

UNDERSTANDING CHEMICAL INTOLERANCE

A personal investigation by [Don Richard Paladin](#)

With some researchers suggesting as many as 15% of our population have chemical intolerance or chemical sensitivity, one wonders why our government has done little to prevent many people from becoming chemically injured. (1)

There are many people world wide who have become disabled by chemicals their bodies do not seem to be able to detoxify.(2) Many of us are involved in a network of chemically injured people world wide who are trying to bring understanding and justice to this issue. [Those](#) who are opposed to recognition of chemical injury are very well organized with access to many resources that have been used to prevent research and recognition of Chemical Intolerance(CI) and chemical injury.(3)(4) They created their own network, it appears to some, to prevent recognition of chemical injury. Because the financial stakes are so high, they have effectively gone after advocates of the chemically injured. The opposition to recognition of CI appear to have the odds in their favor. Unfortunately for them they do NOT have truth on their side. We seek your understanding of chemical intolerance so that we can find safe alternatives to the toxic synthetics man has created and to do no harm to anyone with man-made synthetics.

We would like to focus on a sub-population of the chemically injured because information and understanding about pesticide intolerance to organophosphate poisons will be the crack of light that leads to complete understanding of this issue. Once we acknowledge and understand that pesticides are poisons and not allergens that must be detoxified by enzymes, then we will be closer to explaining why some people cannot tolerate these toxicants.

One of the biggest problems when adding a new level of understanding to accepted conventional knowledge is getting the gatekeepers of conventional knowledge to admit that their systems and understanding are not complete. Unfortunately, many who appoint themselves as GATEKEEPERS of conventional wisdom, are often more oriented to the power of the information they have already learned rather than comprehending and extending knowledge of new information. The allopathic medical diagnostic protocol allows the allopath to sort out illnesses they don't understand from their biomedical model into those generated by the mind. It relieves them of the responsibility of treating someone difficult to treat because they do not respond to the treatment of symptoms by drugs. Since Chemical Intolerance is not an antibody mediated allergy, is not an infection, and is not cancer, then in the allopathic diagnostic protocol it must be psychogenic illness without an external cause. It does not seem to matter to the allopathic scientific community that "psychogenic illnesses" have no known biomarkers. Symptoms are not causes. Symptom based psychobabble does not explain the biochemical mechanism in disorders not understood by the allopathic scientific community.

One difficulty getting others to understand the issues of emerging illnesses like Gulf War Syndrome (GWS) and Multiple Chemical Sensitivity (MCS) or Chemical Intolerance (CI) is that there are many people who are exposed to the same environmental toxicants and seem to be able to effectively detoxify them and not

become injured from these toxic products. The old adage one man's meat is another man's poison seems to apply.

What much of our conventional research does is look at a test population and then statistically average the effects of data gathered. There is NO controlling for what is referred to as a VARIABILITY OF TOLERANCE or BIOCHEMICAL INDIVIDUALITY. Quite simply, not all people react in the same way and at the same level to a variety of different chemicals (both natural and manmade). The new field of pharmacogenetics (5) is just now beginning to address the issue of *variability of tolerance*.

Genelex in Bellevue, Washington does enzyme assays to determine if one may be deficient in the most common enzymes (6) involved in detoxification of drugs. At their site (7) they address the research in the 1998 Journal of the American Medical Association (JAMA) article which begins to address the problem of genetic variation (8) in enzyme levels responsible for detoxifying drugs. "A 1998 medical report estimated that adverse reactions to prescription drugs kill about 106,000 Americans annually, roughly three times as many as are killed in automobile accidents. This makes prescription drugs the fourth leading killer in the U.S., after heart disease, cancer, and stroke. Another 2.2 million Americans experience serious, nonfatal ADRs. Patients on anti-depressants, anti-psychotics, or various heart medicines may be especially vulnerable when they are taking combinations of drugs. Even over-the-counter drugs take their toll, particularly when interacting with prescription drugs."

