NEURO RESEARCH Expanding the Frontiers of knowledge with The Phase 3 Response™

Neurotransmitter Technical Support
NeuroResearch: 877-626-2220
www.NeuroAssist.com

AMINO ACID TREATMENT OVERVIEW

CME
Continuing Medical Education
NeuroResearch is dedicated to providing category I continuing medical education to physicians and category I continuing education units to psychologists.
www.NeuroSupport.com

See page 5
Low Neurotransmitters in the Body Cause

Obesity
- Decreased Life Expectancy
- Diabetes
- Heart Disease
- Increased Rate of Stroke
- Sleep Apnea
- Knee Problems
- Back Problems
- Increased Rehabilitation Time
- Increased Rate of Injuries
- Increases in Gall Stones
- Female Fertility Problems
- Gynecologic Irregularities
- Gouty Arthritis
- High Blood Pressure
- Hiatal Hernia
- High Cholesterol
- Increased Lung Infections
- Increases in Gastric Ulcers
- Chronic Pain
- Fibromyalgia
- Myoclonus

Type II Diabetes
- Decreased Life Expectancy
- Increased Infections
- Diabetic Neuropathy
- Kidney Failure
- Macular Degeneration (blind)
- Heart Disease
- Foot Ulcers
- Vascular Disease
- Therapeutic Amputations
- Disability
- Increased Rate of Stroke
- Impotence

High Blood Pressure and High Cholesterol
- Decreased Life Expectancy
- Heart Disease
- Stroke
- Kidney Failure
- Vascular Disease
- Ischemia

Increases the Risk of Cancer
- Increased Colon Cancer
- Increased Uterine Cancer
- Increased Breast Cancer

“Most patients have 3 or more neurotransmitter dysfunction diseases active at one time.”

Real Treatment: Natural treatment that corrects problems without the risks of side effects from prescription drugs.

Weight Loss: As indicated in the above list, obesity causes many problems

Other Diseases
- Bulimia
- Parkinsonism
- Obesity
- Anorexia
- Depression
- Anxiety
- Panic Attacks
- Migraine Headaches
- Tension Headaches
- Premenstrual Syndrome (PMS)
- Menopausal Symptoms
- Obsessive Compulsive Disorder (OCD)
- Impulsivity
- Obsessionality
- Insomnia
- 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
- Organ System Dysfunction
- Management of Chronic Stress
- Cortisol Dysfunction
- Restless Leg Syndrome

This is not a comprehensive list. Catecholamine and serotonin amino acid precursors have a positive impact on virtually all disease states caused by or associated with serotonin and/or catecholamine dysfunction.
Specific Master Neurotransmitters Implicated in Disease

Norepinephrine can take 2 to 6 weeks to stabilize and epinephrine can take 3 to 6 months to stabilize once serotonin and dopamine levels are in the therapeutic range AND in The Phase 3 Response™. Given the time it takes for norepinephrine and epinephrine to stabilize, the chart below provides a reasonable explanation of why some illnesses (i.e. insomnia) can take 2 to 6 weeks for symptoms to improve after serotonin and dopamine levels are in the therapeutic range and The Phase 3 Response™.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Serotonin</th>
<th>Dopamine</th>
<th>Norepinephrine</th>
<th>Epinephrine</th>
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</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>X</td>
<td></td>
<td>X</td>
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</tr>
<tr>
<td>Depression</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Insomnia</td>
<td>X</td>
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<tr>
<td>ADHD</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Migraine Headaches</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parkinsonism</td>
<td></td>
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<td>X</td>
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<tr>
<td>Hormone Imbalance</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td>Fibromyalgia</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Chronic Fatigue</td>
<td>X</td>
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<td></td>
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<td>Low Cortisol / Low DHEA</td>
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<td></td>
<td>X</td>
</tr>
<tr>
<td>Bulimia / Anorexia</td>
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<td>X</td>
</tr>
<tr>
<td>Anxiety / Panic Attacks</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>PMS</td>
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<tr>
<td>Obsessive Compulsive Disorder</td>
<td>X</td>
<td>X</td>
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<td>X</td>
</tr>
<tr>
<td>Aggression</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Irritable Bowel / Crohn's Disease</td>
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<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Adrenal Dysfunction</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotic Illness</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>
Perspective

The NeuroResearch amino acid treatment protocols are based upon research started in 1995, at the Morgan Park Medical Clinic in Duluth, Minnesota. Our medical doctors began using amino acids with prescription drugs in response to the problem of weight loss prescription drugs developing tachyphylaxis (quit working). Our research quickly turned to the bigger picture of maintaining the effectiveness of prescription drugs used to treat diseases related to the master neurotransmitters (serotonin and/or catecholamines - dopamine, norepinephrine, and epinephrine).

By 1998, the problem of maintaining the effectiveness of prescription drugs used to treat diseases related to the master neurotransmitters had been solved with the use of amino acids. In 1999, an amino acid formulation was perfected that allowed the treatment of master neurotransmitter related illnesses without the use of prescription drugs. In 2001, NeuroResearch began working with urinary neurotransmitter testing in an effort to find a correlation between urinary master neurotransmitters and master neurotransmitter related illnesses. This work resulted in defining the three phases of urinary neurotransmitter response to amino acid therapy. From these findings, it was observed that urinary neurotransmitters in the therapeutic range and in The Phase 3 Response™ correlate highly with the resolution of disease symptoms and an optimal feeling of wellness.

Today, NeuroResearch’s approach is being used by over 800 clinics in the United States and Canada. This approach represents the first real alternative to prescription drugs in the treatment of neurotransmitter dysfunction diseases without the side effects and problems associated with prescribed drugs. Since defining The Phase 3 Response™ in conjunction with the therapeutic range, we have turned our attention to further exploring the many potential applications of our findings and technology.

Applications of NeuroResearch’s AMINO ACID PROTOCOLS

- The amino acid formulas and protocols of NeuroResearch are effective in any disease or illness where improper levels of serotonin and/or the catecholamines (dopamine, norepinephrine, and epinephrine) have been implicated.
- The amino acid formulas and protocols of NeuroResearch, when given in combination with prescription drugs that utilize serotonin and/or catecholamines, ensure that drugs targeting the master neurotransmitters function at optimal efficacy.
- The NeuroResearch database contains documented treatment history over 1 million patient days of treatment. Out of this collection of data, we have documented that the amino acid formulas are safe with ALL prescription drugs.
- The amino acid formulas and protocols of NeuroResearch are effective without prescription drugs for the treatment of neurotransmitter dysfunction diseases and illnesses involving the master neurotransmitters.
- Treatment approaches of amino acid protocols include:
  - Attempting to use amino acids to treat patients without training (least effective).
  - Using properly balanced amino acids formulas without proper training in the treatment of patients.
  - Using properly balanced amino acid formulas in conjunction with laboratory testing to optimize individual and group outcomes.
- Amino acid therapy is not new. NeuroResearch’s approach is innovative because it uses the clinical database to guide treatment to previously unknown levels of efficacy in the treatment of master neurotransmitter dysfunction diseases.

Patent application of technology covered in this manual is under world wide PCT patent filing and under the following U.S. Patents issued and Published Patent Applications in place as of October 15, 2006. (Unpublished patent applications are not listed here.) U.S. Patents 6,384,088, 6,403,657, 6,548,551, 6,660,777, 6,759,437.

U.S. Patent Applications (Published) 20020025972, 20020040054, 20020065311, 2002007537, 20020094969, 20030181509, 20040101575, 20040229285, 2005006590, 20050235008, 2006010325, 20060135567, 20060178423
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CME
CONTINUING MEDICAL EDUCATION

Course Description

This one day multi-disciplinary conference is designed to educate primary care physicians, psychiatrists, psychologists, and allied health professionals on the established and emerging work of treating patients suffering from master neurotransmitter dysfunctions. Overall, the course content will strengthen the participant’s knowledge of clinical care of neurotransmitter dysfunction in patients.

Perspective

These formal scientific education seminars are taught by the inventors of “amino acid therapy guided by noninvasive laboratory neurotransmitter testing in the treatment of master neurotransmitter dysfunction”. The faculties of these medical education seminars are medical doctors who developed this approach while caring for patients in their clinical practices. They will discuss amino acid treatment results only dreamed of in the past. We invite you to take the time to learn of this new approach at one of the formal continuing medical education events. Their approach has been described as a “paradigm shift in medicine” at national medical conferences where their work has been presented.

For conference times and dates, go to www.NeuroSupport.com

Certified for Category I Hours Toward the AMA Recognition Award

Certification is targeted at physicians (MD and DO), nurses, physician assistants, and psychologists. In order to receive credit, participants must attend at least one full session and submit a credit application and evaluation form before leaving. Certificates will be mailed within 6-8 weeks following the activity. This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of Medical Education Collaborative, Inc. (MEC) and NeuroResearch. MEC is accredited by the ACCME to provide continuing medical education for physicians and CEU for psychologists.

Approved for 9.2 contact hour(s) of continuing education for RNs, LPNs, LVNs and NPs.

This program is co-sponsored with Medical Education Collaborative, Inc. (MEC) and NeuroResearch. MEC is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center’s Commission on Accreditation. Provider approved by the California Board of Registered Nursing, Provider Number CEP 12990, for 9.2 contact hour(s). This program is co-sponsored by Medical Education Collaborative, Inc. (MEC) and NeuroResearch. MEC is approved by the American Psychological Association to sponsor continuing education for psychologists. MEC maintains responsibility for this program and its content. The course provides 7.75 hours of credit.

that he/she actually spent in the activity.

