### **Position Statement**

Idiopathic environmental intolerances
AAAAI Board of Directors
January 1999

AAAAI Position Statements and Work Group Reports are not to be considered to reflect current AAAAI standards or policy after five years from the date of publication. For reference only.

The statement below is not to be construed as dictating an exclusive course of action nor is it intended to replace the medical judgment of healthcare professionals. The unique circumstances of individual patients and environments are to be taken into account in any diagnosis and treatment plan. The above statement reflects clinical and scientific advances as of the date of publication and is subject to change.

The condition now called idiopathic environmental intolerances (IEI)<sup>1-3</sup> and formerly known as multiple chemical sensitivities (MCS)<sup>4</sup> or environmental illness was addressed in the AAAI Position Statement on Clinical Ecology published in 1986.<sup>5</sup> Since then, additional research and clinical studies have been reported. This updated position by the AAAAI reflects the current status of this condition as documented in the published scientific literature.

Abbreviations used:

IEI: Idiopathic environmental intolerances

MCS:Multiple chemical sensitivities

## **Definition and Terminology**

The term environmental illness was used for many years to refer to a subjective illness in certain persons who typically describe multiple symptoms, which they attribute to numerous and varied environmental chemical exposures, in the absence of objective diagnostic physical findings or laboratory test abnormalities that define an illness. Other terms, such as universal allergy, 20th-century disease, chemical hypersensitivity syndrome, total allergy syndrome, and cerebral allergy have also been used to describe the same condition. Since the last AAAAI position statement on this subject, the name multiple chemical sensitivities has largely supplanted these names. In February 1996, the invited experts forming a workshop organized by the International Programme on Chemical Safety of the World Health Organization and other organizations, recommended a new name-idiopathic environmental intolerances-because the term MCS "makes an unsupported judgment on causation" (ie, environmental chemicals), does not refer to "a clinically defined disease," and is not based on "accepted theories of underlying mechanisms nor validated clinical criteria for diagnosis." Furthermore, the "relationship between exposures and symptoms is unproven."

### **History of the IEI Phenomenon**

The existence of IEI as a medical illness was first proposed by Randolph, <sup>6-10</sup> who founded a movement known as clinical ecology. He published his theories and numerous case reports in a series of books and articles beginning in the 1950s. He and others <sup>10-15</sup> attributed the illness to a failure of human adaptation to virtually all modern-day (20th century) synthetic chemicals. However, more than a century ago, Beard <sup>16</sup> described the same clinical condition, which he in turn ascribed to certain items and activities introduced into 19th-century living, specifically the telegraph, the sciences, industry, the periodical press, and female education. Shorter <sup>17</sup> has written an excellent treatise with a unique historical and social perspective about related symptom complexes from the late 18th century to the present.

#### Clinical Description of IEI

Because of the varied and subjective nature of the illness, no precise case definition or diagnostic criteria exist. Nevertheless, reports of individual cases and series of cases reveal that the diagnosis is made almost exclusively in adults and primarily in women. Although the "typical" patient has numerous symptoms that appear to involve many organ systems, careful review of case material reveals that IEI has been diagnosed sometimes in persons with few or no symptoms.

The central focus of the diagnosis is the fact that the patient describes symptoms in relation to environmental exposures. As mentioned earlier, there are no physical examination abnormalities in IEI.

The list of environmental chemical exposures triggering symptoms is virtually unlimited. They are usually, although not always, identified by odor. The more common ones cited are perfumes and scented products, pesticides, domestic and industrial solvents, new carpets, car exhaust, gasoline and diesel fumes, urban air pollution, cigarette smoke, plastics, and formaldehyde. In many patients symptoms are triggered also by certain foods, food additives, and drugs and in some cases by electromagnetic fields and mercury in dental fillings. There have been no dose-response studies of this phenomenon, but patients report that these materials provoke symptoms at concentrations at or below commonly encountered ambient levels. Furthermore, symptoms bear no relationship to established toxic effects of the specific chemical and occur at concentrations far below those expected to elicit toxicity. The latent period for response varies considerably. <sup>26,27</sup>

Certain environmental irritants, including some of those mentioned above, are recognized as triggers for patients with asthma and rhinitis. However, this phenomenon differs from that of IEI in that objective changes of bronchial or nasal obstruction and hypersecretion occur rather than subjective symptoms only.

