth floor classrooms and hallways of the water-damaged school (Figs. 1 and 2). The moisture readings of the plaster walls were 70–90% in the third floor stairwell, 80% on the fourth floor exterior wall, and 10–30% on the lower floors. These observations and moisture readings indicated past and current water problems. In the comparison school, there was no visual evidence of on-going water problems with the exception of the band rooms located underneath a patio area.

Demographic and background health data are presented in Table I. Of 119 employees at the water-damaged school, 95 (80%) participated in the evaluation and of 165 employees at the comparison school, 110 (67%) participated. Participants at both schools were similar in sex, age, history of psychiatric disorder, atopy (the predisposition to allergic disease), smoking history, and reporting mold or moisture problems in their home. Some of the relevant differences included a significantly higher percentage of employees at the water-damaged school had hypertension but a significantly higher percentage of comparison school employees reported head injuries. The median number of alcoholic drinks among comparison employees was higher than those at the water-damaged school.

Employees from the water-damaged school had higher prevalences of work-related rash and lower respiratory, upper respiratory, constitutional, and neurobehavioral symptoms in the previous month than employees from the comparison school reported (Table II). Thirteen employees at each school reported currently having asthma, but 69% of the asthmatics at the water-damaged school reported their asthma was worse at work, compared to 23% at the comparison school ($P = 0.02$). We removed employees with current asthma from our analyses for lower respiratory symptoms. Again, the water-damaged school employees had a significantly higher prevalence of work-related cough ($P < 0.01$), wheezing or whistling in the chest, ($P < 0.01$), chest tightness ($P < 0.01$), and unusual shortness of breath ($P < 0.01$). After excluding those employees who reported current, physician-diagnosed asthma, 20 employees at the water-damaged school met the definition of symptoms consistent with work-related asthma.

FIGURE 1. Picture of mold growing along hallway ceiling at the water-damaged school.

FIGURE 2. Picture of classroom wall and ceiling showing active mold growth at the water-damaged school.

Fifteen individuals were excluded from analyses of VCS because of conditions that could affect their VCS such as glaucoma, cataract, LASIK surgery, or retinal surgery (4 from the water-damaged school and 11 from the comparison school). If near visual acuity was ≥20/50 for a particular eye, these specific-eye results were excluded (16 eyes from the water-damaged school and 21 eyes from the comparison school). Three individuals (two from the water-damaged and one from the comparison school) were excluded from analyses because their binocular near visual acuity was ≥20/50. Near monocular and binocular visual acuity did not differ significantly between the remaining employees (80 from the water-damaged school and 81 from the comparison school).

Monocular and binocular VCS values were significantly lower at all spatial frequencies among employees at the water-damaged school ($P < 0.01$). We repeated the analyses excluding diabetics, and results were similar. We compared VCS values between schools among only hypertensives, and again among only nonhypertensives. We found lower values at all frequencies among employees at the water-damaged school in both groups, although the

TABLE I. Selected Characteristics of Participants, by School

Variable	Water-damaged school (n = 91–95[a])	Comparison school (n = 98–109[a])
Age (mean)	46 years	48 years
Tenure (median)	4 years	8 years
Number of alcoholic beverages in the past 30 days (median)	0	3
	Water-damaged school, N (%)	**Comparison school, N (%)**
Female	49 (52)	59 (55)
Mold or moisture problem at home	6 (6)	9 (8)
Ever had asthma	20 (21)	13 (12)
Physician diagnosed asthma	19 (20)	12 (11)
Currently have asthma	13 (14)	13 (12)
Atopy[b]	69 (73)	75 (69)
Diabetes	8 (8)	4 (4)
Hypertension	37 (39)	28 (26)
Physician-diagnosed anxiety	15 (16)	15 (14)
Physician-diagnosed depression	15 (16)	17 (16)
Physician-diagnosed obsessive compulsive disorder	1 (1)	2 (2)
Physician-diagnosed bipolar disorder	3 (3)	1 (1)
History of eye surgery	3 (3)	10 (9)
History of head injury	10 (11)	23 (21)
Ever smoked cigars, cigarettes, or pipes	32 (34)	35 (33)

