CHK MEDICAL FOODS

A superior option when the drugs prescribed for depression do not work well and/or make the cause of the depression worse.

It is a fact that this happens most of the time.

A University of Minnesota Medical School Published Research Project

Neuro Research

AMA Category 1 Continuing Medical Education

CHK Medical Foods as a part of

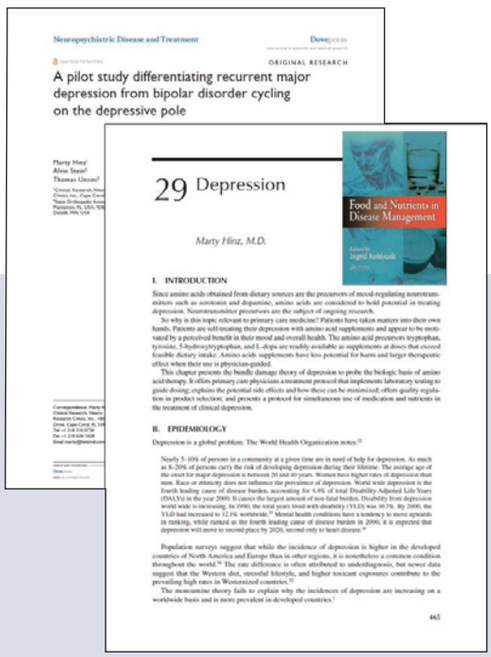
DEPRESSION TREATMENT

Based on NeuroResearch Clinics, Inc. data from over 600 medical clinics with over 2 million patient-days of treatment

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

Depression is a neurotransmitter deficiency disease, not a drug deficiency disease. The only way to increase the total number of neurotransmitter molecules in the brain is with administration of properly balanced nutrients. These medical foods are for the dietary management of disease for which nutrient intake cannot be achieved by the modification of normal diet alone.

BASED ON PEER-REVIEWED SCIENCE



The Medical Foods Approach

For years it has been a dream that amino acid precursors of serotonin and dopamine might be used as an antidepressant. Since 5-HTP became available in the United States in 1995 nobody has been able to produce peer-reviewed scientific studies showing it can be an effective part of a treatment plan until now.

A primary determinant of neurotransmitter levels is the transporters. This approach uses natural amino acids, which are available in the United States without a prescription, and laboratory transporter assay interpretation™ to optimize transporter function™. With this process we can establish the proper levels of amino acids for optimal transporter function and relief of symptoms. This approach has proven highly effective, even for patients with long-term depression lasting many years, even decades.

Depression is not a drug deficiency. It is a neurotransmitter deficiency. Drugs do nothing to increase the total number of neurotransmitter molecules in the brain. The only way to increase neurotransmitter molecules in the brain is with properly balanced amino acids™.

THE PROBLEMS WITH ANTIDEPRESSANT DRUGS

Drugs are not very effective and they deplete neurotransmitters making the cause of depression worse (see pages 2 and 3 of this guide).

INDEX

Medical Foods

CHK NUTRITION

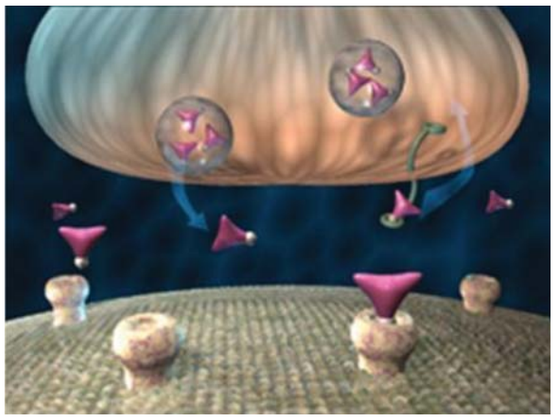
CHK Nutrition
5051 Miller Trunk Highway
Duluth, MN 55811
877-538-8388
www.CHKnutrition.com / www.HinzMD.com

