



MCS America
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Dr. Julie Louise Gerberding, M.D., M.P.H., Director
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Dear Dr. Gerberding;

On September 30, 2005 a letter¹ was submitted via the organization, MCS Awareness, petitioning both the AMA and AAMC to include extensive training in toxicology and environmental illness for all new physicians and to require continuing education in these fields for all currently practicing physicians. We received a reply from Barbara Schneidman, on behalf of Dr. Edward J. Hill, via e-mail on October 19, 2005 which indicated that the issue of an environmental medicine initiative would be brought up and discussed during the next leadership meeting. We have followed-up and are interested in including the Centers for Disease Control in this discussion.

Many of the illnesses in our society today may result from exposure to toxicants in the environment.²⁻¹³ Numerous empirical studies have shown correlations between neurotoxicity and neurodevelopmental disorders such as autism, attention deficit hyperactivity disorder, environmental illness/multiple chemical sensitivities, encephalopathy, and possibly sudden infant death syndrome.¹⁴⁻⁴⁷ Increasing numbers of Americans are developing multiple chemical sensitivity (MCS), chemical injury (CI), environmental illness (EI), asthma, reactive airway disease, autism, attention deficit hyperactivity disorder, fibromyalgia, chronic fatigue syndrome (CFS), and other disorders.⁴⁸⁻⁵⁵ In the United States autism, once a rare condition⁵⁶, has increased from 4 – 5 per 10,000 children in the 1980's to 30 – 60 per 10,000 children in the 1990's, a greater than ten-fold increase.⁵⁷⁻⁵⁸ The prevalence of attention deficit hyperactivity disorder (ADHD) is currently estimated at 8% of the population.⁵⁹ Gliniania suggests an etiological link between metals in particulate air pollution and some forms of sudden

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Multiple **C**hemical **S**ensitivities **A**merica

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infant death syndrome (SIDS).⁶⁰ The prevalence of MCS ranges from 16% to 33% of the population.⁶¹⁻⁶² When compared to the prevalence of diabetes, which is well publicized at 7% of the population, less than half the prevalence of MCS and other environmental illnesses, it is clear environmental illness is under-publicized.⁶³⁻⁷¹ Clearly, MCS requires additional research funding, education, and training of physicians.⁷²⁻⁷³

Multiple chemical sensitivity is an environmental illness which causes negative health effects in multiple organ systems.⁷⁴⁻⁷⁵ Respiratory distress, seizures, cognitive dysfunction, heart arrhythmia, nausea, headache, and fatigue result from exposure to levels of common chemicals such as perfumes, fragrances, cleaners, and pesticides that are normally deemed as safe.⁷⁴⁻⁷⁵ In 1999 consensus criteria were established for the diagnoses and definition of MCS.⁷⁶ The criteria define MCS as symptoms that are reproducible with repeated chemical exposure, are chronic, appear at levels of exposure lower than previously tolerated, improve or resolve when incitants are removed, appear in response to multiple chemically unrelated substances, and which involve multiple organ systems that commonly include the cardiac, pulmonary, and neurological systems.⁷⁷ One of the first studies on MCS focused on possible long term potentiation in the hippocampus and neural sensitization as a central mechanism.⁷⁸ Later studies examined the role of the inflammatory process and found that brain inflammation was correlated with symptoms of MCS.⁷⁹ In 1999 Meggs proposed that MCS is caused by low molecular weight chemicals that bind to chemoreceptors on sensory nerve C-fibers leading to the release of inflammatory mediators.⁸⁰ McKeown-Eyssen showed that polymorphisms in the CYP2D6 allele was responsible for variation in toxicant metabolism pathways that may cause differences in susceptibility to MCS.⁸¹ Pall identified evidence suggesting elevated nitric oxide and peroxynitrite (NO/ONOO-) as the etiology for MCS and several related conditions including fibromyalgia, post traumatic stress disorder, gulf war syndrome, and chronic fatigue syndrome.⁸² Pall has identified organic solvents and related compounds, organophosphorus/carbamate pesticides, organochlorine (chlordane, lindane) pesticides, and the pyrethroid pesticides as initiating the NO/ONOO- cycle of biochemistry leading to MCS.⁸³ With such a large percentage of the population suffering from MCS and a large number of toxicants capable of initiating the NO/ONOO- cycle it is conceivable that a common exposure may initiate or exacerbate MCS.⁸⁴⁻⁸⁵ It is also plausible that a combination of toxicants may be linked to the etiology of MCS.⁸⁶

Various claims that MCS is caused by some ill-defined psychogenic mechanisms have failed to consider the variety of physiological, biochemical, and genetic studies of MCS. Psychogenic advocates have not provided any explanation for the factors distinguishing the chemicals involved in MCS from those that have no role. They have

ignored the prospects for objective biomarker tests for MCS that have been published by Kimata, Millqvist, Bell and Fox and their respective colleagues, each of which is based on measurable physiological changes in response to low level chemical exposures of MCS patients.⁸⁷⁻⁹² Most importantly, they have ignored the genetic data of McKeown-Eyssen and her colleagues and the earlier work of Haley and his colleagues showing that the chemicals initiating MCS act as toxicants, not as odors generating some strictly olfactory response.⁹³⁻⁹⁴ McKeown-Eyssen published data showing that five genetic polymorphisms each have a statistically significant role in determining MCS prevalence.⁹⁵ Each of these genes encode proteins that metabolize chemicals previously implicated in MCS, notably the organophosphorus pesticides (PON1 and PON2 genes) and the organic solvents (CYP2D, NAT1 and NAT2 genes).⁹⁶ These data show that chemicals shown to initiate MCS must be in a specific chemical form to be active; therefore individuals who metabolize them at different rates vary in their susceptibility to MCS.⁹⁷ Haley found similar, confirmatory results with the PON1 gene in studies of the Gulf War syndrome veterans.⁹⁸ Genetic studies coupled with known biochemical functions of the genes involved are the recognized approach to determining biological mechanism.⁹⁹⁻¹⁰⁰ Therefore, these specific studies provided significant confirmation of the toxicogenic roles of chemicals previously implicated in MCS.¹⁰¹⁻¹⁰²