Medical Education Collaborative (MEC) is accredited by the ACCME to provide continuing medical education for physicians and CEU for psychologists.

Approved for 9.2 contact hour(s) of continuing education for RNs, LPNs, LVNs and NPs.

This program is co-sponsored with Medical Education Collaborative, Inc. (MEC) and NeuroResearch. MEC is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center’s Commission on Accreditation. Provider approved by the California Board of Registered Nursing, Provider Number CEP 12990, for 9.2 contact hour(s). This program is co-sponsored by Medical Education Collaborative, Inc. (MEC) and NeuroResearch. MEC is approved by the American Psychological Association to sponsor continuing education for psychologists. MEC maintains responsibility for this program and its content. The course provides 7.75 hours of credit.
The “68” Docs

Since NeuroResearch started working with clinics outside of our own, we have come to refer to a specific type of doctor as a “68” doc. So, what does this mean?

1. After reading information on our work, the typical “68” doc becomes very excited about our work and wants to try amino acids.

2. The typical “68” doc does not take the time to receive proper training on how to use the formulas. In the process, they usually use 6 to 8 bottles of amino acids a month, for 6 to 8 months (hence the term “68” docs). Eventually, they move on and attempt another project. They never fully realize the treatment potential of the amino acids.

3. “68” docs tend to put the cart before the horse. They tend to ask, “How can I try the amino acids to see if I want to attend a continuing medical education seminar?”

4. The typical “68” doc runs into problems from misuse of amino acids. They tend to blame the problems on the amino acids. The real problem is lack of training and improper positioning of patients.

Amino acid treatment is not intuitive. Proper training is needed to master their use. “68” docs have virtually no chance of obtaining the full potential of using amino acids without training. At present, NeuroResearch has hundreds of well trained clinics from across the United States using amino acids every month. The top 20% are treating over 100 patients a month. We rarely see a “68” doc that treats more than 6 to 8 patients. Amino acid training can be a great practice builder.

For several years we have been saying, “Docs trained at a seminar are using 20 to 30 times more amino acids than those who have not been trained. This means 20 to 30 times more patients and 20 to 30 times more revenues.” The fact is, approximately 80% of the patients seen in a medical practice could benefit from amino acid therapy for neurotransmitter dysfunction. Attending a seminar is necessary in order to master proper treatment and management of patients.

FLASH! EDUCATION™

Contains over 40 NeuroResearch seminar slide shows with voice narrative.

The recommended starting point in neurotransmitter clinical education is a live interactive continuing education seminar. Beyond this step is the repetitive training found in our “flash drive education” program. This will allow you to review at your leisure.

Each flash drive contains over 40 NeuroResearch slide shows with voice narrative. Each drive comes in a storage box with a prepaid postage sticker. In the future, as new research materials are added, we will send out a notice to “send in your flash drives”. When you send it in, NeuroResearch will load your drive with the latest work.

Each flash drive is loaded with day 1 talks (neurotransmitters and amino acids) and day 2 talks (management of a high performance clinical weight loss program) plus a PowerPoint viewer.

Call 877-626-2220 for details.

Cost is:
- $100 for physicians who have attended a NeuroResearch training seminar in the past.
- $250 for physicians who have not attended a NeuroResearch seminar.

For more information, go to www.NeuroAssist.com

Contains over 40 NeuroResearch seminar slide shows with voice narrative.
WHAT TO DO IF YOUR AMINO ACIDS ARE NOT WORKING?

NeuroResearch is a medical research company dedicated to providing AMA approved category I medical education to physicians and continuing education to clinical psychologists in the area of amino acids as applied to neurotransmitters. We have been training physicians since 1999.

98% of the comments during technical support phone calls are positive. They attest to the effectiveness of amino acid therapy. Approximately 2% of the calls are from physicians complaining that the amino acids are not working in treatment. In reviewing these phone complaints, the following problem appears to exist. In most cases, the calls are from physicians who have received no formal training on how to properly use the amino acids.

Two major problems stem from attempting to use amino acids without formal training. First, the amino acid dosing is typically improperly adjusted. Secondly, often neurotransmitter lab tests are not ordered at the correct time.

Attending our AMA approved continuing education seminars provides the training needed to achieve positive results. Amino acid therapy guided with lab testing is a powerful tool, but it only works when you understand how to use it.

The only way for this powerful tool to achieve its full potential in your hands is to first obtain proper training at one of our AMA approved continuing medical education seminars.

<table>
<thead>
<tr>
<th>Neurotransmitter Diseases</th>
<th>Neurotransmitter Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Hyperactivity</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Claustrophobia and Other Phobias</td>
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<tr>
<td>Panic attacks</td>
<td>Tension Headaches</td>
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<td>Insomnia</td>
<td>Chronic Pain</td>
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<td>Parkinsonism</td>
<td>Nocturnal Myoclonus</td>
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<td>Bulimia</td>
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<td>Anorexia</td>
<td>Deterioration of Cognitive Functions with Aging</td>
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<td>Premenstrual syndrome</td>
<td>Deterioration of Organ System Innervation</td>
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<td>Menopause</td>
<td>Hormone Dysfunction</td>
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<tr>
<td>Obsessive Compulsive Disorder (OCD)</td>
<td>Problems in Systems</td>
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<td>Impulsivity</td>
<td>Innervated by the Serotonin and/or</td>
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<td>Obsessionalty</td>
<td>Catecholamine Systems.</td>
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<td>Inappropriate Aggression</td>
<td>Cortisol Dysfunction</td>
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<td>Inappropriate Anger</td>
<td>Problems.</td>
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<td>Fibromyalgia</td>
<td>Neurotransmitter Reaction to Chronic Stress</td>
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<td>Chronic Fatigue Syndrome</td>
<td>Obesity</td>
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<tr>
<td>Adrenal Fatigue/Burnout</td>
<td>Restless Leg Syndrome</td>
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<tr>
<td>Dementia</td>
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<td>Alzheimer’s Disease</td>
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<td>Migraine Headaches</td>
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<tr>
<td>ADHD/ADD</td>
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</tbody>
</table>
ON/OFF:

When treating patients with diseases caused by or associated with the master neurotransmitters, the starting point of treatment is to use the protocols found on page 9. 70 to 80% of patients will achieve relief of symptoms by the time the level 3 amino acid dosing is in place for one week. For the remaining 20 to 30% of patients, relief of symptoms requires the guidance of urinary neurotransmitter testing to properly adjust the amino acid dosing. Most patients do not need urinary serotonin and dopamine levels fully established in the therapeutic range and in the Phase 3 Response™ in order to obtain relief of symptoms. Once relief of symptoms is obtained, no further testing is needed and amino acids do not need further adjustments (unless symptoms return).

The response to amino acids is like a light switch. It is either on (relief of symptoms obtained) or off (relief of symptoms is not obtained). In those patients that need urinary neurotransmitter testing, continued testing and adjustments of amino acid doses is needed until symptoms are resolved. If the phase 3 therapeutic response is obtained and disease symptoms do not resolve, please call NeuroResearch for a free consult with one of our licensed medical doctors.

Almost every week during telephone consults physicians say, “I have been treating this patient for 6 weeks. I have done 3 urinary neurotransmitters tests. The patient is not better. Should I keep trying to treat this patient?” The answer to the question is, “yes”. Most patients who need neurotransmitter testing will obtain relief of symptoms by the time the third urinary test is obtained. However, there are examples of patients who need an atypical amino acid dosing. The response to amino acids is like a light switch, it is either on or off. In those patients that need urinary neurotransmitter testing, and adjusting the amino acid dosing should be continued until the light switch comes on and symptoms resolve. With the aid of urinary neurotransmitter testing to adjust amino acid doses, difficult to treat patients will eventually obtain relief of symptoms.
**Important Points of Treatment**

- Start all adult patients on the level 1 dosing. Cysteine and selenium should be divided into three equal doses with the first dose taken at noon.
- When patients return to the clinic after one week, the question to ask is not, “How did you feel this week?” The question should be, “How did you feel yesterday?” It takes 3 to 5 days to get a complete response when starting or changing an amino acid dose. How the patient did yesterday is more indicative of the clinical response to amino acids than asking about the entire week.
- Read the “side effects section” of this booklet starting on page 30.
- If you are experiencing problems with side effects or are not getting the outcomes that we claim, call NeuroResearch at 877-626-2220. Our technical support staff will be glad to answer any questions.

The only way for this powerful tool to achieve its full potential in your hands is to first obtain proper training at one of our AMA approved continuing medical education seminars. (see page 5.)

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**5-HTP/Tyrosine TREATMENT PROTOCOL**

Generic Protocol Presented in Seminar (Cofactors also needed)

<table>
<thead>
<tr>
<th>AM</th>
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</tr>
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<td>---</td>
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</tr>
<tr>
<td><strong>LEVEL 2</strong></td>
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<td>300/1,000</td>
</tr>
<tr>
<td><strong>LEVEL 3</strong></td>
<td>150/1,500</td>
<td>150/1,500</td>
<td>300/1,000</td>
</tr>
</tbody>
</table>

PER DOSE (Milligrams 5-HTP / Milligrams tyrosine)
If the patient’s symptoms remain at level 3, obtain a urinary neurotransmitter test.

All patients need to take a total of 4,500 mg of cysteine with selenium and folate per day. The cysteine, selenium and folate formula should be taken in equally divided doses with the first dose at noon.