The patient may not be able to identify the circumstances surrounding the onset of illness. In those cases involving litigation for workers-compensation benefits or alleged personal injury caused by the actions of a third party, however, the patient typically attributes the disease to a specific initiating exposure event. <sup>23,26,28</sup> IEI has been claimed to arise from silicone breast implants and has been attributed to military service in southwest Asia during the brief 1991 hostilities (Gulf War Syndrome). <sup>29,30</sup>

#### **IEI Theories**

Over the past 40 years, a number of theories have been put forward to address the cause of IEI and the mechanism by which diverse environmental exposures produce symptoms. Immunologic, toxicologic, psychologic, and sociologic theories predominate. Opinions about etiology and pathogenesis are sharply divided.<sup>31</sup>

Immunologic and toxicologic explanations of IEI are favored by clinical ecologists. These physicians place emphasis on the disease being a previously unrecognized form of allergy or immunologic hypersensitivity. This concept was gradually replaced by various immunotoxic theories in which environmental chemicals are believed to cause autoimmunity or immunodeficiency. More recently, a neurotoxic theory of IEI has been introduced. According to this theory, symptoms arise from stimulation of the olfactory-limbic system of the brain and the hypothalamus. The condition has also been ascribed to the effects of oxidative damage to unspecified tissues. IEI has been interpreted by some as an overly sensitive state of the respiratory or nasal mucosa.

Many physicians have proposed that IEI is a manifestation of a psychiatric disease or personality disorder. A comparison with somatoform illness has been noted by some and with panic disorder or mass hysteria by others. Additional psychologic interpretations include atypical posttraumatic stress disorder, he behavioral conditioning, and adult manifestation of childhood abuse. Several investigators have observed a high prevalence of several different psychiatric diagnoses among patient with IEI. Clinical ecologists often interpret the presence of psychopathology in patients with IEI to be the result and not the cause of the illness.

### **Diagnostic Methods**

The diagnosis of IEI is typically made on the basis of the patient's history, without any defining criteria. There are no diagnostic symptoms, and there are no diagnostic objective physical signs. Many different tests and procedures have been proposed, but no single test or combination of tests has been validated as diagnostic. The tests most frequently used by practitioners who diagnose IEI are provocation-neutralization 11,64-68 and a panel of immunologic tests. The latter encompasses measurements of serum immunoglobulins, complement levels, blood lymphocyte subset counts, autoantibodies, and serum antibodies to chemicals. 20,23,26,68-81 Some practitioners obtain blood, urine, or fat levels of environmental chemicals, as well as brain imaging studies, neuropsychologic testing, 24,49,76-78 and

psychologic/psychiatric interviews.<sup>28,50,52,82-84</sup> Studies to date have failed to confirm that any immunologic tests are diagnostic for chemically induced symptomatology.<sup>76,79</sup> The diagnostic validity of the other procedures has yet to be tested.

### **Position Statements**

Several medical societies and organizations have issued position statements pointing out the shortcomings of the IEI diagnosis, the unreliability and misuse of certain diagnostic procedures, and the lack of scientific support for and clinical evidence of the alleged toxic effects from environmental chemicals in these particular patients. In 1986, the AAAI was the first to do so. The American College of Physicians published a position paper in 1989, which was later adopted by the American College of Occupational and Environmental Medicine. The Council on Scientific Affairs of the American Medical Association published a critical review in 1992. The Ministry of Health of the Province of Ontario and the California Medical Association have published results of their investigations of the IEI phenomenon. The US National Academy of Sciences, the World Health Organization, and the International Society of Regulatory Toxicology and Pharmacology have held symposia on the subject. The American Council on Science and Health and the Royal College of Physicians and Royal College of Pathologists in Great Britain have also published reports detailing the unscientific basis for IEI.

