



MCS America

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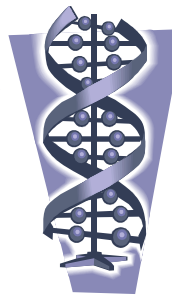
Multiple Chemical Sensitivity (MCS) and Genetics

By Christiane Tourtet, B.A.

Millions of people all over the world, including small children, have developed Multiple Chemical Sensitivity (MCS) upon exposure to toxic chemicals in the environment. Due to the incessantly growing number of the worldwide general population stricken by MCS upon exposure to toxic chemicals, MCS has become one of the most serious and overlooked public health problem in the world. But what causes this terrible hypersensitivity to chemicals and other substances in the environment for millions of people of any race, gender, age, and national origin? Why is it that millions of these people, who lived normal lives, until one day a chemical exposure, ranging anywhere from pesticides, insecticides, fumigants, building renovations, installation of new carpeting, fresh paint... the list could go on almost endlessly, started to develop reactions from mild to severe.

The wide variety of symptoms experienced by persons with MCS has puzzled everybody, including the medical profession, for decades and the medical profession is unprepared to deal with such occurrence of a worldwide epidemic of this environmentally induced disease. How-

ever, regardless of the large variety of symptoms experienced by persons suffering from MCS, there tends to be a definite pattern of neurological, respiratory and cardiac symptoms.¹



There is no doubt that pesticides², especially organophosphates pesticides and insecticides, among so many other neurotoxic chemicals, are playing a major role in triggering MCS. According to the environmental medicine publication, Environmental Health Perspectives, a survey in September 2003 showed indeed, that about one third of persons developed MCS after being exposed to pesticides and another third developed MCS after being exposed to solvents.³

Many research studies have been done on MCS, among them the SPECT brain Scan, with sponsors including the US Department of Health and Human Services, which found that MCS patients exposed to neurotoxic chemicals indicated a random thinning of cortical gray matter (tissue in the cortex of the brain). The researchers made the very important conclusion that symptomatic MCS patients with an history of exposures to chemicals, had significantly diminished cerebral blood flow to the

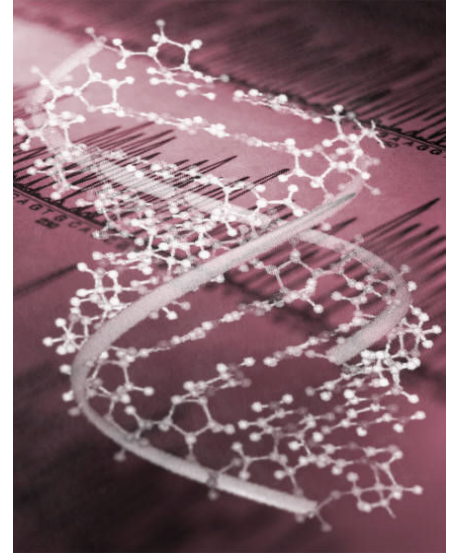
“It also has been found that brain inflammation corresponds with symptoms of MCS.”

brain, and that significant impairment of brain function may last for years after exposure to neurotoxic chemicals has ceased.^{4,5}

Some other studies are indicative that persons with MCS have little or no brain barrier that protects them from damages from low-level chemical exposures due to an excess of peroxynitrite (Pall M. 2001) that breaks down the blood barrier, therefore allowing greater access to the brain of persons afflicted by MCS. The more there is an excess of peroxynitrite, the more there is an inhibition of cytochrome P450, which means that it slows down the body's natural detoxification process. Nitric oxide inhibits cytochrome P450 activity, thus slowing degradation of hydrophobic organic chemicals (Pall, 2003).^{6,7}

These findings can explain the reactivity of persons with MCS to even minute doses of chemical exposures to which the non-MCS population would not react to. The combination of reduced or non-existent blood brain barrier and impaired detoxifying process of persons with MCS may induce long term, and even permanent brain and central nervous system damage, which can include toxic encephalopathy.