**5-HTP/Tyrosine with L-dopa TREATMENT PROTOCOL**

Recommended only for treatment of obesity, Parkinsonism, and Restless Leg Syndrome.

<table>
<thead>
<tr>
<th>AM</th>
<th>NOON</th>
<th>4 PM</th>
<th>7 PM</th>
</tr>
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<tbody>
<tr>
<td><strong>LEVEL 1</strong></td>
<td>150/1,500/60</td>
<td>---</td>
<td>150/1,500/60</td>
</tr>
<tr>
<td><strong>LEVEL 2</strong></td>
<td>150/1,500/60</td>
<td>150/1,500/60</td>
<td>300/1,000/60</td>
</tr>
<tr>
<td><strong>LEVEL 3</strong></td>
<td>150/1,500/60</td>
<td>150/1,500/60</td>
<td>300/1,000/60</td>
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</table>

PER DOSE (Milligrams 5-HTP / Milligrams tyrosine / Milligrams L-dopa)
If the patient’s symptoms remain at level 3, obtain a urinary neurotransmitter test.

All patients need to take a total of 4,500 mg of cysteine with selenium and folate per day. The cysteine, selenium and folate formula should be taken in equally divided doses with the first dose at noon.
The Basics of Urinary Neurotransmitter Testing

Most patients do not need urinary neurotransmitter testing to achieve relief of symptoms. Testing is meant for patients who do not achieve relief of symptoms after being on the level 3 dosing of amino acids for one week. With proper use of amino acids, 70 to 80% of patients will achieve relief of symptoms without testing.

**AT THE FIRST VISIT**

**DO NOT** obtain a urinary neurotransmitter test. Simply start the patient on the level 1 amino acid dosing.

**LEVEL 1**

See the level 1 patient back in 1 week. If symptoms are not under control, increase to the level 2 amino acid dosing.

**LEVEL 2**

See the level 2 patient back in 1 week. If symptoms are not under control, increase to the level 3 amino acid dosing.

See the level 3 patient back in 1 week. If symptoms are not under control, obtain a urinary neurotransmitter test and follow the reported recommendations.

For more information on amino acid dosing levels, call NeuroResearch at 877-626-2220.

Doing It Right

When the adult patient has been on the level 3 amino acid dosing for one week without the relief of symptoms, a neurotransmitter testing is indicated. The goal of urinary neurotransmitter testing is to obtain urinary serotonin and dopamine levels in the therapeutic phase 3 response. The following suggested protocol will help achieve optimal results when using amino acids with urinary neurotransmitter testing.

- Start all patients on the level 1 dosing protocol at the first visit. Formulas containing L-dopa are recommended only as a starting point for treatment of obesity, Parkinsonism and Restless Leg Syndrome.
- When patients return to the clinic after one week, the question to ask is not, “How did you feel this week?” The question should be, “How did you feel yesterday?” It takes 3 to 5 days to get a complete response when starting or changing an amino acid dose. How the patient did yesterday is more indicative of changes in the system than asking about the whole week. If symptoms are not fully under control, increase the patient to the level 2 dosing and have the patient return in one week.
- At the 3rd visit, if symptoms are not under control, increase to the level 3 dosing. If symptoms are under control, simply continue the level 2 dosing and have the patient return in two weeks.
- At the next visit, if symptoms are under control, continue the level 3 dosing. If symptoms are not under control, continue the level 3 dosing and obtain a urinary neurotransmitter test. Follow the amino acid dosing recommendations returned with the test.
- When you obtain a neurotransmitter test, have patients return in one week to discuss amino acid dosing changes.
- Whenever you change an amino acid dosing, have the patient return in one week to evaluate the results. When symptoms are not under control, patients suffer needlessly if you do not see them weekly.
- Of the patients who use neurotransmitter tests, over 60% only need one neurotransmitter test. This is consistent with complete resolution of symptoms after adjusting the amino acid dosing in accordance with the consultant recommendations on the test. The average number of neurotransmitter tests on all patents tested by DBS Labs in the last year (N = 4,168) was 1.81 tests per patient.

For more information on “amino acid dosing levels”, call NeuroResearch at 877-626-2220.
The Three Approaches to Amino Acid Therapy

**Approach #1:**
This approach was popularized in the 1980s and 1990s, when administration of tryptophan, 5-HTP, and other amino acid precursors were simply given individually. It involves administering serotonin and catecholamine amino acid precursors, then hoping for adequate results. In general, 15% of the population reported relief of their symptoms. Another 15% of the population reported some relief of symptoms. The rest reported no relief of symptoms.

**Approach #2:**
This approach uses balanced serotonin and catecholamine amino acid precursors in combination with dosing protocols guided by clinical results. This was the work of NeuroResearch, started in 1997. Database results verified that group outcomes increased markedly when using this approach.

**Approach #3:**
This approach uses urinary neurotransmitter testing to guide amino acid precursors of the serotonin and catecholamine systems in proper balance. Urinary neurotransmitter testing should be used with patients who are receiving proper amino acid therapy, but have not obtained relief of symptoms.

Establishing serotonin and dopamine levels in The Phase 3 Response™ and in the therapeutic range is the patented, patent pending, and trademarked intellectual property of NeuroResearch. Research has shown that establishing urinary serotonin and dopamine levels in The Phase 3 Response™ and in the therapeutic range correlates with the resolution of disease symptoms and an optimal feeling of wellness. To properly recognize and manage the three phases of neurotransmitter responses to amino acid therapy, urine samples must be collected 4 to 6 hours prior to bedtime. In most patients, this is done just prior to taking the 4 PM dosing of amino acids.
The goal of treatment is to optimize relief of symptoms in group treatment. Prior to our work, individual amino acid precursors were used in dosing ranges that our work has defined as the lower end of the group dosing range needs. At best, this approach yields “some results and some relief of symptoms in some patients.” However, to obtain optimal group outcomes, it was necessary to database observations and to perform statistical analysis of treatment responses. This allowed dosing range needs, as applied to group treatment, to be properly defined. The concept of “optimal group outcomes in treatment” is at the heart of our work. Use of only 5-HTP will not provide optimal group outcomes. In fact, using only 5-HTP provides suboptimal group results because it depletes the catecholamines (dopamine, norepinephrine, and epinephrine). 5-HTP must be used in proper balance with dopamine precursors in order for optimal group outcomes to be observed.

DO NOT USE PLAIN 5-HTP

Treatment with Only 5-HTP Will Not Provide Optimal Group Outcomes.
- Plain 5-HTP will not provide optimal group results.
- 5-HTP needs to be given in proper balance with dopamine precursors (tyrosine and/or L-dopa) for optimal group results (N-acetyl-tyrosine is not effective for treatment, see page 57).
- Long-term use of only 5-HTP depletes dopamine, norepinephrine and epinephrine.
- Use of plain 5-HTP does not address the problems driven by or associated with catecholamine dysfunction.
- Only 10% to 15% of patients achieve “good” results using only 5-HTP.
- For all diseases caused by or associated with serotonin and/or catecholamine dysfunction, group treatment that uses only 5-HTP is not as effective as the use of 5-HTP with properly balanced dopamine precursors.
Patients need to be seen weekly and their amino acids need to be adjusted weekly until their symptoms are under control. Prolonging amino acid adjustments by allowing patients to go 2 or 3 weeks between visits will lead to an increase in group drop-out rate due to prolonging the time a patient suffers without relief of symptoms.

Once urinary neurotransmitter test results are received, the amino acid dosing should be changed immediately. The patient should be retested in one week if there is not a complete resolution of symptoms. Waiting several months between tests only prolongs a patient's suffering and increases drop-out rates.

Care givers using initial amino acid doses that are lower than the level 1 dosing (in adults) prolong the suffering and the time it takes to get symptoms under control.

Patients taking tyrosine and/or L-dopa need to take proper levels of cysteine with selenium to prevent sulfur amino acid depletion with associated problems by catecholamine precursors.

In medicine, labs are ordered to assist with treatment decision-making. Neurotransmitter lab testing prior to starting amino acids is of no value. Urinary neurotransmitter testing prior to starting patients on amino acids is a waste of your time and your patients' money.

Neurotransmitters assayed in patients not taking supplemental serotonin and dopamine amino acid precursors are a distinctly different population of neurotransmitters from those seen when the patient is taking supplement amino acid precursors. These are two distinct populations of neurotransmitters that have no correlation on lab testing.

When proper assays of urinary neurotransmitters are performed in patients diagnosed as suffering from one or more neurotransmitter dysfunction diseases, who are not taking supplemental amino acid precursors, 61% assayed in the morning and 87% assayed in the late afternoon have urinary serotonin levels above the reference range reported by the lab.

Urinary neurotransmitter levels have a diurnal variation. The low point of the day occurs 5 to 6 hours before bedtime. For various reasons, other labs advocate urinary neurotransmitter testing in the AM. Urinary neurotransmitter testing for amino acid therapy should be performed 5 to 6 hours before bedtime. This will ensure that neurotransmitter levels do not drop below the levels needed to keep the system symptom free. Urinary neurotransmitter testing in the AM prevents optimal identification of the phase 1 response and The Phase 3 Response™.

Urinary phase 3 serotonin levels greater than 2,400 can lead to a decrease in group outcomes in the treatment of obesity.

Provide patients with only enough pills to make it to the next visit.

