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To: President, St. Bernard Parish 2/22/06
Fire Chief, St. Bernard Parish
Eddie Elovsson, General Manager M/S Scotia Prince
Larry Ingargiola, Homeland Security, St. Bernard Parish

St. Bernard Parish Mold Clinic
February 9,10,11,12
R. Shoemaker MD

Re: Results of health surveys and visual contrast testing

Dear Sirs:

EXECUTIVE SUMMARY:

Patients from five separate groups in St. Bernard Parish were screened for possible biotoxin associated illness 2/9/06-2/12/06 using a self-administered history and a non-invasive test of neurotoxicity (visual contrast sensitivity "VCS"). A formal medical history was not taken and no physical examination was performed. No laboratory data was obtained.

The pertinent findings from the screening project are summarized on the two attached tables named "St. Bernard Parish Roster" and "Katrina Cough." There is no question about the potential for illness caused by biotoxins in this population: the data are overwhelming. In the absence of known other sources of biotoxin illnesses such as that from dinoflagellates, spirochetes and cyanobacteria in these patients, and the known, massive exposure to water-damaged buildings with visible mold growth, we must consider as likely the hypothesis that ongoing exposure to toxigenic molds and water-damaged buildings is making many St. Bernard Parish residents ill. Prompt medical intervention is indicated. As my longstanding experience with over 5000 biotoxin illness patients clearly documents, the longer the illness is untreated, the worse the prognosis becomes.

Our data accumulated over the past eight years demonstrates that use of these screening modalities can confirm with reasonable medical certainty the presence of biotoxin illness, including such illness as acquired following exposure to biotoxins made by organisms residing in water-damaged buildings. The organisms that cause biotoxin-associated illness in water-damaged buildings include fungi, actinomycetes and endotoxin-forming bacteria. The

206 patients examined include an extraordinarily high percentage of affected patients compared to local (M/S Scotia Prince crew) and historical controls.

Intervention measures, including removal from exposure and further delineation of the physiologic parameters of the illness followed by definitive treatment, are strongly recommended to begin before the unprotected exposure exceeds six months. Trailers installed next to contaminated buildings, used by persons with unprotected indoor exposure to those contaminated structures cannot be considered to be a shelter strategy that provides protection from toxigenic elements, including fungi, resident in the contaminated structures.

Background:

Following an invitation from the management of the M/S Scotia Prince and Dr. Diaz, I joined Dr. Richard Lipsey, a noted toxicologist, on board the Scotia Prince in order to address two questions. First, "Is the ship a safe haven for those persons displaced by Hurricane Katrina and subsequent events?" Second, "Is there evidence that the Parish is safe for unrestricted return of its residents beginning the reconstruction process?"

As to the first question - I would note that the only "residential" location in St. Bernard Parish that did not have its "residents" (the Crew) identified with biotoxin-associated illness is the M/S Scotia Prince.[1]

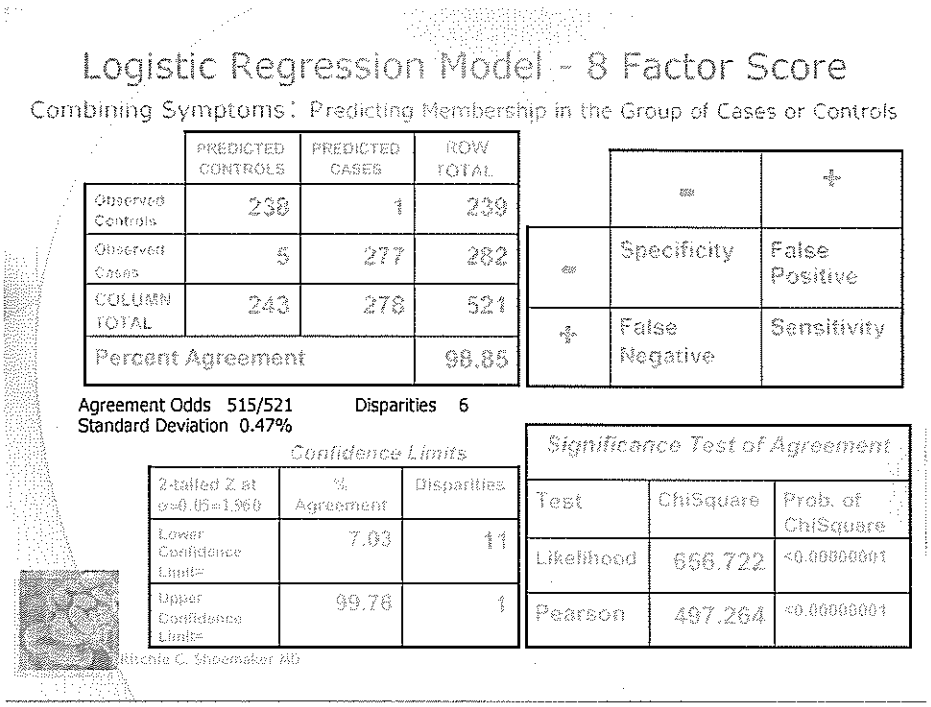
In order to answer these questions, we employed an approach that assessed the potential for exposure to toxigenic elements, including fungi, on board the ship as well as in the surrounding community. Dr. Lipsey obtained multiple environmental samples in both areas. I have seen his preliminary report. First - there is no evidence of microbial contamination on the ship. Second - there is ample evidence of massive microbial growth in the community and initial laboratory analysis indicates that much of it is Stachybotrys. I will await his final report of the cultured samples. That report should then be referenced along with this report and vice versa.