## Treatment Recommendations for Patients with the Diagnosis of IEI

Those physicians who view the symptoms of IEI as arising from the toxic effect of environmental chemicals (and foods) stress an avoidance program that is sometimes extreme. This is usually supplemented with vitamins and minerals, occasionally with intravenous gamma globulin, and often with "neutralizing" administration of chemical and food extracts by injection or sublingual drops. To date, no controlled clinical trial has been carried out to evaluate this approach. There is evidence that such a program may make the patient worse. Others advocate an undocumented form of "detoxification" through induced sweating and the administration of oral minerals and oils.

A psychotherapeutic approach is recommended by those who find evidence for current psychopathology in the patient's history. One study found short-term benefit from a brief course of inpatient psychotherapy, <sup>92</sup> but no long-term studies have yet been reported.

# **Comparison with Other Illness**

Some observers have interpreted IEI as part of a spectrum of nonphysical illnesses characterized by multiple somatic complaints. Others see it as a distinct entity. The so-called Candida hypersensitivity syndrome has been claimed to be a similar illness, <sup>93</sup> but there is no scientific proof that Candida albicans causes such a condition. <sup>94</sup>

Some psychiatrists have pointed out the similarity of IEI to the somatoform/conversion disorders, <sup>19,28,50,51,83</sup> which in the past were called neurasthenia. Myalgic encephalomyelitis and the chronic fatigue syndrome <sup>95</sup> share features in common with IEI, but these patients do not attribute their symptoms to environmental exposures. The influence of social and cultural factors in shaping the interpretation of unexplained somatic symptoms has been discussed <sup>84,96</sup> and could be relevant to IEI because of the current widespread concern about environmental pollution.

IEI is distinct from true environmentally caused diseases. Infectious microorganisms, allergens, toxins, and irritants are responsible for diseases that are clinically well characterized and for which specific diagnostic procedures are available. In a few situations these pathogens have been proven to cause certain building-related illnesses, such as Legionnaire's disease<sup>97</sup> and hypersensitivity pneumonitis.<sup>98</sup> The term sick building syndrome has been applied to a condition of mucous membrane irritation caused by inadequate air-handling systems in new, energy-efficient office buildings.<sup>99</sup> Unlike IEI, however, these patients experience a limited range of symptoms, and they occur in the affected building only. Reactive airways dysfunction syndrome is a persisting asthma-like illness that arises in some persons with no preexisting asthma after an acute exposure to a toxic substance sufficient to induce a chemical bronchitis.<sup>100</sup>

## **Summary**

IEI-also called environmental illness and multiple chemical sensitivities-has been postulated to be a disease unique to modern industrial society in which certain persons are said to acquire exquisite sensitivity to numerous chemically unrelated environmental substances. The patient experiences wideranging symptoms, but evidence of pathology or physiologic dysfunction in such patients has been lacking in studies to date. Because of the subjective nature of the illness, an objective case definition is not possible. Allergic, immunotoxic, neurotoxic, cytotoxic, psychologic, sociologic, and iatrogenic theories have been postulated for both etiology and production of symptoms, but there is an absence of scientific evidence to establish any of these mechanisms as definitive. Most studies to date, however, have found an excess of current and past psychopathology in patients with this diagnosis. The relationship of these findings to the patient's symptoms is also not apparent. Rigorously controlled studies to verify the patient's reported subjective sensitivity to specific environmental chemicals have yet to be done. Moreover, there is no evidence that these patients have any immunologic or neurologic abnormalities. In addition, no form of therapy has yet been shown to alter the patient's illness in a favorable way. A causal connection between environmental chemicals, foods, and/or drugs and the patient's symptoms continues to be speculative and cannot be based on the results of currently published scientific studies.

