Parkinson's, Profit and People: The science money wants hidden

I have some important information to share. We live in a commercial society. Now that I head the *Universal Intelligence Network: Think Net*, I have begun to gather information to construct and propose research and social programs in areas of science which promise the most direct return in health for every dollar invested. I have been shocked, if not in the least surprised, to discover the wealth of information and detailed science that spells out many valuable avenues of treatment and potential cure, which are not known to the public, and again, are by my inference, not brought to the attention of the practicing medical community either, as they are areas which promise little profit for pharmaceutical companies. This is my implication after doing a bit of research. Over and over again, I observe that efficacious, noninvasive, promising and inexpensive treatments are never followed through, I hypothesize, because they are not available for patent, and so, offer no monetary reward. The result, is that people who could be well, are sick, and those who will get sick, in many cases, need not be. I wish to take the next two articles and disclose some of what I have found.

Compounds of low potential profit with scientifically demonstrated efficacy:

Thiamine: http://www.neurores.org/index.php/neurores/article/viewFile/155/155 J Neurol Res • 2012;2(5):211-214. From, "**The Beneficial Role of Thiamine in Parkinson's Disease: Preliminary Report**, Khanh vinh quoc Luong , Lan Thi Hoang Nguyen." Abstract: Parkinson's disease (PD) is the second most common form of neuro-degeneration in the elderly population. PD is clinically characterized by tremors, rigidity, slowness of movement and postural imbalance. A significant association has been demonstrated between PD and low levels of serum thiamine. Five PD patients presented with stone face, right-hand tremors, Parkinsonian gait and bradykinesia with occasional freezing. Two patients presented with sialorrhea and the plasma transkelosase activity was low in one patient. All of the patients received 100 - 200 mg daily doses of parenteral thiamine. Within days of thiamine treatment, the patients had smiles on their faces, walked normally with longer steps, increased their arm swings, and experienced no tremors or sialorrhea. Three patients did not require carbidopa and levodopa without effects on their movements. ***Thiamine may benefit to PD. Further investigation of thiamine in PD patients is needed. [Emphasis added].***

Cinnamon: http://www.ncbi.nlm.nih.gov/pubmed/24946862 (Pub Med). J Neuroimmune Pharmacol. 2014 Sep;9(4):569-81. doi: 10.1007/s11481-014-9552-2. From, **Cinnamon treatment upregulates neuroprotective proteins Parkin and DJ-1 and protects dopaminergic neurons in a mouse model of Parkinson's disease.** Khasnavis S, Pahan K." Abstract: ". . . However, oral treatment of MPTP-intoxicated mice with cinnamon powder and NaB reduced the expression of iNOS and protected Parkin/DJ-1 in the nigra. These findings paralleled dopaminergic neuronal protection, normalized striatal neurotransmitters, and improved motor functions by cinnamon in MPTP-intoxicated mice. ***These results suggest that cinnamon may be beneficial for PD patients.*" *[Emphasis added].***

Cannabis/THC/CBD and the uninvestigated role of pregnenolone:

a. Pregnenolone levels are lowered in an experimental model of nigrostriatal degeneration: http://www.ncbi.nlm.nih.gov/pubmed/21671084 (Pub Med). J Mol Neurosci. 2012 Jan;46(1):177-83. doi: 10.1007/s12031-011-9570-y. From, **Modifications of neuroactive steroid levels in an experimental model of nigrostriatal degeneration: potential relevance to the pathophysiology of Parkinson's disease.** Melcangi RC, Caruso D, Levandis G, Abbiati F, Armentero MT, Blandini F."Among the neuroactive steroid levels assessed (i.e., pregnenolone, progesterone, dihydroprogesterone, tetrahydroprogesterone, isopregnanolone, testosterone, dihydrotestosterone, 3α-diol, dehydroepiandrosterone, 17α-estradiol, and 17β-estradiol), we observed a significant decrease of pregnenolone in the striatum."