The effect of these neurodevelopmental disorders reaches beyond the individual to the parent, the social system, the work force, school curriculum, medical providers, care providers, the welfare system, and therefore affects the pocketbooks of every taxpaying citizen. Cumulative social and economic costs identified in four case studies of illnesses that are candidates for environmental causation totaled between \$568 billion and \$793 billion dollars per year in Canada and the United States.¹⁰³⁻¹⁰⁴ Neurodevelopmental disorders cost the United States \$81.5 to \$167 billion annually and Methylmercury induced toxicity alone is estimated to cost \$8.7 billion dollars in lost productivity in the United States.¹⁰⁵ Sixty-seven percent of chemicals imported into the United States have not been examined for neurotoxicity and could be a further contributing factor.¹⁰⁶ Individuals affected by neurodevelopmental disorders will increasingly become a burden to society for care as they age, giving rise to the essentiality that scientists discover the etiology behind this alarming increase. The costs of reduced IQ in the United States alone in 1987 may have reached \$327 billion.¹⁰⁷

Our additional concern is that the sufferers of these conditions are unable to obtain qualified medical care or rehabilitation services.¹⁰⁸ These patients are often misdiagnosed with psychiatric disorders due to the outward symptoms of physiological neurotoxicity.¹⁰⁹⁻¹¹⁰ While they may be treated with detoxification protocols and/or exposure education, they are too often referred for useless, and in some cases harmful,

psychiatric therapy and medications that exacerbate their symptoms.¹¹¹⁻¹¹² Indeed 80% report no benefit from psychotherapy to treat MCS and 15% report harm from psychotherapy.¹¹³ Though 65% find psychotherapy helpful to cope with the dramatic life changes the condition imposes upon them, psychotherapy is obviously not a cure because MCS is not a psychologically mediated disease.¹¹⁴ Not a single empirical study shows any significant remission rate in the symptoms of environmental illness from counseling. Further, most insurance in our medical system will not cover tests to determine body burden of chemicals or necessary detoxification.¹¹⁵ Even if the patient requests such treatment and pays cash they are often denied the tests and treatment of choice in our medical system.¹¹⁶ Personal choice has been removed from medical care.¹¹⁷ Of special note is that the Medicaid and Medicare that these patients must rely on once they become disabled does not cover the treatments that could very well return the patient to full productivity, thus removing them from the reliance on the disability and social systems.¹¹⁸ This is penny wise and pound foolish and amounts to draconian torture of innocent patients who truly desire qualified medical care and the opportunity to return to full lives and careers.¹¹⁹⁻¹²² Many report they had successful, professional careers prior to becoming ill and reported that they would happily resume their old lives if they could find relief from their MCS.¹²³⁻¹²⁵ Part of this relief would include the recognition and acceptance of MCS as well as funding for additional research so that proper accommodations can be made in the workplace.¹²⁶⁻¹²⁸

SPECT brain scans on individuals with chronic symptoms following toxic exposure to various petrochemical compounds compared to healthy control subjects show reduced blood flow to the brain and reduced ability of the brain to take up the tracer substance in the early phase of injection.¹²⁹⁻¹³¹ Numerous studies document toxic encephalopathy resulting from low level chronic exposure to various chemicals.¹³²⁻¹³⁵ While amounts of individual toxicants that fall within regulatory limits may be safe, studies of product safety often fail to consider the effects of combined exposures in day-to-day living which add to the ever increasing body burden of chemicals in humans that must be utilized, metabolized and excreted, or stored.¹³⁶⁻¹⁶⁷ In the report of the Research Advisory Committee in Gulf War Veterans Illnesses, 28 animal model studies are reviewed each of which have found interactions among stressors, including many chemicals, that are implicated in MCS and related illnesses.¹⁶⁸ It is not currently possible to predict the toxic effects of complex combinations of chemicals, which may be much greater than either their individual or additive effects.¹⁶⁹ Clearly the sheer numbers of those afflicted indicate no one is safe from this mass pandemic.¹⁷⁰⁻¹⁷¹ These illnesses pose a clear threat to our welfare, social security, and disability systems¹⁷²⁻¹⁷³ as patients often have difficulty finding qualified practitioners to diagnose and treat them appropriately.¹⁷⁴⁻¹⁷⁶

A perfect example of this dilemma is the toxic effects of commonly used fragranced items such as air fresheners.¹⁷⁷⁻¹⁸² Many well-established toxicants are emitted during air freshener use including "d-limonene, dihydromyrcenol, linalool, linalyl acetate, and beta-citronellol which were emitted at 35-180 mg/day over 3 days while air concentrations averaged 30-160 microg/m³" as shown in a recent study.¹⁸³ Maternal depression is also significantly associated with air fresheners.¹⁸⁴ Glade, which contains short chain aliphatic hydrocarbons, is shown to cause ventricular fibrillation and is potentially fatal if inhaled.¹⁸⁵ In a 1997 study, emissions of "air freshener at several concentrations (including concentrations to which many individuals are actually exposed) caused increases in sensory and pulmonary irritation, decreases in airflow velocity, and abnormalities of behavior measured by the functional observational battery score".¹⁸⁶ A 2006 study at the University of Colorado and Baylor College of Medicine in Houston concluded that air-freshening chemicals may lead to the formation of cancerous cells by suppression of the enzymes that are essential for regulating normal cell death.¹⁸⁷ There are many acute toxic effects of fragranced products including but not limited to neurotoxicity, sensory irritation, pulmonary irritation, decreasing expiratory airflow velocity, and alterations of functional observational battery.¹⁸⁸ What is absolutely clear is the evidence that pollution and toxicants affect the brain and central nervous system in a negative way and cost taxpayers billions of dollars.¹⁸⁹⁻¹⁹⁰ MCS is not new; it is simply unacknowledged mainly because a clear etiology has yet to be replicated. Many currently recognized conditions including chronic fatigue syndrome (CFS), asthma, sudden infant death syndrome (SIDS), attention deficit hyperactivity disorder (ADHD), autism, diabetes, multiple sclerosis, Parkinson's and cancer have no clear etiology either. Yet they are recognized and their victims supported by social and medical services and research funding. MCS, which affects more of the population than any of the above, needs similar recognition, investigation, and support.¹⁹¹⁻¹⁹²

Since 1998 thirty-two State governors have recognized Multiple Chemical Sensitivity/Toxic Injury Awareness Month by signing proclamations (Appendix B). Sadly, advanced stages of multiple chemical sensitivity can lead to organ failure requiring expensive hospitalization, therapy, and medications at the cost of taxpayers who support disabled Americans on the Medicaid and Medicare system.¹⁹³⁻¹⁹⁴ Unfortunately, the increased exposure to drugs and contaminants used for sanitation and cleaning in a hospital environment also adds insult to injury in these cases making the patient sicker and more reliant on high cost treatments.¹⁹⁵⁻¹⁹⁶ Many observable and empirical, scientific facts can help identify MCS including SPECT scans and chemical encephalopathy, vitamin deficiencies, mineral deficiencies, excess amino acid deficiency, and disturbed lipid and carbohydrate metabolism.¹⁹⁷⁻²⁰⁰

