# Doctor In Italy Astonishingly Quells Parkinson's Disease With Overlooked Vitamin Cure (And The World Pays No Attention)

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In recent weeks the World has learned the news media creates fake news and/or completely shuns significant news stories to match its own politically correct agendas. So an unequivocal cure for a major brain disease goes unreported. Shame on CNN, CBS, ABC, NBC, the BBC, Reuters, Associated Press, *Washington Post*, and the *New York Times.*

For a disease considered incurable, a physician in Italy has begun to provide a common B vitamin to successfully treat a debilitating motor-nerve disease commonly known as Parkinson's disease. The importance of this startling discovery has escaped major news outlets. It should be heralded on television and in newspapers worldwide. But it has only been reported by an obscure European news source.[i]

## History of Vitamin B1 and Parkinson's

In 1817, physician James Parkinson first described a "shaking palsy."[ii] Today, 200 years later, Parkinson's disease is still considered an incurable disease.

Parkinson's disease emanates from the loss of dopamine-producing cells in the brain. Approximately 60-80% of dopamine-producing cells are damaged before symptoms arise. Dopamine is a nerve-transmitting chemical in the brain.

It has taken two centuries for a vitamin-phobic medical profession to hesitantly begin to treat Parkinson's disease with Vitamin B1 (thiamin). Historically, the link between Parkinson's disease and thiamin deficiency has been agonizingly slow to develop.

Thiamin, or Vitamin B1, was the first vitamin to be discovered. Vitamin B1 was first synthesized in 1936.[iii] So, dietary supplementation was possible from that point forward.

It took until 1967 for the first published report to appear showing that a decline in brain dopamine levels of pigeons was due to experimentally induced thiamin deficiency.[iv]

A link between thiamin deficiency and low dopamine levels was discussed in 1987 in an experiment that attempted to determine why rats tend to eat mice (muricide). Low dopamine levels induced by a shortage of thiamin in the diet were linked to this abnormal animal behavior.[v]

In 1988, researchers noted a thiamin-deficient diet decreased dopamine concentration and synthesis in the brain (striatum). The provision of alcohol to lab animals also decreased dopamine levels. The brain region most susceptible to damage (the hypothalamus) in thiamin-deficient animals is the very same region of the brain that produces dopamine.[vi]

In 1999, it was observed that low levels of thiamin in the cerebrospinal fluid were related to Parkinson's disease.[vii] In 2013, researchers reviewed all prior, published scientific reports and concluded that thiamin plays a role in Parkinson's disease.[viii]

## First Therapeutic Trials Report

In 2013, the first reports were published demonstrating the use of high-dose thiamin among Parkinson's disease sufferers resulted in considerable improvement in measured motor function (31.3% to 77.3%). Injection of high-dose thiamin was effective in reversing symptoms.[ix]

Then, another study published in 2015 confirmed that injectable thiamin treatment (100 mg twice a week) improves motor-nerve function among Parkinson's patients.[x]

In 2016, researchers in Italy reported on the successful use of high-dose thiamin among Parkinson's patients. Notably, all of the patients had normal blood levels of thiamin yet thiamin therapy led to significant improvement in Parkinson's symptoms. There were no adverse events.[xi]

## Loss of Sense of Smell Is Earliest Sign

Finally, in January of 2017, researchers noted that the sense of smell declines years prior to the onset of Parkinson's disease. Scented strips were used to test scent among individuals with Parkinson's disease. Almost half of the individuals tested (47%) scored low on this test and dietary thiamin was also low in these subjects.

The main finding of the study was an association between low thiamin in the diet reported 2-8 years prior to the onset of symptoms and diagnosis. Impairment of olfaction (sense of smell) is a characteristic and early feature of Parkinson's disease, these researchers wrote. Involvement of many B- family vitamins appears to be involved in the decline of smell. Therefore, a decline in the sense of smell may serve as an early screening tool for Parkinson's disease.[xii]

Because of a change in Western diets, Dr. Derrick Lonsdale, the reigning clinical authority on Vitamin B1, says the high sugar/carbohydrate diet of today results in a

return of the scourge of beri beri. The problem is, modern medicine observes symptoms of this nutrient deficiency disease and treats those instead of its cause.