Do not let your amino acid stock drop below a two week supply. If your supplies fall below this level and there is a problem with the trusted shipping process, you may not have the amino acid treatments needed to keep symptoms of disease under control.

If amino acids appear to quit working after the patient has been on them for several weeks or months, the patient may have missed one or more doses during the week. Have the patient journal (write down) all pills taken for one week. If symptoms have not resolved in one week, order a urinary neurotransmitter test.
Beyond the Key Hole
The amino acid therapies of the past are like looking at a room on the other side of a door through the key hole. We could tell there was something there but the results, when applied to group treatment, were not very effective or consistent in the relief of symptoms. In retrospect, the problem was that we did not understand the amino acid dosing ranges needed to provide optimal group treatment and optimal relief of symptoms. Unless you are prepared to properly establish each patient’s individual amino acid dosing, you will not obtain optimal results with your patients.

Amino Acid Dosing Ranges Needed For Optimal Group Treatment
Individual amino acid dosing needs vary greatly. The following are the dosing ranges for individual amino acid components that are needed to obtain optimal group results:
- **5-HTP** 37.5 to 1,600 mg per day
- Tyrosine 375 to 12,000 mg per day
- L-dopa 120 to 12,000 mg per day.

It is important that 5-HTP be used in proper balance with the dopamine precursors tyrosine and/or L-dopa. DO NOT use only one precursor. Amino acid dosing at the higher end of the above ranges should only be prescribed under the guidance of neurotransmitter laboratory testing.

Establishing Individual Amino Acid Doses in Treatment
The goal of treatment is the optimal relief of symptoms. In order to obtain optimal group outcomes, each patient needs to have serotonin and catecholamine amino acid precursors established in the ranges on page 42.

See Page 9 for Amino Acid Dosing Levels
- All adult patients should be started on the level 1 treatment. Patients should return to the clinic in one week. If symptoms are not under control, adjust amino acids to level 2. Patients should return in one week. If symptoms are not under control, adjust amino acids to level 3. Most patients will have found relief of symptoms by level 3.
- If, after one week at level 3, symptoms are not under control, obtain a urinary neurotransmitter test. Follow the recommendations until symptoms resolve.
- Do not prescribe over 900 mg per day of 5-HTP or 5,000 mg per day of tyrosine without using a neurotransmitter test to first establish the need.
Use of Tyrosine with L-Dopa

Perspective
Initially, our work used tyrosine with 5-HTP and cofactors. In 2001, a natural nonprescription L-dopa in the form of Mucuna Pruriers came into the picture. At that time, based on preliminary research, we added L-dopa (Mucuna Pruriens) to the tyrosine/5-HTP formula. We then compiled the results. As time progressed, we were asked, “Why is tyrosine in the L-dopa formula? L-dopa probably shuts down the tyrosine hydroxylase enzyme via the norepinephrine feedback loop. This would likely leave tyrosine ineffective.”

Our answer has been, “We never developed data showing that you could remove tyrosine without decreasing group performance.” Currently, the results of our research are very clear. L-dopa needs to be used in combination with an L-dopa precursor and tyrosine for optimal results.

Lab Based Research Findings
Our work supports the observations that shutting down the tyrosine hydroxylase enzyme by norepinephrine is not an absolute or complete process. In lab results, we have seen that even when large amounts of L-dopa are administered, there continues to be two sources of L-dopa synthesized into dopamine. One source is from the L-dopa being administered. The second source is from the L-dopa that continues to be synthesized from tyrosine by the tyrosine hydroxylase enzyme.

Administration of only L-dopa is not subject to the regulation of the norepinephrine/tyrosine hydroxylase feedback loop. It has the ability to raise dopamine levels infinitely high, if infinitely high levels are administered. We have observed, through serial laboratory assays, when only L-dopa is administered that dopamine levels have the ability to fluctuate wildly. This can make it almost impossible to obtain stable dopamine levels in some subjects.

Our research has found that for optimal results and control of dopamine, L-dopa must be used in combination with L-tyrosine. In order to obtain optimal results and to control the synthesis of dopamine due to administration of L-dopa, the underlying stream of dopamine being synthesized from tyrosine must be addressed through the proper administration of tyrosine in combination with L-dopa.

Lab Based Research Findings
Tyrosine is converted to L-dopa by the tyrosine hydroxylase enzyme. L-dopa is then converted to dopamine. Dopamine is converted to norepinephrine. Norepinephrine regulates the tyrosine hydroxylase enzyme and L-dopa synthesis.
The underlying stream of dopamine synthesized from tyrosine needs to be properly addressed with the co-administration of proper levels of tyrosine. In some patients, if this does not occur, dopamine synthesized from the administered L-dopa tends to fluctuate widely as the underlying stream of dopamine from tyrosine fluctuates. Therefore, achieving stable levels of dopamine (and other catecholamines) when administering L-dopa requires that proper levels of tyrosine be administered in combination with the L-dopa.

### USE OF TYROSINE WITH L-DOPA, continued ...

The underlying stream of dopamine synthesized from tyrosine needs to be properly addressed with the co-administration of proper levels of tyrosine. In some patients, if this does not occur, dopamine synthesized from the administered L-dopa tends to fluctuate widely as the underlying stream of dopamine from tyrosine fluctuates. Therefore, achieving stable levels of dopamine (and other catecholamines) when administering L-dopa requires that proper levels of tyrosine be administered in combination with the L-dopa.

Failure to stabilize the underlying dopamine being synthesized from tyrosine can result in total dopamine levels that fluctuate wildly during the administration of supplemental L-dopa. This was the subject of our June 2006 patent application. In order to obtain stable dopamine levels when administering supplemental L-dopa, you need to fully control the dopamine in the system that is still being synthesized from tyrosine. The optimal way to affect this is with the co-administration of tyrosine and L-dopa as guided by urinary neurotransmitter testing.

The approach of co-administration of tyrosine with L-dopa leads to the following clinical impact. In the past, when using only L-dopa, we have seen it take 6 to 8 lab tests to stabilize urinary dopamine levels in some patients. By laying down a proper tyrosine base and adding a low dose L-dopa, we are now able to position the urinary dopamine of most patients into the “phase 3 therapeutic response” within 2 to 3 tests.

### The “Tyrosine Base”

When administering supplemental L-dopa, a proper “tyrosine base” needs to be established in all patients. In the past, the irreversible side effects of dyskinesias have been associated with long-term, high dose L-dopa administration. Research of the past has clearly demonstrated that this side effect is clearly dose and time dependent. This means that the higher the L-dopa dosing and the longer it is given, the more likely a patient is to develop dyskinesias. The benefits of preventing dyskinesias through co-administration of tyrosine with L-dopa as guided by urinary neurotransmitter testing are profound. The overall dosing of L-dopa needed to achieve desired clinical outcomes is markedly less when a proper tyrosine base has been established. A proper “tyrosine base” prevents the fluctuations of dopamine seen when only L-dopa is used. Establishing a proper “tyrosine base” can only be affected with the guidance of urinary neurotransmitter testing.

In most patients, a proper “tyrosine base” is laid down after serotonin is stabilized with tyrosine/5-HTP and an additional 6,000 mg per day of tyrosine in equally divided doses with low dose L-dopa. The general population of patients would not tolerate this type of tyrosine dosing, but with lab guidance establishing the need for a tyrosine base during treatment, patients identified as needing L-dopa with a tyrosine base tolerate the dosing well.

For more information on neurotransmitter testing or for tech support, call: NeuroResearch at 877-626-2220.
Expectations of Treatment

Safety
Based on the analysis of over 1 million patient days of treatment, research has shown that the amino acid protocols of NeuroResearch are safe and effective. The NeuroResearch amino acid protocols can be given safely with any prescription drug. If patients are managed properly, there are no reasons patients should have to stop the NeuroResearch amino acid protocols on a long-term basis. If you experience problems that are difficult to manage, call NeuroResearch at 877-626-2220. Our technical support staff is willing to assist you.

Efficacy
Optimizing the effectiveness of the amino acid formulas depends upon the clinical expertise of the care giver, which starts with proper training in the use of the treatment protocols developed by NeuroResearch. Care givers who have been trained to use the groundbreaking approaches of NeuroResearch’s protocols tend to experience a learning curve. Over time, they become more effective at treating patients with amino acids as a result of experience.

Typically, it takes the average doctor who has attended one of our continuing education training seminars, one to two years to master amino acid therapy. Training is absolutely necessary for success.

The efficacy results that are reported in this catalog, such as a first month average group weight loss of 16.9 pounds, 100% resolution of depressive symptoms, and 85% of migraine headaches resolving, are the results of experienced clinicians who have mastered amino acid therapy.

As with any medical treatment modality, concerns with treatment are primarily focused on two areas - safety and efficacy.
Depression

Classically, bipolar disorder is characterized by mood swings between depression and mania (or hypomania). It is obvious in our work that there is a subgroup of patients with bipolar disorder who simply cycle on the depressive side. Some patients with depressive bipolar disorder may have long cycles of depression and short cycles of hypomania/mania. From a clinical standpoint, it appears they are always depressed and never cycle into hypomania/mania.

Start the depressed patient on the level 1 amino acid dosing. Follow the 3 level protocol on page 9 of this catalog. If depression has not improved one week after the level 3 amino acid dosing has been established, obtain a urinary neurotransmitter test and follow the recommendations when reported. The goal of treatment is to continue testing and adjusting the amino acid dosing until symptoms are resolved or until urinary serotonin and dopamine levels are in the phase 3 therapeutic response.