#### References

- 1. Conclusions and recommendations of a workshop on multiple chemical sensitivities (MCS). Reg Toxicol Pharmacol 1996;24:S188-9.
- 2. Lessof M. Meetings report: Report of multiple chemical sensitivities (MCS) workshop, Berlin, Germany, 21-23 February 1996. PCS/96.29 IPCS, Geneva, Switzerland. Human Exp Toxicol 1997;16:233-4.
- 3. Report of multiple chemical sensitivities (MCS) workshop: International Programme on Chemical Safety (IPCS)/German workshop on multiple chemical sensitivities: Berlin, Germany, 21-23 February 1996. Int Arch Occup Environ Health 1997:69:224-6.
- 4. Cullen MR. The worker with multiple chemical sensitivities: an overview. Occup Med State Art Rev 1987;2:655-61.
- 5. American Academy of Allergy and Clinical Immunology Executive Committee. Position statement: Clinical ecology. J Allergy Clin Immunol 1986;78:269-71.
- 6. Randolph TG. The specific adaption syndrome. J Lab Clin Med 1956;48:934.
- 7. Randolph TG. Human ecology and susceptibility to the chemical environment. Springfield (IL): Charles C Thomas: 1962.
- 8. Randolph TG. Clinical ecology as it affects the psychiatric patient. Int J Soc Psychiatry 1966;12:245-54.
- 9. Randolph TG. Both allergy and clinical ecology are needed. Ann Allergy 1977;39:215-6.
- Randolph TG, Moss RW. An alternative approach to allergies. New York: Lippincott and Crowell;
   1980.
- 11. Dickey LD, editor. Clinical ecology. Sprigfield (IL): Charles C Thomas; 1976.
- 12. Rea WJ. Chemical sensitivity. Boca Raton (FL): Lewis Book Publishers; 1992.
- 13. Bell IR. Clinical ecology: a new medical approach to environmental illness. Bolinas (CA): Common Knowledge Press; 1982.
- 14. Ashford NA, Miller CS. Chemical exposures: low levels and high stakes. New York: Van Nostrand Reinhold; 1991.
- 15. Levin AS, Byers VS. Multiple chemical sensitivities: a practicing clinician's point of view. Clinical and research findings. Toxicol Ind Health 1992;8:95-109.
- 16. Beard GM. American nervousness, its causes and consequences: a supplement to nervous exhaustion (neurasthenia). New York: GP Putnam's Sons; 1881.
- 17. Shorter E. From paralysis to fatigue: a history of psychosomatic illness in the modern era. New York: MacMillen, Inc; 1992.
- 18. Black DW, Rathe A, Goldstein RB. Environmental illness. A controlled study of 26 subjects with '20th century disease.' JAMA 1990:264:3166-70.
- 19. Brodsky CM. Allergic to everything: a medical subculture. Psychosomatics 1983;24:731-42.