[a]Denominators vary due to missing information.
[b]Atopy is a self-reported history of asthma, allergic rhinitis or hay fever, or eczema. Atopy signifies a predisposition to allergic disease.

differences were not statistically significant among the hypertensives at spatial frequencies 1.5 cpd ($P = 0.12$) or 18 cpd ($P = 0.07$) in the left eye or spatial frequencies 12 cpd ($P = 0.12$) and 18 cpd ($P = 0.16$) binocularly among nonhypertensives. A significantly higher percentage of employees at the water-damaged school had scores at all spatial frequencies in the both the right eye and left eye that fell below the average performance for 90% of the general population (i.e., in the lower 10th percentile) than employees at the comparison school (Table III).

We also assessed whether VCS deficits were associated with upper or lower respiratory symptom complexes (Table IV). Persons meeting the designation of lower respiratory symptom complex had significantly lower mean VCS values at all spatial frequencies than those reporting no lower respiratory symptoms. In addition, persons reporting one or more lower respiratory symptom had significantly higher prevalences of VCS scores below 90% of the population (i.e., in the lower 10th percentile) at all spatial frequencies in the left eye, and the majority of frequencies in the right eye and binocularly (Table V).

The environmental analyses and observations (Figs. 1 and 2) revealed widespread evidence of water-damage and mold contamination in the water-damaged school. MSQPCR analysis of the dust showed that nearly every one of the 36 molds tested for was detected in this school (Table VI). In addition, the Environmental Relative Moldiness Index (ERMI) values were high, ranging from 13.8 to 19.1 (The ERMI scale ranges from about -10 to 20, lowest to highest; Table VI). Fifteen of the 16 sticky-tape samples contained abundant mold spores, primarily *Cladosporium*. More culturable mold and spores were seen in the air samples from the water-damaged school (Table VII) than the comparison school (Table VIII).

At the comparison school, no water-damage or mold growth was observed except in the band rooms. Six of the eight sticky tape samples showed no mold spores and the other two samples, collected in the band rooms had only a few mold spores and hyphae. Air samples from the comparison school for spores, culturable molds, and those analyzed by MSQPCR showed low levels of airborne molds (Table VIII). The outdoor mold concentration was much lower in Cincinnati than NO (Tables VII and VIII).

Lead concentrations in the peeling paint chips collected in seven rooms in the water-damaged school averaged 937 μg lead per gram dust (range: 11–1,730 μg). Since there was no peeling paint in the comparison school, no comparison samples were obtained for lead analysis.

DISCUSSION

Visual observation of flaking paint and extensive mold growth, high moisture readings, and the results from analytical tests for molds documented the environmental

TABLE II. Prevalence of Work-Related Symptoms in the Last Month, by School

Symptom	Water-damaged school (n = 81–88[a]), N (%)	Comparison school (n = 102–107[a]), N (%)	Prevalence ratio (95% confidence interval)	*P*-value
Lower respiratory				
Cough	35 (43)	11 (10)	4.16 (2.26, 7.68)	<0.01
Wheezing or whistling in chest	19 (23)	2 (2)	12.13 (2.91, 50.62)	<0.01
Chest tightness	22 (27)	0	+inf (7.69, +inf)[b]	<0.01
Unusual shortness of breath	19 (24)	4 (4)	6.22 (2.20, 17.56)	<0.01
Upper respiratory				
Sinus problems	27 (33)	14 (13)	2.44 (1.37, 4.35)	<0.01
Dry or irritated eyes	16 (20)	12 (11)	1.72 (0.86, 3.44)	0.12
Nosebleeds	3 (4)	1 (1)	3.70 (0.53, 47.02)	0.33
Sore or dry throat	21 (24)	13 (13)	1.95 (1.04, 3.67)	0.03
Frequent sneezing	17 (20)	4 (4)	5.23 (1.83, 14.96)	<0.01
Stuffy nose	25 (29)	10 (10)	3.09 (1.57, 6.07)	<0.01
Runny nose	22 (25)	7 (7)	3.87 (1.73, 8.62)	<0.01
Constitutional				
Fever or sweats	14 (16)	4 (4)	4.10 (1.40, 12.01)	<0.01
Aching all over	12 (14)	4 (4)	3.71 (1.24, 11.08)	0.01
Unusual tiredness or fatigue	25 (31)	18 (17)	1.78 (1.04, 3.03)	0.03
Headache	30 (35)	21 (20)	1.74 (1.08, 2.81)	0.02
Neurobehavioral				
Difficulty concentrating	15 (18)	4 (4)	4.63 (1.60, 13.44)	<0.01
Confusion or disorientation	8 (10)	2 (2)	5.05 (1.25, 29.56)	0.02
Trouble remembering things	15 (17)	5 (5)	3.59 (1.36, 9.47)	<0.01
Irritability	19 (22)	15 (14)	1.51 (0.82, 2.80)	0.18
Depression	6 (7)	2 (2)	3.74 (0.87, 20.82)	0.14
Change in sleep patterns	16 (19)	4 (4)	4.99 (1.73, 14.37)	<0.01
Rash, dermatitis, or eczema (on face, neck, arms, or hands)	12 (14)	4 (4)	3.70 (1.24, 11.06)	0.01