It also has been found that brain inflammation corresponds with symptoms of MCS. In 1999, Meggs came to the conclusion that Multiple Chemical Sensitivity (MCS) is potentially caused by low molecular weight chemicals, binding to chemoreceptors on sensory nerve C-fibers, thus



leading to the release of inflammatory mediators.⁸

Others studies also showed certain vitamins and minerals deficiencies, abnormal lipid and carbohydrate metabolism.⁹ In 2005, Gibson suggested limbic kindling, neural sensitization, and neurogenic inflammation upon exposures to various pesticides, volatile organic compounds, solvents and other toxic compounds.¹⁰

However, all these very interesting findings are rather secondary effects of MCS in a vulnerable population, and do not really reveal the true cause of MCS. So what is the real cause of Multiple Chemical Sensitivity? The real cause of MCS appears to lie in the susceptibility of genes. This is why, even though we are all exposed daily to toxic chemicals in the environment, millions of people have developed MCS, while millions of others not only did not develop MCS, but have been totally unaffected by exposures to toxic/neurotoxic chemicals in the environment.

“Because of their genetic makeup some people are more susceptible to chemical exposures than others.”

An individual with normal healthy genes will be very resistant to exposures to toxic/neurotoxic chemicals in the environment, while an individual with abnormal, weak genes will have very little resistance to exposures to toxic/neurotoxic chemicals in the environment, which explains why millions of people all over the world have developed MCS versus millions of others who have not. This determines how the body responds to exposures to chemicals. Certain segments of the population are generally more sensitive including the very young, the old and people with compromised immune systems or livers. Men and women may respond differently to chemical exposures and are at risk for different adverse health effects.¹¹

Because of their genetic makeup some people are more susceptible to chemical exposures than others. The adverse health effect of chemicals depends on their toxicity and how people are exposed to the chemicals, as well as individual susceptibility. Exposures to toxic chemicals can lead to a very wide range of health effects which may take place immediately or may take very long time to develop, which include impairment of the immune system, genetic damage (mutagenicity) and inhibition of the body's ability to breakdown chemicals.^{13, 14, 16}

There is no doubt, that persons with MCS have a genetic susceptibility to toxic chemicals found in the environment. In other words

they have abnormal genes that make them vulnerable to exposures to toxic chemicals in the environment. These abnormal genes impair the detoxifying process in the MCS population, which in turn has the consequence of bio-accumulation of toxic chemicals in the body (body burden). Once the body has reached its saturation point, any subsequent exposure to any chemicals will trigger the symptoms of Multiple Chemical Sensitivity. The accumulation of toxic/neurotoxic chemicals in the body of persons with MCS explains the characteristics of the symptoms of neurotoxicity experienced by all persons afflicted by severe MCS.

There lies the true answer to the puzzle of Multiple Chemical Sensitivity. Now, let's look scientifically into how genetics apply to Multiple Chemical Sensitivity. Genetic factors seem to contribute to virtually every human disease. The majority of diseases are influenced by multiples genes and environmental factors. Genetic variation plays a role in whether an individual has a higher or lower risk for getting a particular illness.¹²



“Genes are pieces of DNA that hold instructions for building a particular protein.”



The DNA sequence in every human being is 99.9 % percent identical to the one of every other human. The slight variation in our genes is known as single nucleotide polymorphisms or SNPS. It is estimated by scientists that there are about 1.4 million locations on the genome, where SNPS can possibly occur in humans. Small variations contribute to individual differences and can also be triggered by toxic substances in the environment. Some SNPS can cause or predispose a person to disease or influence the person's response drugs.¹¹

Some human illnesses and defects are directly or indirectly caused by genetics abnormalities. Some other illnesses however are caused by complex interacting genetics and environmental factors that cannot be explained by inheritance patterns,¹¹ We may inherit a specific condition, as well as the particular ability to respond to environmental stresses such as bacteria, viruses and environmental toxins.^{11,12} Understanding how DNA can influ-

ence every health aspect will hopefully and eventually lead to effective ways to prevent, treat, and cure thousands of diseases afflicting humankind.

