

More Evidence of the Organic Nature of Multiple Chemical Sensitivity

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Attention is drawn to a recent paper which serves to confirm that multiple chemical sensitivity (MCS), a well-documented component of myalgic encephalomyelitis (ME), is not a somatoform disorder or any other kind of psychiatric disorder as asserted by certain psychiatrists, most notably those of the Wessely School.

The paper (Biological definition of multiple chemical sensitivity from redox state and cytokine profiling and not from polymorphisms of xenobiotic-metabolizing enzymes; De Luca C et al: Toxicol. Appl. Pharmacol 2010; doi:10.1016/j.taap.2010.04.017) supports the work of Professor Emeritus of Biochemistry and Basic Medical Sciences at Washington State University, Martin Pall, who proposed that MCS is caused by toxic chemical exposure leading to toxic brain injury.

This recent work was conducted by a research group based in Rome and was supported by grants from the Italian Ministry for Health, the Italian Ministry for University and Research, the Swedish Medical Society and the Swedish Research Council.

The authors note that the number of subjects affected by MCS has been growing steadily (reaching up to 15% of the US population) and that patients report recurring multi-organ symptoms affecting the nervous, cardiovascular, gastrointestinal, respiratory, musculo-skeletal, skin and ocular systems and that, following the primary triggering event, such symptoms occur after subsequent exposure to virtually negligible concentrations of everyday odours such as perfume, new paint, household cleaning chemicals, newsprint, new carpets etc.

These researchers sought to prove their working hypothesis about the inherited and/or acquired dysfunction of the chemical defence system as a molecular basis for MCS, focusing on genetic and metabolic markers of the chemical defence and immune systems exerted through cytokine dysregulation.

GST (glutathione S-transferase) belongs to a family of conjugating enzymes that play a key role in cellular processes of inflammation and the authors note that the GST isozyme polymorphisms have

been implicated in other environment-related pathologies such as systemic lupus erythematosus as well as in MCS. Both reduced and oxidised forms of glutathione were severely depleted in MCS subjects compared with healthy controls. In addition, the authors found highly elevated levels of pro-inflammatory IFNgamma, IL-8 and MCP-1 (macrophage chemotactic protein) in the plasma of MCS patients compared with healthy controls, as well as higher than normal levels of IL-10, PDGF (platelet derived growth factor) and VEGF (vascular endothelial growth factor).

The authors note that high levels of IFNgamma suggest the prevalence of activated Th1 lymphocytes, whereas up-regulated IL-10 may represent the persistent effort of regulatory T cells to counteract Th1 activation, noting that differentiation of T helper cells in the direction of Th1 and regulatory T cells is characteristic of an autoimmune response and that these findings in MCS patients provide a promising link between impairment of immune and chemical defensive systems in such patients.

Based on the results obtained, the authors suggest that the serious and multiple dysfunctions of the chemical defence system found in MCS patients may not depend on genetic defects but on non-genetic modifications of metabolising/antioxidant enzyme expression and/or activity. They conclude that MCS is characterised by a number of biochemical and immunological disturbances and that these metabolic and immunological parameters should be taken into consideration in both the biological and clinical/laboratory diagnosis of MCS.