CHK MEDICAL FOODS™

The general approach to

Transporter Optimization™

Medical foods for the dietary management of disease for which nutrient requirements cannot be achieved by the modification of normal diet alone.

Monoamine Neurotransmitter Diseases
(Partial listing)

- Parkinsonism
- Obesity
- Bulimia
- Anorexia
- Depression
- Anxiety
- Panic Attacks
- Migraine Headaches
- Tension Headaches
- Premenstrual Syndrome (PMS)
- Menopause Symptoms
- Obsessive Compulsive Disorder (OCD)
- Compulsivity
- Insomnia
- Impulsivity
- Obsessionality
- Inappropriate Aggression
- Inappropriate Anger
- Psychotic Illness
- Fibromyalgia
- Chronic Fatigue Syndrome
- Adrenal Fatigue/Burnout
- Hyperactivity
- ADHD/ADD
- Hormone Dysfunction
- Adrenal Dysfunction
- Dementia
- Alzheimer's Disease
- Traumatic Brain Injury
- Phobias
- Chronic Pain
- Nocturnal Myoclonus
- Irritable Bowel Syndrome
- Crohn's Disease
- Ulcerative Colitis
- Cognitive Deterioration
- Functional Deterioration
- Increased Mortality Rate
- Organ System Dysfunction
- Chronic Stress
- Cortisol Dysfunction
- Hormone Dysfunction
- Restless Leg Syndrome

- **DEVELOPED BY MDs IN THEIR PRIVATE CLINICS**
- **NOW PUBLISHED IN PEER-REVIEWED SCIENCE**

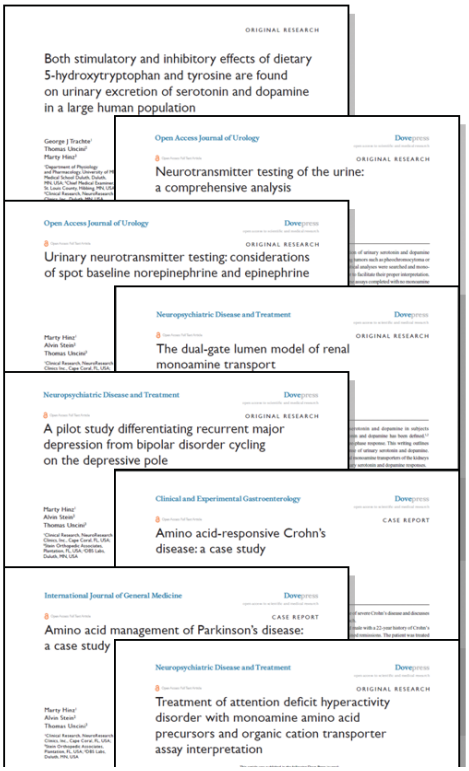
A primary determinant of neurotransmitter levels everywhere in the body is, "Transporter Functional Status™".

GENERAL APPLICATIONS OF "AMINO ACID TRANSPORTER OPTIMIZATION™" DOCUMENTED IN PEER-REVIEWED SCIENCE:

1. Treatment of chronic monoamine neurotransmitter diseases under the "Monoamine or Bundle Damage Theories".
2. Prevent depletion of neurotransmitters by drugs.
3. Proper use of amino acids will keep the drugs that work with neurotransmitters functioning properly, avoiding tachyphylaxis.
4. Any process where serotonin and/or any of the catecholamines (dopamine, norepinephrine, epinephrine) have been implicated.

"Everything used in this treatment approach (including L-dopa) is recognized by the FDA as GRAS (generally regarded as safe) and available over the counter without a prescription in the United States." (Hinz et. al. 2011c)

The medical approach presented in peer-reviewed literature



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Medical Foods

CHK NUTRITION

CHK Nutrition
5051 Miller Trunk Highway
Duluth, MN 55811
877-538-8388
www.CHKnutrition.com / www.HinzMD.com