Most patients with depression obtain relief of symptoms prior to establishing the urinary serotonin and dopamine fully in the phase 3 therapeutic response. Approximately 2% of patients treated do not experience relief of symptoms when urinary serotonin and dopamine levels are in the phase 3 therapeutic response. These patients appear to be suffering from depressive bipolar disorder. The proper approach, once urinary serotonin and dopamine levels are in the phase 3 therapeutic response, is to continue the amino acid dosing and start a bipolar drug such as Lithium, Depakote, Lamictil, etc. Once the starting dose of a bipolar drug is in place, the majority of patients find relief of symptoms within one week.

For the most part, it appears that patients with depressive bipolar disorder (who are taking a proper amino acid dosing) respond to subtherapeutic drug doses for treatment of bipolar illness. For example, in starting these patients on Lithium 300mg, twice a day, relief is obtained in one week as long as a proper amino acid dosing is in place. Certainly, in the treatment of bipolar disorder, Lithium 300mg twice a day does not establish serum levels high enough in most patients to achieve the classic therapeutic serum levels recommended for treatment of bipolar disorder. But, with amino acids, it does produce relief of symptoms. In observing urinary neurotransmitter lab results on a large scale, it is obvious that many of these patients with depressive bipolar disorder need 6 to 8 neurotransmitter tests to establish the phase 3 therapeutic response. In working with the depressed patient who does not have full relief of symptoms, it is important to establish the urinary serotonin and dopamine in the phase 3 therapeutic response prior to starting a bipolar drug to ensure that the patient indeed is suffering from depressive bipolar disorder. We have seen some patients who were not suffering from depressive bipolar disorder and only obtained relief of depression symptoms by establishing urinary serotonin and dopamine levels in the phase 3 therapeutic response.
Treating depression with amino acids requires the same techniques and treatment approaches as treating depression with prescription drugs. The only difference is the pharmacologic medium (in the form of amino acid) has changed. A clinical understanding of and experience with treating depression are necessary for successful treatment of patients.

Thoughts on Depression ...

**Diagnosis:** Made by the standard medical approached using the DSM IV criteria with usual supporting labs such as testing to rule out anemia and hypothyroidism in making the diagnosis of depression.

**Treatment:** Start 5-HTP/tyrosine amino acids at level 1 (see page 9) with the cysteine selenium, and folate formula.

**Follow-Up Treatment:** Patients should be seen weekly until symptoms are under control. If symptoms do not resolved after one week, increase to the level 2 dosing protocol. When patients return, do not ask, “How did you feel last week?” Instead inquire, “How did you feel yesterday?” It takes 3 to 5 days for the results of starting or changing an amino acid dosing to take effect. Therefore, asking about the previous day is more indicative of the changes that are occurring in the system.

**When to Get Neurotransmitter Testing:**
- If a patient’s symptoms are not fully resolved by the time they have been on the level 3 treatment protocol for a week, obtain a urinary neurotransmitter test and follow the recommendations in order to optimize results.

**Pearls of Treatment:**
- Urinary neurotransmitter testing prior to treatment has no benefit. Testing determines the response of the kidneys to administered amino acids precursors.
- The master urinary neurotransmitters found in the urine when patients are taking and not taking supplemental amino acids are both synthesized by the kidneys. However, from a practical stand point, it appears that they represent two distinct and separate populations of neurotransmitters. There is no correlation between these two populations as demonstrated by laboratory testing.
- If a depressed patient has obtained urinary serotonin AND dopamine levels “in the therapeutic range and The Phase 3 Response™” without complete relief of symptoms, consider the possibility of “depressive bipolar disorder”. Leave the amino acids, as well as any other drugs being used, in place and start a bipolar drug (such as Lithium, Depakote, Lamictil, etc). Approximately 2% of depressed patients do not respond to amino acids. But when a bipolar drug is added, virtually 100% of patients will obtain relief of symptoms.
- Anxiety is driven by GABA. GABA is controlled by the master neurotransmitters (serotonin, norepinephrine, epinephrine, and dopamine). Treat patients by establishing serotonin and dopamine levels in “the therapeutic range AND in The Phase 3 Response™”. Virtually all patients with anxiety can be treated effectively and GABA can be properly controlled without GABA precursors.
- In general, panic attacks require laboratory guidance for control of symptoms once level 3 amino acid dosing is obtained (see page 9). Be prepared to use lab testing if relief of symptoms is not seen after the level 3 amino acid dosing has been established for one week. Over 98% of patients with panic attacks can find natural relief of symptoms when treated properly.
Insomnia

Treatment Expectations
Sleep is controlled by melatonin. Melatonin is synthesized from serotonin via a long chemical pathway. At the end of this pathway, norepinephrine regulates the synthesis of melatonin.

Once urinary serotonin and dopamine levels are in “the therapeutic range and The Phase 3 Response™”, approximately 70% of patients will experience relief of symptoms. The rest may take 2 to 6 weeks for relief of symptoms. (The amount of time it takes for norepinephrine to stabilize once urinary serotonin and dopamine levels are in the therapeutic range and in The Phase 3 Response™.)

Hypersomnia in the Insomniac:
If a patient upon starting amino acid treatment complains of extreme exhaustion, take a sleep history. Many patients who complain of hypersomnia when starting amino acids have poor sleep histories prior to treatment. They will have to pay back the acquired “sleep debt” prior to being able to attain normal sleep. In extreme cases, the patient may have to stop their amino acid dosing and restart the amino acids on a Friday. Instruct the patient to plan on sleeping all weekend. Patients with insomnia have a “sleep debt” that prevents them from sleeping normally. When trying to achieve normal sleep habits, changes in lifestyle and the new allocation of time may also pose a challenge to patients.
Parkinsonism

The Parkinson model has profound implications for all neurotransmitter dysfunction diseases. The system damage model in humans exposed to the neurotoxin MPTP, which in turn induces Parkinsonism by damaging the dopamine bundles of the substantia niagra, has profound implications for the rest of the master neurotransmitter dysfunction diseases. The cause of chronic suffering due to other neurotransmitter diseases may also be permanent neuron bundle damage of the other master neurotransmitter as is found in Parkinsonism.

Treatment of Parkinsonism with amino acid precursors of the master neurotransmitters needs to be guided by urinary neurotransmitter testing. Treating Parkinsonism is different from caring for other neurotransmitter diseases. Dopamine levels need to be established in The Phase 3 Response™ at a level of 6,000 to 8,000 micrograms per gram of creatinine versus 300 to 600 micrograms of dopamine per gram of creatinine for other diseases. Serotonin needs to be in The Phase 3 Response™ at a level of 800 to 2,400 micrograms per gram of creatinine. In rare cases, patients have required urinary dopamine levels in The Phase 3 Response™ as high as 14,000 to achieve relief of symptoms.

To start treatment, begin the patient on level 1 of the 5-HTP/tyrosine/L-dopa protocol (see page 9). In one week, start Mucuna Pruriens - 40% standardized 2 pills, 3 times a day with a properly established tyrosine base. On the third visit, obtain a neurotransmitter test and follow the recommendations.

In general, it takes 2 to 4 months and the use of 4 to 8 urinary neurotransmitter tests to stabilize a Parkinson’s patient. The use of amino acid precursors and urinary neurotransmitter testing has led to clinical outcomes and long-term patient positioning that is superior to other current clinical approaches.

Managing Parkinsonism More Effectively Than L-dopa / Carbidopa

A combination of L-dopa and Carbidopa are the primary medicines used in the treatment of Parkinsonism. Our research suggests that most of the problems and side effects encountered with long-term L-dopa/Carbidopa use are related to mismanagement of the amino acids, L-dopa and Carbidopa.

- Using plain L-dopa depletes serotonin. This results in serotonin driven depression in Parkinson patients that responds to highly selective serotonin drugs, such as Citalopram. Whenever supplemental L-dopa is administered, it needs to be in proper balance with a serotonin precursor (5-HTP is preferred) in proper amounts in order to prevent depletion of serotonin by unopposed L-dopa during long-term use.
- Using plain L-dopa causes depletion of the sulfur amino acid system. To properly manage, administer the cysteine, selenium, and folate formula 2 pills, 3 times a day with doses at noon, 4 PM and bedtime. Problems associated with sulfur amino acid depletion include:
  - Dyskenesias
  - Depletion of glutathione
  - Depletion of SAMe and epinephrine
- Long-term use of L-dopa leads to tachyphylaxis of L-dopa. Our research suggests that this is primarily due to the depletion of the serotonin system by L-dopa.
- Carbidopa (used in combination with L-dopa to treat most Parkinson patients) inhibits the peripheral synthesis of serotonin and dopamine, leading to:
  - Weakness
  - Muscle fasciculation
Specific Disease Considerations

**Parkinsonism:** Start the Parkinson patient on the level 1 amino acid protocol with L-dopa (Mucuna Pruriens, see page 9). The patient should return to the clinic in one week.

At the second visit, add 2 capsules of Mucuna Pruriens, 3 times a day with proper tyrosine base. The patient should return to the clinic in one week. At which time, a urinary neurotransmitter test should be ordered. The patient should return in 7 to 10 days to discuss the results of the test. After the neurotransmitter results are returned, follow the recommendations until the patient’s symptoms are under control or until urinary serotonin and dopamine levels are in the desired phase 3 range.