- 20. Kipen HM, Fiedler N, Maccia C, Yurkow E, Todaro J, Laskin D. Immunologic evaluation of chemically sensitive patients. Toxicol Ind Health 1992;8:125-35.
- 21. Selner JC, Staudenmayer H. Neuropsychophysiologic observations in patients presenting with environmental illness. Toxicol Ind Health 1992;8:145-55.
- 22. Simon GE. Epidemic multiple chemical sensitivity in an industrial setting. Toxicol Ind Health 1992;8:41-6.
- 23. Sparks PJ, Simon GE, Katon WJ, Ayars GH, Johnson RL. An outbreak of illness among aerospace workers. West J Med 1990;153:28-33.
- 24. Staudenmayer H, Selner ME, Selner J. Adult sequelae of childhood abuse presenting as environmental illness. Ann Allergy 1993;71:538-46.
- 25. Stewart DE, Raskin J. Psychiatric assessment of patients with "20th-century disease" ("total allergy syndrome"). Can Med Assoc J 1985;133:1001-6.
- 26. Terr Al. Environmental illness. A clinical review of 50 cases. Arch Intern Med 1986;146:145-9.
- 27. Terr Al. Clinical ecology in the workplace. J Occup Med 1989;31:257-61.
- 28. Brodsky CM. Psychological factors contributing to somatoform diseases attributed to the workplace: the case of intoxication. J Occup Med 1983;25:459.
- 29. US Department of Defense. Conduct of the Persian War: final report to Congress. Washington (DC): US Government Printing Office; 1992.
- 30. National Institutes of Health Technology Assessment Workshop Panel. The Persian Gulf experience and health. JAMA 1994;272:391-6.
- 31. Sparks PJ, Daniell W, Black DW, Kipen HM, Altman LC, Simon GE, et al. Multiple chemical sensitivity syndrome: a clinical perspective. I. Case definition, theories of pathogenesis, and research needs. J Occup Med 1994;36:718-30.
- 32. Cotterill JA. "Total allergy syndrome." Lancet 1982;1:628-9.
- 33. Green MA. "Allergic to everything": 20th century syndrome. JAMA 1985;253:842.
- 34. Klein GL, Ziering RW, Girsh L, Miller RH. The allergic-irritability syndrome: four case reports and a position statement from the Neuroallergy Committee of the American College of Allergy. Ann Allergy 1985:55:22-4.
- 35. Broughton A, Thrasher JD. Antibodies and altered cell mediated immunity in formaldehydeexposed humans. Common Toxicol 1988;2:155-74.
- 36. McGovern JJ, Lazaroni JA, Hicks MF, Adler CJ, Cleary P. Food and chemical sensitivity: clinical and immunologic correlates. Arch Otolaryngol 1983;109:292-7.
- 37. Levin AS, Byers VS. Environmental illness: a disorder of immune regulation. Occup Med State of the Art Rev 1997;2:669-82.
- 38. Bell IR, King DS. Psychological and physiological research relevant to clinical ecology: overview of the literature. Clin Ecol 1982;1:15.
- 39. Bell IR, Miller CS, Schwartz GE. An olfactory-limbic model of multiple chemical sensitivity syndrome: possible relationships to kindling and affective spectrum disorders. Biol Psychiatry 1992;32:218-42.
- 40. Miller CS. Possible models for multiple chemical sensitivity: conceptual issues and role of the limbic system. Toxicol Ind Health 1992;8:181-202.
- 41. Levine SA. Oxidants/anti-oxidants and chemical hypersensitivities (Part 1). J Biosoc Res 1983;4:51-4.
- 42. Levine SA. Oxidants/anti-oxidants and chemical hypersensitivities (Part 2). J Biosoc Res 1983;4:102-4.
- 43. Levine SA, Reinhart JH. Biochemical pathology initiated by free radicals, oxidant chemicals and therapeutic drugs in the etiology of chemical hypersensitivity disease. J Orthomol Psychiatry 1983;12:166-83.
- 44. Bascom R. Multiple chemical sensitivity: A respiratory disorder? Toxicol Ind Health 1992;8:221-8.
- 45. Doty RL, Deems DA, Frye RE, Pelberg R, Shapiro A. Olfactory sensitivity, nasal resistance, and autonomic function in patients with multiple chemical sensitivities. Arch Otolaryngol Head Neck Surg 1988;114:1422-7.
- 46. Meggs WJ. Multiple chemical sensitivities and the immune system. Toxicol Ind Health 1992;8:203-14.
- 47. Meggs WJ. Neurogenic inflammation and sensitivity to environmental chemicals. Environ Health Perspect 1993;101:234-8.