THE BASIS FOR TREATMENT

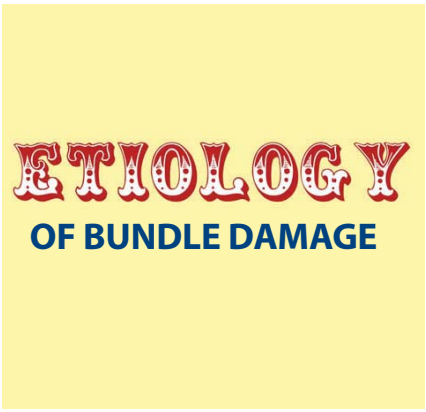
THE BUNDLE DAMAGE THEORY

Published 2009

"Neurotransmitter dysfunction disease symptoms, such as symptoms of depression, develop when the electrical flow through the neuron bundles that regulate function is compromised by damage to the individual neurons or the neuron components composing the neuron bundle which conducts electricity to regulate or control function. In order to optimally restore neuron bundle regulatory function, synaptic neurotransmitter levels of the remaining viable neurons must be increased to levels higher than is normally found in the system, which restores adequate electrical outflow resulting in relief of symptoms and optimal regulatory function." (Hinz et al 2009)

"Bundles of monoamine neurons can be impaired from neurotoxin exposures, trauma, or biological insult. Neurotoxin exposures are poorly defined and ongoing exposures are in contrast to the MPTP study model of Parkinsonism. The most comprehensive listing located reveals 1179 known neurotoxins. Susceptibility of individuals based on genetic predisposition, environmental influences, synergy between chemicals or other predisposing factors suggest that some individuals may experience neurotoxicity from many unlisted substances and at lower than threshold doses of known neurotoxins, and so was not considered. Under the bundle damage theory it is assumed that neurotoxins are the leading cause of monoamine bundle damage leading to the following speculation:

> The bundle damage's theory of repeated insult during a lifetime can explain the lack of efficacy seen in the treatment of elderly with reuptake inhibitors who presumably have greater cumulative lifetime effects from neurotoxins and other events that cause neuron damage. In the end these patients need to have neurotransmitter levels established that are much higher than can be achieved with reuptake inhibitors alone." (Hinz et al 2009)



LITERATURE NOTES: "The only way to increase neurotransmitter levels in the central nervous system is to administer amino acid precursors that cross the blood-brain barrier and are then synthesized into neurotransmitters. Increasing neurotransmitter levels in the synapse is analogous to increasing the voltage in an electrical wire, whereby turning up the voltage you get more electricity out of the other end of the wire. Turning up the voltage increases the electrical potential (pressure) of the electrons entering a partially damaged wiring connection, leading to more electrons (electricity) flowing out of the other end. In the case of neurotransmitter disease where the neurons of the neuron bundles are damaged to the point that the electricity flowing out of the neuron bundles is diminished disease develops. (Hinz et al 2009)

"Increasing neurotransmitter levels will effectively increase voltage in the remaining viable neurons in the bundle, causing electrical flow out of the damaged neuron bundles to increase to the point that normal regulation and/or control is once again observed. In this state, from a clinical standpoint, the symptoms of disease are under control." (Hinz et al 2009)

INADEQUATE LEVELS OF NEUROTRANSMITTERS CAUSE DISEASE

The states of transporter optimization

Endogenous state:
Taking no amino acids

ENDOGENOUS STATE: Most notably, urinary monoamine neurotransmitter testing in the endogenous state is of no value, results differ significantly from day to day making daily testing a random event.

Competitive inhibition state:
Taking significant amounts of serotonin and dopamine amino acid precursors

COMPETITIVE INHIBITION STATE: This occurs when significant amounts of both serotonin and dopamine precursors are taken. Physiologically this is a distinctly different state from the endogenous state and the physiological effects are markedly different.

Transporter optimization:
Organic cation transporter optimization guided by laboratory testing

TRANSPORTER OPTIMIZATION: Starts when urinary transporter testing is initiated in the competitive inhibition state and ends when the patient experiences relief of symptoms or both the serotonin and dopamine in the phase 3 therapeutic ranges.

THE GOAL: Urinary serotonin and dopamine both in the phase 3 therapeutic ranges.

Baseline testing prior to starting monoamine amino acid precursors is of no value

"While there have been attempts to integrate spot baseline urinary monoamine assays into treatment of peripheral or central neurotransmitter-associated disease states, diagnosis of neurotransmitter imbalances, and biomarker applications, significant differences in day-to-day reproducibility make this impossible given the known science as it exists today . . . Spot urinary neurotransmitter testing, due to lack of reproducibility, does not have a place in clinical practice for identifying biomarkers of peripheral or central nervous system function and disease states."

END POINT DOSING VALUES ARE INDIVIDUALIZED

Imagine a bull's eye with the center as the goal of treatment: the urinary serotonin and dopamine in the phase 3 therapeutic ranges.