The goal of treatment is to establish urinary serotonin in the phase 3 response and therapeutic range (800 to 2,400 micrograms of serotonin per gram of creatinine). Urinary dopamine should be established in the range of 6,000 to 8,000 micrograms of dopamine per gram of creatinine (although we have seen rare patients who need urinary dopamine levels as high as 14,000 to experience relief of symptoms).

In general, it take 6 to 8 urinary neurotransmitters tests to obtain relief of symptoms in the Parkinson patient.

**ADHD/ADD:** In children with ADHD and ADD, start the patient at 1/2 the adult level, found on page 9. In one week, obtain a urinary neurotransmitter test and follow the reported recommendations. Variance in the response to amino acids is too great in children to define a second step of amino acid dosing without first performing a urinary neurotransmitter testing. Therefore, the recommendation for treatment of pediatric ADHD and ADD patients is to simply start them on the initial amino acid dosing. The patient should return in one week to obtain a urinary neurotransmitter test of the master neurotransmitters. The patient should return in one week to discuss test results.

Pediatric amino acid dosing is defined as children 16 years of age or less. Although, when urinary neurotransmitter testing is used, it is obvious that the age lines can blur. For example, we have seen 10- and 11-year old children who ultimately appear to have adult dosing needs and 20-year olds that appear to have pediatric dosing needs (based on testing results).

When treating adults with ADD, start patients on the level 1 dosing. If symptoms do not improve, weekly increase dosing levels until level 3. If symptoms are not under control after a week at level 3, obtain a neurotransmitter test and follow the recommendations until the patient is stabilized.
Pediatric Patients

Amino acids are safe and non-toxic. We have an abundance of experience in treating pediatric patients. Physicians have treated patients as young as 2-years-old without adverse reactions or problems. When treating pediatric patients, it is recommended to use the 5-HTP/tyrosine protocol (see page 9). The starting dose for pediatric patients is 1/2 the adult dose (i.e. 2 pills of 5-HTP/tyrosine twice a day with 1 pill, 3 times a day of the cysteine, selenium, and folate formula).

Do not adjust the dosing in one week (as is done in adults patients who have not experienced relief of symptoms after one week). Instead, obtain a neurotransmitter test and follow the recommendations. Pediatric dosing needs vary too greatly to be able to do an amino acid dosing adjustment on the second visit without lab guidance. Testing is necessary in order to adjust amino acids. Continue testing and adjusting amino acids until the patient’s symptoms are under control or until the patient’s serotonin and dopamine levels arrive at the phase 3 therapeutic response.

For more information on amino acid dosing levels, contact NeuroResearch at 877-626-2220.

(ADHD) Attention Deficit Hyperactivity Disorder

Baseline urinary neurotransmitter testing is not recommended for patients prior to treatment. It has no diagnostic value. It is unable to determine the needed amino acid dosing. However, the unique starting point of ADHD holds the following considerations.

In studying ADHD patients (adult and pediatric) who are not under treatment with amino acids, a very distinct pattern emerges in urinary testing. Urinary catecholamines, in general, are markedly elevated. This pattern is so distinct that it leaves us questioning whether the patient would actually have ADHD if the urinary catecholamines had not been elevated prior to treatment. The problem is that the kidneys are manufacturing dopamine and norepinephrine, which are then inappropriately dumped into the urine, instead of into the system. Amino acid therapy effectively deals with and controls this problem. Treat pediatric ADHD by starting the patient on the level 1 protocol, 5-HTP/tyrosine treatment (2 pills, twice a day) with cysteine, selenium, and folate formula (1 pill, 3 times a day). Obtain a urinary neurotransmitter test in one week and follow the recommendations.
ADHD Drugs
Adderall®  Ritalin®  Concerta®

Prescription drugs Adderall®, Ritalin®, and Concerta®, which are now used extensively in the treatment of Attention Deficit Hyperactivity Disorder (ADHD) are of concern. Adderall® is an amphetamine. Ritalin® and Concerta® are dopamine/norepinephrine reuptake inhibitors. Of the three drugs, it is our opinion that Adderall® is the least desirable for use due to its potential long-term permanent effects.

In general, amphetamines are associated with neurotoxicity (permanent damage to neurons of the central nervous system). Studies performed by NeuroResearch have demonstrated an increased incidence of neurotransmitter dysfunction diseases in patients with a history of amphetamine ingestion. Ritalin® and Concerta® are dopamine/norepinephrine reuptake inhibitors. These drugs do not increase the number of dopamine and norepinephrine molecules in the central nervous system. They work by moving dopamine and norepinephrine molecules from the vesicles of the pre-synaptic neuron to the synapse.

In the process, they facilitate a more effective firing of the electrical impulses across the synapse. But, there is no free lunch. While in the pre-synaptic vesicles, dopamine and norepinephrine are not exposed to the enzymatic breakdown of the COMT and MAO enzyme systems. Once outside the vesicles, these neurotransmitters are exposed to the COMT and MAO enzymes, causing an increase in the metabolism of dopamine and norepinephrine.

Over time, the overall number of dopamine and norepinephrine molecules in the central nervous system is further depleted. If you need to use Ritalin® or Concerta® to treat patients, they should be given serotonin and dopamine amino acid precursors in proper balance. This will properly balance the neurotransmitters to prevent further depletion by these drugs.

At present, we have a study under way with the cooperation of a large school district in Texas. Preliminary studies in treating children with ADHD using only properly balanced amino acid precursors have shown great promise.

Research has shown that properly using balanced amino acid precursors of dopamine and serotonin is a highly effective means of treatment. Most importantly, there are no neurotoxicity issues or further depletion of neurotransmitters as occurs when prescription drugs are used to treat ADHD.

The starting point for treatment of ADHD in children is the tyrosine/5-HTP with cofactor formula developed by NeuroResearch (see page 9). It is recommended that children (16 years of age or less) be started on one half the adult dosing (2 pills in the AM and at 4 PM). The following week, obtain a urinary neurotransmitter test and follow the recommendations. Adjusting amino acid doses in children beyond the starting dose without a urinary neurotransmitter test is not recommended. The response of children to amino acids varies greatly and is not as predictable as in adults.

For more information on ADHD Treatment, call 877-626-2220.
Migraine Headaches

85% of migraine headaches resolve with amino acid therapy. In studying this problem, our research suggests that the remaining 15% who do not experience relief have been misdiagnosed in the past or have adopted the diagnosis on their own. They do not have true migraine headaches.

In general, migraine headaches resolve on a level 1 or 2 dosing of amino acids. The response to amino acids is so consistent that if a patient does not achieve complete relief of migraine headaches by the level 3 dosing level, it is recommended that the care giver look for another etiology to explain the headaches.

Anti-Aging Applications

Virtually all eight systems addressed by anti-aging medicine are controlled or impacted by the master neurotransmitters. The first step to a comprehensive approach for anti-aging treatment is to optimize the master neurotransmitters. Baseline testing of the eight systems addressed by anti-aging medicine prior to optimizing the master neurotransmitter levels, when that is the goal, is a waste of money and time. These systems should not be addressed until after the master neurotransmitters are optimized. In the process of optimizing the master neurotransmitters, baseline labs performed on these systems have no correlation with lab testing once the master neurotransmitters have been optimized.

Chronic Pain

In general, chronic pain, chronic stress, chronic disease, and chronic illnesses deplete neurotransmitters. In patients suffering from chronic states, there is an increased incidence of other neurotransmitter diseases and illnesses occurs.

The first step in managing chronic pain is to optimize neurotransmitters with amino acids (guided by laboratory testing, as needed). Once neurotransmitters are optimized, the patient’s ability to deal with pain and associated stress increases greatly. This results in easier management of pain with pharmacologic methods. The power of neurotransmitter optimization for chronic pain management can not be understated.

In general, 85% of migraine headaches resolve with amino acid therapy. In studying this problem, our research suggests that the remaining 15% who do not experience relief have been misdiagnosed in the past or have adopted the diagnosis on their own. They do not have true migraine headaches.

In general, migraine headaches resolve on a level 1 or 2 dosing of amino acids. The response to amino acids is so consistent that if a patient does not achieve complete relief of migraine headaches by the level 3 dosing level, it is recommended that the care giver look for another etiology to explain the headaches.

Anti-Aging Applications

Virtually all eight systems addressed by anti-aging medicine are controlled or impacted by the master neurotransmitters. The first step to a comprehensive approach for anti-aging treatment is to optimize the master neurotransmitters. Baseline testing of the eight systems addressed by anti-aging medicine prior to optimizing the master neurotransmitter levels, when that is the goal, is a waste of money and time. These systems should not be addressed until after the master neurotransmitters are optimized. In the process of optimizing the master neurotransmitters, baseline labs performed on these systems have no correlation with lab testing once the master neurotransmitters have been optimized.

Chronic Pain

In general, chronic pain, chronic stress, chronic disease, and chronic illnesses deplete neurotransmitters. In patients suffering from chronic states, there is an increased incidence of other neurotransmitter diseases and illnesses occurs.

The first step in managing chronic pain is to optimize neurotransmitters with amino acids (guided by laboratory testing, as needed). Once neurotransmitters are optimized, the patient’s ability to deal with pain and associated stress increases greatly. This results in easier management of pain with pharmacologic methods. The power of neurotransmitter optimization for chronic pain management can not be understated.
Hormones

The hormone system is one of the eight systems addressed by anti-aging medicine. From a master neurotransmitter standpoint, hormone synthesis is controlled by norepinephrine, among other factors.

