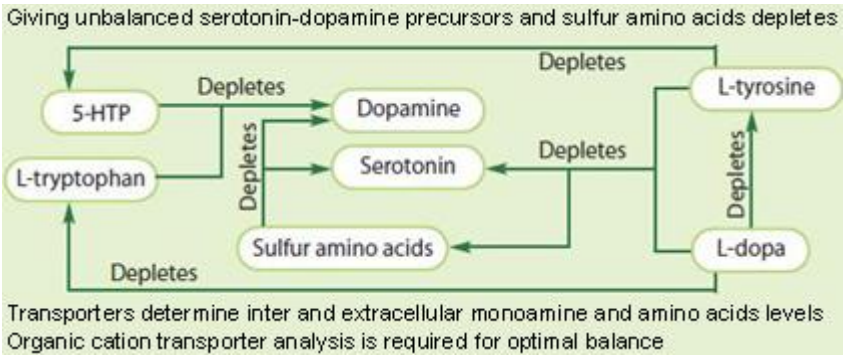

Transporter Signaling and Encoding

Prior to my research no one had ever developed a method for determining the status of organic transporters in live humans. Organic cation transporter assay analysis 1) defines the phase of monoamine transport 2) status of transporter entrance gates 3) transporter lumen saturation status 4) transport balance status between the monoamines and their amino acid precursors, all of which are critical to determining the relative concentrations of neurotransmitters at the systemic and synaptic levels.

There are three primary forces that determine concentrations of intercellular, extracellular, and synaptic levels of neurotransmitters (serotonin, dopamine, norepinephrine, and epinephrine) and their amino acid precursors; 1) synthesis 2) metabolism 3) transporter function. Of these three forces transporter function dominates and controls the other two, synthesis and metabolism.

Signaling and encoding of transporters has been known to science for many years that chemical or electrical signal go throughout the body encoding the transporters to regulate and react in a specific way. For example, if the body is suffering from inadequate levels of dopamine a signal goes out that encodes the transporters to conserve dopamine. In turn all transporters that are identical and homologous will conserve dopamine in an identical manner.

The following hypothesis has evolved from the research. When post-synaptic neuron damages occur leading to electrical defect associated disease, such as found in Parkinson disease and other neurotransmitter related diseases, a signal goes throughout the body which encodes the organic cation transporters to conserve and balance the transport of neurotransmitters in a manner to compensate for the damage. When damage is too great causing a need for neurotransmitter levels greater than can be achieved by dietary modification a relative nutritional deficiency develops.

The organic cation transporters of the kidneys, liver, bowels, and brain are “identical and homologous” meaning as a signal generated secondary to post-synaptic damage goes throughout the body these transporters react and control neurotransmitter with precursors transport in an identical manner.

Determining the functional status of kidney OCT allows for determination of status of the OCT in the brain and other sites. The when chronic neurotransmitter disease due to post-synaptic damage occurs the needed levels of monoamine neurotransmitters to correct the problem is encoded in the transporters of the body. Proper administration of amino acid precursors as guided by OCT status analysis can define the amino acid dosing values needs to compensate for the post-synaptic electrical problem.

In the world of neurotransmitters nothing is as simple or intuitive as the approach that most physicians take. If you believe that there is only a direct relationship (phase 3) between 5-HTP and L-dopa dosing values with serotonin and dopamine concentrations respectively, you are wrong and have over looked the inverse (phase 1) and no (phase 2) relationships that exist. In the majority of applications what sounds good and logical on the surface it is not the proper way to optimize the serotonin and catecholamine system. For years I have observed physicians who are sure that giving only 5-HTP to increase serotonin is the way to go. In the process 5-HTP’s well known ability to deplete dopamine is over looked (see above) and those physicians do not realize how properly balanced serotonin and dopamine precursors with sulfur amino acids improves and optimizes group outcomes significantly.

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