b. Cannabis improves symptoms: www.ncbi.nlm.nih.gov/pubmed/24614667 (Pub Med). Clin Neuropharmacol. 2014 Mar-Apr; 37(2):41-4. doi: 10.1097/WNF.0000000000000016. From, **Cannabis (medical marijuana) treatment for motor and non-motor symptoms of Parkinson disease: an open-label observational study.** Lotan I, Treves TA, Roditi Y, Djaldetti R. "**RESULTS:** Mean (SD) total score on the motor Unified Parkinson Disease Rating Scale score improved significantly from 33.1 (13.8) at baseline to 23.2 (10.5) after cannabis consumption ($t = 5.9$; $P < 0.001$). ***Analysis of specific motor symptoms revealed significant improvement after treatment*** in tremor ($P < 0.001$), rigidity ($P = 0.004$), and bradykinesia ($P < 0.001$). **CONCLUSIONS:** ***There was also significant improvement of sleep and pain scores. No significant adverse effects of the drug were observed. The study suggests that cannabis might have a place in the therapeutic armamentarium of PD. Larger, controlled studies are needed to verify the results. [Emphasis added].***

c. THC substantially increases the synthesis of pregnenolone in the brain: http://www.sciencemag.org/content/343/6166/94 *Science 3 January 2014: Vol. 343 no. 6166 pp. 94-98 DOI: 10.1126/science.1243985* From, **Pregnenolone Can Protect the Brain from Cannabis Intoxication** Monique Vallée, et al.: "***Pregnenolone is considered the inactive precursor of all steroid hormones, and its potential functional effects have been largely uninvestigated.*** The administration of the main active principle of *Cannabis sativa* (marijuana), Δ^9-tetrahydrocannabinol (THC), substantially increases the synthesis of pregnenolone in the brain via activation of the type-1 cannabinoid (CB_1) receptor." [Emphasis added].

d. There are antioxidant effects and others ascribed to CBD as well. See, http://www.ncbi.nlm.nih.gov/pubmed/21545415 Br J Pharmacol. 2011 Aug;163(7):1365-78. doi: 10.1111/j.1476-5381.2011.01365.x. **Prospects for cannabinoid therapies in basal ganglia disorders.** Fernández-Ruiz J[1], Moreno-Martet M, Rodríguez-Cueto C, Palomo-Garo C, Gómez-Cañas M, Valdeolivas S, Guaza C, Romero J, Guzmán M, Mechoulam R, Ramos JA. This CB(2) receptor up-regulation has been found in many neurodegenerative disorders including HD and PD, which supports the beneficial effects found for CB(2) receptor agonists in both disorders. In conclusion, the evidence reported so far supports that ***those cannabinoids having antioxidant properties and/or capability to activate CB(2) receptors may represent promising***

therapeutic agents in HD and PD, thus deserving a prompt clinical evaluation. [Emphasis added].

Also: http://www.ncbi.nlm.nih.gov/pubmed/17196181 Brain Res. 2007 Feb 23;1134(1):162-70. Epub 2006 Dec 28. **Evaluation of the neuroprotective effect of cannabinoids in a rat model of Parkinson's disease: importance of antioxidant and cannabinoid receptor-independent properties.** García-Arencibia M, González S, de Lago E, Ramos JA, Mechoulam R, Fernández-Ruiz J. In summary, our results indicate that those cannabinoids having antioxidant cannabinoid receptor-independent properties provide neuroprotection against the progressive degeneration of nigrostriatal dopaminergic neurons occurring in PD. In addition, the activation of CB2 (but not CB1) receptors, or other additional mechanisms, might also contribute to some extent to the potential of cannabinoids in this disease.

Also: http://www.ncbi.nlm.nih.gov/pubmed/15837565 Neurobiol Dis. 2005 Jun-Jul;19(1-2):96-107. **Cannabinoids provide neuroprotection against 6-hydroxydopamine toxicity in vivo and in vitro: relevance to Parkinson's disease.** Lastres-Becker I, Molina-Holgado F, Ramos JA, Mechoulam R, Fernández-Ruiz J. In summary, our results support the view of a potential neuroprotective action of cannabinoids against the in vivo and in vitro toxicity of 6-hydroxydopamine, which might be relevant for PD. Our data indicated that these neuroprotective effects might be due, among others, to the antioxidant properties of certain plant-derived cannabinoids, or exerted through the capability of cannabinoid agonists to modulate glial function, or produced by a combination of both mechanisms.

Next time K(2), Vitamin D, glutathione, Diltiazem, calcium channel blockers, case histories and compounds, speculative new ideas, and demonstrated scientific mechanisms of cure.