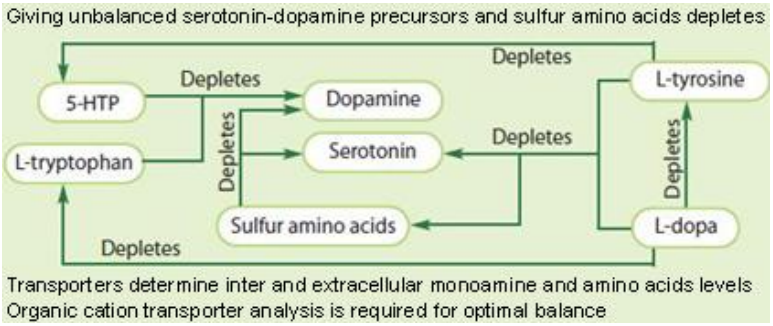

Parkinson RND

The centrally acting monoamines serotonin, dopamine, norepinephrine, and epinephrine do not cross the blood brain barrier. When the total number of their molecules needs to be increased in the central nervous system the only way to affect this is with administration of properly balanced nutrients (serotonin and dopamine amino acid precursors) which cross the blood brain barrier then are synthesized into new monoamines.

Parkinson disease is not due to a drug deficiency. It is due to post-synaptic neuron damage with associated relative nutritional deficiencies which are induced secondary to post-synaptic dopamine neuron damage and management attempts. This damage induces a defect in the flow of electricity in the substantia nigra, specifically the control of fine motor control.

Components of Parkinson disease being classified as a nutritional deficiency is based on the fact that L-dopa (the nutrient and amino acid precursor of dopamine) is required in amounts greater than can be obtained from the diet in order to increase dopamine levels high enough to achieve the flow of electricity needed to relieve symptoms.

There a numerous drugs used to treat Parkinson that are stand in stark contrast to L-dopa. Drugs like agonists, antagonists, MAO inhibitors, etc. are not nearly as effective as the much superior results of L-dopa. In terms of effectiveness L-dopa wins hands down in the management of Parkinson disease. But, side effects associated with long term use of L-dopa leave it almost dead last in the race. As noted in the attached peer-reviewed Parkinson disease paper **virtually all of the side effects and problems associated with L-dopa administration are due mismanagement of the multiple relative nutritional deficiencies associated with the disease itself, improper L-dopa administration, and/or administration of carbidopa.**

Below are the 5 categories of relative nutritional deficiency associated problems that can negatively affect or derail attempts to get the symptoms of Parkinson disease under control with L-dopa. These categories are expanded and discussed on page 172 of the attached peer-reviewed manuscript.

Category 1: Problems caused by depletion of serotonin by L-dopa
Category 2: Problems caused by imbalance of serotonin and dopamine
Category 3: Problems caused by dopamine fluctuations due to inadequate tyrosine levels
Category 4: Problems caused by depletion of sulfur amino acids by L-dopa
Category 5: Peripheral problems caused by peripheral depletion of serotonin and catecholamines by carbidopa

	Status in Parkinson's disease	Status with L-dopa Rx	Status with Carbidopa Rx
Serotonin (Central)	Depleted	Further Depleted	
Dopamine (Central)	Depleted		
Norepinephrine (Central)	Depleted		
Epinephrine (Central)	Depleted		
Serotonin (Peripheral)	Depleted	Further Depleted	Further Depleted
Dopamine (Peripheral)	Depleted		Further Depleted
Norepinephrine (Peripheral)	Depleted		Further Depleted
Epinephrine (Peripheral)	Depleted		Further Depleted
L-tyrosine		Depleted	
Tyrosine Hydroxylase	Depleted		
L-tryptophan		Depleted	
5-Hydroxytryptophan		Depleted	
Sulfur amino acids		Depleted	

Parkinsonism is known to induce a relative nutritional deficiency associated with serotonin, the catecholamines and tyrosine hydroxylase yet it is standard practice to ignore these nutritional deficiencies and start the patients on something other than L-dopa because of side effects that occur when L-dopa is improperly administered.

If L-dopa is not administered with proper levels of serotonin precursors, L-tyrosine and sulfur amino acids a progressive relative nutritional deficiency is observed along with an abundance of side effects (see pages 171 and 172 of the attached). In the course of this research we have established that some Parkinson patients need as much as 25,000 mg (25 grams) of L-dopa per day to get symptoms under control. This is a dosing value never documented before because it was inaccessible due to relative nutritional deficiency induced side effects. Simply giving 25 grams of L-dopa cannot be done. In general an L-dopa dosing barrier is encountered with group administration of 3,000 to 4,000 mg of L-dopa per day. Attempts to increase the L-dopa dosing values higher met with an abundance of symptoms associated with side effects. The chief symptom is L-dopa induced nausea. This can be dealt with easily if proper nutritionally driven OCT testing is in place during amino acid administration. The nausea is caused by a serotonin related 5-HTP relative nutritional deficiency induced by L-dopa. The only way to get this under control is with testing since serotonin levels that are too high or too low both cause severe nausea and there is no way from a clinical stand point to empirically differentiate where the serotonin levels are at.

PROTOCOL: Call DBS Labs for lab test kits, information, and protocols 877-476-7229

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