A commonly understood example of an enzyme deficiency is lactose intolerance.(9) This is NOT a milk allergy. It is a deficiency of the natural enzyme in some people (a large number of people) who cannot effectively break down milk products so that they can be digested effectively. People with lactose intolerance can take an enzyme called *lactase* to help them break down milk products. Once one understands the difference between an *allergy* and an *intolerance*, one soon realizes that this model will help explain how someone with GWS/MCS cannot tolerate a chemical like pesticides but not have conventional antibody mediated allergic responses. (10)

In August of 1998 Dr. Clement Furlong of the University of Washington was interviewed by Andrew Wineke of The Everett Herald in Washington State for an article about MCS.(11) Dr. Furlong explains how one person may be very sensitive to pesticides while others may be able to detoxify and tolerate pesticides. About the same time Dr. Robert Haley did Gulf War Syndrome research (12)(13) validating Dr. Furlong's hypothesis that some people with a low level of serum paraoxonase (PON) may be chemically injured from this enzyme deficiency.

PON is an enzyme that helps mammals (both humans and lab rats) detoxify organophosphates like sarin or Dursban. If one has a low level of this enzyme, then it will be difficult for the person with the lower level to detoxify the poison. Like lactose intolerance, this seems to be genetically predisposed. One may assume the chemical companies do not want the public to know that a portion of the population will not be able to effectively detoxify their products .

There are thousands of enzymes ... and the ability to metabolize using them is as variable as is the human race. The scientific researchers who produce "industry"

research do not usually control for the statistically deviant population who may have any particular low level of any given enzyme involved in the detoxification process. So, the data the American EPA, the FDA, and all the other supposed regulatory agencies receive from the researchers often NEVER look for any at risk population. Industry just creates products and then the marketplace becomes the lab to test their products.

When Vietnam vets tried to report illnesses from Agent Orange exposures, industries and the science community that benefitted from these products rejected this information. Eventually scientific research validated chemical injury from exposure to Agent Orange.

When Gulf War vets reported illnesses from exposures to toxic substances, again industries and the scientific community that benefit from production of products in their protocols rejected the complaints of the soldiers. Research by Robert Haley, M.D., and others has been out for several years indicating that soldier exposed to low levels of organophosphate pesticide and/or nerve gas agents like sarin were, in fact, intolerant of low level exposures. Researchers have known for years that not all people have the same level of tolerance to chemicals and that children may be especially at risk because their tolerances are lowest.

Unfortunately, those who benefit from the present scientific system are unwilling to accept negative feedback about the failures of the complete understanding in their system of synthetic product creations. Pesticides and many other synthetic creations of man were never a result of the natural order of things, or, a creation in God's design. Pesticides are, by manmade design, created to interfere with life. Because someone does not die immediately from low levels of these poisons does not mean that they are not harming humans. It is NOW accepted that Vietnam vets who were exposed to Agent Orange and developed diabetes and other illnesses developed them as a result of exposures to once thought supposedly safe poisons. (14)

In "*AIR FORCE STATEMENT ON RESULTS FROM THE 1997 PHYSICAL EXAMINATION OF THE RANCH HAND STUDY*" (15) Dr. Joel Michalek, senior principal investigator for the Air Force Health Study on Agent Orange, states, "While the Air Force Health Study indicates that adult-onset diabetes and cardiovascular disease seem most likely related to herbicide exposure, biological processes relating herbicide exposure with diabetes or cardiovascular disease have not been described, and until such relationships are found, these statistical findings may not reflect cause and effect." In other words, the research needs to be done.

It is not until Dr. Robert Haley did his serum paraoxonase research, that a likely relationship between enzymes that detoxify particular poisons (OP's) and diabetes and cardiovascular disease exists may be recognized. It is likely there is not much research on toxicant effects of pesticides on the hypochondrium organ systems (liver, pancreas, spleen, and stomach) because research for registration of pesticides and other toxicants regulated by regulatory agencies do not require research on those effects. Although Agent Orange was not made up of organophosphate pesticides, the similarities in the pattern of these disorders suggests there may be a common mechanism.