Western populations are paying a high price for poor absorption or depletion of thiamin due to consumption of alcohol, drugs like diuretics (water pills), refined sugar, carbohydrates, and even coffee and tea.[xiii] The ordeal of undetected thiamin deficiency is only exacerbated by the modern paradigm of treating symptoms of disease as if they emanate from the drug deficiency, not a nutrient deficiency.

So, we now have 60,000 Americans diagnosed with Parkinson's disease annually and 10 million worldwide living with the disease. More than 23,000 individuals die of Parkinson's disease annually in the United States. And Levodopa, the main drug prescribed for Parkinson's disease, costs approximately $2500 per year.[xiv]

As of 2014, there were 23 medicines under development for Parkinson's disease. Drugs only serve as a distraction for a disease that may emanate from a vitamin deficiency.

## Alzheimer's Too

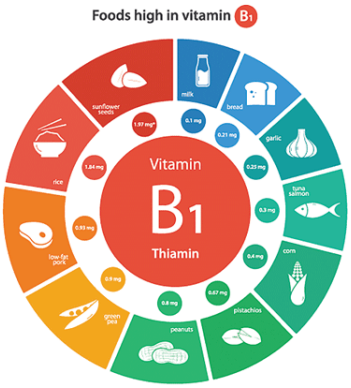
Thiamin deficiency is associated with other age-related brain diseases such as Alzheimer's and Huntington's disease.[xv] This author penned an earlier report linking Alzheimer's disease to thiamin deficiency.[xvi]

The earliest sign of Alzheimer's disease is also a decline in the sense of smell.[xvii] That was first reported in 1974.[xviii]

The startling problem with Alzheimer's disease is that there is no proven cure for this brain disease either and only 1 in 244 drugs undergoing trials for this brain disease have achieved FDA approval.

Accumulation of beta amyloid plaque in the brain begins up to 20 years before symptoms occur. With so many Americans now living into their eighth, ninth, and tenth decade of life and 44% of age 75-84 adults affected, it is too late to conduct a long-term controlled study to head off this anticipated Alzheimer's plague. We are going to have close to half of the senior adult population living with Alzheimer's memory loss and the other half of that population taking care of them!

## Rx: B Vitamins



The provision of the entire family of supplemental B vitamins, in particular thiamin (B1), pyridoxine (B6), folic acid (B9), and cobalamin (B12) appears to be important in the theoretical model of these diseases. Disturbed sense of smell has been reversed with Vitamin-B12 supplementation.[xix]

The best way to achieve this proposed prophylaxis is either with a B-vitamin complex or a multivitamin. Unfortunately, in most instances the provision of B vitamins in these formulas is marginal, often not exceeding the outdated Daily Value or the Recommended Daily Allowance.

A progressive fall-off in the absorption of these B vitamins due to a decline in secretion of stomach acid with advancing age, or in the case of thiamin the blockage of absorption by drugs, antacids, alcohol, sugar and carbohydrates and even presumably healthy beverages like tea and coffee, serve as sufficient evidence that doses of B vitamins need to be updated in most multivitamins if a universal attempt is to be made to head off Parkinson's and Alzheimer's disease epidemic.

Because of poor absorption of water-soluble forms of B1, Vitamin B1 in fat-soluble form (Benfotiamine) is the preferred form in dietary supplements but is generally not provided in B-complex or multivitamins.[xx] Fat-soluble benfotiamine is almost six times more biologically available than water-soluble thiamin hydrochloride, the common form used in dietary supplements.[xxi]

Furthermore, gut bacteria has now been shown to regulate movement disorders like Parkinson's disease in laboratory mice and represents a risk factor.[xxii] Ditto for Alzheimer's disease.[xxiii] Multivitamins need to incorporate ingredients that promote healthy gut bacteria.

