



An international study has linked an industrial solvent to Parkinson's disease. Researchers found a six-fold increase in the risk of developing Parkinson's in individuals exposed in the workplace to **trichloroethylene (TCE)**. Although many uses for TCE have been banned around the world, the chemical is still used as a degreasing agent. While the exact cause of the disease is still unknown, research to date suggests a mix of genetic and environmental factors, and a link has previously been made with pesticide use.

—Neil Bowdler, Health Reporter,  
BBC News, WakeUp-World.com

# Is Parkinson's Disease

Dr. Hans Conser, DC

## Caused by Impaired Detoxification?

New studies indicate that if you are trying to hold off Parkinson's disease, you might want to take a hard look at science-based detoxification. Medical doctors rarely consider detoxification in relation to Parkinson's; however, this may change very soon.

It may be worth altering your diet and supplementation strategy a bit if you are concerned about Parkinson's, and let me tell you why.

First, I define Parkinson's disease. Then I delve into the new scientific case for the toxicity model for the cause of Parkinson's disease. I discuss the biphasic detoxification model, endogenous and exogenous toxins, and I close by offering specific recommendations for preventing Parkinson's disease.

### What is Parkinson's Disease?

Parkinson disease (PD) is a progressive and irreversible degenerative neurological disease characterized by a subtle onset of symptoms of flat facial affect, slowed movement, speech changes, tremor, and in the later stages, dementia. It afflicts about 1% of the population over 60, and about 4% of the population over 80.

### Searching for the Cause of PD

The search for the cause of PD has been focused on two main cell abnormalities found in dopamine

producing cells of PD sufferers. These are impaired mitochondrial function, and protein clumps called Lewy Bodies.

However, there is a deeper mechanism at play that may be the underlying cause of both the protein clumping and mitochondrial dysfunction associated with PD, and that something is called endogenous toxicity.

### Endogenous Toxicity

Endogenous toxicity means toxicity of an internal origin. In this case, the theory is that the Phase I metabolite of Dopamine itself is causing the toxicity, dysfunction, and death of the dopamine producing neurons. Dopamine is broken down into a toxic metabolite called DOPAL. DOPAL is then broken down to non-toxic metabolites by the Phase II enzyme Aldehyde Dehydrogenase (ALDH for short). These two phases together are known as "biphasic detoxification."

It appears that ALDH activity is deficient in the brains of PD patients, leading to DOPAL toxicity in the dopamine producing neurons. Basically, PD patients have impaired Phase II detoxification, leading to a buildup of the toxic Phase I dopamine metabolite, DOPAL. DOPAL toxicity leads to cell aberrations like protein clumping, mitochondrial dysfunction and eventually cell death. Mitochondria are the powerhouse organelles of the cell.

The reduced mitochondrial function in PD is why nutritional ingredients like CoEnzyme Q10 and Creatine are being studied for their possible positive effect on PD.

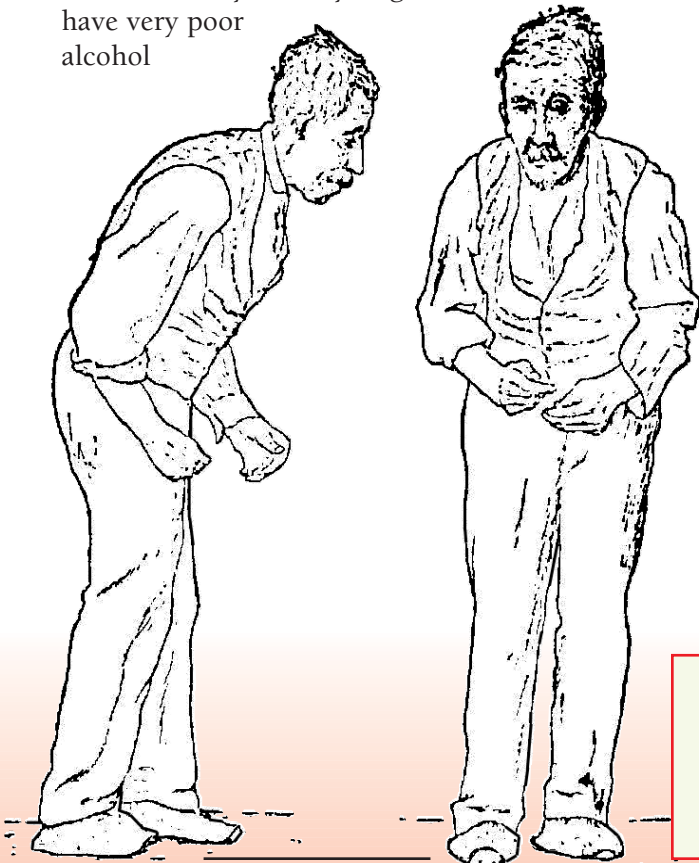
### Scientific Evidence for the DOPAL Toxicity in Parkinson's