THE MECHANISM OF ACTION

HOW REUPTAKE INHIBITORS DEPLETE NEUROTRANSMITTERS

Illustrations courtesy of the National Institute on Drug Abuse

Pre-treatment state

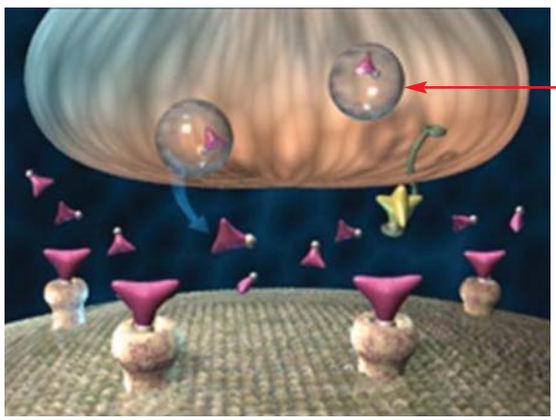


"The endogenous state" found prior to treatment. Synaptic levels of neurotransmitters are not high enough, contributing to symptoms of disease.

WHAT DOES NEUROTRANSMITTER DEPLETION DUE TO REUPTAKE INHIBITORS LOOK LIKE IN THE CLINIC?

From a clinical standpoint, when reuptake inhibitors stop working and symptoms of disease return, the drug has just depleted the neurotransmitters it is working with causing the neurotransmitter levels to fall below what is needed for the drug to function properly.

Early treatment with reuptake inhibitors



Vesicle

With reuptake blocked, neurotransmitters move from the pre-synaptic vesicles to the synapse.

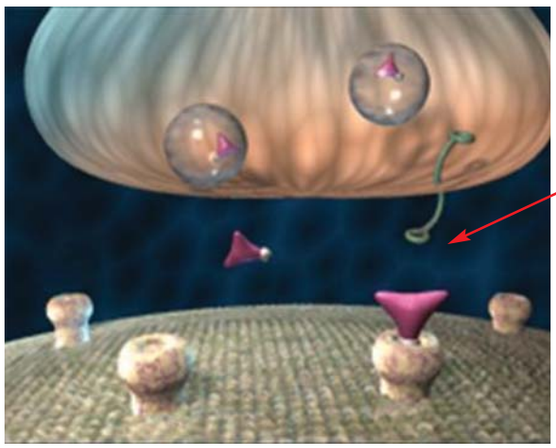
Drugs do nothing to increase the total number of neurotransmitter molecules in the brain™.

Drugs work by moving neurotransmitters from one place to another™.

The only way to increase the total number of neurotransmitter molecules in the brain is through administration of amino acid precursors which cross the blood-brain barrier then are synthesized into new neurotransmitters™.

Administration of improperly balanced serotonin and dopamine amino acid precursors will deplete neurotransmitters™.

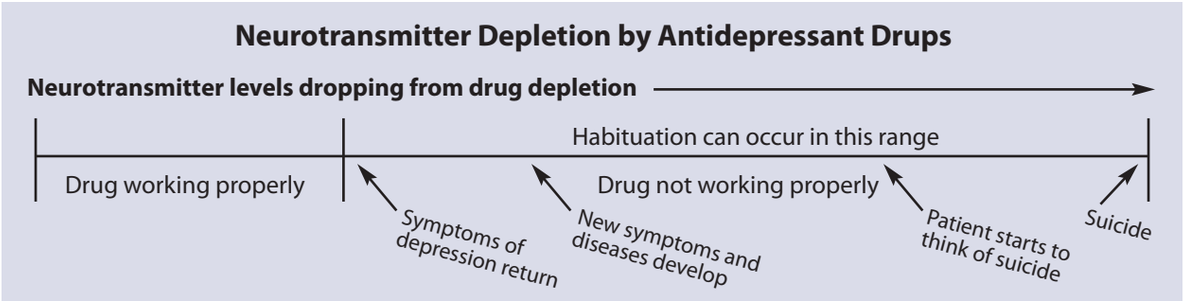
Depletion from reuptake inhibitors



While in the pre-synaptic vesicles, neurotransmitters are safe from metabolism. Once they move to the synapse, the neurotransmitters are exposed to the MAO and COMT enzymes which catalyze metabolism, leading to neurotransmitter depletion.