In the late 70s the marketplace discovered that some people with asthma were intolerant to sulfites used on salads to preserve them. Eventually it was recognized that people with an enzyme deficiency of *sulfite oxidase* (16) made them ill. When enough people reported illness from sulfites in salad bars, they took the preservative that was causing the problem out of salads and some other foods. The enzyme sulfite oxidase helps transform toxic sulfite into nontoxic sulfate.

The collective ignorance of our scientists to the biochemical uniqueness of the population they create products for continues. Modern man has created so many artificial products that never were a function or creation of the natural order of nature. Sarin did not exist until the Nazis created it in 1939. For thousands and thousands of years there were cultures that never ate or drank milk products. And, all these synthetic food colorings and food preservatives never existed until industry created them to help market and maintain the shelf lives of their products. It makes one wonder why anyone with an ounce of common sense would be shocked that there are many, many people unable to metabolize all these synthetic creations of man. It is time to recognize and understand the problem. Are we overloading our detoxification systems with all kinds of STUFF that never was designed by nature to be detoxified?

Are MCS and GWS disorders both manifestations of the yet universally recognized premise that there is VARIABILITY OF TOLERANCE of all chemicals because there is a variable level of enzymes within the human continuum involved in the detoxification and metabolizing process? As long as there is no universally accepted explanation for MCS/GWS, then products like pesticides that injure many people will continue to be made without seeking safer alternatives. Can thousands of reports of illness from toxic products like pesticides be hysteria and stress? Does that mean we should believe that all the critters that die from pesticide exposures are dying from a psychogenic illness? We think not!

At this point there are no commercially available tests to measure PON deficiency in humans. Several people with MCS have made contact with Dr. Clem Furlong at University of Washington in Seattle to have lab work done. Both these ladies were exposed to pesticides and became chemically sensitive. Both tested positive for low levels of PON enzyme comparable to those Gulf War vets who were tested by Dr. Robert Haley in his research on PON .

Barbara Rubin of New York State has been in contact with Dr. Clem Furlong who did her lab work because she could find no commercial lab to test for a PON deficiency. A case history written by her follows. We have also included a summary of a second case who had her PON Q assay done at the University of Washington.

Barb Rubin's History

Briefly, I was completely normal and healthy until the age of 21 when my first Dursban exposure to a fogger used in my apartment for fleas occurred. I developed a rash and GI effects which passed fairly quickly followed by recurrent upper respiratory infections which may or may not

have been associated with that exposure. Six years later, another landlord subjected me to exposures to a Dursban fogger (I was actually in the apartment briefly since I entered while it was running - no advance notice). I collapsed that night with GI pain, nausea, weakness and sweats. I do not recall if respiratory symptoms occurred. Doctors did not relate the exposure to the symptoms and diagnosed a viral illness. I was bedridden for the better part of three weeks and recovered very slowly. I was unable to tolerate all but two foods (turkey and sweet potatoes) and filtered water. I became symptomatic from a multisystem standpoint when exposed to dry cleaned clothes, newsprint, perfumes, pesticides, new construction, adhesives, paints, synthetic fabrics and a number of drugs. Reactions were occasionally "allergic" in type as for sulpha drugs, certain fruits etc. The majority of reactions were systemic but non-IGE mediated. Doctors were unable to find the cause but I found an allergist with EI experience in 1990 who was able to understand the constellation of symptoms. Treatment for hypothyroidism, candida and avoidance of triggers improved my health significantly and I lived and worked productively with accommodation plans.

In 1995, I changed jobs and began working in a midtown office building housing a private school. Within three years, I had chronic asthma and it was discovered to be a "sick building" with no fresh air entering the ventilation system. I was transferred and the asthma subsided into a reactive form of the illness.