[a]Denominators vary because of missing information.
[b]Positive infinity or undefined.

problems in the water-damaged school. The ERMI scale was based on the MSQPCR analysis of 36 molds in dust samples from a random national sampling of homes in the US [Vesper et al., 2007a]. Of these 36 molds, there are 26 Group 1 molds associated with mold growth in water-damaged buildings and 10 Group 2 molds that are found commonly in buildings, even without water damage [Vesper et al., 2011; Vesper, 2011]. High ERMI values (e.g., ERMI > 5) in homes have been associated with childhood development of wheeze, rhinitis, asthma, and asthma exacerbation [Vesper et al., 2006, 2007b; Reponen et al., 2011]. Although the ERMI scale was developed for homes in the United States, finding very high ERMI values in other buildings would indicate that these buildings were water-damaged [Meklin et al., 2004; Yap et al., 2009].

Some employees of the water-damaged school reported work-related symptoms shown to be associated with occupancy in damp and/or moldy buildings, including upper and lower respiratory symptoms and possibly development of asthma. Twenty of these employees without current, physician-diagnosed asthma met our case definition of work-related asthma, and employees at the water-damaged school who reported current asthma were significantly more likely to report that their asthma was worse at work than were employees with asthma at the comparison school. Employees at the water-damaged school had significantly elevated prevalences of constitutional symptoms such as fever, body aches, and unusual tiredness, which, along with cough and shortness of breath, could indicate a history of hypersensitivity pneumonitis.

We attempted to address methodological limitations of prior studies of VCS and water-damaged buildings. We compared VCS scores and symptoms of employees in the water-damaged school to those of employees in a school without water-damage. All employees were asked to participate. Participation was relatively high at both schools,

TABLE III. Prevalence of Visual Contrast Sensitivity Scores Below 90% of the General Population

	Water-damaged school (n = 80), N (%)	Comparison school (n = 81), N (%)	*P*-value
Left eye (cycles per degree)			
1.5	10 (13)	1 (1)	<0.01
3	11 (14)	1 (1)	<0.01
6	23 (29)	4 (5)	<0.01
12	23 (29)	3 (4)	<0.01
18	22 (28)	2 (3)	<0.01
Right eye (cycles per degree)			
1.5	7 (9)	0 (0)	<0.01
3	10 (13)	1 (1)	<0.01
6	21 (26)	2 (3)	<0.01
12	22 (28)	4 (5)	<0.01
18	23 (29)	6 (7)	<0.01
	Water-damaged school (n = 86), N (%)	**Comparison school (n = 95), N (%)**	***P*-value**
Both eyes (cycles per degree)			
1.5	0 (0)	0 (0)	—
3	2 (2)	0 (0)	0.22
6	11 (13)	1 (1)	<0.01
12	10 (12)	2 (2)	0.01
18	11 (13)	1 (1)	<0.01

so that not only persons who had significant health issues made up our study population. Although the significant decrements in VCS scores in the water-damaged school may be due to mold contamination, these effects also may be the result of other factors. We did not examine all possible exposures that may be present in damp buildings, and it is still unclear exactly what exposures in damp buildings are responsible for health effects [WHO, 2009]. Dust mites, bacteria, and chemical emissions can be present in damp buildings.