When neurotransmitters become depleted enough, drugs that work with neurotransmitters stop working™.

ANTIDEPRESSANTS: *THE ONLY CLASS OF DRUGS THAT MAKE THE CAUSE OF THE DISEASE WORSE*



For a discussion on habituation and its management, see page 5.

Effectiveness of Reuptake Inhibitor Antidepressants

- **ADULTS:** Double-blind, placebo-controlled studies indicate that only 7% to 13% of adult patients achieve relief of symptoms greater than placebo with antidepressant drugs. This means that 87% to 93% of patients taking antidepressant drugs get relief that is no greater than a sugar pill and are exposed to the drug side effects that include risk of suicide. (Hinz et al 2009)
- **ELDERLY:** All studies to date of patients 60 years old and older indicate that use of antidepressants leads to results that are no better than taking sugar pills for any patient. There is no response in treatment of depression in patients 60 years and older. This means none of the patients in this age group receive relief of symptoms but are exposed to drug side effects including risk of suicide. (Hinz et al 2009)

Low levels of neurotransmitters are a primary cause of depression. These antidepressant drugs set up conditions that deplete neurotransmitters making the cause of the depression worse. (Hinz et al 2009)

Since 2005 reuptake inhibitor medications have become embroiled in controversy with the FDA and others relating to pulmonary hypertension in infants and birth defects.

Why do these drugs make patients suicidal?

Low levels of neurotransmitters are thought to be a primary cause of depression. As the reuptake inhibitor drugs further deplete neurotransmitters depression can become more severe. The most severe form of depression is when the patient starts to think about committing suicide, and then acts on it.

It is no coincidence that all of these drugs carry a suicide warning. These drugs can deplete neurotransmitters. When the depletion is great enough the risk of suicidal thoughts and behavior develops. (Hinz et al 2009)

SOME OF THE DRUGS REQUIRED BY THE FDA TO CARRY A "BLACK BOX SUICIDE RISK WARNING"

PAXIL PAROXETINE HCl
ONCE-DAILY VENLAFAXINE HCl EFFEXOR XR EXTENDED RELEASE CAPSULES
Celexa citalopram HBr
zoloft (sertraline HCl)
PROZAC fluoxetine hydrochloride
Lexapro escitalopram oxalate
Pristiq desvenlafaxine
Wellbutrin SR (bupropion HCl)
Cymbalta duloxetine HCl

ANTIDEPRESSANT DRUGS HAVE THE RISK OF SUICIDE ASSOCIATED WITH THEM

"BLACK BOX WARNING" — THE MOST SERIOUS WARNING REQUIRED BY THE FDA

THE "BLACK BOX SUICIDE RISK WARNING" REQUIRED BY THE FDA

Suicidality and Antidepressant Drugs

Antidepressants increased the risk compared to placebo of suicidal thinking and behavior (suicidality) in children, adolescents, and young adults in short-term studies of major depressive disorder (MDD) and other psychiatric disorders. Anyone considering the use of Zoloft or any other antidepressant in a child, adolescent, or young adult must balance this risk with the clinical need. Short-term studies did not show an increase in the risk of suicidality with antidepressants compared to placebo in adults beyond age 24; there was a reduction in risk with antidepressants compared to placebo in adults aged 65 and older. Depression and certain other psychiatric disorders are themselves associated with increases in the risk of suicide. Patients of all ages who are started on antidepressant therapy should be monitored appropriately and observed closely for clinical worsening, suicidality, or unusual changes in behavior. Families and caregivers should be advised of the need for close observation and communication with the prescriber. Zoloft is not approved for use in pediatric patients except for patients with obsessive compulsive disorder (OCD). (See Warnings: Clinical Worsening and Suicide Risk, Precautions: Information for Patients, and Precautions: Pediatric Use)

About 90% of patients taking antidepressants have no hope of attaining results better than a sugar pill, but are exposed to side effects including risk of suicide.