In 1999, my accommodation plan requiring me to have advance notice of pesticide applications was violated. I was subjected to six months of pyrethroid exposures and developed severe neurological problems by the second month of exposure. Toxicological assessment of floor swipes revealed high levels of residues for cypermethrin and lambda-cyhalothrin. The MSDS sheets support the kinds of symptoms and adverse effects I exhibited. Brain damage was diagnosed in May of 2000 and my job duties had to be reduced due to impaired functioning. I was released from spending too many hours on site and eventually transferred.

The following September (2000), I had a severe OP exposure to dichlorvos foggers used below my apartment by new tenants. This lasted for three weeks until I collapsed and wound up in an ER with GI and neuro symptoms that had waxed and waned for the entirety of the exposure. Toxicological testing of my clothing and bedding showed the contamination and all my possessions had to be discarded. I became unable to work and was bedridden for most of the next several months. My hypothyroidism shifted to hyperthyroidism for a year. I have not worked since and been awarded Long Term Disability on the basis of poisoning (Toxic Effects of Chemicals, non-medicinal - 989.9). I was then awarded SSD on the basis of brain damage (amnesic syndrome) as my short term memory deficits affect my ability to work.

I have advanced MCS and so show some of the hallmark test patterns from

repeated pesticide poisonings. These include:

Deficient Paroxonase (PON 1): Likely the strongest causal factor for my MCS given my two early Dursban exposures (1979 and 1985) which precipitated the onset of chemical sensitivities and food intolerances.

Suppression of Acetylcholinesterase levels: Baseline level had been supranormal while post-dichlorvos results were clinically low and have remained at significantly low levels since that time.

Cortical atrophy as revealed by MRI scan (no contrast used).

Neuropsychological assessment: Loss of 24 IQ points with attendant deficits in working memory; cognition, language, sensori-motor functions. Neurotoxicity was considered the source of the damage (fairly rapid onset following pyrethroid exposures). Right-left asymmetries are present as well.

Periodic low blood sugar levels and vitamin deficiencies from poorly balanced diet. Foods high in sulphur, sugars, vitamin A and E are all poorly tolerated. No foods which have come in contact with pesticides or chemical/hormonal/antibiotic additives are tolerated. I am lactose and gluten intolerant. No food restrictions were present prior to my second Dursban exposure. Fatty acid analysis is very abnormal.

Chronically elevated SED rate.

Autoimmune findings of smooth muscle antibodies and fibromyalgia.

Positive ANA values for 10 months post dichlorvos poisoning. It shifted back to negative after that point and is periodically retested.

Reduced pulmonary function with reactive airway. Cannot tolerate asthma medications but acupuncture has had positive effects.

Severe multisystemic reactions to many synthetic products. Forced to live in my car frequently during 2001 due to inability to tolerate chemicals used on apartment premises and lack of suitable hotel accommodations.

Needed workups at this point: Cardiac, ophthalmology, continuing neurological monitoring.

These are the major issues in my history. Please let me know if you have any further questions or comments. You may pass this on as you deem appropriate for educational purposes.

Barbara Rubin

S's Case Summary - Tests positive for low levels of PON Q.

S is the college educated wife of an executive with an aerospace company in the South. She is.

chemically sensitive. In 1986 a lawnspray company accidentally sprayed her .She suffered cholinesterase inhibition.

In 1998 She was re-exposed to Dursban and Diazanon. She had alerted her neighbors prior to buying her home that she was chemically sensitive ALL neighbors agreed not to spray. One neighbor sprayed 22 times in 1998. Another sprayed Dursban twice. She became completely disabled upon re-exposure. A serum paraoxonase assay from the University of Washington showed that her PON-1 levels rank in the lowest group and that her reaction to Dursban would have probably been predictable. Four Gulf-War researchers have tested her. Although she did not serve in the Gulf War, she has many similarities to those individuals exposed to Dursban.