For some patients simply starting the amino acids leads to relief of symptoms in one week. Other patients need to hit the bull's eye and have both the urinary serotonin and dopamine established into the phase 3 therapeutic ranges.

The bull's eye metaphor is good. Some patients merely need to hit the edge of the target for relief of symptoms while others need to hit the bull's eye.

TREATMENT DOES NOT HAVE A SET TIMELINE OR END POINT, IT IS A PROCESS

The statistics have been validated; it is true that a significant number of patients achieve relief of symptoms on the starting dose of the amino acids in less than a week, but the fact is that some patients need 2 to 4 months of weekly visits to get symptoms under control. All patients starting the amino acids should be screened to make sure they can participate in weekly visits for as long as 4 months (if needed) to ensure that time and expenses invested in the process are not wasted by unrealistic expectations.

RESPONSE is like a light switch

Some patients get relief of symptoms in one week while others may not get relief of symptoms until 2 or 3 months into treatment. Along the way most patients do not get better gradually with each amino acid dosing value adjustment. For most the response to amino acids is like a light switch, "on or off", with no relief of symptoms occurring until the proper amino acid dosing value is found secondary to several urinary transporter assays being performed.

PLACING THE PATIENT IN THE COMPETITIVE INHIBITION STATE

ADULT PROTOCOL-1

For all states except Restless Leg Syndrome and Parkinson's disease

	AM	NOON	4 PM	7 PM
LEVEL 1	4 NeuroReplete	—	4 NeuroReplete	—
LEVEL 2	4 NeuroReplete	4 NeuroReplete	4 RepleteExtra	—
LEVEL 3	4 NeuroReplete	4 NeuroReplete	4 RepleteExtra	4 RepleteExtra

If symptoms of depression are still present one week after starting level 3, obtain a urine sample then submit it for transporter interpretation (see pages 5 and 8).

ADULT PROTOCOL-2

Restless Leg Syndrome and Parkinson's disease

	AM	NOON	4 PM
LEVEL 1	4-D5	—	4-D5
LEVEL 2	4-D5 2-D5 Mucuna 40%	2-D5 Mucuna 40%	4-D5 2-D5 Mucuna 40%

After one week on level 2, obtain a urine sample and submit it for transporter optimization analysis™.

PEDIATRIC PROTOCOL

All diseases and Parkinson's disease

	AM	NOON	4 PM
LEVEL 1	2 NeuroReplete	—	2 NeuroReplete

If symptoms of depression are still present after one week of treatment, obtain a urine sample then submit it for transporter interpretation.

PROPERLY BALANCED AMINO ACIDS

The amino acid precursors of serotonin and dopamine need to be properly balanced. Improper balance depletes neurotransmitters. Giving only 5-HTP depletes dopamine. Giving only L-tyrosine or L-dopa depletes serotonin.

CysReplete

All patients need to take CysReplete to prevent depletion of sulfur amino acids by L-tyrosine and L-dopa.

- **PEDIATRIC DOSING:** 1 pill of CysReplete 3 times a day (with the first dose at noon).
- **ADULT DOSING:** 2 pills of CysReplete 3 times a day (with the first dose at noon).

SUGGESTED TREATMENT:

- See patients weekly until stable.
- There is no benefit in waiting longer than one week to see if there is further improvement prior to obtaining a urine sample.
- While many patients stabilize in 1 to 6 weeks, at the start of treatment ensure all patients are able to participate in weekly visits for as long as 2 to 4 months.

If you don't change it, at least change insure to ensure.

- If a drug side effect develops treat it as a drug side effect and do not decrease or stop the amino acids.
- Read pages 6 and 7 of the brochure for management of treatment problems.
- If the patient has problems swallowing a capsule, twist it open and put the powder in juice or other water soluble liquid.

MEDICAL FOODS STATEMENT: 1) These medical foods are specially formulated and processed products for partial feeding of the patient by means of oral intake. 2) These medical foods are intended for the dietary management of a patient who has other special medically determined nutrient requirements needing increased levels of monoamine neurotransmitters, the dietary management of which cannot be achieved by the modification of normal diet alone. 3) These medical foods are intended to be used under active and ongoing medical supervision wherein the patient requires medical care on a recurring basis for, among other things, instructions on the use of the medical food. 4) These medical foods provide nutritional support specifically modified for the management of the unique (distinctive) nutrient needs that result from the specific disease or condition as determined by medical evaluation.