As for the doctor in Italy who has reported on the successful use of thiamin/Vitamin B1 for Parkinson's disease symptoms, we can only say *Bravo* and wonder when the Nobel Prize committee will take notice.

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[i] "Videos Parkinson's Patients before and after treatment," *Ultima Edizione.eu*, at http://www.ultimaedizione.eu/videos-parkinsons-patients-treatment/. (Be sure to click the before-and-after video tabs.)

[ii] Christopher G. Goetz, "The History of Parkinson's Disease: Early Clinical Descriptions and Neurological Therapies," *Cold Spring Harb Perspect Med*, 2011 Sep; 1(1): a008862.

doi: 10.1101/cshperspect.a008862,
at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3234454/.

[iii] Heinz M. Wuest, "The History of Thiamine," *Annals of the NY Academy of Sciences*, DOI: 10.1111/j.1749-6632.1962.tb30561.x (April 1962), at THIS LINK.

[iv] Linét O, Widhalm S, Meriting G, “The influence of experimental thiamine-avitaminosis on cathecholamine levels in hearts and brains of pigeons,” *Int’l Journal of Neuropharmacology*, Vol. 6, No. 4, pp. 337-339 (July 1967).

[v] Abe Y, Tadano T, Yonezawa A, Kisara K, “Suppressive effects of intraventricular injected dopamine and nomifensine on muricide induced by thiamine deficiency,” *Pharmacol Biochem Behav*, 1987 Jan;26(1):77-81.

[vi] Sjöquist B, Johnson HA, Neri A, Lindén S, “The influence of thiamine deficiency and ethanol on rat brain catecholamines,” *Drug Alcohol Depend*, 1988 Dec;22(3):187-93, at https://www.ncbi.nlm.nih.gov/pubmed/3234241.

[vii] Jiménez-Jiménez FJ, Molina JA, Hernánz A, *et al.*, “Cerebrospinal fluid levels of thiamine in patients with Parkinson's disease,” *Neurosci Lett*, 1999 Aug 13;271(1):33-6, at https://www.ncbi.nlm.nih.gov/pubmed/10471207.

[viii] Luong KV, Nguyễn LT, “The beneficial role of thiamine in Parkinson disease,” *CNS Neurosci Ther*, 2013 Jul;19(7):461-8. doi: 10.1111/cns.12078. Epub 2013 Mar 6, at https://www.ncbi.nlm.nih.gov/pubmed/23462281.

[ix] Costantini A, Pala MI, Compagnoni L, Colangeli M, “High-dose thiamine as initial treatment for Parkinson's disease,” *BMJ Case Rep*, 2013 Aug 28;2013. pii: bcr2013009289. doi: 10.1136/bcr-2013-009289, at https://www.ncbi.nlm.nih.gov/pubmed/23986125.

[x] Costantini A, Pala MI, Grossi E, *et al.*, “Long-Term Treatment with High-Dose Thiamine in Parkinson Disease: An Open-Label Pilot Study,” *J Altern Complement Med*, 2015 Dec;21(12):740-7. doi: 10.1089/acm.2014.0353. Epub 2015 Oct 27, at https://www.ncbi.nlm.nih.gov/pubmed/26505466.

[xi] Costantini A, Fancellu R, “An open-label pilot study with high-dose thiamine in Parkinson's disease,” *Neural Regen Res*, 2016 Mar;11(3):406-7. doi: 10.4103/1673-5374.179047, at https://www.ncbi.nlm.nih.gov/pubmed/27127471.

[xii] Håglin L, Johansson I, Forsgren L, Bäckman L, “Intake of vitamin B before onset of Parkinson's disease and atypical parkinsonism and olfactory function at the time of diagnosis,” *Eur J Clin Nutr*, 2017 Jan;71(1):97-102. doi: 10.1038/ejcn.2016.181. Epub 2016 Oct 5, at https://www.ncbi.nlm.nih.gov/pubmed/27703161.