In this report, I will use the term, "mold illness" to refer to those persons with a complex, multisystem, multi-symptom illness acquired following exposure to buildings with a history of water intrusion. While others may call the illness, "Sick Building Syndrome" or something else, I feel that mold illness will suffice to describe the typical biotoxin-associated illness seen in patients with exposure to water-damaged buildings (WDB) and not seen in controls. The use of the term mold illness does not imply that only molds cause the illness; indeed, other toxigenic microbes, including actinomycetes and bacteria are important toxigenic organisms in WDB as well.

As a treating physician, I am on record as insisting that the only reliable marker for a "Sick Building," is a sick person. I agree, however, that we must combine carefully obtained environmental samples with a complete health analysis to assess the potential for acquisition of adverse health effects following exposure to WDB. The assessment of health effects

[1] One crew member presented as a ciguatera case – most likely the result of eating contaminated fish several months previously at his home in the Caribbean.

involves use of a screening medical history and VCS, a neurotoxicological test of vision. Our data on over 5000 patients with biotoxin illnesses, including those caused by exposure to WDB when compared to over 4000 patients in various control groups, clearly shows the accuracy of these two tests in developing reasonable medical certainty separating those with a biotoxin illness from those without such illness. The accuracy exceeds 98.5%. I have included a graphic that demonstrates the statistical basis of this statement.



Such medical certainty provides us with the basis to make statements about the cohort of patients seen on the Scotia Prince, but it does not provide 100% absolute medical certainty. Our diagnostic laboratory findings and the process of differential diagnosis serve that function. Given the ongoing political and economic battles regarding the disaster relief programs, government should support an effort to expand our diagnostic accuracy using the tests that are readily available from nationally certified, high complexity laboratories such as LabCorp and Quest.

Following collection of a database of laboratory studies that will provide accurate delineation of the illness, an objective group of findings will be present that either supports the diagnosis of mold illness or not. If we find additional objective confirmation of the results of screening then we should begin to treat affected patients using protocols I have published in peer-reviewed journals and have employed for over eight years. Our protocols are both safe and effective, with a target of greater than 75% reduction of symptoms achieved in over 92% of 3000 patients with mold illness.

Case definition:

The case definition for human illness caused by exposure to toxigenic organisms, including fungi, mandates two tiers. First, there must be (1) the potential for exposure, (2) presence of multiple symptoms from multiple systems and (3) absence of confounding exposures. The second tier includes the requirement that there also must be three of six of the following elements: (1) presence of deficit in visual contrast sensitivity (VCS); (2) presence of a susceptible HLA DR genotype, as analyzed by PCR; (3) elevated levels of matrix metalloproteinase-9 (MMP9); (4) dysregulation of simultaneously measured ACTH/cortisol; (5) dysregulation of simultaneously measured ADH/osmolality; and (6) reduction of levels of MSH. This case definition was derived by identifying biomarkers present in all those patients with illness and none of control patients without illness. It was first presented in a peer-reviewed paper on September 12, 2003. To date, the case definition continues to maintain an unheard of accuracy of 100%.