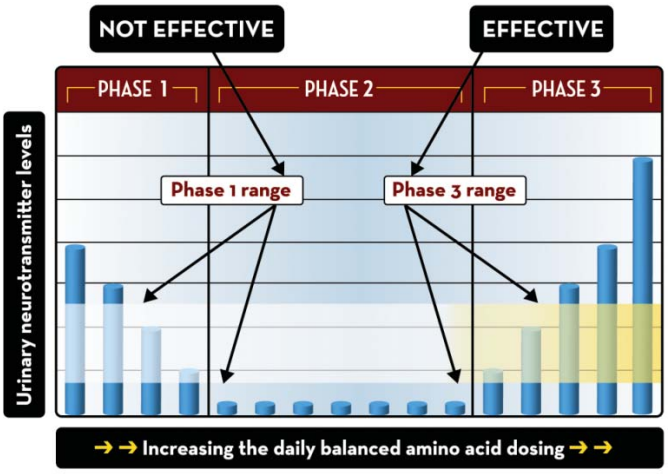
ORGANIC CATION TRANSPORTER ASSAY INTERPRETATION

(Continued on page 8)

DBS Labs
8723 Falcon Street
Duluth, MN 55808
877-476-7229
www.DBSlabs.com

The source of urinary monoamine neurotransmitters

"The serotonin and dopamine filtered at the glomerulous are metabolized by the kidneys, and significant amounts do not make it to the final urine. Serotonin and dopamine found in the urine are monoamines synthesized in the proximal convoluted renal tubule cells and have never been found in the central nervous system or peripheral system. Serotonin and dopamine that are newly synthesized by the kidneys meet one of two fates. Urinary serotonin and dopamine levels are primarily dependent on the interaction of the basolateral monoamine transporters (OCT2s) and the apical monoamine transporters (OCTN2s) of the proximal convoluted renal tubule cells of the kidneys. The OCTN2s of the proximal convoluted renal tubule cells transport serotonin and dopamine that is not transported by the OCT2. While in the competitive inhibition state, serotonin and dopamine not transported by the OCT2s are found in the final urine as waste. Although there are numerous other forces that interact with the newly synthesized renal monoamines, they are small compared with the effects of these transporters. Proper interpretation of urinary serotonin and dopamine levels in the competitive inhibition state determines the functional status of the OCT2s of the proximal convoluted renal tubule cells of the kidneys, known as OCT assay interpretation. The OCT2s exist in three different phases dependent on the status of the entrance gate and lumen saturation." (Hinz et al 2011)



ORGANIC CATION TRANSPORTER RESPONSE

The goal of this approach is to achieve the:

- urinary serotonin in the phase 3 therapeutic range of 80 to 240 µg serotonin/g of creatinine
- urinary dopamine in the phase 3 therapeutic range of 475 to 1,100 µg dopamine/g of creatinine (Hinz et al 2011)

The 3 phase response of the organic cation transporters

Serotonin and dopamine exist in two states. "The endogenous state" is found when no amino acid precursors are being administered. "The competitive inhibition state" is found when significant amounts of amino acid precursors of both serotonin and dopamine are administered simultaneously.

"The basis for OCT assay interpretation requires that the entire system be placed into the competitive inhibition state and then two or more urinary serotonin and dopamine assays performed while taking serotonin and dopamine amino acid precursors at significantly varied dosing values. The results are then compared in order to determine the change in urinary serotonin and dopamine levels in response to the change in amino acid precursor dosing values. Urinary serotonin and dopamine values found on assay were reported in micrograms of monoamine per gram of creatinine in order to compensate for fluctuations in urinary specific gravity. A urinary serotonin or dopamine value less than 80 or 475 µg of monoamine per 1 g of creatinine, respectively, is defined as a Phase 2 response. A urinary serotonin or dopamine value greater than 80 or 475 µg of monoamine per 1 g of creatinine, respectively, is interpreted as being in Phase 1 or Phase 3. Differentiation of Phase 1 from Phase 3 is a follows. If a direct relationship is found between amino acid dosing and urinary assay response, it is referred to as a Phase 3 response. An inverse relationship is referred to as a Phase 1 response. The Phase 3 therapeutic range for urinary serotonin is defined as 80–240 µg of serotonin per g of creatinine. The Phase 3 therapeutic range for urinary dopamine is defined as 475–1100 µg of dopamine per g of creatinine." (Hinz et al 2011)

PROPER MANAGEMENT OF AMINO ACIDS

PATIENT ORIENTATION:

At the first visit all patients need to be instructed as follows: **"If there are any problems in the first week of treatment, stop the amino acids until you get back to clinic and I will tell you what to do."** Failure to properly orient patients at the first visit will result in patients dropping out of care if problems are experienced in the first week of treatment.