TABLE IV. Comparison of Binocular VCS Values Between Participants From Both Schools Who Reported One or More Symptoms of the Upper Respiratory or Lower Respiratory Symptom Complex and Those Who Reported No Symptoms

Work-related symptom complex	Spatial frequency (cycles per degree)	Both schools[a] (n = 159–164), *P*-value
Upper respiratory	1.5	<0.01
	3	<0.01
	6	0.07
	12	0.15
	18	0.09
Lower respiratory	1.5	<0.01
	3	<0.01
	6	<0.01
	12	<0.01
	18	<0.01

[a]All findings in anticipated direction (i.e., mean VCS scores were lower for those with symptom complex).

We postulated that upper and lower respiratory symptoms and their potential treatment may have led to constitutional and neurobehavioral symptoms and the lower VCS scores among these employees. Studies have clearly demonstrated that some persons with allergic rhinitis or asthma complain of fatigue, sleepiness, poor concentration, poor work or school performance, poor sleep, and irritability [Bender, 2005; Leander et al., 2009; Williams et al., 2009]. In addition, objective evidence of cognitive impairment (impaired mood, decreased reaction time, attention, and memory) has been demonstrated in persons with allergic rhinitis and asthma [Fitzpatrick et al., 1991; Weersink et al., 1997; Bender, 2005].

We do not recommend using VCS testing in a clinical setting to diagnose illness in occupants of water-damaged buildings because of its nonspecificity. VCS is adversely affected by a multitude of conditions that are common in general population. We were able to detect significant differences in VCS between these two groups of employees, but most employees at the water-damaged school had normal contrast sensitivity, that is, that which would be seen in 90% of the population. VCS testing is part of a panel of neurobehavioral tests recommended by the Agency for Toxic Substances and Disease Registry (ATSDR) for use in community studies of residents exposed to neurotoxins, as a nonspecific screening tool [Amler et al., 1996; Sizemore and Amler, 1996]. ATSDR also stated there is no evidence that the tests will identify past exposures to neurotoxins, but they "will detect, without specificity, subtle neurobehavioral changes that may be consequent to many insults" [Amler et al., 1996]. VCS testing has not been validated as a standalone test for diagnosing neurobehavioral deficits in individuals. In this study, lower VCS or abnormal VCS was mostly related to lower respiratory symptoms. Because asthma and other respiratory symptoms are known to be associated with occupancy in water-damaged buildings, it is more important to remove affected individuals from the building until remediation is complete, and to diagnose and treat their respiratory symptoms or asthma using standard methods such as spirometry and peak flows.

There were several limitations to this evaluation. It is possible that the use of a comparison school from a different region of the country biased our study. This makes it difficult to use outdoor samples for comparisons. The higher outdoor air mold concentrations from NO compared to Cincinnati probably reflects the sub-tropical compared to temperate climates. We had planned a third visit

TABLE V. Prevalence of Visual Contrast Sensitivity (VCS) Scores Below 90% of the General Population Between Those Reporting One or More Work-Related Lower Respiratory Symptom and Those Reporting No Lower Respiratory Symptoms

Spatial frequency in cycles per degree	≥1 work-related lower respiratory symptom	VCS below 90% of population in left eye	VCS below 90% of population in right eye	VCS below 90% of population in both eyes
1.5	Yes	8/52 (15)[a]	4/53 (8)	0/58 (0)
	No	3/94 (3)	1/94 (1)	0/105 (0)
3	Yes	7/52 (14)[a]	6/53 (11)[a]	2/58 (3)
	No	3/94 (3)	2/94 (2)	0/105 (0)
6	Yes	14/52 (27)[a]	12/53 (23)[a]	7/58 (12)[a]
	No	8/94 (9)	6/94 (6)	2/105 (2)
12	Yes	15/52 (29)[a]	13/53 (25)[a]	8/58 (14)[a]
	No	7/94 (7)	7/94 (7)	2/105 (2)
18	Yes	14/52 (27)[a]	14/53 (26)[a]	8/58 (14)[a]
	No	6/94 (6)	10/94 (11)	2/105 (2)

[a]Statistically significantly higher prevalence.