For patients age eighteen and younger, the case definition necessarily excludes pituitary hormone abnormalities. We include two separate assays of autoimmunity, anticardiolipins and antigliadins as additional parameters of diagnostic significance because we have documented the unusual increase in autoimmune factors in children with mold illness compared to children without exposure and without illness. Our case definition for children must also account for the delay in maturation of the neurons involved with development of the neurologic function of vision involved in contrast detection. We include in our case definition in children five elements on the second tier, two of which must be present, including HLA DR, antigliadins, anticardiolipins, MMP9 elevation and MSH deficiency. This case definition was presented 12/14/05 at the ASTMH meetings in Washington, DC.

Results:

1. Please review the data summarized by the St. Bernard Parish Roster table. Based on population studies across various ethnic groups, we would expect that 24% of all persons with exposure to WDB would become ill once their HLA-DR based susceptibility was expressed. This "unveiling" of susceptibility follows a significant cytokine illness or massive exposure to toxigenic organisms. If the exposure to the toxigenic organisms is massive, we have seen illness prevalence that exceeds 24% in affected populations. In smaller cohorts, the expected prevalence of illness can be elevated beyond the expected 24% by reasons of chance. Larger samples (populations) won't show the effects of small sample size and then 24% becomes a criterion for consideration of potential chronic illness.

2. The finding of illness findings consistent with our case definition and previously reported logistical regression analysis of symptoms, in all subsets of patients supports our hypothesis that there are many persons with exposure to toxigenic organisms, including molds, in St. Bernard Parish. These people should undergo complete medical evaluation and treatment. Our control population, taken from ship's crew members who did not have exposure to indoor environments on land, show that the ship is a safe haven and that there is no evidence of cross-contamination of ship's

crew by individuals coming on board the ship with clothing and possessions that might be contaminated with spores, fungal fragments or other toxigenic materials. The ship employs an air filtration system that may be a factor in prevention of acquisition of illness on board.

3. We found that age, gender and race did not show any predisposition to acquisition of illness. There was only one exposure to a known biotoxin other than WDB, that being a ship's crew member who became ill following consumption of fish while in tropical reef areas suggestive of ciguatera, a dinoflagellate illness.

4. Symptoms in affected patients were no different from known cases; symptoms in ship's crew were no different from known controls. Symptoms in non-cases in the St. Bernard Parish cohort were no different from known controls.

5. Distribution of symptoms (frequency) was no different from putative cases and non-cases in this cohort compared to known cases and known controls.

6. Visual contrast (VCS) scores were no different in putative cases from St. Bernard Parish from known cases of biotoxin illnesses. VCS scores were no different in ship's crew from other known controls and were no different in putative non-cases from known controls.

7. There has been discussion in the media of the "Katrina cough," but there has been no systematic study of the origin of that persistent respiratory abnormality. Without a case definition of Katrina cough, we simply analyzed those persons who acknowledged that cough was a symptom they had on a daily basis. We have already presented materials on mechanisms involved in acquisition of cough following exposure to WDB. We know that low MSH, high MMP9, elevated C4a, low levels of VIP and low VEGF all contribute to cough in patients with normal IgE. As an aside, low levels of IgE, a reliable screening marker for allergy when elevated, are dominant in biotoxin-associated illnesses. We found that cough in the Katrina cohort was slightly higher in prevalence from cough in other biotoxin-associated illness cohorts, but that the number of associated illness symptoms had not quite reached the levels seen in patients with illness that typically is of much longer duration. Having said that, anyone with a cough and 12 other symptoms, as the Katrina cough persons report, cannot be considered to only have a respiratory illness.

Discussion:

These data support the need for more screening and case-finding in this population of mold-exposed people. Based on discussions with health care providers in the parish, there is little awareness of the diagnostic process and treatment protocols used for patients with illness from WDB. These providers expressed an interest in learning more about what to do for their patients as they are overloaded with sickened residents of St. Bernard parish at this time. The greatest risk for these patients is additional delay in proper diagnosis and

treatment. The longer the inflammatory basis of this illness persists, the greater the number of additional biological cascades of "downstream" events that will occur.