There is a wealth of scientific evidence incriminating DOPAL. DOPAL has been shown to increase the protein clumps in the brains of rats.<sup>1</sup> DOPAL has been shown to be toxic to dopamine producing neurons.<sup>2-5</sup> DOPAL injected into the brains of rats creates PD-like symptoms.<sup>5</sup>

Researchers have shown that the brains of Parkinson's patients have over 4 times the DOPAL levels of non-Parkinson's brains.<sup>6</sup> N-Acetyl-Cysteine, which increases ALDH activity, has been shown to stop toxicity-induced death of dopamine producing neurons in mice.<sup>7,8</sup>

### The Hangover Chemical

Aldehyde conjugate molecules are common byproducts of the first stage (Phase I) of detoxification of various toxins like alcohol, nicotine, and many others. Acetyl-Aldehyde, the toxic Aldehyde conjugate of alcohol, is known as the "hangover chemical." People who have a less effective mutated form of Aldehyde Dehydrogenase have very poor alcohol



tolerance, and increased incidence of PD and coronary heart disease.<sup>9,10</sup>

If you get hangovers from even moderate amounts of alcohol, this is a warning sign that your Phase II detoxification may be impaired.

### Exogenous Toxic Load Creating Endogenous Toxicity

ALDH requires "thiol" groups to detoxify Aldehyde molecules. Thiols are sulfur-bearing molecules that are supplied by amino acids like Cysteine, Methionine, and Taurine. If you run out, then your Aldehyde detoxification is impaired. It seems very likely that exogenous toxin exposures like herbicides, pesticides, solvents, and metals may use up your sulfur-bearing amino-acids and then leave you vulnerable to damaging your dopamine producing neurons with toxic DOPAL build-up.

### Factors that Decrease Aldehyde Detoxification

ALDH activity is decreased by the following factors: Fasting, a low-protein diet (less than 5%), Vitamin A deficiency, and excess isothiocyanate intake.<sup>11-15</sup> Isothiocyanates are toxic molecules found in the cruciferous vegetable family (broccoli, cabbage, etc.), actually healthy in small amounts, but unhealthy in large amounts. I generally recommend that crucifers make up 20% or less of your vegetable intake. Another natural component of food that inhibits ALDH activity is Citral, a citrus-smelling compound found in lemons, limes, oranges, and lemongrass, among others. This is one reason why I recommend people showing signs of impaired detoxification stop citrus fruit intake completely. Also, there is long list of exogenous agricultural toxins that inhibit ALDH activity, they include pesticides, herbicides, growth modification agents, and fungicides.<sup>16-18</sup>

### Factors that Increase Aldehyde Detoxification

These factors include Vitamin A, Vitamin B3 (Niacin), Methionine, Cysteine, N-Acetyl-Cysteine, Taurine, and Magnesium. An odd one is Vitamin E deficiency. Perhaps this is a good argument for not taking high levels of Vitamin E.

**Sir William Richard Gowers, MD**, neurologist, researcher and artist, drew this illustration in 1886, as part of his documentation of Parkinson's disease. The image appeared in his book, *A Manual of Diseases of the Nervous System*, and is still used today by medical professionals as a primary reference for this disease.

## How to Prevent Parkinson's Disease

Do everything you can to fight both internal and external toxicity. Limit your exposure to agricultural industry toxins. Eat plenty of protein, vegetables and legumes. Don't eat excessive amounts of cruciferous vegetables or citrus fruits. Supplement your diet with broad-spectrum of detox-enhancing nutrients that emphasize Phase II support. Take fish oil. Get plenty of Vitamin D from sunlight. If you want to fast, be sure to support it with a supplement that has plenty of detox nutrition. Some of my favorite supplements for this are Detox I & II, UltraClear Plus, and concentrated green foods. ■