[xiii] Derrick Lonsdale, “A Review of the Biochemistry, Metabolism and Clinical Benefits of Thiamin(e) and Its Derivatives,” *Evid Based Complement Alternat Med*, 2006 Mar; 3(1): 49-59.

Published online 2006 Feb 1. doi: 10.1093/ecam/nek009, at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1375232/.

[xiv] “Statistics on Parkinson’s,” Parkinson’s Disease Foundation, at http://www.pdf.org/en/parkinson_statistics.

[xv] Richard L. Doty, "Smell and the Degenerating Brain," *The Scientist*, October 1, 2013, at http://www.the-scientist.com/?articles.view/articleNo/37603/title/Smell-and-the-Degenerating-Brain/.

[xvi] Bill Sardi, "Commentary: The B-Vitamin Alzheimer's Cure," *Knowledge of Health*, July 8, 2013, at http://knowledgeofhealth.com/commentary-the-b-vitamin-alzheimers-cure/.

[xvii] Yong-ming Zou, Da Lu, *et al.*, "Olfactory dysfunction in Alzheimer's disease," *Neuropsychiatr Dis Treat*, 2016; 12: 869-875; Published online 2016 Apr 15. doi: 10.2147/NDT.S104886, at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4841431/.

[xviii] Waldton S, "Clinical observations of impaired cranial nerve function in senile dementia," *Acta Psychiatr Scand*, 1974;50(5):539-47, at https://www.ncbi.nlm.nih.gov/pubmed/4460686.

[xix] Mundt B, Krakowsky G, Röder H, Werner E, "Loss of smell and taste within the scope of vitamin B 12 deficiency," *Psychiatr Neurol Med Psychol (Leipz)*, 1987 Jun;39(6):356-61 (in German), at https://www.ncbi.nlm.nih.gov/pubmed/3659193.

[xx] Park WS, Lee J, Hong T*, et al.*, "Comparative Pharmacokinetic Analysis of Thiamine and Its Phosphorylated Metabolites Administered as Multivitamin Preparations," *Clin Ther*, 2016 Oct;38(10):2277-2285. doi: 10.1016/j.clinthera.2016.08.009; Epub 2016 Oct 1, at https://www.ncbi.nlm.nih.gov/pubmed/27707509; Greb A, Bitsch R, "Comparative bioavailability of various thiamine derivatives after oral administration," *Int J Clin Pharmacol Ther*, 1998 Apr;36(4):216-21, at https://www.ncbi.nlm.nih.gov/pubmed/9587048.

[xxi] Xie F, Cheng Z, Li S, *et al.*, "Pharmacokinetic study of benfotiamine and the bioavailability assessment compared to thiamine hydrochloride," *J Clin Pharmacol*, 2014 Jun;54(6):688-95. doi: 10.1002/jcph.261; Epub 2014 Jan 22, at https://www.ncbi.nlm.nih.gov/pubmed/24399744.

[xxii] Sampson TR, Debelius JW, Thron T, *et al.*, "Gut Microbiota Regulate Motor Deficits and Neuroinflammation in a Model of Parkinson's Disease," *Cell*, Vol 167, No 6, p1469-1480.e12 (1 December 2016), at http://www.cell.com/fulltext/S0092-8674(16)31590-2; Mulak A & Bonaz B, "Brain-gut-microbiota axis in Parkinson's disease*," World J Gastroenterol*, 2015 Oct 7; 21(37): 10609–10620, Published online 2015 Oct 7. doi: 10.3748/wjg.v21.i37.10609, at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4588083/.

[xxiii] Hu X, Wang T, Jin F, "Alzheimer's disease and gut microbiota," *Sci China Life Sci*, 2016 Oct;59(10):1006-1023; Epub 2016 Aug 26, at https://www.ncbi.nlm.nih.gov/pubmed/27566465.