- 48. Rosenberg SJ, Freedman MR, Schmaling KB, Rose C. Personality styles of patients asserting environmental illness. J Occup Med 1991;33:737-9.
- Staudenmayer H, Camazine M. Sensing type personality, projection and universal "allergic" reactivity. J Psychol Type 1989;18:59-62.
- 50. Brodsky CM. Multiple chemical sensitivities and other "environmental illness"; a psychiatrist's view. Occup Med State of the Art Rev 1987;2:695-704.
- 51. Stewart DE. The changing faces of somatization. Psychosomatics 1990;31:153-8.
- 52. Dager SR, Holland JP, Cowley DS, Dunner DL. Panic disorder precipitated by exposure to organic solvents in the workplace. Am J Psychiatry 1987;144:1056-8.
- 53. Binkley KE, Kutcher S. Panic response to sodium lactate in patients with multiple chemical sensitivity syndrome. J Allergy Clin Immunol 1997;99:570-4.
- 54. Leznoff A. Provocative challenges in patients with multiple chemical sensitivity. J Allergy Clin Immunol 1997;99:438-42.
- 55. Faust HS, Brilliant LB. Is the diagnosis of "mass hysteria" an excuse for incomplete investigation of low-level environmental contamination? J Occup Med 1981;23:22-6.
- 56. Krug SE. Mass illness at an intermediate school: Toxic fumes or epidemic hysteria? Pediatr Emerg Care 1992;8:280-2.
- 57. Davidoff LL. Models of multiple chemical sensitivities (MCS) syndrome: using empirical data (especially interview data) to focus investigations. Toxicol Ind Health 1992;8:229-47.
- 58. Schottenfeld RS, Cullen MR. Occupation-induced posttraumatic stress disorder. Am J Psychiatry 1985;142:198.
- 59. Shusterman D, Balmes J, Cone J. Behavioral sensitization to irritants/odorants after acute exposure. J Occup Med 1988;30:565-7.
- 60. Bolla-Wilson K, Wilson RJ, Bleecker MJ. Conditioning of physical symptoms after neurotoxic exposure. J Occup Med 1988;30:684-6.
- 61. Black DW. Environmental illness and misdiagnosis-a growing problem. Reg Toxicol Pharmacol 1993;18:23-31.
- 62. Post RM. Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. Am J Psychiatry 1992;149:999-1010.
- 63. King DS. Can allergic exposure provoke psychological symptoms: a double blind test. Biol Psychiatry 1981;16:3-17.
- 64. American Academy of Allergy and Clinical Immunology Executive Committee. Position statement: Controversial techniques. J Allergy Clin Immunol 1981;67:333.
- 65. California Medical Association Scientific Board Task Force on Clinical Ecology. Clinical ecology-a critical appraisal. West J Med 1986;144:239-45.
- 66. Grieco MH. Controversial practices in allergy. JAMA 1985;253:842.
- 67. Jewett DL, Fein G, Greenberg MH. A double-blind study of symptom provocation to determine food sensitivity. N Engl J Med 1990;323:429-33.
- Terr AI. Unconventional theories and unproven methods in allergy. In: Middleton E Jr, Reed CE, Ellis EF, et al, editors. Allergy: principles and practice. 4th ed. St Louis: CV Mosby; 1993. p 1778-80.
- Madison RE, Broughton A, Thrasher JD. Immunologic biomarkers associated with an acute exposure to exothermic byproducts of a urea-formaldehyde spill. Environ Health Perspect 1991;94:219-23.
- 70. Grammar LC, Harris KE, Shaughnessy MA, Sparks PJ, Ayars GH, Altman LC. Clinical and immunological evaluation of 37 workers exposed to gaseous formaldehyde. J Allergy Clin Immunol 1990;86:177-81.
- 71. Patterson R, Dykewicz MS, Grammer LC, Pruzansky JJ, Zeiss CR, Harris KE. Creating an indoor environmental problem from a nonproblem: a need for cautious evaluation of antibodies against hapten-protein complexes. N Engl Reg Allergy Proc 1985;6:135-9.
- 72. Patterson R, Beltrani VS, Singel M, Zeiss CR, Harris KE. Formaldehyde reactions and the burden of proof. J Allergy Clin Immunol 1978;79:705-6.
- 73. Rea WJ. Environmentally triggered cardiac disease. Ann Allergy 1978;40:243-51.
- 74. Rea WJ. Environmentally triggered thrombophlebitis. Ann Allergy 1976;37:101-9.
- 75. Rea WJ. Environmentally triggered small vessel vasculitis. Ann Allergy 1977;38:245-51.

