Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Idiopathic Environmental Intolerance

Discussion paper prepared for

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Prepared by:

Dr. Anthony Weinberg

Professor University of Ottawa Faculty of Medicine
Chief, Division of Internal Medicine
University of Ottawa and The Ottawa Hospital

Dr. Anthony Weinberg graduated in 1965 from the University of Durham Medical School in England. He obtained membership of Royal College of Physicians in 1968 and became a Fellow in 1993. He immigrated to Canada in 1976 and became a Fellow of the Royal College of Physicians of Canada in1979. He has been teaching at the Faculty of Medicine of the University of Ottawa since 1979 and holds de rank of full Professor. He has received numerous awards, including an Alpha Omega Alpha, Honors Society (1983), a nomination for PAIRO award for clinical teaching (1987 and 1992), a PAIRO award for excellence in clinical teaching (1988) and, in 2004, the Andre Peloquin award for excellence in clinical education. His experience includes extensive committee work for both hospital and university. He served as a Medical Assessor at the Tribunal for a number of years before being appointed a Counsellor in 1998. He is currently Head & Chair of the Division of General Internal Medicine at the University of Ottawa and at the Ottawa Hospital, an Internist and an Attending Physician on the Clinical Teaching Unit of The Ottawa Hospital.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that It is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

IDIOPATHIC ENVIRONMENTAL INTOLERANCE

Idiopathic Environmental Intolerance is the preferred term[1] for what is otherwise known as Environmental illness or Multiple Chemical Sensitivity.

It is not a recognized disease[2] with demonstrable pathology, but a state of ill health characterized by a multiplicity of symptoms and attributed to environmental toxins. Symptoms vary but include headache, breathlessness, fatigue, impaired concentration and anxiety. Sufferers may be so disabled that they cannot work and they often attribute cause of the condition to the workplace. Symptoms however generally extend into the non-work environment and are complained of in a variety of settings.

No unique reproducible physical or biochemical abnormality has been demonstrated and the mechanism by which the symptoms are induced is still subject to debate. Indeed the very existence of the condition remains controversial and there is no universally accepted case definition. Nevertheless some insights have been gained from observational studies.

In the absence of demonstrable disease I.E.I. is attributed by sufferers to environmental exposure to many chemically unrelated substances in concentrations at which the majority of the population would be unaffected. The condition is thus distinguished from demonstrably toxic exposures to which the majority of the population would suffer injury or from demonstrable allergic reactions such as asthma. Despite the lack of objective findings the condition may cause major life disruptions. Environmental intolerance sufferers make an average of twenty-three healthcare visits per annum.[3]

The triggering substances may be identified or not, but are generally volatile chemicals. They may be single triggers but most often symptoms are evoked by multiple, chemically unrelated odors. In some, the complaints begin with an identified exposure but in at least 40% no such exposure is recalled. Women are disproportionately represented[4] and there are substantial overlaps with chronic fatigue,[5] fibromyalgia and other symptom-defined[6] conditions. The true prevalence is unknown but estimates range from 0.2% to 4.0% of a general population. Experimentally the symptoms can be induced by lactate infusion[7] or CO2 inhalation[8] in susceptible subjects and resemble panic disorder. These are characteristics of persons with chronic anxiety. Affected individuals are also characteristically liable to a varity of somatization even before I.E.I. is complained of.[9]

Many theories have been advanced to explain the etiology of Idiopathic Environmental Intolerance (I.E.I.). One for which there is considerable evidence, is that I.E.I. is a manifestation of chronic anxiety. There is a tendency for sufferers to catastrophization and to adopt a particular attributional style. The contrary opinion is that the symptoms are due to an as yet undefined physiologic mechanism though to date no such mechanism has been reliably demonstrated. Postulated viral, immunology and endocrinologic hypotheses abound. Thus these theories fall into two broad categories: physical/toxicologic and behavioural or psychologic.

Physical/toxicological theories that have implicated include (a) Immunologic, (b) Non-specific inflammatory and (c) Neurotoxic mechanisms. Well-controlled studies have failed to find evidence for immunologic abnormality[10] and satisfactorily controlled studies of other mechanisms have not been performed. The relationship between the symptoms of I.E.I. and the claimed exposure does not fit the basic tenets of toxicity, i.e. dose dependency, reproducibility, consistency and predictability. Bradford Hills criteria for causality are notable for their absence.[11]

Behavioural/psychological theories include behavioural conditioning[12] and attributional style, with risk perception being a major determinant.[13] The concept of I.E.I. as a phobic disturbance is supported by studies of subjects with I.E.I. having been infused with sodium lactate[14] and other substances[15] then exhibit symptoms identical with panic disorder. This has led to the notion that I.E.I. is in effect “olfactory panic”.[16]

Others have claimed that I.E.I. may be a misdiagnosed psychiatric illness.[17] Certainly there is a body of evidence to suggest that a history of somatization and psychiatric morbidity was the strongest predictor of I.E.I., resulting from workplace exposure to chemicals.[18]

Finally there is speculation supported by the commonality of complaints between Chronic Fatigue, Fibromyalgia, Sick-Building and Gulf-War Syndromes that I.E.I. is simply the most contemporary cultural expression of psychosomatic illness.[19]

In conclusion, though no view of the etiology of I.E.I. is universally accepted the evidence for psychological/psychiatric/cognitive etiologies is increasingly more robust than the purely physical alternative. This step forward in acceptance has important implications for both prevention and treatment strategies as well as socio-political implication.[20]

It is an important concern for workplace compensation schemes as well as health care systems. As with the chronic fatigue syndrome, causal hypotheses will most probably be validated by successful treatment programs based upon them.

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[1] ISRTP Board Conclusions. Reg. Toxicology. Pharmacology 18:79, 1993

[2] JOEM 41(11) 940-941, 1999

[3] Bell IR, Schwartz GE, Baldwin CM et al. 1. Environmental. Health Perspect. 105 (Supple #2). 457-466. 1997

[4] Bell IR et al. Am.J. Med. 105(3A), 745-825, 1998

[5] Sparks PJ, Occupational. Med. 15(3), 497-510, 2000

[6] Buchwald D & Garrity D.

[7] Leznoff A, J Allergy & Clinical Immunology 99(4), 438-442, 1997

[8] Poonai N et al, J. Allergy & Clinical. Immunology, 106(2), 358-363, 2000

[9] Simon G, Katon WJ, Sparks PJ, Am. J. Psychiatry, 147 : 901-906, 1990

[10] Simon G, Danell W, Stockbridge H. et al., Annals. Internal. Med. vol 119: 97-103, 1993

[11] Hill AB, Proc Roy Soc Med. 58: 295-300, 1965

[12] Bella-Wilson K, Wilson RJ, Bleeker ML.

[13] McClelland GH, et al 1. Risk Analysis 10: 485-497, 1990

[14] Binkley KE, Krutcher S.J. Allergy Clinical Immunology 99(4) 570-574, 1997

[15] Leznoff A. J Allergy Clinical Immunology 99(4), 483-442, 1997

[16] Binkley KE, Krutcher S.J. Allergy Clinical Immunology 99(4) 570-574, 1997

[17] Black DW, Rhaths A, Goldstein RB., JAMA 264 : 3166-3170, 1990

[18] Simon G, Katon WJ, Sparks PJ., Am.J. Psychiatry 147, 901-906, 1990

[19] Brodsky CM, Occupational Med. 2 : 695-704, 198

[20] Deyo RA, NEJM 336(16) : 1176-1179, 1997