Genes are pieces of DNA that hold instructions for building a particular protein. Proteins are most essential for all aspects of life. Through these proteins, our genes dictate how we look, how we respond to infections, how well we process our foods, and detoxify poisons. Harmful DNA variations, called mutations, can cause or constitute many different disease and conditions. It depends on their size and location, and they can be devastating;^{11, 12}

An interesting scientific study done in Israel, “A transcription –activating polymorphism in the ACHE promoter associated with acute sensitivity to anti-acetylcholinesterases”, by Michael Shapira et. al, showed that chemical hypersensitivity to xenobiotics causes adverse responses to normally subacute levels of a specific chemical or a group of chemicals. Persons afflicted with this abnormality may suffer from an exaggerated immune response showing itself as inflammation of mucosal and epithelial tissues. They may also alternatively show abnormal capacity for scavenging, modifying or degrading relevant chemicals.¹⁷

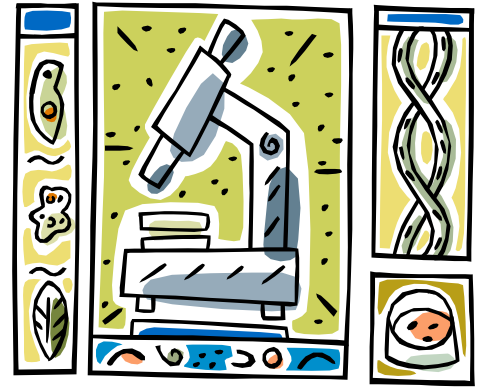
The aberrantly processed chemicals may in turn cause toxicological stress in target tissues with many symptoms of varying nature and timing according to the tissue, the type of exposure, and the actual permeability of the chemicals.

“The ability of the PON1 enzyme to protect the body from the toxicity of pesticides is determined by genetics...”

The mutations that lead to such faulty chemical metabolism were identified largely within the coding regions and consequently affecting the detoxifying protein properties. However, the impaired transcriptional activation of genes responsible for detoxification, because of their regulatory sequences, may be an equally important cause of chemical hypersensitivity. For instance, the metal-chelating metallothioneins, and some of the members of the cytochrome P450 chemical-modifying enzyme family, respond to exposure to xenobiotics by transcriptional activation, which is responsible for increased protection. The most significant finding thus, is that impaired transcriptional activation due to promoter polymorphisms in these genes would be then the cause of chemical hypersensitivity.¹⁷

In another extremely interesting scientific study, researchers at the University of California, Berkley and the University of Washington have found that some newborns can be 26 to 50 times more susceptible to certain organophosphates pesticides and 65 to 130 times more sensitive than adults.¹⁶ The study also revealed much greater variability in susceptibility to pesticides, than it was previously predicted. The researchers used the activity levels of paraoxonase I (PON1) measured in blood samples as a marker for pesticides susceptibility.¹⁶

PON1 is an enzyme whose function is to break down the toxic metabolites of organophosphate



pesticides. Research Professor, Clement Furlong in the Department of Medicine and Genome Sciences at the University of Washington and co-lead author of the paper, had found the significance of PON1 in prior animal studies.¹⁶ In these studies, mice that lacked the PON1 enzyme died when exposed to low levels of organophosphates pesticides, while the other mice with normal levels of the enzyme PON1 and given the same dose did not develop any symptoms at all. Both the quality and quantity of the PON1 enzyme is very important in determining the ability to detoxify pesticides.¹⁶

What is extremely significant for the MCS population is that the ability of the PON1 enzyme to protect the body from the toxicity of pesticides is determined by genetics, more specifically, whether a person has the Q or R form of the PON1 gene at position 192 on the chromosome.¹⁶

In addition to the factors that affect the type of PON1 enzyme, there are also additional genetic variants that affect the levels of enzyme available. People have remarkable differences in enzymes that defend their health from toxic pesticides exposures.¹⁶

“The result showed evidence of significant differences... in regard to the distribution of NAT2, GSTM1, and GSTT1 gene variants.”