Conclusion

A PON deficiency may not be a definitive biomarker for either MCS or GWS. It points to the direction that research must go (the crack of light) to find out the causes of these disorders. Those opposing recognition of chemical injury or those looking for the definitive biomarker will correctly point out that a deficiency of ONE enzyme involved in detoxification of organophosphates in many enzymes involved in detoxification will not be deficient in ALL people with MCS/GWS. Without educating the people who can bring about recognition of the problem, MCS and GWS will continue to be held hostage by the old "psychogenic" canard used by some industry advocates to prevent recognition of the problem. We need research that will bring complete understanding of the problem so that we can create safe alternative products to the ones that are injuring so many. We also need those entrusted with the regulatory responsibilities related to toxic chemicals like pesticides to take a precautionary approach so that not one man, woman or child is injured from pesticide sprays and other toxic synthetics.

* More on Chemical Intolerance can be found on <http://wsmcsn.s5.com/hubpage.htm>

REFERENCES & NOTES

1. Kreutzer R, Neutra RR, Lashuay N. Prevalence of people reporting sensitivities to chemicals in a population-based survey. *Am J Epidemiol.* 1999;150:1-12. At <http://aje.oupjournals.org/cgi/content/abstract/150/1/1?maxtoshow=&HITS=10&hits>

[=10&RESULTFORMAT=&author1=Kreutzer%2C+R&titleabstract=Prevalence+of+People+Reporting+Sensitivities+to+Chemicals&searchid=QID_NOT_SET&stored_search=&FIRSTINDEX=&journalcode=amje](#)

2..The Wall of Personal Testimony at <http://www.herc.org/wall/>

3. Multiple Chemical Sensitivity Under Siege by Ann MacCampbell, M.D., Townsend Letter for Doctors & Patients, January 2001, #210, p. 20 - 27 at <http://www.getipm.com/personal/mcs-campbell.htm> in which she writes. "When confronted by the harm they have caused, corporations typically blame the victims, deny the problem, and try to avoid responsibility for the harm caused. The corporate response to MCS has been no different."

4. THE CHEMICAL MANUFACTURERS ASSOCIATION'S ENVIRONMENTAL ILLNESS BRIEFING PAPER, 1990 , in which they state: "Because it has the potential to impact many segments of society, many groups have an interest in placing environmental illness in its proper perspective. ... Because environmental illness is a health issue, the only people who can legitimize it are physicians, and they have not. Should environmental illness arise as an issue, a coalition with the state medical association is absolutely necessary. " .

<http://users.lanminds.com/~wilworks/books/cmaeibri.htm>

[In Washington State, a doctor of Environmental and Occupational Medicine, writes anti-MCS peer reviewed literature, does Independent Medical Exams (IME's) for the WA State Labor and Industry and other insurance claims, does medical post graduate education inservice on the issue of MCS presenting the industry line, was (and may still be) on the WASHINGTON STATE MEDICAL ASSOCIATION committee that advises Labor & Industry about such issues as chemical injury, testifies in court as an expert witness against people in chemical injury cases, and is on industry contact lists as expert media consultants on issues of things like pesticides (eg. Durban being banned ... the pesticide industry used her as the expert for the media to contact.).... and so on. dp]

5. Scientific American: In Focus: Personal Pills: October 1998 at <http://www.sciam.com/1998/1098issue/1098infocus.html>

6. Genetic Testing Checks For Drug Reactions
<http://komotv.com/healthwatch/story.asp?ID=10875>

7. Drug Reaction Testing at <http://www.healthanddna.com/drugreactiontest.html>

8. Cytochrome P450 enzymes: Introduction (Psychotropic Research) at <http://www.psychotropic.com/notes/316.html> "There is great genetic variation in the activity level of most cytochrome P450 enzymes both between persons (and genetic groups)." and "The incidence of serious and fatal adverse drug reactions is high in hospital patients. This causes an estimated 100,000 deaths per year in the US, making it the 5th most frequent cause of death. Genotyping for cytochrome P450 enzymes may avoid as many as 20% of these deaths."