Scientific research for MCS requires immediate and sustained attention and support, including a strong focus on identifying biomarkers, developing and testing efficacious treatments, and expanding genetic studies on susceptibility to MCS. In the long run it would be more cost effective and socially responsible to identify genetically susceptible individuals prior to onset rather than treating the secondary conditions such as heart problems, respiratory disorders, and seizures that result from a deteriorating physiological state once onset has taken place.²⁰¹⁻²⁰² Members of the work force needed to support a healthy economy are being lost to the productive sector.²⁰³⁻²⁰⁹ In essence, the current approach is reactionary in that the medical fires of medical emergency and health disaster are fought as they occur, but the underlying etiology is not identified or remedied to prevent the fires from igniting. It is necessary to become proactive. It is critical to global economies and global well-being in the upcoming century to have practitioners, health care service administrators, and public health officials well-educated in the new chemical paradigm of illness, toxicology, and environmental medicine so that patients are treated and returned to the workforce.²¹⁰⁻²¹⁶ Indeed, while infectious disease was formerly the main threat to health, it has now been over compensated for in our zeal to kill germs with potentially toxic chemical disinfectants and pesticides. This has generated a new disease paradigm in which anthropogenic chemicals are becoming an increasingly ominous threat to health as even more diseases are revealed to be of chemical origin or are chemically aggravated. It is interesting to note that the Centers for Disease Control (CDC) recently recognized chemical sensitivity as a symptom of chronic fatigue syndrome (CFS).²¹⁷ But physicians and health care systems lack education and training in chemical injury, toxicology, and environmental controls and are ill prepared to serve this population of patients.²¹⁸⁻²²⁰

“The Centers for Disease Control and Prevention (CDC) serves as the national focus for developing and applying disease prevention and control, environmental health, and health promotion and health education activities designed to improve the health of the people of the United States”.²²¹ Since our first petition many more empirical studies have been completed and published regarding what may prove to be the largest breakthrough in medical history once the results have been replicated. We have mentioned a mere few here. Many ICD10 codes are already in place (Appendix A). The only logical choice is to support further investigation, endorse the full recognition of MCS as a physiological medical condition, and to educate practitioners and other health care providers about MCS and environmental illness for the betterment of public health. With up to 30% of the population currently affected by some level of MCS, this is a crisis situation!⁶¹ We look forward to your reply regarding your position and future plans for MCS recognition!

Sincerely,

Lourdes Salvador
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Also in the names of:

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1. Salvador, Lourdes (2005). Letter to the American Medical Association by Lourdes Salvador on behalf of MCS Awareness. Retrieved January 1, 2007, from MCS America Web site: <http://mcs-america.org/ama.pdf>
2. Hodgson, MB, et al. (1989) Encephalopathy and vestibulopathy following short-term hydrocarbon exposure. *Journal Occupational Medicine*. 31: 51-54.
3. Morrow, LA, et al. (1990) Alternations in cognitive and psychological functioning after organic solvent exposure. *Journal of Occupational Medicine*. 32:444-450.
4. Foo, SC, et al. (1990). Chronic neurobehavioral effects of toluene. *Journal of Industrial Medicine*. 47:480-44.
5. Orbaek, P, Lindgren, M (1988). Prospective clinical and psychometric investigation of patients with chronic toxic encephalopathy induced by solvents. *Scandinavian Journal of Work & Environmental Health*, 14:37-44.
6. Stollery, BP, Flindt, MLH (1988). Memory sequelae of solvent intoxication. *Scandinavian Journal of Work & Environmental Health*. 14:45-48.
7. Kilburn, KH (1998). Chemical Brain Injury. Van Nostrand Reinhold.
8. Gregersen, P, et al.(1987). Chronic toxic encephalopathy in solvent-exposed painters in Denmark 1976-1980: Clinical cases and social consequences after a 5-year follow-up. *American Journal of Industrial Medicine*. 11: 399-417.
9. Bruhn, P, et al. (1981). Prognosis in chronic toxic encephalopathy: A two year follow-up study in 26 house painters with occupational encephalopathy. *Acta Neurology Scandinavia*. 64: 259-272.
10. Rasmussen, H, et al. (1985). Risk of encephalopathia amongst retired solvent-exposed workers. *Journal of Occupational Medicine*. 27:581-565.
11. Rasmussen, H, et al. (1985). Risk of encephalopathia amongst retired solvent-exposed workers. *Journal of Occupational Medicine*. 27:581-565.
12. Edling, C, et al. (1990). Long-term follow-up of workers exposed to solvents. *British Journal of Industrial Medicine* 47:75-82.
13. Stollery, BP, Flindt, MLH (1988). Memory sequelae of solvent intoxication. *Scandinavian Journal of Work & Environmental Health*. 14:45-48.
14. Bonham, AC, Chen, CY, Mutoh, T, Joad, JP (2001). Lung c-fiber CNS reflex: role in the respiratory consequences of extended environmental tobacco smoke exposure in young guinea pigs. *Environmental Health Perspectives*. 109:4, 573-578.
15. Booker, S (2001). NIEHS investigates links between children, the environment, and neurotoxicity. *Environmental Health Perspectives*. 109:6, A258-A261.
16. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
17. Burbacher, TM, Shen, DD, Liberto, N, Grant, KS, Cernichiari, E, & Clarkson, T (2005). Comparison of blood and brain mercury levels in infant monkeys exposed to methylmercury or vaccines containing thimerosal. *Environmental Health Perspectives*. 113:8, 1015-1021.
18. Clarkson, TW (2002). The Three Modern Faces of Mercury. *Environmental Health Perspectives*. 110:1, 11-23.
19. Colborn, T (2004). Neurodevelopment and endocrine disruption. *Environmental Health Perspectives*. 112:9, 944-949.
20. Gibson, P (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111:12, 1498 – 1504.
21. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
22. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
23. Goth, SR, Chu, RA, Gregg, JP, Cherednichenko, G, & Pessah, IN (2006). Uncoupling of ATP-mediated calcium signaling and dysregulated interleukin-6 secretion in dendritic cells by nanomolar thimerosal. *Environmental Health Perspectives*. 114:7, 1083-1091.

24. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.
25. Hertz-Picciotto, I, Croen, L, Hansen, R, Jones, C, Van De Water, J, & Pessah, I (2006). The CHARGE study: an epidemiologic investigation of genetic and environmental factors contributing to autism. *Environmental Health Perspectives*. 114:7, 1119-1125.
26. Joffres, MR, Sampalli, T, & Fox, Roy (2005). Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomized controlled blinded pilot booth study. *Environmental Health Perspectives*. 113:9, 1178-1183.
27. Losiniecki, A, & Prahlow, JA (2006). Sudden Infant Death Due to Neurofibromatosis Type 1. *American Journal of Medical Pathology*. 27:4, 317-319.
28. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
29. Pall, M (2003). Elevated nitric oxide/peroxynitrite theory of multiple chemical sensitivity: central role of N-methyl-D-aspartate receptors in the sensitivity mechanism. *Environmental Health Perspectives*. 111:12, 1461-1464.
30. Pall, M (2006). Novel disease paradigm produces explanations for a whole group of illnesses. Washington State University, Department of Biochemistry and Basic Medical Sciences, Retrieved December 3, 2006, from: http://molecular.biosciences.wsu.edu/Faculty/pall/pall_main.htm
31. Schettler, T (2001). Toxic threats to neurologic development of children. *Environmental Health Perspectives*. 109:6, 813-816.
32. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
33. Walker, SJ, Segal, J, & Aschner, M (2006). Cultured lymphocytes from autistic children and non-autistic siblings up-regulate heat shock protein RNA in response to thimerosal challenge. *Neurotoxicology*. 27:5, 685-692.
34. Wasley, A, Lepine, L, Jenkins, R, Rubin, C (2002). An investigation of unexplained infant deaths in houses contaminated with methyl parathion. *Environmental Health Perspectives*. 110:6, 1053-1056.
35. Windham, GC, Zhang, L, Gunier, R, Croen, LA, & Grether, JK (2006). Autism spectrum disorder in relation to distribution of hazardous air pollutants in the San Francisco bay area. *Environmental Health Perspectives*. 114:9, 1438-1444.
36. Hodgson, MJ, et al. (1989). Encephalopathy and vestibulopathy following short-term hydrocarbon exposure. *Journal of Occupational Medicine*. 31: 51-54.
37. Morrow, LA, et al. (1990) Alternations in cognitive and psychological functioning after organic solvent exposure. *Journal of Occupational Medicine*. 32:444-450.
38. Foo, SC, et al. (1990). Chronic neurobehavioral effects of toluene. *Journal of Industrial Medicine*. 47:480-44.
39. Orbaek, P, Lindgren, M (1988). Prospective clinical and psychometric investigation of patients with chronic toxic encephalopathy induced by solvents. *Scandinavian Journal of Work & Environmental Health*, 14:37-44.
40. Stollery, BP, Flindt, MLH (1988). Memory sequelae of solvent intoxication. *Scandinavian Journal of Work & Environmental Health*. 14:45-48.
41. Kilburn, KH (1998). Chemical Brain Injury. Van Nostrand Reinhold.
42. Gregersen, P, et al.(1987). Chronic toxic encephalopathy in solvent-exposed painters in Denmark 1976-1980: Clinical cases and social consequences after a 5-year follow-up. *American Journal of Industrial Medicine*. 11: 399-417.
43. Bruhn, P, et al. (1981). Prognosis in chronic toxic encephalopathy: A two year follow-up study in 26 house painters with occupational encephalopathy. *Acta Neurology Scandinavia*. 64: 259-272.
44. Rasmussen, H, et al. (1985). Risk of encephalopathia amongst retired solvent-exposed workers. *Journal of Occupational Medicine*. 27:581-565.
45. Rasmussen, H, et al. (1985). Risk of encephalopathia amongst retired solvent-exposed workers. *Journal of Occupational Medicine*. 27:581-565.

46. Edling, C, et al. (1990). Long-term follow-up of workers exposed to solvents. *British Journal of Industrial Medicine* 47:75-82.
47. Stollery, BP, Flindt, MLH (1988). Memory sequelae of solvent intoxication. *Scandinavian Journal of Work & Environmental Health*. 14:45-48.
48. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
49. Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 1996;Jul-Aug;51(4):275-82.
50. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.
51. Hertz-Picciotto, I, Croen, L, Hansen, R, Jones, C, Van De Water, J, & Pessah, I (2006). The CHARGE study: an epidemiologic investigation of genetic and environmental factors contributing to autism. *Environmental Health Perspectives*. 114:7, 1119-1125.
52. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
53. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
54. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
55. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
56. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.
57. Hertz-Picciotto, I, Croen, L, Hansen, R, Jones, C, Van De Water, J, & Pessah, I (2006). The CHARGE study: an epidemiologic investigation of genetic and environmental factors contributing to autism. *Environmental Health Perspectives*. 114:7, 1119-1125.
58. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
59. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
60. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
61. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
62. Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 1996;Jul-Aug;51(4):275-82.
63. American Diabetes Association (2006). Diabetes Statistics. Retrieved January 1, 2007, Web site: <http://www.diabetes.org/diabetes-statistics.jsp>
64. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
65. Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 1996;Jul-Aug;51(4):275-82.
66. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.