I have added additional information regarding our protocols and lab testing in the enclosed Appendix "A". In an Excel workbook I have attached Appendices "B" through "L". These are various important data sets that resulted from the clinic onboard the Scotia Prince. Of particular note are the data in the first table set out below. We know from our work that there are two routes to biotoxin associated illness – (i) genetic susceptibility found in 24% of the population; and (ii) massive exposure. The St. Bernard sample size is small at 189 persons examined but the overall positive "Case" rating of 54.5%, being more than double the level due to genetic susceptibility, argues persuasively for massive exposure.

The above observations are based on a reasonable degree of medical certainty and are submitted without bias. I am willing to assist you in any way.

Sincerely,

"Ritchie C. Shoemaker"

Ritchie C. Shoemaker, MD

Location	N =	# Positive	% Positive
Firemen	31	14	45.2%
Parish Employees	16	11	68.8%
Health Workers	6	2	33.3%
Homeless Adults	126	71	56.3%
Homeless Children	10	5	50.0%
Total SB Parish	189	103	54.5%
Controls (Ship)	23	1	4.3%
Historical Controls	239	0	0

	N=	Total Symptoms	Av. Sx per person	Total Cough	% with cough	Total Sx/Cough	Sx/person with cough
Historic Mold Cases	594	10,276	17.3	345	58	6,580	19.1
Historic Normal Controls	239	645	2.7	41	17	126	3.1
Total Parish Residents							
Case	101	1,443	14.3	80	79%	1,211	15.2
Non-case	82	142	1.7	21	**25%**	54	2.6
Total	183	1585	8.7	101	55%	1265	12.5
Ship's Crew	23	33	1.43	6	26	15	2.5
Fireman							
Case	14	183	13.1	11	78	153	14
Non-case	17	43	2.52	5	29	7	1.4
Homeless							
Adult case	71	1,086	15.3	57	80	932	16.4
Adult non- case	55	83	1.5	13	23	35	2.8
Child case	5	52	10.4	4	80	44	11.0
Child non-case	5	15	3.0	3	60	12	4.0
Parish Employee							
Case	11	121	11.0	8	73	83	10.3
Non-case	5	2	0.4	0	0	0	0

Appendix A to RCS Letter February 22, 2006

Vision Tests & Analyses

All subjects who normally wore corrective lenses for near-point viewing were asked to wear them during vision testing. The visual acuity and VCS tests were administered monocularly to each eye; an eye occluder was held over one eye while the other eye was tested. All vision tests were administered under illumination from a "daylight" illuminator (fluorescent source with a correlated color temperature of approximately = 6500E K; color rendering index > 90; intensity = 1150 lux; luminance approximately 70 foot-lamberts) in a clinical unit with normal background lighting. A light meter was used to insure that luminance remained constant throughout the test sessions. A test card holder, consisting of a face rest placed just under the cheek bones or chin as comfort provided, and connected by a calibrated

rod to a card holder on the distal end, was used to position the acuity and VCS test cards at a constant distance, previously standardized, from the eyes (acuity - 36 cm (14 inches); contrast sensitivity - 46 cm (18 inches)).

Near Visual Acuity

The acuity test card (MIS Pocket Vision Guide, © 1997 MIS, Inc.) contained 10 rows of numbers in which the size of the numbers progressed from a larger size in the top row to a smaller size in the bottom row. Participants were asked to first read the numbers in a middle row. Testing proceeded to the next lower row if all numbers were correctly identified or to the next higher row if an error occurred. The Snellen visual acuity of the row (20/20 or 20/30, for example) with the smallest numbers each identified correctly was recorded as the visual acuity score. Two-tailed Student t-tests 0.05 were performed, using the mean score of each participant's two eyes, to determine if scores differed significantly between cohorts.

Contrast Sensitivity (VCS)

The contrast sensitivity test card (Functional Acuity contrast Test, (FACT), Stereo Optical Co., Chicago, IL, a Gerber-Coburn Co.) contained a matrix (5 x 9) of circles filled with sinusoidal gratings (dark and light bars). Spatial frequency (1.5, 3, 6, 12 and 18 cycles/degree of visual arc) increased from top to bottom, and contrast decreased from left to right in steps of approximately 0.15 log units. The grating bars were oriented either vertically, or tilted 15 degrees to the left or right. As the investigator called out each circle from left to right, row by row, subjects responded by saying either: vertical, left, right or blank. Participants were encouraged to name an orientation if they had any indication that the bars could be seen. Participants were given the option to point in the direction to which the top of the grating was tilted if they felt any difficulty in verbalizing the orientation; none needed this assistance. The contrast sensitivity score for each row (spatial frequency) was recorded as the contrast of the last test patch correctly identified on that row following verification by repeated testing of that patch and the subsequent patch. The procedure was repeated for each row in descending order. The a priori criterion for the inclusion of data in analyses was that the eye has a visual acuity (Snellen Distance Equivalent Score) of 20:50 or better, in order to avoid confounding of the VCS results by excessive optical-refraction error. All eyes include in data analyses met the visual acuity criterion.

