

ABC OF ALLERGOLOGY

IDIOPATHIC ENVIRONMENTAL INTOLERANCE (IEI)

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Is IEI an organic disease initiated by environmental chemical exposure or a psychological disorder?

This controversial condition first described by USA 'clinical ecologist' Theron Randolph in the 1940s, was called 'environmental illness'. Randolph hypothesised that the body was like 'a barrel filling up with chemicals' until a critical point is reached after which it reacts to any further chemical exposure. With numerous household chemicals and everyday agents suspected in the aetiology, the label multiple chemical sensitivity (MCS) was coined by Cullen in the 1980s.¹ MCS enjoyed wide acceptance as a medical condition in the popular lay media, among alternative practitioners, and in those individuals with non-specific symptoms who self-diagnosed themselves with MCS.



Up to 16% of the general population report some form of 'unusual sensitivity' to common everyday chemicals (a phenomenon which is culturally restricted to North America and Europe). This prompted the National Institute of Environmental Health (NIEH) in the USA to develop a consensus statement in 1999.² The NIEH defined multiple chemical sensitivity as a 'chronic recurring disease caused by a person's inability to tolerate an environmental chemical or class of foreign chemicals' and proposed the preferred medical term Idiopathic environmental intolerance (IEI) with 6 diagnostic criteria (Table I).

Symptoms attributed to MCS/IEI are diffuse and include: recurrent headaches, sinus and nasal congestion, itchy eyes and throat, with abdominal symptoms such as nausea and vomiting, diarrhoea or constipation and bloating. Additional symptoms and signs include

Table I. Idiopathic environmental intolerance (IEI) diagnostic criteria

1. Symptoms recur and are reproducible.
2. The condition is chronic.
3. Low levels of exposure (lower than previously or commonly tolerated) result in manifestations of the syndrome (i.e. increasing sensitivity occurs over time).
4. The symptoms improve, or resolve when the triggering chemicals are removed from the environment.
5. Responses often occur to multiple unrelated chemicals.
6. Symptoms involve multiple-organs (runny nose, itchy eyes, headache, scratchy throat, nausea and/or diarrhoea, abdominal cramping, aching joints, etc.).

non-specific rashes, breathing difficulties, muscle and joint aches, bladder problems, fatigue and flu-like symptoms, irritability, anxiety, depression, malaise, inability to concentrate, stupor ('brain fog'), hypotension, sleepiness, insomnia, mania, tremor and even paraesthesia. These symptoms are attributed to a chronic prolonged and low-dose environmental chemical exposure.

Overlap has been proposed with other environment-linked conditions such as sick-building syndrome (SBS), food intolerance syndrome (FIS) and even Gulf War illness (GWI). It is postulated that trigger chemicals include formaldehyde found in furnishings and plastics, ink in newsprint and printers, volatile organic compounds (VOCs) in cleaning materials, Freon in poorly ventilated buildings, sulphites, pesticides and other chemicals in wine and processed food products. Those affected may also be prone to chronic fatigue syndrome, fibromyalgia, irritable bowel syndrome, connective tissue disorders after silicone breast implants, reactive hypoglycaemia, drug-induced hepatitis, reactions related to living near toxic waste dumps and electromagnetism from power lines, dental amalgam disease and reactions to the petrol additive MTBE (methyl-tert-butyl ether).



Immunological mechanisms (mainly non-IgE) have been proposed but none have been satisfactorily proven. Terr³ extensively reviewed the immune theories proposed by the clinical ecologists and found none could be substantiated. Aetiological theories are usual-

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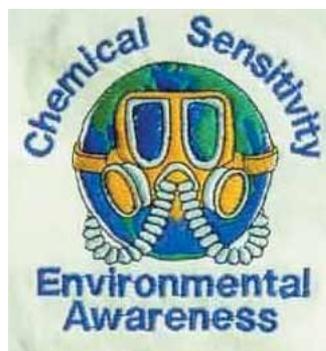
ly based on empiric hypotheses drawn mostly from poor-quality studies published in the fringe medical press. These have failed to establish any convincing evidential link between ill-health and day-to-day low-level chemical exposure.

A genetic predisposition with heightened sensitivity and abnormal olfactory or 'odorant' receptors has also been proposed but never confirmed in clinical studies. In studies where the chemical odour was masked or suppressed by menthol, there was no difference between the study and control group symptomatology.

Many physicians believe that the symptoms of IEI are psychophysiological in nature and those affected are prone to panic responses which enhance their symptoms. Binckley and Kutcher⁴ demonstrated that individuals with self-identified chemical sensitivities responded with typical panic attacks when challenged with intravenous lactate (in a similar manner to individuals with underlying panic disorders). In Lenzoff's⁵ (Query: spelling? Diff in ref) study, all the MCS patients who responded to a challenge with their trigger substances developed symptoms and signs of acute anxiety with hyperventilation and a rapid fall in PCO₂, while their lung function remained normal.

In addition, IEI-prone individuals appear to have a heightened olfactory awareness (hyperosmia) and a profound odour aversion (cacosmia) to otherwise harmless volatile organic compounds found in many household products, aerosols and even from trees. A recent systematic review of provocation studies for MCS by Das-Munshi, Rubin and Wessely⁶ drew negative conclusions. Thirty-seven provocation studies testing 784 persons reporting MCS and 547 controls were assessed in their meta-analysis. Blinding was found to be inadequate in many studies, as most did not conceal the implicated chemical's odour from their subjects. Only 3 studies used olfactory masking agents and in these none found associations between provocations and response. They concluded that persons with MCS/IEI do react to chemical challenges if they can smell the chemical but do not react when the odour is masked. This suggests that their response was not to the chemical itself, but rather related to their 'expectations and prior beliefs'.

Psychological conditioning has been proposed as the underlying abnormality in IEI, but is complicated by a high incidence of pre-morbid psychological trauma



(including childhood physical and sexual abuse). This may lead to profound long-term effects on mood and affect, cognitive processing, hypervigilance and entrenched beliefs of victimisation.⁷ It is possible for patients with IEI to react to an olfactory sensation by amplifying the perceived

effects into a full-blown panic attack and even trigger off underlying idiopathic anaphylaxis.

In the final analysis, after many years of investigation, there appears to be no convincing evidence in the medical literature for the existence of MCS or IEI. The underlying cause for the IEI symptom complex is unlikely to be a direct reaction to everyday chemicals, but rather a masked stress disorder with heightened olfactory awareness (hyperosmia) and associated unresolved psychological issues.

Declaration of conflict of interest

The author declares no conflict of interest.

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