Press Release:

Gene Variants in CS

"A genetic variant makes suffers of multiple chemical sensitivity (MCS) more likely to develop the condition."

For Immediate Release

Three recent studies show that a genetic variant makes sufferers of multiple chemical sensitivity (MCS) more likely to develop the condition. In 2004, McKeown-Eyssen studied 203 MCS sufferers and 162 controls and found that genetic differences relating to detoxification processes were present more often in those with MCS than those without. The study concluded that "a genetic predisposition for MCS may involve altered biotransformation of environmental chemicals. Haley found similar, confirmatory results in a 1999 study with the PON1 gene in Gulf War syndrome veterans.

A new study by Schnakenberg et al (2006) confirmed the genetic variation previously found by McKeown-Eyssen and Haley. A total of 521 unrelated individuals participated in the study. Genetic variants of four genes were analyzed: NAT2, GSTM1, GSTT1, and GSTP1. The researchers concluded that individuals who are NAT2 slow acetylators and those with homozygously deleted GSTM1 and GSTT1 genes are significantly more likely to develop chemical sensitivity.

"According to the study the glutathione S-transferases act to inactivate chemicals so people without these GSTM1 and GSTT1 genes are less able to metabolize environmental chemicals," said Lourdes Salvador, Founder and President of MCS America. "If a person cannot metabolize chemicals, they build up in the body and cause disturbances in normal body function," added Salvador who is also the chief editor of MCS America News. Schnakenberg and fellow researchers explain that "glutathione S-transferases play an important role in the detoxification of chemicals... the deletion of this gene may be an important step in the early onset of diseases" which is a critical discovery that provides a biological basis behind the etiology of multiple chemical sensitivity.

The researchers also noted that diseases such as non-Hodgkin's lymphoma, hepatocellular and prostate carcinoma, and Alzheimer's disease have been associated with the common chemicals metabolized by GSTP1. The deletion of the GSTP1 gene leaves individuals more susceptible to developing these diseases, as lack of these genes means a loss of protection from oxidative stress. "This discovery is crucial to being able to diagnose and treat those who suffer from multiple chemical sensitivity and other toxic injuries," said Salvador. "It is the first step toward understanding and explaining the cause of chemical injury and resulting sensitivities so that treatments can be developed."

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A cross-sectional study of self-reported chemical-related sensitivity is associated with gene variants of drug-metabolizing enzymes.

Schnakenberg E,
Fabig KR,
Stanulla M,
Strobl N,
Lustig M,
Fabig N,
Schloot W.

Institute for Pharmacogenetic and Genetic Disposition, Ostpassage 7, D-30853 Langenhagen, Germany. es@ipgd.org

BACKGROUND: N-acetyltransferases (NAT) and glutathione S-transferases (GST) are involved in the metabolism of several ubiquitous chemical substances leading to the activation and detoxification of carcinogenic heterocyclic and aromatic amines. Since polymorphisms within these genes are described to influence the metabolism of ubiquitous chemicals, we conducted the present study to determine if individuals with self-reported chemical-related sensitivity differed from controls without self-reported chemical-related sensitivity with regard to the distribution of genotype frequencies of NAT2, GSTM1, GSTT1, and GSTP1 polymorphisms. METHODS: Out of 800 subjects who answered a questionnaire of ten items with regard to their severity of chemical sensitivity 521 unrelated individuals agreed to participate in the study. Subsequently, genetic variants of the NAT2, GSTM1, GSTT1, and GSTP1 genes were analyzed. RESULTS: The results show significant differences between individuals with and without self-reported chemical-related sensitivity with regard to the distribution of NAT2, GSTM1, and GSTT1 gene variants. Cases with self-reported chemical-related sensitivity were significantly more frequently NAT2 slow acetylators (controlled OR = 1.81, 95% CI = 1.27-2.59, P = 0.001). GSTM1 and GSTT1 genes were significantly more often homozygously deleted in those individuals reporting sensitivity to chemicals compared to controls (GSTM1: controlled OR 2.08, 95% CI = 1.46-2.96, P = 0.0001; GSTT1: controlled OR = 2.80, 95% CI = 1.65-4.75, P = 0.0001). Effects for GSTP1 gene variants were observed in conjunction with GSTM1, GSTT1 and NAT2 gene. CONCLUSION: The results from our study population show that individuals being slow acetylators and/or harbouring a homozygous GSTM1 and/or GSTT1 deletion reported chemical-related hypersensitivity more frequently.

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