TABLE VI. Fungal Spore Equivalents in Dust Samples (Cells per Milligram Dust) From the Water-Damaged School and Their Calculated Environmental Relative Moldiness Index (ERMI)[a] Value

Fungal species	Room 321	Room 317	Room B	Room 322	Room 400
Group 1[b]					
Aspergillus flavus	6	5	31	7	ND
Aspergillus fumigatus	14	3	94	13	7
Aspergillus niger	9	2	16	7	22
Aspergillus ochraceus	ND	ND	ND	ND	ND
Aspergillus penicillioides	743	211	351	47	55
Aspergillus restrictus	ND	ND	63	ND	ND
Aspergillus sclerotiorum	76	38	196	104	ND
Aspergillus sydowii	1,174	214	67	ND	113
Aspergillus unguis	4,504	237	375	52	2,631
Aspergillus versicolor	ND	ND	ND	ND	ND
Aureobasidium pullulans	754	1,273	5,036	431	2,288
Chaetomium globosum	49	2	21	14	51
Cladosporium sphaerospermum	2,464	1,758	2,647	578	1,119
Eurotium group	512	409	762	173	224
Paecilomyces variotii	31	11	174	82	148
Penicillium brevicompactum	39	14	48	ND	ND
Penicillium corylophilum	ND	ND	ND	ND	36
Penicillium group 2	ND	ND	ND	ND	ND
Penicillium purpurogenum	ND	ND	ND	ND	3
Penicillium spinulosum	ND	ND	ND	ND	ND
Penicillium variabile	12	4	37	16	95
Scopulariopsis brevicaulis	4	18	2	ND	10
Scopulariopsis chartarum	ND	ND	ND	ND	ND
Stachybotrys chartarum	ND	ND	11	ND	ND
Trichoderma viride	10	5	16	135	64
Wallemia sebi	75	43	250	42	57

(*Continued*)

TABLE VI. (*Continued*)

Fungal species	Room 321	Room 317	Room B	Room 322	Room 400
Group 2[c]					
Acremonium strictum	ND	ND	4	ND	ND
Alternaria alternate	ND	22	841	72	159
Aspergillus ustus	252	62	88	138	392
Cladosporium cladosporioides-1	611	502	1,820	556	453
Cladosporium cladosporioides-2	19	3	12	6	17
Cladosporium herbarum	36	12	619	42	86
Epicoccum nigrum	20	16	499	19	95
Mucor group	25	6	8	3	7
Penicillium chrysogenum type 2	112	114	37	43	400
Rhizopus stolonifer	7	21	45	ND1	21
Sum of the Logs Group 1	32.66	29.32	39.05	27.88	33.39
Sum of the Logs Group 2	13.62	14.24	19.93	14.05	18.1
ERMI values	19.0	15.1	19.1	13.8	15.3

ND, not detected.
[a]ERMI values are calculated by subtracting the sum logs of Group 2 molds from the sum logs of Group 1 molds.
[b]Molds associated with mold growth in water-damaged buildings.
[c]Molds that are found commonly in buildings, even without water damage.

to NO to perform similar testing at a local school that did not have water intrusion or mold growth. However, Hurricane Katrina and the subsequent flooding prevented access to an appropriate comparison school in NO. Therefore, an alternative school and location were needed.

Although the schools were of a similar age (both built around 1930) and made of similar materials (stone, brick, and concrete), the comparison was not optimal. The ventilation systems in were different. The NO school was originally designed with a natural ventilation system using large exterior windows and windows at the top of the interior walls adjacent to the hallways to provide cross-ventilation. In 2002, individual ventilation units (Scholar QV^{TM}, Marvair Cordele, GA) were installed in each classroom and hallway. Each unit was placed on an exterior wall; the ventilation unit's louvered outside air intake replaced an existing window. In the comparison school, the ventilation system consisted of steam heat in the winter and air conditioning in the summer.