Data Analysis:

The units of analysis for the VCS test were the mean scores of the participant's two eyes at each spatial frequency. Standard error of the mean was calculated for each group of measurements. The VCS data were analyzed using multivariate analyses of variance (MANOVA, with the Wilks' lambda statistic) procedures suitable for repeated measures with $\pm = 0.05$. The factors in the model were group and spatial frequency. A factor for gender was not included since there aren't any gender differences in susceptibility to biotoxin-induced effects shown as yet, and no gender differences in VCS have been reported. Results that showed a significant group-by-spatial frequency interaction were further analyzed in the

step-down, two-tailed Student t-tests ($\pm = 0.05$), the equivalent of a univariate ANOVA to determine which spatial frequencies accounted for the overall effect.

Laboratory:

LabCorp, Inc., Quest Diagnostics, and Specialty Laboratories, Inc., each CLIA approved, high complexity, national laboratory facilities.

MSH: alpha melanocyte stimulating hormone (MSH) is a 13 amino acid compound formed in the ventromedial nucleus (VMN) of the hypothalamus, solitary nucleus and arcuate nucleus by cleavage of proopiomelanocortin (POMC) to yield beta-endorphin and MSH. MSH exerts inductive regulatory effects on production of hypothalamic endorphins and melatonin. MSH has multiple anti-inflammatory and neurohormonal regulatory functions, exerting regulatory control on peripheral cytokine release as well as on both anterior and posterior pituitary function. Deficiency of MSH, commonly seen in biotoxin-associated illnesses, is associated with impairment of multiple regulatory functions and dysregulation of pituitary hormone release. Symptoms associated with MSH deficiency include chronic fatigue and chronic, unusual pain syndromes. Normal values of MSH in commercial labs (Esoterix and LabCorp) are 35-81 pg/ml.

Leptin: leptin is a 146 amino acid adipocytokine produced by fat cells in response to rising levels of fatty acids. Leptin has peripheral metabolic effects, promoting storage of fatty acids, as well as central effects in the hypothalamus. Following binding by leptin to a long isoform of the leptin receptor in the VMN, a primordial gp-130 cytokine receptor, a JAK signal causes transcription of the gene for POMC, which is in turned cleaved to make MSH. Peripheral cytokine responses can cause phosphorylation of a serine moiety (instead of threonine) on the leptin receptor, creating leptin resistance and relative deficiency of MSH production. Normal values in commercial labs show differences between males (5-8 ng/ml) and females (8-18 ng/ml), with levels of leptin correlated with BMI.

ADH/osmolality: abnormalities in ADH/osmolality are recorded as absolute if ADH is < 1.3 or > 8 pg/ml; or if osmolality is >295 or <275 mOsm/kg. Abnormalities are recorded as relative if simultaneous osmolality is 292-295 and ADH ≤ 2.3; or if osmo is 275-278 and ADH ≥ 4.0. Symptoms associated with dysregulation of ADH include dehydration, frequent urination, with urine showing low specific gravity; excessive thirst and sensitivity to static electrical shocks; as well as edema and rapid weight gain due to fluid retention during initial correction of ADH deficits.

ACTH/cortisol: abnormalities in ACTH/cortisol are absolute if AM cortisol > 19 ug/ml or $<$ 8 ug/ml; or if AM ACTH is >60 pg/ml or < 10 pg/ml. Abnormalities are recorded as dysregulation if simultaneous cortisol is > 15 and ACTH is > 15, or if cortisol is < 8 and ACTH <40. Early in the illness, as MSH begins to fall, high ACTH is associated with few symptoms; a marked increase in symptoms is associated with a fall in ACTH. Finding simultaneous high cortisol and high ACTH may prompt consideration of ACTH secreting tumors, but the reality is that the dysregulation usually corrects with therapy.