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.

Adult Depression Protocol

	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 and submit it for transporter interpretation (see pages 8 and 9).

Pediatric Depression Protocol (<17 years of age)

	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 and submit it for transporter interpretation.

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 one to six weeks, ensure all patients are able to participate in weekly visits for two to four months if needed at the start of treatment.
- Continue all drugs the patient is taking at the start of treatment.
- 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 child 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.

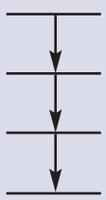
WHEN YOU START, GET THE JOB DONE

DEPRESSION TREATMENT CONSIDERATIONS

This approach is based on one of the peer-reviewed medical studies on pages 10 through 16 of this brochure. There have been no reported treatment failures with depression as long as the approach is applied properly. The benefit even extends to patients who have suffered from depression for many years without previous treatment success.

STAGES IN DEPRESSION TREATMENT

Treatment should continue until symptoms are under control. There is no need to progress through the stages of treatment further once symptoms are under control.



Start of treatment

Amino acids level 3 dosing value

Urinary serotonin and dopamine both phase 3 therapeutic ranges

Serum Depakote® or lithium carbonate in the therapeutic ranges

Do not use lithium ornitate — it has not been calibrated for serum therapeutic ranges.

The following stages for adult treatment should be followed:

Adult Stage 1: Place the patient fully in the competitive inhibition state. The goal is relief of symptoms or level 3 dosing, whichever comes first.

Adult Stage 2: submit urine sample then follow the amino acid dosing change recommendations when they are received. The goal of treatment is to continue until relief of symptoms are relieved or both the urinary serotonin and dopamine are in the phase 3 therapeutic ranges.

Adult Stage 3: continue amino acids from step 2 and start a mood stabilizing drug, either Depakote 250 mg three times a day or lithium carbonate 300 mg twice a day. Adjust the selected drug until serum levels are in the therapeutic range as defined by the laboratory being used. The goal of treatment is relief of symptoms.

As patients progress through the stages of treatment, an increasing number will find relief of symptoms requiring no further manipulation of the amino acids or drugs. Approximately 80% of patients will find relief of symptoms in stage 1 without stage 2 urine testing. As noted in the scientific article found on pages 10 to 16, only 7.3% of patients who start urine testing need stage 3 treatments with Depakote or lithium carbonate in combination with the amino acids. It has been found that 100% of patients who still have symptoms with the urinary serotonin and dopamine in the phase 3 therapeutic range are ultimately suffering from depression dominant bipolar disorder. It is not uncommon to see patients who have suffered with bipolar depression for for more than 40 years finally achieve relief of symptoms for the first time in their lives with this approach.

HABITUATION

Habituation is a decrease in response to a stimulus after repeated presentations. In the case of the reuptake inhibitors, the decrease in response is due to these drugs setting up conditions that deplete neurotransmitters.

Drugs that work with neurotransmitters do not work if there are not enough neurotransmitters in the system™.

The patient gets stuck on the drug. After taking the drug for a while, neurotransmitter levels are depleted and the patient feels worse when the drug is stopped because the neurotransmitter levels in the synapses of the brain drop lower than before treatment due to depletion. Even though the drug does not work most of the time, when the patient attempts to stop the drug, synaptic neurotransmitter levels drop lower than prior to treatment; the drugs have made the problem worse and the patient feels worse when attempts are made to stop the pills.