### NOTES:

1. Acta Neuropathol. 2008 Feb;115(2):193-203. Aggregation of alpha-synuclein by DOPAL, the monoamine oxidase metabolite of dopamine.
2. Brain Res. 2003 Nov 7;989(2):205-13. 3,4-Dihydroxyphenylacetaldehyde is the toxic dopamine metabolite in vivo: implications for Parkinson's disease pathogenesis.
3. Brain Res Mol Brain Res. 2001 Sep 10;93(1):1-7. 3,4-Dihydroxyphenylacetaldehyde and hydrogen peroxide generate a hydroxyl radical: possible role in Parkinson's disease pathogenesis.
4. Neurodegeneration. 1995 Sep;4(3):271-81. An endogenous dopaminergic neurotoxin: implication for Parkinson's disease. Mattammal MB, Haring JH, Chung HD, Raghu G, Strong R.
5. PLoS One. 2010 Dec 13;5(12):e15251. The neurotoxicity of DOPAL: behavioral and stereological evidence for its role in Parkinson disease pathogenesis.
6. Eur J Neurol. 2011 May;18(5):703-10. doi: 10.1111/j.1468-1331.2010.03246.x. Epub 2010. Nov 12. Catechols in post-mortem brain of patients with Parkinson disease.
7. Ann Neurol. 2011 Mar;69(3):509-20. doi: 10.1002/ana.22162. N-acetylcysteine prevents loss of dopaminergic neurons in the EAAC1<sup>-/-</sup> mouse.
8. PLoS One. 2010 Aug 23;5(8):e12333. Oral N-acetylcysteine attenuates loss of dopaminergic terminals in alpha-synuclein overexpressing mice.
9. Clin Chim Acta. 2007 Jul;382(1-2):43-7. A Glu487Lys polymorphism in the gene for mitochondrial aldehyde dehydrogenase 2 is associated with myocardial infarction in elderly Korean men.
10. Mov Disord. 2000 Sep;15(5):813-8. Alcohol dehydrogenase alleles in Parkinson's disease.
11. Arch Oral Biol. 2008 May;53(5):423-8. Epub 2007 Dec 21. Salivary aldehyde dehydrogenase--reversible oxidation of the enzyme and its inhibition by caffeine, investigated using fluorimetric method.
12. Acta Pharmacol Toxicol (Copenh). 1977 Jan;40(1):134-44. Effect of a low-protein diet on acetaldehyde metabolism in rats.
13. Biochem Pharmacol. 1979 Aug 1;28(15):2313-20. Enzymatic and metabolic modification of hepatic ethanol and acetaldehyde oxidation by the dietary protein level.
14. J Nutr Sci Vitaminol (Tokyo). 1994 Dec;40(6):547-55. Comparative study of alcohol metabolism in stroke-prone spontaneously hypertensive rats and Wistar-Kyoto rats fed normal or low levels of dietary protein.
15. J Pharmacol Exp Ther. 1995 Oct;275(1):79-83. Phenethyl isothiocyanate, a new dietary liver aldehyde dehydrogenase inhibitor.
16. Chem Res Toxicol. 2010 Nov 15;23(11):1843-50. Epub 2010 Oct 18. Relative inhibitory potency of molinate and metabolites with aldehyde dehydrogenase 2: implications for the mechanism of enzyme inhibition.
17. Neurotoxicology. 2007 Jan;28(1):143-9. Epub 2006 Sep 1. Inhibition of aldehyde detoxification in CNS mitochondria by fungicides.
18. Mol Pharmacol. 2004 Dec;66(6):1372-82. Epub 2004 Aug 26. Oxidative stress and mitochondrial aldehyde dehydrogenase activity: a comparison of pentaerythritol tetranitrate with other organic nitrates.
19. Chem Res Toxicol. 2004 Feb;17(2):258-67. Characterization of S-(N,N-Dialkylaminocarbonyl)cysteine Adducts and Enzyme Inhibition Produced by Thiocarbamate Herbicides in the Rat.
20. Life Sci. 1994;55(20):1537-44. Aldehyde dehydrogenase of mice inhibited by thiocarbamate herbicides.



*Dr. Hans Conser, DC, Bozeman's BodyMind Chiropractor, offers Network Chiropractic and science-based, individualized supplementation, weight-loss and detoxification programs at his office, Amazing Touch Chiropractic, in downtown Bozeman. Dr. Hans can be reached at 595-1928. Schedule appointments online at [www.BozemanChiropractic.com](http://www.BozemanChiropractic.com).*