GI UPSET → ON START UP

THE PROBLEM: Approximately 1% to 2% of patients (higher in some medical practices where patients have been exposed at a higher rate to drugs that deplete neurotransmitters) experience GI upset or nausea on starting the amino acids. Typically, this starts with the first dose and builds with every dose until the third day, at which point the patient can no longer tolerate the symptoms.

THE CAUSE: The patients who are most depleted of neurotransmitters experience GI upset or nausea on starting the amino acids. *These are the very patients who need the amino acids the most*.

MANAGEMENT: Restart the NeuroReplete taking only one pill at bedtime. Bedtime is when the patient is ready to get in bed and go to sleep, not when the patient gets in bed and reads a book for an hour. If the patient can fall asleep within 20 minutes after taking the one pill of NeuroReplete there should be no problems with GI upset. After 3 or 4 nights with no GI problems increase the NeuroReplete to 2 pills at bedtime. When the patient is able to take 2 pills at bedtime with no problems, start 1 pill in the AM then increase to 2 pills after 3 or 4 days of no problems. In adults, when the patient is taking 2 pills of NeuroReplete twice a day, submit a urine sample for transporter evaluation.

GI UPSET → CARBOHYDRATE INTOLERANCE

THE PROBLEM: Once the proper dosing values of the amino acids have been established, patients may experience transient nausea lasting 45 to 60 minutes periodically during the day. The etiology of this problem is distinctly different than "GI upset on start up" discussed above.

THE CAUSE: With optimization of neurotransmitter transporters, the way the body reacts to specific carbohydrates may change causing nausea.

MANAGEMENT: Usually only one food needs to be changed. Most of the time, it is a bread, cereal, or noodle that needs to be changed. Examples of effective management include 1) Changing from white to whole wheat bread. 2) Changing from one type of noodle to another. 3) Changing from one cereal to another, for example changing from Wheaties to Shredded Wheat. At times identification of the food can be difficult. One case of carbohydrate intolerance was tracked down to the breading on chicken eaten with most lunches.

HEARTBURN (PYROSIS)

THE PROBLEM: Intense substernal or epigastric burning or nausea after taking the pills.

THE CAUSE: When the veggie caps are simply gulped down with some water the surface does not liquefy properly causing the pills to stick in the esophagus and dissolve, at which time an intense substernal and/or epigastric burning is experienced.

MANAGEMENT: Hold the pills in the mouth with water for 10 to 15 seconds before swallowing so that the capsule surface starts to liquefy and slides down easily.

PRESCRIPTION DRUG SIDE EFFECTS

THE PROBLEM: The recommendation is to leave all drugs in place when starting the amino acids. Side effects not associated with the amino acids may occur in 3% to 5% of patients while starting or changing the amino acid dosing.

THE CAUSE: Drugs that work with neurotransmitters not only become more effective as neurotransmitter levels increase with amino acid administration; the side effects of these drugs may display as well.

MANAGEMENT: Proper management is to treat the event like a drug side effect. DO NOT stop the amino acids. Tapering or stopping the drug causing the side effect is proper management.

Amino acid only side effect profile

Dry mouth	2.1%
Insomnia	0.9%
Headache	0.7%
Nausea	0.6%
Dizziness	0.4%
Constipation	0.4%

All other side effects occur at a rate less than 1 in 500 visits

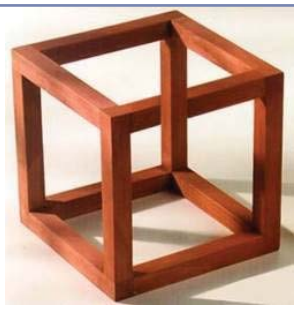
WHEN AMINO ACIDS STOP WORKING

THE PROBLEM: The patient's symptoms are under control, then it appears that the pills stopped working and symptoms of disease return.