Another difference in the schools was the occurrence of flaking paint in the NO but not in the Cincinnati school. It may have been present in both schools based on the age of the two buildings, but no peeling paint was observed at the comparison school. It would have been ideal to have been able to separate water-damage and flaking paint exposures but this was not possible. However, lead paint in buildings generally poses a risk to children because of ingestion, and not adults (except from improper removal exposures). We do not think that lead exposures were the source of the VCS deficits, although it cannot be ruled-out. We did not observe any other obvious contaminants that might explain the VCS results.

Another limitation of our study was the difference in racial and socioeconomic status of the two employee populations. Unfortunately, we did not collect information on our specific participant employees because we had planned to use another school in NO as our comparison. However, some city-wide comparisons were available

TABLE VII. Results of Air Sampling for Mold at the Water-Damaged School

	Room 205	Room 316	Room 420	Room 427	Outside
Culturable analysis	Average number of colony forming units per cubic meter of air				
Molds present in order of prevalence (*Cladosporium, Penicillium, Alternaria, Aureobasidium, Aspergillus*)	888	321	246	1,000	1,179
Spore trap analysis	Average number of mold spore counts per cubic meter of air				
Molds present in order of prevalence (*Cladosporium, Alternaria, Epicoccum Penicillium/Aspergillus*)	1,311	388	98	1,911	4,000

TABLE VIII. Results of Air Sampling for Mold at the Comparison School

	Room 102	Room 333	Library	Room 260	Outside
Culturable analysis	Average number of colony forming units per cubic meter of air				
Molds present in order of prevalence	8	82	159	12	140
(*Cladosporium, Penicillium, Epicoccu, Aureobasidium, Aspergillus*)					
Spore trap analysis	Average number of mold spore counts per cubic meter of air				
Molds present in order of prevalence (*Cladosporium, Penicillium/Aspergillus*)	27	187	253	93	520
Mold-specific quantitative polymerase chain reaction analysis	Spores per cubic meter of air				
Group 1 Molds					
Aspergillus fumigates	ND	ND	34	ND	35
Aspergillus niger	ND	ND	7	ND	ND
Eurotium group	ND	61	20	ND	210
Penicillium brevicompactum	ND	ND	ND	23	13
Group 2 Molds					
Cladosporium cladosporioides type 1	1	4	ND	ND	ND
Penicillium chrysogenum type 2	ND	ND	5	ND	ND

ND, none detected.

from databases. Cincinnati was 84% white and 13% black and New Orleans was 55% white and 37% black [CensusScope, 2010]. The salaries for teachers in the NO School District ranged from $25,439 to $41,478 and those of Cincinnati Public School teachers ranged from $34,888 to $69,609 [US Cities, 2009]. This implies a lower socio-economic status for the NO school employees, which may be associated with poorer health overall and perhaps substandard housing. However, a similar proportion of employees at both schools reported mold or moisture problems at home (6–8%). Another limitation is the possibility of reporting bias, meaning that those participants in the water-damaged school may be more likely to report symptoms than those in the comparison school. Finally, the cross-sectional nature of our evaluation does not allow for determination of cause–effect relationships.

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REFERENCES

Amler RW, Gibertini M, Lybarger JA, Hall A, Kakolewski K, Phifer BL, Olsen KL. 1996. Selective approaches to basic neurobehavioral testing of children in environmental health studies. Neurotoxicol Teratol 18(4):429–434.

Atkin A, Bodis-Wollner I, Wolkstein M, Moss A, Podos SM. 1979. Abnormalities in central contrast sensitivity in glaucoma. Am J Ophthalmol 88(2):205–211.

Bartgis J, Lefler EK, Hartung CM, Thomas DG. 2009. Contrast sensitivity in children with and without attention deficit hyperactivity disorder symptoms. Dev Neuropsychol 34(6):663–682.

Bender BG. 2005. Cognitive effects of allergic rhinitis and its treatment. Immunol Allergy Clin N Am 25(2):301–312.

Boeckelmann I, Pfister EA. 2003. Influence of occupational exposure to organic solvent mixtures on contrast sensitivity in printers. J Occup Environ Med 45(1):25–33.

Brinkman NE, Haugland RA, Wymer LJ, Byappanahalli M, Whitman RL, Vesper SJ. 2003. Evaluation of a rapid, quantitative real-time PCR method for cellular enumeration of pathogenic *Candida* species in water. Appl Environ Microbiol 69(3):1775–1782.