A very important, most recent, cross-sectional study¹⁹ of self-reported - related sensitivity, conducted by Eckart Schnakenberg, Institute for Pharmacogenetic and Genetic Disposition, Langenhagen, Germany, Karl-Rainer Fabig, Nathalie Fabig, Clinical Practice for Toxicology and Environmental Medicine, Hamburg, Germany, Martin Stanulla, Children's Hospital, Pediatric Hematology and Oncology, Hannover Medical School, Hannover, Germany, Nils Strobl, Michael Lustig, Werner Schloot, Center for Human Genetics and Genetic Counseling, University of Bremen, Bremen, Germany, analyzed genetic variants of the NAT2, GSTM1, GSTT1, and GSTP1 genes.

The result showed evidence of significant differences between individuals with and without self-reported chemical-sensitivity in regard to the distribution of NAT2, GSTM1, and GSTT1 gene variants. Cases with self-reported chemical-related sensitivity were

quite significantly more frequently NAT2 slow acetylators. GSTM1 and GSTT1 genes were very significantly more often homozygously deleted in the individuals who were reporting sensitivity to chemicals in comparison to controls, and the effects for GSTP1 gene variants were observed in conjunction with GSTM1, GSTT1 and NAT2 gene.

In conclusion, the results from this study population show that individuals who are slow acetylators and/or having a homozygous GSTM1 and/or GSTT1 deletion reported more frequently chemical-related hypersensitivity, thus providing the genetic evidence of MCS.

Brenda Eskenazi of the University of California, Berkley, Professor of Epidemiology and Director of the Center for Children's Environmental Health Research said “Our next step is to look at the relationship between pesticides exposure and neurodevelopment, specifically for young children and genetically susceptible populations”.



“CYP2D6 is responsible for encoding enzymes that metabolizes many toxic chemicals as well as therapeutic drugs.”

One of the most significant scientific genetic findings for the MCS population is the Case-control study of genotypes in Multiple Chemical Sensitivity: CYP2D6, NAT2, PON1 and MTHFR, conducted by: Gail Mckeown-Eyssen,¹⁸ Department of Public Health Sciences, University of Toronto, Department of Nutritional Sciences, University of Toronto, Toronto, Canada, Cornelia Baines, Nicole Railey, Vartouhi Jazmaji, Department of Public Health Sciences, University of Toronto, Toronto, Canada, David E.C. Cole, Department of Laboratory Medicine and Pathology, University of Toronto, Department of Medicine, University of Toronto, Toronto, Department of Pediatrics (Genetics), University of Toronto, Toronto, Canada. Rachel F.Tyndale, Centre for Addiction and Mental Health, Toronto, Department of Pharmacology, University of Toronto, Toronto, Canada, Lynn Marshall, Environmental Health Clinic Unit, Women’s College Hospital Toronto, Canada, Ambulatory care Center, Sunnybrook and Women’s College Health Sciences Centre, Toronto, Canada.

The research team collected the blood samples of the 203 cases and 162 controls participating in the research for genetic analysis. They isolated genomic DNA and identified specific polymorphisms in order to determine the frequency of occurrence of certain polymorphisms, in cases compared to controls, for several genes that play major roles in how the body responds to chemicals. In their testing for different allele frequencies



in specific genes, they found quite significant differences for the CYP2D6 (Cytochrome P450 2D6) gene and a marginally significant difference for the NAT2 gene.¹⁸

CYP2D6 is responsible for encoding enzymes that metabolizes many toxic chemicals as well as therapeutic drugs. Cytochrome P450 enzymes are a large group of enzymes that include a quite important part of the body’s instruments to get rid of potentially harmful substances.¹⁸

NAT1 and NAT2 (N-acetyltransferases) are responsible for encoding enzymes needed for metabolizing toxic chemicals, different drugs, aromatic amines. PON (paraoxonase) are genes code for the products of proteins that react with toxins such as pesticides and nerve agents.¹⁸

MTHFR (methylenetetrahydrofolate reductase) is responsible for encoding a key enzyme involved in the metabolism of some B vitamins (including folate and B 12). MTHFR – C677T gene was studied because impaired vitamin B12 metabolism can contribute to neurological symptoms.¹⁸

“These differences were found in genes that are crucial to detoxifying toxic compounds.”