9. Pacific Food Got distress? Maybe you're lactose intolerant By Lynn Jacobson
Seattle Times staff reporter, Wednesday, May 16, 2001
http://seattletimes.nwsourc.com/html/lifestyles/134295679_milk16.html

10. Enzyme key to reaction, scientists say By Andrew Wineke, Everett Herald,
Tuesday, August 4, 1998, p C1 & C2 <http://wsmcsn.s5.com/Enzyme.htm>

11 . PON Q Research at <http://www3.ncbi.nlm.nih.gov/htbin-post/Omim/dispim?168820>

[A good review of the research on PON Q]

12. "A review of the scientific literature as it pertains to Gulf War Illness" at
<http://www.rand.org/publications/MR/MR1018.2>

13 UT Southwestern researcher finds genetic cause for Gulf War syndrome at
http://irweb.swmed.edu/newspub/newsdetl.asp?story_id=144 AND .Research on Gulf War- Associated Neurologic Illness , by the Division of Epidemiology, UT Southwestern Medical Center, Robert W. Haley, M.D.

14. Agent Orange Air Force Study at
<http://www.brooks.af.mil/AFRL/HED/hedb/afhs/afhs.shtml>

15. Study: Agent Orange, Diabetes Link, *By ROBERT BURNS, AP Military Writer*
,YahooNews, March 29, 200

"The study found a 47 percent increase in diabetes among veterans with the highest levels of dioxin in their bloodstream. Dioxin is the compound in Agent Orange linked to health effects in laboratory animals. The result is based on 1997 physical examinations of 1,000 Air Force veterans who were exposed to Agent Orange during the nine years that it was used as a defoliant and crop killer in Vietnam. " AND "The study found no consistent evidence that Agent Orange is related to cancer." [On the Net: An executive summary of the Air Force report is available at <http://www.brooks.af.mil/AFRL/HED/hedb/afhs/afhs.shtml>]

16. AIR FORCE STATEMENT ON RESULTS FROM THE 1997 PHYSICAL EXAMINATION OF THE RANCH HAND STUDY Dr. Joel Michalek, senior principal investigator_for the Air Force Health Study on Agent Orange, <http://www.brooks.af.mil/AFRL/HED/hedb/afhs/pressconfstmt.html> " **At the end of 15 years of follow-up, we have still found no consistent evidence in the Ranch Hand population that dioxin exposure is related to cancer "**. AND **"A 47 percent increase in diabetes was seen in those with the highest levels of dioxin. This is particularly strong evidence, since dioxin is the component of Agent Orange linked to many health effects in laboratory animals."** AND **"While the Air Force Health Study indicates that adult-onset diabetes and cardiovascular disease seem most likely related to herbicide exposure, biological processes relating herbicide exposure with diabetes or cardiovascular disease have not been described, and until such relationships are found, these statistical findings may not reflect cause and effect."** [How much research into the effects of cardiovascular disease and diabetes from pesticides does The EPA require????? dp]

17. Sulfite Oxidase Deficiency at
<http://www.digitalnaturopath.com/cond/C488289.html>

18. Chemical Sensitivity Due to Genetic Differences at
<http://ace.orst.edu/info/extoxnet/faqs/senspop/genetic.htm>

More notes:

Variability of TOLERANCE note: Benzene Risk Assessment: Transgenic Animals and Human Variability , CIIT Activities (Vol.17, No.7), *Chemical Industry Institute of Toxicology (CIIT)*, 1997 at
<http://www.ciit.org/ACT97/ACTIVITIESJUL97/jul97.html>