67. Hertz-Picciotto, I, Croen, L, Hansen, R, Jones, C, Van De Water, J, & Pessah, I (2006). The CHARGE study: an epidemiologic investigation of genetic and environmental factors contributing to autism. *Environmental Health Perspectives*. 114:7, 1119-1125.
68. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
69. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
70. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
71. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
72. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
73. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
74. Gibson, P (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111:12, 1498 – 1504.
75. Rea, WJ, Johnson, AR, Ross, GH, Butler, JR, Fenyses, EJ, Griffiths, B, & Laseter, J (2006). Considerations for the Diagnosis of Chemical Sensitivity. Retrieved January 1, 2007 from <http://www.aehf.com/articles/A55.htm>
76. Joffres, MR, Sampalli, T, & Fox, Roy (2005). Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomized controlled blinded pilot booth study. *Environmental Health Perspectives*. 113:9, 1178-1183.
77. Joffres, MR, Sampalli, T, & Fox, Roy (2005). Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomized controlled blinded pilot booth study. *Environmental Health Perspectives*. 113:9, 1178-1183.
78. Pall, M (2003). Elevated nitric oxide/peroxynitrite theory of multiple chemical sensitivity: central role of N-methyl-D-aspartate receptors in the sensitivity mechanism. *Environmental Health Perspectives*. 111:12, 1461-1464.
79. Pall, M (2003). Elevated nitric oxide/peroxynitrite theory of multiple chemical sensitivity: central role of N-methyl-D-aspartate receptors in the sensitivity mechanism. *Environmental Health Perspectives*. 111:12, 1461-1464.
80. Meggs, WJ (1999). Mechanisms of allergy and chemical sensitivity. *Toxicology and Industrial Health*. 15:3-4, 331-338.
81. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
82. Pall, M (2003). Elevated nitric oxide/peroxynitrite theory of multiple chemical sensitivity: central role of N-methyl-D-aspartate receptors in the sensitivity mechanism. *Environmental Health Perspectives*. 111:12, 1461-1464.
83. Pall, M (2006). Novel disease paradigm produces explanations for a whole group of illnesses. Washington State University, Department of Biochemistry and Basic Medical Sciences, Retrieved December 3, 2006, from: http://molecular.biosciences.wsu.edu/Faculty/pall/pall_main.htm
84. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
85. Pall, M (2006). Novel disease paradigm produces explanations for a whole group of illnesses. Washington State University, Department of Biochemistry and Basic Medical Sciences, Retrieved December 3, 2006, from: http://molecular.biosciences.wsu.edu/Faculty/pall/pall_main.htm
86. Pall, M (2006). Novel disease paradigm produces explanations for a whole group of illnesses. Washington State University, Department of Biochemistry and Basic Medical Sciences, Retrieved December 3, 2006, from: http://molecular.biosciences.wsu.edu/Faculty/pall/pall_main.htm

87. Kimata H. (2004). Effect of exposure to volatile organic compounds on plasma levels of neuropeptides, nerve growth factor and histamine in patients with self-reported multiple chemical sensitivity. In *J Hyg Environ Health* 207:159-163.
88. Millqvist E, Ternesten-Hasseus E, Stahl A, Bende M. (2005). Changes in levels of nerve growth factor in nasal secretions after capsaicin inhalation in patients with airway symptoms from scents and chemicals. *Environ Health Perspect* 113:849-852.
89. Millqvist E, Bengtsson U, Lowhagen O. (1999). Provocations with perfume in the eyes induce airway symptoms in patients with sensory hyperreactivity. *Allergy* 54:495-499.
90. Bell IR, Schwartz GE, Baldwin CM, Hardin EE. (1996). Neural sensitization and physiological markers in multiple chemical sensitivity. *Regul Toxicol Pharmacol* 24:S39-S47.
91. Bell IR, Baldwin CM, Schwartz GE. (1998). Illness from low levels of environmental chemicals: relevance to chronic fatigue syndrome and fibromyalgia. *Am J Med* 105:74S-82S.
92. Joffres MR, Sampalli T, Fox RA. (2005). Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomized controlled blinded pilot booth study. *Environ Health Perspect* 113:1178-1183.
93. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
94. Haley, RW Billecke, S, & La Du, BN (1999). Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans. *Toxicology and Applied Pharmacology* 157(3):227-33.
95. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
96. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
97. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
98. Haley, RW, Billecke, S, & La Du, BN (1999). Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans. *Toxicology and Applied Pharmacology* 157(3):227-33.
99. Binkley, K, King, N, Poonai, N, Seeman, P, Ulpian, C, Kennedy, J (2001). Increased prevalence of panic disorder-associated cholecystokinin B receptor allele 7. *Journal of Allergy and Clinical Immunology*. 107:887-890.
100. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
101. McKeown-Eyssen, G, Baines, C, Cole, D, Riley, N, Tyndale, R, Marshall, L, & Jazmaji, V (2004). Case-control studies of genotypes in multiple chemical sensitivity: CYP2D, NAT1, NAT2, PON1, PON2 and MTHFR. *International Journal of Epidemiology* 33, 1-8.
102. Haley, RW Billecke, S, & La Du, BN (1999). Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans. *Toxicology and Applied Pharmacology* 157(3):227-33.
103. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
104. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
105. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.

106. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
107. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
108. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
109. Gibson, PR, Elms, AN, Ruding, LA (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111(12), 1498-1504.
110. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
111. Gibson, PR, Elms, AN, Ruding, LA (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111(12), 1498-1504.
112. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
113. Gibson, PR, Elms, AN, Ruding, LA (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111(12), 1498-1504.
114. Gibson, PR, Elms, AN, Ruding, LA (2003). Perceived treatment efficacy for conventional and alternative therapies reported by persons with multiple chemical sensitivity. *Environmental Health Perspectives*. 111(12), 1498-1504.
115. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
116. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
117. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
118. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
119. Koch, L (2004). Multiple chemical sensitivity and rehabilitation planning implications. Kent State University Center for Disability Studies.
120. Gibson, PR, Placek, E, Lane, J, Brohimer, SO, & Earehart Lovelace, AC (2005). Disability induced identity changes in persons with multiple chemical sensitivity. *Qualitative Health Research*. 15:4, 1-23.
121. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
122. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
123. Koch, L (2004). Multiple chemical sensitivity and rehabilitation planning implications. Kent State University Center for Disability Studies.
124. Gibson, PR, Placek, E, Lane, J, Brohimer, SO, & Earehart Lovelace, AC (2005). Disability induced identity changes in persons with multiple chemical sensitivity. *Qualitative Health Research*. 15:4, 1-23.
125. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
126. Koch, L (2004). Multiple chemical sensitivity and rehabilitation planning implications. Kent State University Center for Disability Studies.
127. Gibson, PR, Placek, E, Lane, J, Brohimer, SO, & Earehart Lovelace, AC (2005). Disability induced identity changes in persons with multiple chemical sensitivity. *Qualitative Health Research*. 15:4, 1-23.
128. Gibson, PR, Cheavens, J, & Warren, ML Chemical injury chemical sensitivity and life disruption. James Madison University.
129. Callender, TJ, et al. (1993). Three-dimensional brain and metabolic imaging in patients with toxic encephalopathy. *Environmental Res*. 60: 295-319.