If hormone problems need to be addressed, it is recommended that the master neurotransmitters be optimized prior to testing or addressing hormones. The master neurotransmitters control hormone production. They have a powerful effect on baseline testing. Prioritize treatment and address one system at a time, instead of trying to treat everything at once. Treatment involving hormones and neurotransmitters that is not prioritized will result in unpredictable changes that are difficult to control. Once serotonin and dopamine are at desired levels, there is no further manipulation of their amino acid precursors. It takes 2 to 6 weeks for norepinephrine to stabilize. Meaning, it takes 2 to 6 weeks for hormone levels (whose synthesis is controlled by norepinephrine) to stabilize.

Adrenal Burnout

Prolonged stress has been implicated as a cause of adrenal burnout. In patients diagnosed with one or more neurotransmitter dysfunction diseases, urinary testing prior to treatment indicates that 79% have low epinephrine levels. Norepinephrine is the precursor of epinephrine. Norepinephrine controls the synthesis of Cortisol. Cortisol controls the synthesis of PNMT, the enzyme that converts norepinephrine to epinephrine. The synthesis of epinephrine is dependant on two limiting factors - PNMT and SAMe. Proper use of amino acids, as guided by urinary neurotransmitter testing, restores epinephrine levels to normal. Once serotonin and dopamine levels are in the therapeutic range and in The Phase 3 Response™, it can take 3 to 6 months for the epinephrine levels to be restored to normal. Once epinephrine levels are restored to normal, it can be assumed that cortisol and SAMe are once again functioning normally.
Obsessive Compulsive Disorder (OCD), Bulimia and Anorexia

The symptoms of OCD, Bulimia, and Anorexia are relatively easy to control with amino acid therapy. The problem in treating all three of these diseases is that the relapse rate is very high if proper psychotherapy is not provided. Controlling the symptoms is just the first step to recovery from these complex diseases. The other step is providing proper psychotherapy in order to allow patients to properly adjust and reintegrate back into a normal life.

OCD

In general, the repetitive behaviors of OCD will need amino acids to be adjusted to the level 3 protocol followed by urinary neurotransmitter testing in order to achieve symptom resolution. Amino acid therapy needs to be used in conjunction with neurotransmitter testing to fine-tune dopamine levels. NeuroResearch has found that resolution of the repetitive symptoms of OCD is relatively easy to achieve. In order to prevent patients from dropping out of treatment or from relapsing, proper psychotherapy must be provided. The profound changes that occur in the patient’s life as symptoms resolve often lead to personal and social conflicts.

Bulimia

The repetitive binge and purge cycles of Bulimia are relatively easy to control with amino acid therapy, when guided by laboratory testing. As with OCD, the profound changes that patients experience can lead to personal conflict. When personal conflict is not managed properly with psychotherapy, many patients stop therapy or relapse.

Anorexia

Anorexia has been called “the flip side” of Bulimia. Patients starve themselves, rather than overeat. While amino acid therapy was originally examined and used for weight loss, research has shown that it is also effective in treating other eating disorders. Amino acid therapy corrects the unbalanced system back to normal. An unbalanced system can produce a variety of diseases and disorders, which is why amino acid therapy has so many applications.

Anorexia is a deadly disease. NeuroResearch recommends that only experienced caregivers, with formal training in the treatment of Anorexia, use amino acids with psychotherapy and other needed medical support to provide care to these patients.
Obesity

The appetite center of the brain is controlled by serotonin and/or norepinephrine. This assertion is based on the fact that the only drugs in medicine which control appetite and allow patients to comfortably eat less food are those drugs that work with serotonin and/or norepinephrine.

Obesity is the most difficult neurotransmitter disease to treat. It involves resolving symptoms of hunger by balancing the neurotransmitters, as well as introducing new lifestyle choices to patients. Obesity is responsible for numerous secondary diseases and illnesses. The following two pages are an overview of the five most crucial factors needed for optimal weight loss. They do not represent all of the factors needed.

NeuroResearch’s approach to medical weight loss was designed based on the extensive statistical analysis of our research database. It contains information on the clinical treatment of weight loss patients. The NeuroResearch protocols were developed and refined with the goal of easily and effectively maximizing group weight loss.

16.9 lbs. = average weight loss in the first month.
8.4 lbs. = average monthly weight loss for the first 12 months of treatment.

To obtain these results, the following five factors must be in place (listed in order of importance).

- Patient motivation
- On-time clinic visits
- Monitoring caloric intake
- Properly taking pills
- Using computer printouts properly

**Patient Motivation**

The most important indicator of success is motivation. Patients are the most motivated during the first month of treatment. In the first month, patients must learn to perfect the skills needed to ensure that they are able to meet the requirements for success. Patients must attend scheduled clinic appointments, properly monitor and journal their caloric intake, and properly take their pills. It takes perfecting these skills in the first month to face the final months, which are the most difficult because weight loss slows down and motivation is tested. The average patient in the NeuroResearch database desired to lose 94 pounds at the start of treatment. It takes motivation and commitment to be able to endure the months of treatment that are required to lose almost 100 pounds.

Improperly adjusted amino acids can cause a patient to feel hungry, which can wear out a patient’s willpower and deplete their motivation. If a patient is still hungry one week after an amino acid dosing change occurs, the amino acid dosing needs to be adjusted again.

As a caregiver, you can nurture motivation, but you cannot give it to the patient. Patients who are struggling at the end of the first month are not properly positioned to make goal weight. A heart-to-heart talk may be needed to determine the patient’s level of motivation. With poor motivation, even properly positioned appetite suppression can be unconsciously overridden.

**On-Time Clinic Visits**

In the first month, patients need to be seen every week until their weight loss and appetite are under control. Once the first month has passed, patients whose appetites are under control and are properly losing weight may come every 2 weeks. If, at any visit, the patient complains of hunger or is not losing enough weight, they need to be seen in one week. The best way to get patients back into the clinic on-time is to give them only enough pills to make it to the next visit. This is why 56 pills bottles are available. This is enough to provide one patient for one week of treatment.
Monitoring Caloric Intake
To achieve goal weight, patients will have to eat the appropriate number of calories. Patients need to be properly positioned from the start of treatment to eat at the appropriate caloric level. Retraining patients to eat at a lower level is very difficult, if not impossible after the first month of treatment. To truly be counting calories, patients need to use measuring cups, weigh their food, and read labels. The calories in food need to be measured, guessing at a food’s calorie count will not be sufficient. This is the only way for patients to know exactly how many calories they are consuming.

Patients who eyeball their portions rather than measuring exactly tend to consume 400 to 600 more calories per day. This will prolong the time it takes to make goal weight, which will make maintaining motivation more difficult. This could make losing the last 10 pounds nearly impossible. For patients that are struggling, it may be necessary to recommend a dietician or some form of behavioral modification therapy.

\[ \text{CALRX} = (\text{goal weight} \times 10) - 500 \]

This calorie prescription (CALRX) formula positions patients to lose the last 10 pounds of weight at a rate of 1 to 1 1/4 pounds per week.

When patients eat the exact number of prescribed calories per day, it takes patients 2.1 months to lose the last 10 pounds of weight. It takes only a few extra calories to greatly slow the rate of weight loss. For example, if a patient with a goal weight of 140 pounds is placed on a CALRX of 900 calories per day, it will take this patient 2.1 months to lose the last 10 pounds. If the same patient eats an additional 150 calories, three times a day (for a total caloric intake of 1,350 calories per day) the time to lose the last 10 pounds increases to 12.8 months.

The NeuroResearch computer program is free of charge. It is served over the website at [www.usaweightloss.com](http://www.usaweightloss.com). It helps caregivers ensure that their patients will always be properly positioned to make goal weight.

Taking Pills Properly
It generally takes 3 to 5 days for the effects of starting or changing an amino acid dosing to be observed. To allow the patient’s system to properly adjust, dosing changes should not be made until 7 days have passed since the last change.

Patients should be on a consistent number of pills. There should not be different doses for different days. This causes the patient’s system to fluctuate, which makes appetite control impossible.

Missing one or two doses of pills can leave the patient hungry for 3 to 5 days until the neurotransmitter levels return to their previous state. Patients whose appetites were under control and return to the clinic complaining of hunger have likely missed one or more doses. For the next week, these patients will need to journal all of their pills taken with the times that they were taken. After one week, patients that have properly taken all of their pills and return complaining of hunger will need a urinary neurotransmitter test. This allows the caregiver to see if the patient’s needs have changed.

Computers in Weight Loss
Proper use of the computer program increases group weight loss by 60%. Using printouts and graphs, the program allows caregivers to quickly determine if the patient is properly positioned to make goal weight, which allows them to make changes accordingly.

Use of the computer program is taught by NeuroResearch over the telephone (by appointment only). The program is served at the website, [www.usaweightloss.com](http://www.usaweightloss.com), which is password protected. The computer program is free of charge, but it is only available to clinics that have been trained at one of our seminars.

In the past NeuroResearch taught a two-day training program with the second day dedicated to weight loss. We have now gone to a one-day format. We have a free teaching disk with learning materials available covering all the weight loss slides and topics of the second day of training. Call NeuroResearch to order a copy of these training materials (see page 6).
Neurotransmitter drugs do not work if there are not enough neurotransmitters with which to work. Drugs that redistribute neurotransmitters from one place in the brain to another, such as reuptake inhibitors and excretors, deplete neurotransmitters in most patients as a result of long-term use. When this happens, the drugs can no longer work effectively and symptoms return.