THE CAUSE: Missing one or more doses of amino acids can cause symptoms of disease to return. It then may take three to five days for symptoms to get back under control once the pills are taken correctly. If the patient misses one pill dosing every three to four days multiple times, it may appear that the amino acids have quit working.

MANAGEMENT: In 99% of patients for whom the pills stop working it is a compliance issue relating to taking the pills properly. Have the patient journal (write down) all pills taken for 7 to 10 days. After journaling if the patient's symptoms are not under control, submit a urine sample for transporter assay. Only 1% of patients experience a change in dosing needs during treatment and retesting can manage this problem.

PARADOXICAL REACTIONS



THE PROBLEM: A specific amino acid dosing value is started or changed and the patient complains of an exacerbation of symptoms related to one or more monoamine neurotransmitter related diseases (see page 1). For example, depression may get worse, sleep may get worse, the patient may get more agitated, anxiety may get worse, migraines may get worse, etc.

THE CAUSE: The exact cause of paradoxical reactions is unknown, but it is known that there is a dosing range within which they do occur.

MANAGEMENT: When a paradoxical reaction is identified, it is an indication that the amino acid dosing value needs to be increased, at which point the paradoxical symptoms will resolve in one to two days. If the dose is lowered in hopes of increasing the dosing values slowly, the patient's suffering will be unnecessarily prolonged as the patient is subjected to the dosing value range where symptoms occur for an extended period of time.

DIZZINESS

THE PROBLEM: Dizziness (vertigo) may be caused by many different things when taking amino acids. As with paradoxical reactions, in many cases it is an indication to increase the amino acid dosing.

THE CAUSE: While complaints of dizziness may have many etiologies, dizziness associated with inadequate amino acid dosing is responsive to food intake. If the patient complains of dizziness which resolves after eating a small amount of carbohydrate such as a candy, cookie, pastry, etc. it is a carbohydrate-dependent vertigo which can develop during amino acid treatment.

MANAGEMENT: Management of "carbohydrate-dependent vertigo" involves increasing the amino acid dosing to the level where symptoms will resolve in one to two days. Any patient taking amino acids with complaints of dizziness needs to be properly evaluated for "carbohydrate-dependent vertigo".

HYPERSOMNOLENCE

THE PROBLEM: The patient recently started on amino acids may present at the clinic complaining of excess sleepiness to the point of having problems staying awake at work or during other daily activities.

THE CAUSE: In general, these patients were suffering from poor sleep prior to treatment and have a "sleep debt" that needs to be repaid prior to feeling optimal again.

MANAGEMENT: The first thing to do when complaints of excessive tiredness are encountered is to take a medical history to determine whether the cause is an imbalance in the amino acids or if the patient is suffering from a sleep debt that needs to be repaid. In patients with very poor sleep prior to treatment (3 to 4 hours per night), stop the amino acids and restart them on a Friday if the patient has the weekend off, telling the patient to sleep all weekend. If sleep was not a problem prior to treatment, cut the amino acid dosing in half, then obtain a urinary transporter analysis in order to determine the proper level of amino acids needed.

Two heads are better than one, and experience trumps all. There are no problems that should require long-term stoppage of the amino acids other than the patient quitting treatment.

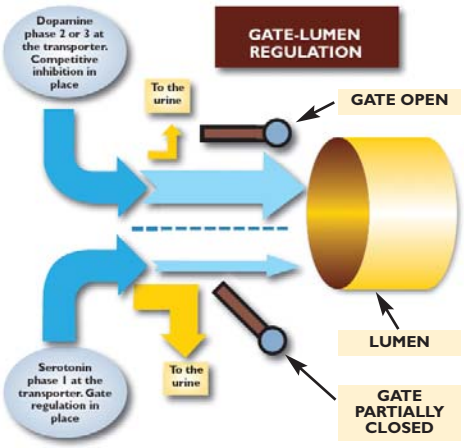
TRANSPORTER FUNCTIONAL STATUS DETERMINATION

(Continued from page 5)

Neurotransmitter levels for all systems are primarily dependent on the functional status of the organic cation transporters.

GATE-LUMEN TRANSPORTER STATUS FOR EACH PHASE

	Gate	Lumen
Phase 1	Partially closed	Unsaturated
Phase 2	Open	Unsaturated
Phase 3	Open	Saturated



**Dual-gate lumen organic cation transporter model developed by Hinz et al 2010.*

As noted in numerous peer-reviewed scientific writings, determination of transporter functional status can only be done in the "competitive inhibition state" found when significant amounts of serotonin and dopamine amino acid precursors are being taken simultaneously. Assays in the endogenous state performed while taking no amino acids are meaningless.