130. Callender, TJ, et al. (1995). Evaluation of chronic neurological sequelae after acute pesticide exposure using SPECT brain scans. *Journal Toxicology & Environmental Health*. 41:275-284.
131. Heuser, G, et al. (1994). Neurospect findings in patients exposed to neurotoxic chemicals. *Toxicology & Industrial Health*. 10:561-571.
132. Elofsson, S, et al. (1980). Exposure to organic solvents. *Scandinavian Journal of Work & Environmental Health*. 6:239-273.
133. Seppalainen, AM, et al. (1978). Neurophysiological effects of long-term exposure to a mixture of organic solvents. *Scandinavian Journal of Work & Environmental Health*. 4: 304-314.
134. Jonkman, EJ, et al. (1992). Electroencephalographic studies in workers exposed to solvents or pesticides. *Electro Clinical Neurophysiology*. 82: 439-444.
135. Bokina, AI, et al. (1976). Investigation of the mechanism of action of atmospheric pollutants on the central nervous system and comparative evaluation of methods of study. *Environmental Health Perspectives*. 13: 37-42.
136. Rea, WJ, Johnson, AR, Ross, GH, Butler, JR, Fenyves, EJ, Griffiths, B, & Laseter, J (2006). Considerations for the Diagnosis of Chemical Sensitivity. Retrieved January 1, 2007 from <http://www.aehf.com/articles/A55.htm>
137. Body burden: the pollution in people, (2007). Retrieved January 2, 2007, from Environmental Working Group Web site: <http://www.ewg.org/reports/bodyburden/>
138. Dickey, LD (1976). *Clinical Ecology*. Springfield, IL: Charles C. Thomas.
139. Rea, WJ, & Mitchell, MJ *(1982). Chemical sensitivity and the environment. *Immunology Allergy & Practice* Sept/Oct:21-31.
140. Randolph, T.G. *Human Ecology and Susceptibility to the Chemical Environment*. Springfield, IL: Charles C. Thomas, 1962 (sixth printing 1978).
141. Ader, R, (1987). Behavioral Influences of Immunity. *Proceedings of American Academy of Environmental Medicine. Annual Meeting*. Nashville, TN.
142. Brostoff, J, & Challacombe, S (1987). *Food Allergy and Intolerance*. London: Bailliere Tindall/W.B. Saunders.
143. Calabrese, EJ (1977). Pollutants and high risk groups. *The Biological Basis of Increased Human Susceptibility to Environmental and Occupational Pollutants*. New York: Wiley Interscience.
144. Clayson, CD, Amdur, MO, Doull, J. *Casarett and Doull's Toxicology: The Basic Science of Poisons*, MacMillan Publishing Co., New York, NY.
145. Schnare, D, & Shields, M (1954). Body burden reduction of PBS's, PBB's and chlorinated pesticides in human subjects. *Ambio*. 13:378-80.
146. Davidson, M, & Fienleib, M (1972). Disulfide poisoning: A review. *American Heart H* 83:100.
147. Dodson, RF, Atkinson, MA (2006). Measurements of asbestos burden in tissues. *Annals of the New York Academy of Sciences*. 1076:281-91.
148. DeRosa, CT, Hicks, HE, Ashizawa, AE, Pohl, HR, Mumtaz, MM (2006). A regional approach to assess the impact of living in a chemical world. *Annals of the New York Academy of Sciences*. 1076:829-38.
149. Freedman, BJ (1980). Sulphur dioxide in foods and beverages: Its use as a preservative and its effect on asthma. *Br Journal Dis Chest* 74(2):128-134.
150. Furst, P (2006). Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk. Levels, correlations, trends and exposure through breastfeeding. *Molecular Nutrition & Food Research*. 50(10):922-33.
151. Gilpin, A (1978). *Air Pollution*, 2nd edition. St. Lucia, Queensland: University of Queensland Press.
152. Hill, J (1987). Peptides and their receptors as the biochemicals of emotion. *Proceedings of the American Academy of Environmental Medicine. Annual Meeting*. Nashville, TN.
153. Innami, S, Tojo, H, Utsuja, S, Nakamura, A, & Nagazama, S (1974). PCB toxicity and nutrition. *PCB Toxicity and Vitamin A*. *Japanese Journal of Nutrition* 32:58-666.
154. King, DS (1981). Can allergic exposure provoke psychological symptoms? A double-blind test. *Biological Psychiatry* 16:3-10.

155. Kerger, BD, Leung HW, Scott, P, Paustenbach, DJ, Needham, LL, Patterson, DG Jr., Gerthoux, PM, Mocarelli, P (2006). Age- and concentration-dependent elimination half-life of 2,3,7,8-tetrachlorodibenzo-p-dioxin in Seveso children. *Environmental Health Perspectives*. 114(10):1596-602.
156. Larsen, JC (2006). Risk assessments of polychlorinated dibenzo- p-dioxins, polychlorinated dibenzofurans, and dioxin-like polychlorinated biphenyls in food.. *Molecular Nutrition & Food Research*. 50(10):885-96.
157. Li, QQ, Loganath, A, Chong, YS, Tan, J, Obbard, JP (2006). Levels of persistent organic pollutant residues in human adipose and muscle tissues in Singapore. *Journal Toxicology & Environmental Health A*. 69(21):1927-37.
158. Laseter, JL, DeLeon, IR, Rea, WJ, & Butler, JR (1983). Chlorinated hydrocarbon pesticides in environmentally sensitive patients. *Clinical Ecology*. 2(1):3-12.
159. National Research Council. *Indoor Pollutants*. Washington, D.C.: National Academy Press, 1981.
160. Pan, Y, Johnson, AR, & Rea, WJ (1987-88). Aliphatic hydrocarbon solvents in chemically sensitive patients. *Clinical Ecology*. 5(3):126-131.
161. Rea, WJ, Johnson, AR, Smiley, RE, Maynard, & Dawkins-Brown, O (1986). Magnesium deficiency in patients with chemical sensitivity. *Clinical Ecology* 4(1):17-20.
162. Rea, WJ, et al. (1987). Toxic volatile organic hydrocarbons in chemically sensitive patients. *Clinical Ecology* 5(2):70-74.
163. Stokinger, HE (1965). Ozone toxicology: A review of research and industrial experience, 1954-1962. *Archives of Environmental Health* 110:719.
164. Villeneuve, DC, Grant, DL, Phillipos, WEJ, Clark, ML, & Clegg, DJ (1971). Effects of PCB administration on microsomal enzyme activity in pregnant rabbits. *Bull Environmental Contamination & Toxicology*. 16:120.
165. Winslow, SG (1981). *The effects of environmental chemicals on the immune system: Selected Bibliography with Abstracts*. Oak Ridge, TN: Toxicology Information Response Center, Oak Ridge National Laboratory.
166. Yaktine, AL, Harrison, GG, Lawrence, RS (2006). Reducing exposure to dioxins and related compounds through foods in the next generation. *Nutrition Reviews*. 64(9):403-9.
167. Tretjak, Z, et al. (1990). PCB reduction and clinical improvement by detoxification, *Human Experiment Toxicology*, 9:235-44.
168. Binns JH, Cherry N, Golomb BA, et al (2004). [Research Advisory Committee on Gulf War Veterans' Illnesses]. Report and Recommendations.
169. Binns JH, Cherry N, Golomb BA, et al (2004). [Research Advisory Committee on Gulf War Veterans' Illnesses]. Report and Recommendations.
170. Gibson, P (2005). *Understanding & accommodating people with multiple chemical sensitivity in everyday living*. Houston, TX: Independent Living Research Utilization.
171. Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 1996;Jul-Aug;51(4):275-82.
172. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
173. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
174. Treatment efficacy, a survey of 305 MCS patients. *The CFIDS Chronicle*, Winter 1996, 52-53.
175. Davis, TH, Jason, LA, & Banghart, MA (1995). The effect of housing on individuals with multiple chemical sensitivities. *Archives of Environmental Health*, 50:425-431.
176. Miller, CS (1995). Multiple chemical sensitivity syndrome. *Journal of Occupational and Environmental Medicine*. 37:13-23.
177. Singer, BC, Destailats, H, Hodgson, AT, Nazaroff, WW (2006). Cleaning products and air fresheners: emissions and resulting concentrations of glycol ethers and terpenoids. *Indoor Air*. 16(3):179-91.