Broadwell DK, Darcey DJ, Hudnell HK, Otto DA, Boyes WK. 1995. Work-site clinical and neurobehavioral assessment of solvent-exposed microelectronics workers. Am J Indust Med 27(5):677–698.

Bubl E, Van Elst LT, Gondan M, Ebert D, Greenlee MW. 2009. Vision in depressive disorder. World J Biol Psychiatry 10(4):377–384.

CensusScope. (http://www.censusscope.org/us/metro_rank_race_black africanamerican.html). Date accessed: February 2012.

Fitzpatrick MF, Engleman H, Whyte KF, Deary IJ, Shapiro CM, Douglas NJ. 1991. Morbidity in nocturnal asthma: Sleep quality and daytime cognitive performance. Thorax 46(8):569–573.

Frenette B, Mergler D, Bowler R. 1991. Contrast sensitivity loss in a group of former microelectronics workers with normal visual acuity. Optom Vis Sci 68(7):556–560.

Ginsburg AP. 1993. Functional Acuity Contrast Test F.A.C.T.® Instructions for use. Chicago, IL: Stereo Optical Company, Inc.

Gong Y, Kishi R, Kasai S, Katakura Y, Fujiwara K, Umemura T, Kondo T, Sato T, Sata F, Tsukishima E, Tozaki S, Kawai T, Miyama Y. 2003. Visual dysfunction in workers exposed to a mixture of organic solvents. Neurotoxicology 24(4–5):703–710.

Hammond SD Jr, Puri AK, Ambati BK. 2004. Quality of vision and patient satisfaction after LASIK. Curr Opin Ophthalmol 15(4):328–332.

Haugland RA, Brinkman NE, Vesper SJ. 2002. Evaluation of rapid DNA extraction methods for the quantitative detection of fungal cells using real time PCR analysis. J Microbiol Methods 50(3):319–323.

Haugland RA, Varma M, Wymer LJ, Vesper SJ. 2004. Quantitative PCR of selected *Aspergillus*, *Penicillium* and *Paecilomyces* species. Syst Appl Microbiol 27(2):198–210.

Helsel DR. 2005. Nondetects and data analysis, statistics for censored environmental data. Hoboken, NJ: Wiley and Sons Inc.

Hitchcock EM, Dick RB, Krieg EF. 2003. Visual contrast sensitivity testing: A comparison of two F.A.C.T. test types. Neurotoxicol Teratol 26(2):271–277.

Leander M, Cronqvist A, Janson C, Uddenfeldt M, Rask-Andersen A. 2009. Non-respiratory symptoms and well-being in asthmatics from a general population sample. J Asthma 46(6):552–559.

Meklin T, Haugland RA, Reponen T, Varma M, Lummus Z, Bernstein D, Wymer LJ, Vesper SJ. 2004. Quantitative PCR analysis of house dust can reveal abnormal mold conditions. J Environ Monitor 6(7):615–620.

NIOSH. NIOSH manual of analytical methods (NMAM®). 4th edition. Schlecht PC, O'Connor PF, editors. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for. Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH). Publication 94-113 (August, 1994); 1st Supplement Publication 96–135, 2nd Supplement Publication 98–119; 3rd Supplement 2003-154. [http://www.cdc.gov/niosh/nmam/]. Date accessed: February 2012.

Nomura H, Ando F, Niino N, Shimokata H, Miyake Y. 2003. Age-related change in contrast sensitivity among Japanese adults. Jpn J Ophthalmol 47(3):299–303.

Pearson P, Timney B. 1998. Effects of moderate blood alcohol concentrations on spatial and temporal contrast sensitivity. J Stud Alcohol 59(2):163–173.

Pearson P, Timney B. 1999. Alcohol does not affect visual contrast gain mechanisms. Vis Neurosci 16(4):675–680.

Regan D. 1989. Human brain electrophysiology. New York: Elsevier.

Reponen T, Vesper S, Levin L, Johansson E, Ryan P, Burkle J, Grinshpun SA, Zheng S, Bernstein DI, Lockey J, Villareal M, Khurana Hershey GK, LeMasters G. 2011. High environmental relative moldiness index during infancy as a predictor of asthma at 7 years of age. Ann Allergy Asthma Immunol 107(2):120–126.