- Simon GE, Daniell W, Stockbridge H, Claypoole K, Rosenstock L. Immunologic, psychological, and neuropsychological factors in multiple chemical sensitivity. A controlled study. Ann Intern Med 1993;119:97-103.
- 77. Staudenmayer H, Selner J. Post-traumatic stress syndrome (PTSS). Escape in the environment. J Clin Psychol 1987;43:156-7.
- 78. Staudenmayer H, Selner JC. Neuropsychophysiology during relaxation in generalized, universal 'allergic' reactivity to the environment: a comparison study. J Psychosom Res 1990;34:259-70.
- 79. Terr Al. Immunological issues in "multiple chemical sensitivities." Reg Toxicol Pharmacol 1993;18:54-60.
- 80. Thrasher JD, Madison R, Broughton A, Gard Z. Building-related illness and antibodies to albumin conjugates for formaldehyde, toluene diisocyanate, and trimellitic anhydride. Am J Ind Med 1989;15:187-95.
- 81. Ziem GE. Multiple chemical sensitivity: treatment and follow-up with avoidance and control of chemical exposures. Toxicol Ind Health 1992;4:73-86.
- 82. Black DW, Rathe A, Goldstein RB. Measures of distress in 26 "environmentally ill" subjects. Psychosomatics 1993;34:131-8.
- 83. Brodsky CM, Green MA, Ogrod ES. Environment illness: Does it exist? Patient Care 1989;11/15:41-59.
- 84. American College of Physicians. Position paper: Clinical ecology. Ann Intern Med 1989;111:168-78.
- Council on Scientific Affairs, American Medical Association. Clinical Ecology. JAMA 1992;268:1634-5.
- 86. Committee on Environmental Hypersensitivities. Report of the ad hoc committee on environmental hypersensitivities disorders. Toronto (Ontario): Ministry of Health; 1985.
- National Research Council. Multiple chemical sensitivities. Washington (DC): National Academy Press; 1992.
- 88. Board of the International Society of Regulatory Toxicology and Pharmacology. Report of the ISRTP Board. Regul Toxicol Pharmacol 1993;18:79.
- 89. Barrett S. MCS: multiple chemical sensitivity. New York: American Council on Science and Health; 1994.
- Royal College of Physicians and Royal College of Pathologists. Good allergy practice-standards
  of care for providers and purchasers of allergy services within the National Health Service. Clin
  Exp Allergy 1995;25:586-95.
- 91. Root DE, Katzin DB, Schnare DW. Diagnosis and treatment of patients presenting subclinical signs and symptoms of exposure to chemicals which bioaccumulate in human tissue. In: Proceedings of the National Conference on Hazardous Wastes and Environmental Emergencies. Silver Springs (MD): Harzardous Materials Control Institute; 1985 May 14-16; p. 150-3.
- 92. Haller E. Successful management of patients with "multiple chemical sensitivities" on an inpatient psychiatric unit. J Clin Psychiatry 1993;54:196-9.
- 93. Crook WJ. The yeast connection: a medical breakthrough. Jackson (TN): Professional Books; 1983.
- 94. Blonz ER. Is there an epidemic of chronic candidiasis in our midst? JAMA 1986;256:3138.
- 95. Abbey SE, Garfinkel PE. Neurasthenia and chronic fatigue syndrome: the role of culture in the making of a diagnosis. Am J Psychiatry 1991;148:1638-46.
- 96. Terr Al. Clinical ecology. J Allergy Clin Immunol 1987;79:423-6.
- 97. Edelstein PH. Legionnaire's disease. Clin Infect Dis 1993;16:741.
- 98. Fink JN. Hypersensitivity pneumonitis. In: Middleton E Jr, Reed CE, Ellis EF, Adkinson NF Jr, Yunginger JW, Busse WW, editors. Allergy: principles and practice. 4th ed. St Louis: Mosby-Year Book; 1993.p. 1415-31.
- Marks PJ, Daniel EB. The sick building syndrome. Immunol Allergy Clin North Am 1994;14:521-35.
- 100. Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome. Case reports of persistent airways hyperreactivity following high-level irritant exposures. Am Rev Respir Dis 1985;27:473-6.

AAAAI Position Statements and Work Group Reports are not to be considered to reflect current AAAAI standards or policy after five years from the date of publication. For reference only. January 1999.