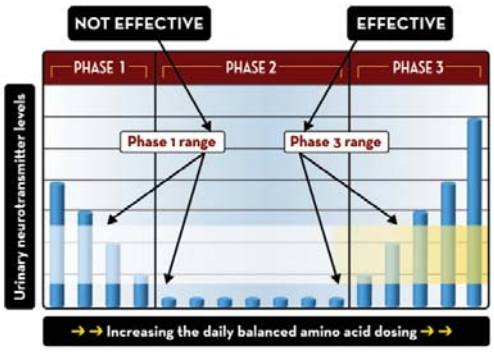
The goal of treatment is to get both the serotonin and dopamine into the phase 3 responses with urinary levels just over the phase 2 threshold (80 to 240 µ of serotonin per g of creatinine and 475 to 1,100 µ of dopamine per g of creatinine).

Interpretation of urinary data for phase determination rests on some simple concepts, but actual interpretation can become quite complex.

With each urine sample submitted to DBS Labs, a formal lab report is generated, followed the next day by a phase interpretation with suggested recommendations for moving serotonin and dopamine closer to the desired phase 3 ranges.

The phases of serotonin and dopamine occur independent of each other. Serotonin and dopamine can be in any of the three phases independent of each other.

Relief of symptoms is like shooting at a target, where urinary serotonin and dopamine in the phase 3 therapeutic ranges is the bull's eye. Some patients are symptom free by merely hitting the edge of the target; others need to be fully into the phase 3 therapeutic ranges before relief of symptoms is seen.



When administering monoamine amino acid precursors in the competitive inhibition state everything learned while administering just one amino acid precursor has to be abandoned because response in the competitive inhibition state is not intuitive.

In the competitive inhibition state amino acid side effects are not caused by one amino acid, they are caused by an imbalance between the serotonin and dopamine (catecholamine) systems. Amino acid dosing values required to get symptoms of disease under control are not dependent on getting amino acid levels high enough. Achieving proper balance between serotonin and dopamine can, in many cases, be accomplished by lowering the amino acid dosing.

The organic cation transporters of the kidneys, brain, liver, and bowels are "identical and homologous". Once the amino acids and neurotransmitters are at equilibrium, determining the functional status of one set of transporters will give insight into other sets of transporters.

TECH SUPPORT:
877-626-2220
www.HinzMD.com

MEDICAL FOODS based on scientific writings found in the following peer-reviewed scientific research:
Hinz, M. 2009 Depression, In: Kohlstadt I. editor. Food and Nutrients in Disease Management CRC Press; 465-481.
Hinz, M. Stein, A, Uncini T. 2010a The dual-gate lumen model of renal monoamine transport Neuropsychiatric Disease and Treatment 6 387–392
Hinz, M. Stein, A, Trachte, G, Uncini T. 2010b Neurotransmitter testing of the urine; a comprehensive analysis. Open Access Journal of Urology 2010:2 177–183
Hinz, M. Stein, A. Uncini T. 2010c A pilot study differentiating recurrent major depression from bipolar disorder cycling on the depressive pole, NeuroPsychiatric Disease and Treatment Neuropsychiatric Disease and Treatment:6 741–747
Hinz, M. Stein A. Uncini T. 2011a Treatment of attention deficit hyperactivity disorder with monoamine amino acid precursors and organic cation transporter assay interpretation Neuropsychiatric Disease and Treatment 2011:7 31–38
Hinz, M. Stein, A. Uncini T. 2011b Urinary neurotransmitter testing: considerations of spot baseline norepinephrine and epinephrine Open Access Journal of Urology 2011:3 19–24
Hinz, M. Stein, A. Uncini T. 2011c Amino acid management of Parkinson's disease: A case study International Journal of General Medicine 2011:4 1–10
Stein, A. Hinz, M. Uncini T. Amino acid responsive Crohn's disease, a case study. Clinical and Experimental Gastroenterology 2010:3 171–177
Trachte, G. Uncini, T. Hinz, M, 2009 Both stimulatory and inhibitory effects of dietary 5-hydroxytryptophan and tyrosine are found on urinary excretion of serotonin and dopamine in a large human population Neuropsychiatric Disease and Treatment:5 227–235