Roquelaure Y, Gargasson LE, Kupper S, Girre C, Hispard E, Dally S. 1995. Alcohol consumption and visual contrast sensitivity. Alcohol Alcohol 30(5):681–685.

Schreiber JS, Hudnell HK, Geller AM, House DE, Aldous KM, Force MS, Langguth K, Prohonic EJ, Parker JC. 2002. Apartment residents' and day care workers' exposure to tetrachloroethylene and deficits in visual contrast sensitivity. Environ Health Perspect 110(7): 655–664.

Shamshinova AM, Arakelian MA, Rogova SIU, Adasheva TV, Silakova OL. 2007. Classification of the forms of hypertensive retinopathy on the basis of estimation of the bioelectrical retinal potential and contrast sensitivity. Vestn Oftalmol 123(1):24–28.

Shoemaker RC, House DE. 2005. A time-series study of sick building syndrome: chronic, biotoxin-associated illness from exposure to water-damaged buildings. Neurotoxicol Teratol 27(1):29–46.

Shoemaker RC, House DE. 2006. Sick building syndrome and exposure to water-damaged buildings: Time series study, clinical trial and mechanisms. Neurotoxicol Teratol 28(5):573–588.

Sizemore OJ, Amler RW. 1996. Characteristics of ATSDR's adult and pediatric environmental neurobehavioral test batteries. Neurotoxins 17(1):229–236.

Sokol S, Moskowitz A, Skarf B, Evans R, Molitch M, Senior B. 1985. Contrast sensitivity in diabetics with and without background retinopathy. Arch Ophthalmol 103(1):51–54.

Trick GL, Burde RM, Gordon MO, Santiago JV, Kilo C. 1988. The relationship between hue discrimination and contrast sensitivity deficits in patients with diabetes mellitus. Ophthalmology 95(5):693–698.

US Cities. (http://www.city-data.com/us-cities/The-South/New-Orleans-Education-and-Research.html). 2009. Date accessed: February 2012.

Vesper S. 2011. Traditional mould analysis compared to a DNA-based method of mould analysis. Crit Rev Microbiol 37(1): 15–24.

Vesper SJ, McKinstry C, Yang C, Haugland RA, Kercsmar CM, Yike I, Schluchter MD, Kirchner HL, Sobolewski J, Allan TM, Dearborn DG. 2006. Specific molds associated with asthma. J Occup Environ Med 48(8):852–858.

Vesper S, McKinstry C, Haugland R, Wymer L, Bradham K, Ashley P, Cox D, Dewalt G, Friedman W. 2007a. Development of an environmental relative moldiness index for US homes. J Occup Environ Med 49(8):829–833.

Vesper SJ, McKinstry C, Haugland RA, Iossifova Y, LeMasters G, Levin L, Hershey GKK, Villareal M, Bernstein DI, Reponen T. 2007b. Relative moldiness index as predictor of childhood respiratory illness. J Expo Science Environ Epi 17:88–94.

Vesper S, Wakefield J, Ashley P, Cox D, Dewalt G, Friedman W. 2011. Geographic distribution of environmental relative moldiness index (ERMI) molds in U.S. homes. J Environ Pub Health Article ID 242457:11 (doi: 10.1155/2011/242457).

Weersink EJ, van Zomeren EH, Koëter GH, Postma DS. 1997. Treatment of nocturnal airway obstruction improves daytime cognitive performance in asthmatics. Am J Respir Crit Care Med 156(4 Pt 1): 1144–1150.

World Health Organization. 2009. WHO guidelines for indoor air quality: dampness and mould. Copenhagen, Denmark: WHO Regional Office for Europe.

Williams SA, Wagner S, Kannan H, Bolge SC. 2009. The association between asthma control and health care utilization, work productivity loss, and health-related quality of life. J Occup Environ Med 51(7): 780–785.

Yap J, Toh ZA, Goh V, Ng LC, Vesper S. 2009. Assessment of mould concentrations in Singapore shopping centers using mould specific quantitative PCR (MSQPCR) analysis. Indian J Microbiol 49(2):290–293.