Androgens: total testosterone, androstenedione and DHEA-S provide measurements regarding the effectiveness of gonadotrophin secretion as influenced adversely by MSH deficiency. Normal ranges of these hormones in males are 75-205 ng/ml for androstenedione, 350-1030 ng/ml for testosterone and 70-218 ug/ml for DHEA-S. Normal values for pre-menopausal women are 60-245, 10-55 and 48-247, respectively. Post-menopausal normal ranges are 30-120, 7-40 and 48-247, respectively.

HLA DR by PCR: LabCorp offers a standard HLA DR typing assay of 10 alleles using a PCR sequence specific chain reaction technique. As opposed to serologic assays for the HLA DR genotypes, the PCR gives far greater specificity in distinguishing individual allele polymorphisms. Linkage disequilibrium is strong in these genotypes, with multiple associations made to inflammatory and autoimmune disease. These genes are part of the human major histocompatibility complex (MHC), also called the HLA complex, located on the short arm of chromosome 6. Relative risk was calculated, susceptible genotypes identified, compared within each group to location and exposure.

MMP9: matrix metalloproteinase 9 (gelatinase B) is an extracellular zinc-dependent enzyme produced by cytokine-stimulated neutrophils and macrophages. MMP9 is involved in degradation of extracellular matrix; it has been implicated in the pathogenesis COPD by destruction of lung elastin, in rheumatoid arthritis, atherosclerosis, cardiomyopathy, and abdominal aortic aneurysm. Cytokines that stimulate MMP9 production include IL-1, IL-2, TNF, IL-1B, interferons alpha and gamma. MMP9 is felt to play a role in central nervous system disease including demyelination, by generation of myelin peptides, as it can break down myelin basic protein. MMP9 "delivers" inflammatory elements out of blood into subintimal spaces, where further delivery into solid organs (brain, lung, muscle, peripheral nerve and joint) is initiated. Normal ranges of MMP9 have a mean of 150, with range of 85-322 ng/ml.

C3a and C4a: Split products of complement activation, often called anaphylatoxins. Each activates inflammatory responses, with spillover of effect from innate immune response to acquired immune responses and hematologic parameters. These short-lived products are re-manufactured rapidly, such that an initial rise of plasma levels is seen within 12 hours of exposure and sustained elevation is seen until definitive therapy is initiated. The components increase vascular permeability, release inflammatory elements from macrophages, neutrophils and monocytes, stimulate smooth muscle spasm in small blood vessels and disrupt normal apoptosis.

Anticardiolipins IgA, IgM and IgG: autoantibodies often identified in collagen vascular diseases such as lupus and scleroderma; often called anti-phospholipids. These antibodies in high titers are associated with increased intravascular coagulation requiring treatment with heparin and coumadin. Lower levels titers are associated with hypercoagulability. An increased risk of spontaneous fetal loss in the first trimester of pregnancy is not uncommonly seen in women with presence of cardiolipin antibodies. This problem does not have the same "dose-response" relationship seen with levels of autoantibodies and illness as does the anti-phospholipid syndrome. Anticardiolipins are found in over 33% of children with biotoxin associated illnesses.

Antigliadin IgA and IgG: Antibodies thought at one time to be specific for celiac disease. With the advent of testing for IgA antibodies to tissue transglutaminase (TTG-IgA), gliadin antibodies are most often seen in patients with low levels of MSH. Ingestion of gliadin, the 22-amino acid protein found in gluten (found in wheat, oats, barley and rye; often added to processed foods) will initiate a release of pro-inflammatory cytokines in the tissues lining the intestinal tract. This cytokine effect will often cause symptoms within 30 minutes of ingestion that mimic attention deficit disorder, often leading to an incorrect diagnosis. Antigliadin antibodies are found in over 58% of children with biotoxin-associated illnesses.

Vasoactive intestinal polypeptide (VIP): neuroregulatory hormone with receptors in suprachiasmatic nucleus of hypothalamus. This hormone/cytokine regulates peripheral cytokine responses, pulmonary artery pressures and inflammatory responses throughout the body. Deficiency is commonly seen in mold illness patients, particularly those with dyspnea on exertion.