The distribution of genotypes between cases and controls were significantly different for both the CYP2D6 and NAT2. The women who were homozygous for the active form of the gene CYP2D6, had over three times the risk of getting MCS compared to the women who were homozygous for the inactive form of CYP2D6. The women who were heterozygous for the active form of the gene CYP2D6, also showed an increased risk of developing MCS.¹⁸

This suggests a “gene-dose effect”, with the risk of getting Multiple Chemical Sensitivity (MCS) increasing in respect to the number of active CYP2D6 alleles. The women who were homozygous for the fast form of the gene NAT2, (the 4 allele) called “rapid acetylators” had over four times the risk of getting Multiple Chemical Sensitivity (MCS) in comparison to slow acetylators

who have a lack of the 4 allele of the gene NAT2. As for the women who were heterozygous, intermediate and had only one 4 allele, they did not show an increased risk.¹⁸

The most striking finding was that the women with genes that encode rapid metabolism by both enzymes CYP2D6 homozygous active and NAT2 “rapid acetylator” genotypes, were over 18 times more likely to get MCS in comparison to the women with the slow metabolic forms.¹⁸

So in resume, in this epidemiology study, the researchers have reported an extremely important finding for the MCS population of several genetic differences between cases and controls in the genes involved in the detoxifying process of contaminants and that basically a genetic predisposition for MCS involves altered biotransformation of environmental chemicals.

Not only did they discover that indeed differences do exist, but also these differences were found in genes that are crucial to detoxifying toxic compounds, and that MCS patients are more likely to have the type of genes that would impair their ability to detoxify toxics themselves. When they looked at two genes at the same time, they found that women with MCS were over 18 times more likely than controls to have a very specific combination of forms of two separate genes.



**“Further
scientific
genetic
research must
urgently be
done.”**

Their data is the first of a kind to demonstrate such genetic differences in enzymes crucial for the detoxification of contaminants by comparing people with MCS to those who do not have MCS. It is thus providing strong genetic evidence for the physically- based occurrence of MCS.

The studies of Haley²⁰ and Furlong²⁰, of the Gulf War Veterans, which implicated the gene PONI, of Shapira¹⁷ et al, of Schnakenberg¹⁹ et al involving three genes, and of Mc Keown-Eyssen¹⁸ and colleagues implicating five genes, show that one or more genes controlling the metabolism of compounds previously implicated in Multiple Chemical Sensitivity, control the occurrence of chemical sensitivity, thus providing the physical evidence of the genetic origin of Multiple Chemical Sensitivity (MCS) upon exposures to chemicals.

Due to the increasing magnitude of this pandemic environmentally induced disease affecting millions of people all over the world, further scientific genetic research must urgently be done in order to fully identify and understand all the genes variations and mechanisms that make the MCS population so susceptible to toxic chemicals and other substances in the environment.

Research into possible treatment, such as gene therapy, would hopefully be on its way to possibly prevent and even cure the millions of people from all over the world, suffering from the devastating health effects of MCS, and would be the scientific turning point for a better world for future generations.

- Christiane Tourtet

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Christiane Tourtet is a well-known freelance, award winning Medical, Sciences, and Environmental writer, photo-journalist, photographer, and devoted Multiple Chemical Sensitivity (MCS) activist for over 20 years. She is the Founder and President of International MCS Awareness, and USA and Florida State Coordinator for MCS-Global. Her biography has been included in numerous world wide publications, notably in Who's Who in America and Who's Who in the World.

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