Benzene, a ubiquitous environmental chemical and a human leukemogen, has been the subject of active research at the Chemical Industry Institute of Toxicology (CIIT) for a number of years. The results of recent studies conducted at CIIT using genetically engineered animals indicate that CYP2E1 is the primary isozyme responsible for benzene metabolism in vivo. These studies have also demonstrated that metabolic activation of benzene is required for the development of both cytotoxicity and genotoxicity following benzene exposure. These observations are important because humans vary in their expression of CYP2E1 activity and, as we have demonstrated at CIIT, in their ability to metabolically activate benzene. In vitro rates of benzene metabolism correlate with measured CYP2E1 activity. However, assessing the risk due to exposure to benzene is more complicated than simply measuring CYP2E1 activity because detoxication rates also vary across humans. Thus the balance between activation of benzene to its toxic metabolites and detoxification of these metabolites ultimately determines potential risk of humans exposed to benzene

AND Variability in Human Metabolism of Benzene

The role of CYP2E1 in benzene metabolism in humans was investigated using subcellular fractions from 10 human livers in which CYP2E1 activity was found to vary 14-fold. Results were compared with data obtained in rats and mice (Figure 6). The variation seen in human samples is high compared with that seen in mice and rats, but this is not surprising. The rats and mice used in these studies tend to be genetically similar and are maintained on identical diets, whereas humans are a much more heterogeneous population and eat a much more varied diet. Both genetic variation and diet contribute to large variations in CYP2E1 activity. Benzene metabolism correlated with measured CYP2E1 activity across species and individuals. As the measured CYP2E1 activity increased, the percentage of benzene metabolized increased. A mathematical model was developed in which CYP2E1 activity was the only difference between individuals or species (Seaton et al., 1994)

Journal of Pesticide Reform v.108, n.3 Fall98 rev.Oct00 14. Hietanen, E., K. Linnainmaa, and H. Vainio. 1983. Effects of phenoxy herbicides and glyphosate

on the hepatic and intestinal biotransformation activities in the rat. *Acta Pharma. et Toxicol.* 53:103-112.

""Comments on Paraoxonase and MCS" [Letter, accepted May 1999, published August 1999 *Environmental Health Perspectives*] S.C. Rowat ©©1999

Second Draft, June 22, 1999 at

PON Q Note:

Obscure Enzyme May Play Major Role In Heart Disease, NEW YORK, NY and HAIFA, ISRAEL -- April 15, 1998, P\S\L Consulting Group Inc, at <http://pslgroup.com/dg/6c926.htm>

"The real function of the enzyme has been something of a mystery since it was discovered more than 40 years ago. Its previously known function was to break down organophosphates, chemicals that are used as insecticides and poison gases. That activity was obviously not the complete story of paraoxonase, as humans do not normally contain these substances in their blood, Aviram explained" .

American Society for Technion-Israel Institute of Technology, press release, April 14, 1999, Copyright ©1996 - 1999 Center for Cardiovascular Education, Inc., New Providence, NJ USA.

"Paraoxonase is located in the blood on the HDL, the 'good' cholesterol, and it can break down oxidized LDL to non-harmful products," explains Aviram, adding that the discovery of this enzyme's activity opens a possible new route to prevention of heart diseases. "

Aviram, M. " Paraoxonase Inhibits High-density Lipoprotein Oxidation and Preserves its Functions. A Possible Peroxidative

Role for Paraoxonase," *Journal of Clinical Investigation*, Volume 101, Number 8, 1581-1590;

"Diabetics, heart attack patients, and people with very high cholesterol often have low concentrations of PON in their blood, other studies have shown. They

may be the first to benefit from efforts, now underway, to find a compound that helps stimulate PON in humans, says Aviram" [at SCIENCE REPORTER -]

**[YES, THERE IS A RELATIONSHIP BETWEEN DIABETES,
CARDIOVASCULAR DISEASE AND DETOXIFICATION OF
PESTICIDES!]**

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