178. Farrow, A, Taylor, H, Northstone, K, Golding, J (2003). Symptoms of mothers and infants related to total volatile organic compounds in household products. *Archives of Environmental Health*. 58(10):633-41.
179. Lovechio, F, & Fullton, SE (2001). Ventricular fibrillation following inhalation of Glade Air Freshener. *European Journal of Emergency Medicine*. 8(2):153-4.
180. Anderson, RC, & Anderson, JH (1998) Toxic effects of air freshener emissions. *Archives of Environmental Health*. 52(6):433-41.
181. Air-freshening chemicals may lead to cancerous cells (2006) *Denver Post*. Nation: World News. Retrieved May 15, 2006 from: http://www.denverpost.com/search/ci_3823832
182. Anderson, RC, & Anderson, JH (1998) Acute toxic effects of fragrance products. *Archives of Environmental Health*. 53(2):138-46.
183. Singer, BC, Destailats, H, Hodgson, AT, Nazaroff, WW (2006). Cleaning products and air fresheners: emissions and resulting concentrations of glycol ethers and terpenoids. *Indoor Air*. 16(3):179-91.
184. Farrow, A, Taylor, H, Northstone, K, Golding, J (2003). Symptoms of mothers and infants related to total volatile organic compounds in household products. *Archives of Environmental Health*. 58(10):633-41.
185. Lovechio, F, & Fullton, SE (2001). Ventricular fibrillation following inhalation of Glade Air Freshener. *European Journal of Emergency Medicine*. 8(2):153-4.
186. Anderson, RC, & Anderson, JH (1998) Toxic effects of air freshener emissions. *Archives of Environmental Health*. 52(6):433-41.
187. Air-freshening chemicals may lead to cancerous cells (2006) *Denver Post*. Nation: World News. Retrieved May 15, 2006 from: http://www.denverpost.com/search/ci_3823832
188. Anderson, RC, & Anderson, JH (1998) Acute toxic effects of fragrance products. *Archives of Environmental Health*. 53(2):138-46.
189. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
190. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
191. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
192. Meggs WJ, Dunn, KA, Bloch, RM, Goodman, PE, & Davidoff, AL (1996). Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 51(4):275-82.
193. Rea, WJ, Peters, DW, Smiley, RE, et al. (1981). Recurrent environmentally triggered thrombophlebitis. *Ann Allergy* 47:338-344.
194. Jollow, DJ, et al. (1977). Biological reactive intermediates: Formation, toxicity, and inactivation. New York.
195. Rea, WJ, Peters, DW, Smiley, RE, et al. (1981). Recurrent environmentally triggered thrombophlebitis. *Ann Allergy* 47:338-344.
196. Jollow, DJ, et al. (1977). Biological reactive intermediates: Formation, toxicity, and inactivation. New York.
197. Rea, WJ, Johnson, AR, Ross, GH, Butler, JR, Fenyves, EJ, Griffiths, B, & Laseter, J (2006). Considerations for the Diagnosis of Chemical Sensitivity. Retrieved January 1, 2007 from <http://www.aehf.com/articles/A55.htm>
198. Ziem, G (2001). Medical Evaluation and Treatment of Patients with Chemical Injury and Sensitivity. National Institute of Environmental Health Sciences.
199. Callender, TJ, et al. (1995). Evaluation of chronic neurological sequelae after acute pesticide exposure using SPECT brain scans. *Journal Toxicology & Environmental Health*. 41:275-284.
200. Heuser, G, et al. (1994). Neurospect findings in patients exposed to neurotoxic chemicals. *Toxicology & Industrial Health*. 10:561-571.

201. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
202. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
203. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
204. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
205. Meggs, WJ, Dunn, KA, Bloch, RM, Goodman, PE, & Davidoff, AL (1996). Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 51(4):275-82.
206. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.
207. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
208. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
209. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
210. Muir, T, & Zegarac, M (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*. 109:6, 885-903.
211. Gibson, P (2005). Understanding & accommodating people with multiple chemical sensitivity in everyday living. Houston, TX: Independent Living Research Utilization.
212. Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. Prevalence and nature of allergy and chemical sensitivity in the general population. *Archives of Environmental Health*. 1996;Jul-Aug;51(4):275-82.
213. Goodman, LR, & Koduru, S (2000). Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives*. 108:S3, A412-A413.
214. Szpir, M (2006). New thinking on neurodevelopment. *Environmental Health Perspectives*. 114:2, A100-A107.
215. Braun, JM, Robert SK, Froehlich, T, Auinger, P, & Lanphear, BP (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*. 114:12, 1904-1909.
216. Glinianaia, SV, Rankin, J, Bell, R, Pless-Mulloli, T, & Howel, D (2004). Does particulate air pollution contribute to infant death? A systemic review. *Environmental Health Perspectives*. 112:14, 1365-1370.
217. Chronic Fatigue Syndrome, (2006). Symptoms. Retrieved January 2, 2007, from Centers for Disease Control Web site: <http://www.cdc.gov/cfs/cfssymptomsHCP.htm>
218. Treatment efficacy, a survey of 305 MCS patients. *The CFIDS Chronicle*, Winter 1996, 52-53.
219. Davis, TH, Jason, LA, & Banghart, MA (1995). The effect of housing on individuals with multiple chemical sensitivities. *Archives of Environmental Health*, 50:425-431.
220. Miller, CS (1995). Multiple chemical sensitivity syndrome. *Journal of Occupational and Environmental Medicine*. 37:13-23.
221. CDC Functional Mission Statement (2006). Centers for Disease Control and Prevention. Retrieved on January 8, 2007 from: <http://www.cdc.gov/od/funcmiss.htm>