NOT MERELY GOING TO THE HEALTH FOOD STORE

It is no secret that this documented scientific approach uses the nutrients 5-HTP, tyrosine, levodopa, and cysteine for treatment of patients with depression. Proper use of these ingredients in medical treatment is not simple. Occasionally a patient will say, "Why do I want to take that, I can go to a health food store and buy it?" Attempting to achieve success by buying nutrients in a health food store is like going to an art store and buying a bunch of oil paints then going home and expecting to paint a master piece seen in an art gallery even though there is no previous painting experience. These nutrients have tremendous potential due to their chemical properties. This potential is only fully realized in the hands of the trained professional using transporter optimization testing. It is not just giving a nutrient pill. It is the whole treatment approach trained physicians use to insure that that treatment is on track to get symptoms under control.

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 two 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

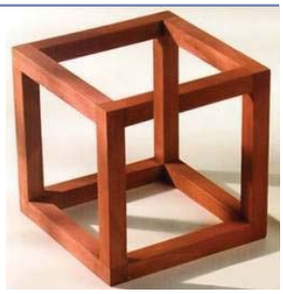
WHEN AMINO ACIDS STOP WORKING

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 8). 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 a prolonged 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 that 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.

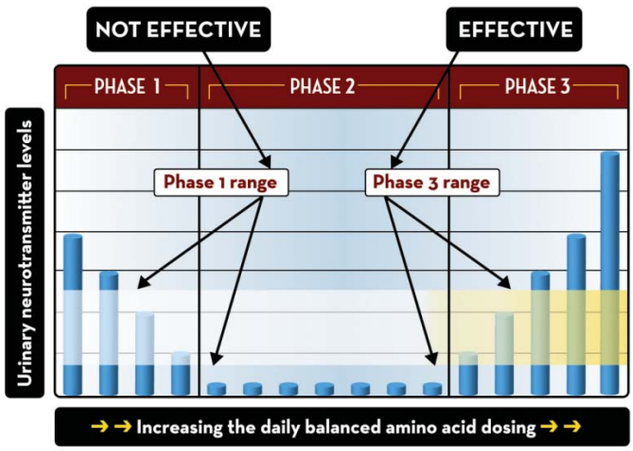
Two heads are better than one, and experience trumps all. There are no problems that should require long-term stoppage of the amino acids other than the patient quitting treatment.

ORGANIC CATION TRANSPORTER ASSAY INTERPRETATION

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)

RESPONSE is like a light switch

Some patients get relief of symptoms in one week while others may not get relief of three 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.

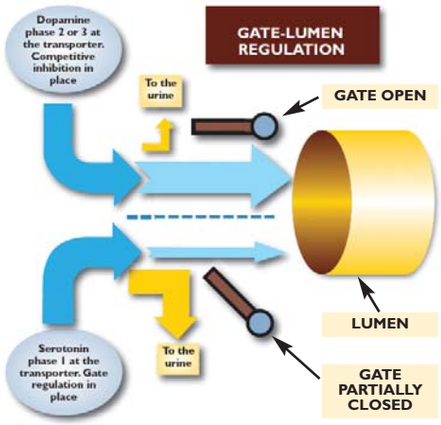
TRANSPORTER FUNCTIONAL STATUS DETERMINATION

(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.

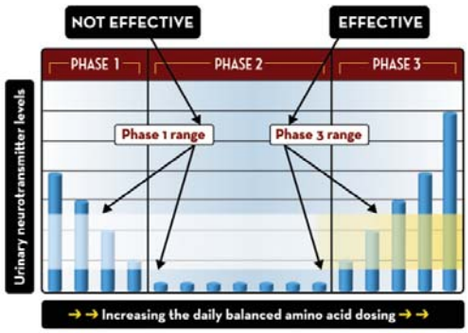
The goal of treatment is to get both the serotonin and dopamine into the phase 3 responses with urinary levels just over the phase 2 threshold (80 to 240 µ of serotonin per g of creatinine and 475 to 1,100 µ of dopamine per g of creatinine).

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.



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.

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

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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.

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