Appendix B

Category Name	Symptoms (Sx) Average
Controls	2.7
JAMA Cases	18.2
Ship Crew	1.4
Fireman 0-5 Sx	2.5
Firemen 6+ Sx	12.6
Homeless Adults 0-5 Sx	2.6
Homeless Adults 6+ Sx	15.6
Parish Government 0-5 Sx	1.4
Parish Government 6+ Sx	11.8
Homeless children 0-5 Sx	3
Homeless children 6+ Sx	10.4

Appendix C

	St. Bernard Parish February 2006				
	Homeless Adults - Cases	Parish Employees- Cases	Firemen - Cases	JAMA SBS Draft Paper	Controls
Number	71	11	15	288	239
Fatigue	66	91	73	83	6
Weakness	48	36	33	70	5
Ache	62	64	53	68	8
Cramp	35	18	20	56	2
Unusual pain	23	9	20	51	1
Ice pick	25	18	20	41	1
Headache	66	45	66	66	9
Light	42	9	33	66	2
Red	35	18	20	48	3
Blurred	35	36	20	56	1
Tearing	41	18	20	48	3
Sinus	76	64	87	65	8
Cough	76	64	87	53	7
SOB	58	45	47	63	11
Abdominal pain	24	18	0	39	3
Diarrhea	27	9	13	39	4
Joint	58	36	53	53	11
Morning	52	18	47	44	6
Memory	51	55	87	6	2
Concentration	60	36	87	62	1
Confusion	39	9	53	57	3
Word	41	45	27	66	1
Decreased Assimilation	21	9	13	65	2
Disorientation	25	0	13	40	3
Skin sensitivity	38	36	20	ND	ND
Increased thirst	47	18	27	69	1
Increased urination	47	36	13	66	0
Static Shocks	35	9	27	41	0
Mood Swings	65	45	87	65	1
Appetite	34	18	33	58	1
Sweats (Night)	39	9	47	54	1
Reg Body Temp	24	36	13	60	2
Numbness	38	9	7	44	0
Tingling	34	36	7	51	1
Vertigo	30	36	7	48	2
Metallic Taste	21	18	7	36	0
Tremor	13	9	13	ND	ND

Appendix D

M/S Scotia Prince Crew VCS			
	Average	SD	SEM
A	71.8	24.1	14.4
B	106.3	32.8	21.3
C	117.6	36.7	23.5
D	58.2	18.3	11.6
E	44.0	11.2	8.8

Appendix E

SBS Firemen 0-5 Sx VCS Scores			
	Average	SD	SEM
A	68.0		16.2
B	97.0		23.1
C	128.0		30.5
D	60.0		14.3
E	9.0		2.1

Appendix F

SBS Firemen 6+ Sx VCS Scores			
	Average	SD	SEM
A	53.1		18.8
B	89.2		24.3
C	89.3		21.8
D	42.9		16.0
E	21.7		14.0

Appendix G

Homeless Adults 0-5 Sx VCS Scores			
	Average	SD	SEM
A	66.8	26.1	21.7
B	103.9	36.3	30.3
C	111.4	43.0	35.8
D	57.7	36.9	30.8
E	22.6	19.6	16.3

Appendix H

Homeless Adults 6+ Sx VCS Scores			
	Average	SD	SEM
A	48.6	17.6	2
B	72.7	27.8	3.2
C	62.3	33.7	3.9
D	27.8	16.8	8.0
E	10.6	8.0	0.9

Appendix I

Parish 0-5 Sx VCS Scores			
	Average	SD	SEM
A	45.8	5.6	15.0
B	84.4	17.3	28.3
C	101.4	15.2	37.5
D	61.6	19.4	17.9
E	18.6	15.2	4.2

Appendix J

Parish 6+ Sx VCS Scores

	Average	SD	SEM
A	44.9	20.6	18.7
B	64.4	16.7	26.9
C	52.1	24.1	21.7
D	23.2	16.2	9.7
E	23.2	16.2	9.7

Appendix K

Children 0-5 Sx VCS Scores

	Average	SD	SEM
A	52.2	19	12.9
B	101.6	23.3	45.4
C	114.0	50.8	40.0
D	56.6	33.4	12.5
E	25.2	16.3	6.3

Appendix L

Children 6+ Sx VCS Scores			
	Average	SD	SEM
A	45.2	8.4	18.8
B	69.2	36.7	28.8
C	116.6	27	48.6
D	39.4	11.1	16.4
E	14.4	7.5	6