Appendix A

ICD-10 Codes

G70.1	Toxic myoneural disorders
G72.2	Myopathy due to other toxic agents
G92	Toxic encephalopathy
J68	Respiratory conditions due to inhalation of chemicals, gases, fumes and vapours
J68.0	Bronchitis and pneumonitis due to chemicals, gases, fumes and vapours
J68.1	Acute pulmonary oedema due to chemicals, gases, fumes and vapours
J68.2	Upper respiratory inflammation due to chemicals, gases, fumes and vapours, not elsewhere classified
J68.3	Other acute and subacute respiratory conditions due to chemicals, gases, fumes and vapours
J68.4	Chronic respiratory conditions due to chemicals, gases, fumes and vapours
J68.8	Other respiratory conditions due to chemicals, gases, fumes and vapours
J68.9	Unspecified respiratory condition due to chemicals, gases, fumes and vapours
L23.5	Allergic contact dermatitis due to other chemical products
L24.5	Irritant contact dermatitis due to other chemical products
L25.3	Unspecified contact dermatitis due to other chemical products
L51.2	Toxic epidermal necrolysis [Lyell]
L53.0	Toxic erythema
M34.2	Systemic sclerosis induced by drugs and chemicals
N14.4	Toxic nephropathy, not elsewhere classified
P04.6	Fetus and newborn affected by maternal exposure to environmental chemical substances
P93	Reactions and intoxications due to drugs administered to fetus and newborn
T37	Poisoning by other systemic anti-infectives and antiparasitics
T45	Poisoning by primarily systemic and haematological agents, not elsewhere classified
T46	Poisoning by agents primarily affecting the cardiovascular system
T47	Poisoning by agents primarily affecting the gastrointestinal system
T48	Poisoning by agents primarily acting on smooth and skeletal muscles and the respiratory system
T51-T65	Toxic effects of substances chiefly nonmedicinal as to source
T52	Toxic effect of organic solvents
T53	Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons
T54	Toxic effect of corrosive substances
T55	Toxic effect of soaps and detergents
T56	Toxic effect of metals
T57	Toxic effect of other inorganic substances
T58	Toxic effect of carbon monoxide
T59	Toxic effect of other gases, fumes and vapours
T60	Toxic effect of pesticides

Appendix A, Continued

T61	Toxic effect of noxious substances eaten as seafood
T62	Toxic effect of other noxious substances eaten as food
T64	Toxic effect of aflatoxin and other mycotoxin food contaminants
T65	Toxic effect of other and unspecified substances
T65.8	Toxic effect of other specified substances
T65.9	Toxic effect of unspecified substance
T78.1	Other adverse food reactions, not elsewhere classified
T90-T98	Sequelae of injuries, of poisoning and of other consequences of external causes
X40-X49	Accidental poisoning by and exposure to noxious substances
X45	Accidental poisoning by and exposure to alcohol
X46	Accidental poisoning by and exposure to organic solvents and halogenated hydrocarbons and their vapours
X47	Accidental poisoning by and exposure to other gases and vapours
X48	Accidental poisoning by and exposure to pesticides
X49	Accidental poisoning by and exposure to other and unspecified chemicals and noxious substances
X69	Intentional self-poisoning by and exposure to other and unspecified chemicals and noxious substances
X89	Assault by other specified chemicals and noxious substances
X90	Assault by unspecified chemical or noxious substance
Y15	Poisoning by and exposure to alcohol, undetermined intent
Y16	Poisoning by and exposure to organic solvents and halogenated hydrocarbons and their vapours, undetermined intent
Y17	Poisoning by and exposure to other gases and vapours, undetermined intent
Y18	Poisoning by and exposure to pesticides, undetermined intent
Y19	Poisoning by and exposure to other and unspecified chemicals and noxious substances, undetermined intent
Y36.7	War operations involving chemical weapons and other forms of unconventional warfare
Z03.6	Observation for suspected toxic effect from ingested substance
Z57.4	Occupational exposure to toxic agents in agriculture
Z57.5	Occupational exposure to toxic agents in other industries

Appendix B

State Governor Proclamations

Since 1998, 32 states have issued MCS/Toxic Injury Awareness Proclamations

Alabama	2006
Alaska	2006
Connecticut	1998, 1999, 2000, 2001, 2002, 2003, 2004, 2005, 2006
District Columbia	2006
Florida	2000, 2001, 2002, 2003, 2004, 2005, 2006
Florida (Broward County)	2000, 2001, 2002, 2003, 2004, 2005, 2006
Georgia	2006
Hawaii	2001, 2006
Idaho	2006
Illinois	1999, 2001, 2004, 2006
Iowa	2006
Kansas	2001, 2002, 2006
Kentucky	2006
Louisiana	2004, 2005, 2006
Maine	2006
Maryland	2006
Massachusetts	2000, 2003
Michigan	2000
(Detroit, Ann Arbor, Livonia)	2001, 2004, 2005, 2006
Minnesota	1999
Missouri	1998, 1999, 2001, 2002, 2003, 2004
Mississippi	2000, 2001, 2002, 2006
Missouri	2006
Nebraska	2006
New Hampshire	2000, 2006
New Jersey	2006
New Mexico	1998, 1999, 2006
New York	2006
North Carolina	1998, 1999, 2000, 2001, 2002, 2003, 2004, 2006
Ohio	2001
Oregon	2006
Washington State	1999, 2000, 2001, 2002, 2004, 2005, 2006
West Virginia	2006
Wisconsin	2006