The recommendation for treatment is to leave patients on any prescription drugs they may be on at the start of amino acid therapy. Once patients are stabilized on balanced amino acids, it is up to the discretion of the caregiver whether or not a change in prescription drug dosing is needed. Many patients, once their systems are optimized, are able to have their prescription drug dosing lowered or stopped.

When patients with diminished effects of prescription drugs are started on amino acid therapy, not only will the effects of the drugs once again become fully evident, but the side effects of the drugs may also become evident. This occurs in approximately 5% to 10% of patients. The emergence of a prescription drug’s side effects is more prominent in patients taking drug doses that are higher than the starting dose. Caregivers should use a PDR to determine a prescription drug’s side effects. Most of the side effects, other than those listed at the left, in patients taking prescription drugs that work with neurotransmitters are due to the prescription drug, not the amino acids. Therefore, the recommendation is usually to lower the daily dosing of the prescription drug rather than cutting back on the amino acid dosing.

### Amino Acid Side Effect Profile

#### Statistical Analysis of the side effect profile
- Side effects are based on surveys of patients during clinic visits.
- Analysis is based on the random grouping of 494 patients.
- Analysis represents 1,604 patient visits.
- Patients used for analysis were only being treated with amino acids.
- Patients were randomly selected from practices that have mastered amino acid therapy.
- The data represents over 50 patient-years of treatment.

<table>
<thead>
<tr>
<th>Side Effect</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry mouth</td>
<td>34 (2.1%)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>14 (0.9%)</td>
</tr>
<tr>
<td>Headache</td>
<td>12 (0.7%)</td>
</tr>
<tr>
<td>Nausea</td>
<td>10 (0.6%)</td>
</tr>
<tr>
<td>Dizziness</td>
<td>6 (0.4%)</td>
</tr>
<tr>
<td>Constipation</td>
<td>6 (0.4%)</td>
</tr>
</tbody>
</table>

The following side effects were reported at the rate of 0.2% or less, which is 4 or less reports from 1,604 patient visits. The numbers in parentheses represent the number of incidences.

- Cold extremities (1)
- Depression (1)
- Abdominal Burning (1)
- Abdominal Pain (1)
- Spots before Eyes (1)
- Flushed Face (1)
- Fingers Tingling (1)
- Hypoglycemia (1)
- Drowsy (2)
- Irritability (2)
- Sweats (2)
- Jittery (2)
- Flatulence (2)
- Light Headed (2)
- Thirst (2)
- Sore Tongue (Glossitis) (2)
- Non-Specific Dermatitis (2)
- Moodiness (2)
- Cravings (4)
- Diarrhea (4)
- Fatigue (4)
- Palpitations (4)

### Prescription Drug Side Effects

Neurotransmitter drugs do not work if there are not enough neurotransmitters with which to work. Drugs that redistribute neurotransmitters from one place in the brain to another, such as reuptake inhibitors and excretors, deplete neurotransmitters in most patients as a result of long-term use. When this happens, the drugs can no longer work effectively and symptoms return.

The recommendation for treatment is to leave patients on any prescription drugs they may be on at the start of amino acid therapy. Once patients are stabilized on balanced amino acids, it is up to the discretion of the caregiver whether or not a change in prescription drug dosing is needed. Many patients, once their systems are optimized, are able to have their prescription drug dosing lowered or stopped.

#### Amino Acid Side Effect Profile

If amino acids are used properly, patients should not need to stop amino acid therapy on a long-term basis.

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If amino acids are used properly, patients should not need to stop amino acid therapy on a long-term basis.
Managing Side Effects and Adverse Reactions

Management of Start-Up Problems

GI upset upon starting amino acids occurs in patients that have severely depleted neurotransmitters. Severely depleted patients need amino acid therapy. Patients who have been on drugs that deplete neurotransmitters (in the last 18 to 24 months) may experience an increased rate of GI upset. A nutrient poor diet may also contribute to GI upset. There are also several less common reasons for GI upset. The easiest way to prevent patients from dropping out of treatment (as a result of GI upset) is to instruct patients at the beginning that if they experience GI upset, they should stop taking their pills until they are able to return to the office. When a patient returns complaining of GI upset, or other start-up problems, proper management involves cutting the patient’s dose back to one pill at bedtime. Instruct the patient that bedtime is when they are ready to fall asleep, not when they are getting into bed to read a book. Patients that take one pill and fall asleep within 15 to 20 minutes tend to report that their GI upset has disappeared. Some patients have reported that eating one or two soda crackers with their pills helps prevent start-up GI upset.

Management of Start-Up Problems

After a patient has been free of their start-up complaints for 3 to 4 days, add an additional pill at bedtime. Continue to increase the dosing in a similar manner every 3 to 4 days until the patient is taking 4 pills at bedtime. Once the bedtime dosing reaches 4 pills, start adding one pill in the morning every 3 to 4 days, as long as the patient is complaint-free. Continue to add pills until the patient is taking 4 pills in the morning and 4 pills in the evening. Once the patient is taking 8 pills a day, amino acid dosing can be altered using the standard amino acid protocols with the aid of clinical observations and laboratory testing.

It generally takes 3 to 4 weeks for depleted patients to reach the standard starting dose of amino acids (8 pills a day). Statistical analysis has found that the incidence of GI upset at the start-up of amino acid therapy is 0.5%. Practices that have a higher than average number of patients with depleted neurotransmitters will see a higher incidence of GI upset. The most common example is clinics that are treating drug addicts, since using drugs depletes neurotransmitters.
Managing Side Effects and Adverse Reactions

Carbohydrate Intolerance

As our research led us to using higher amino acid doses, we began seeing an increase in the number of patients who were experiencing GI upset several weeks or months after relief of symptoms was experienced. Initially, we thought that the GI upset was the result of neurotransmitter depletion. However, this explanation did not seem logical. We wondered, “Why would patients whose pills were properly adjusted be experiencing signs of neurotransmitter depletion later in treatment?” It made more sense that they would experience GI upset as a result of neurotransmitter depletion at the start of amino acid therapy. Our intensive research led us to the correct answer.

Patients who were fully adjusted on their amino acids and experiencing GI upset were suffering from carbohydrate intolerance. This form of GI upset is most commonly seen in weight loss patients. They tend to have severely depleted neurotransmitters and need higher levels of amino acid doses. As weight loss patients obtain appetite suppression, many experience changes in their physical responses to food. Often, one of the greatest changes is the patient’s response to carbohydrates.

If patients are properly managed, side effects should not require patients to stop amino acids on a long-term basis. If you have a patient with difficult to manage side effects, call NeuroResearch at 877-626-2220. We can help.

Patients do not experiencing GI upset from all carbohydrates. Carbohydrate intolerance is selective. It usually results from one carbohydrate (usually a carbohydrate that is eaten regularly). The most common carbohydrates that cause problems are breads, noodles, and cereals. If patients make note of what they have eaten prior to experiencing GI upset, it is often easy to identify which carbohydrates are creating a problem.

GI upset is more likely to occur in the morning, usually around 2 to 3 hours after breakfast. Although, GI upset can be seen anytime during the day. The most common foods to cause GI upset are breads, noodles, and cereals. The easiest way to treat this form of GI upset is to have the patient remove or change the food(s) that causes GI upset.

Usually changes like switching from white bread to whole wheat bread, changing the type of noodle eaten, or changing a cereal that is eaten regularly will allow the patient’s GI upset to be managed successfully.
Managing Side Effects and Adverse Reactions

The amino acid formulas and protocols of NeuroResearch were formulated to optimize group efficacy AND minimize side effects. Using amino acid combinations outside of the NeuroResearch amino acid protocol, unless guided by laboratory testing, will result in a decrease in efficacy and an increase in side effects.

GI Upset from Cysteine
Cysteine may cause GI upset. 20% of patients taking cysteine in the morning experience GI upset. The mechanism of action is unknown. But our research has found that if patients wait until noon to take their first dose, GI upset can be eliminated. The remaining doses of the cysteine/selenium/folate formula should be taken at 4 or 5 PM and again at bedtime. Therefore, the recommendation is that all patients on the cysteine/selenium/folate formula take three evenly divided doses (with doses at noon, 4 PM, and bedtime).

Paradoxical Reactions
A paradoxical reaction occurs when neurotransmitter disease symptoms become more pronounced at the start of amino acid therapy or when amino acids are adjusted. For example, a weight loss patient might complain of increased hunger, a patient with migraine headaches might complain of increased headaches, or a patient with depression might complain of increased depression. There are numerous examples of paradoxical reactions, that relate to increased neurotransmitter disease symptoms. Many caregivers respond to paradoxical reactions by lowering the patient's amino acid dose. However, patients actually need an increase in their dose to get through the paradoxical reaction and achieve relief of disease symptoms. By decreasing the dose and then slowly increasing amino acids, patients will experience prolonged suffering with their symptoms. This will increase the time that the patient experiences distress, as well as increase the probability that the patient will drop out of treatment.
Paradoxical Reactions

When an amino acid dosing is started or increased, some patients experience an increase in their neurotransmitter disease symptoms. This is known as a “Paradoxical Reaction.” Some common examples of paradoxical reactions are:

- Depression becomes worse.
- Sleep becomes worse.
- The weight loss patient’s appetite increases.
- Anxiety becomes worse.
- Migraine headaches become worse.

Any neurotransmitter dysfunction disease can display a paradoxical reaction during treatment.

If you encounter a paradoxical reaction in your patients, treat by increasing the amino acid dosing to the next level of treatment. Physicians are trained to decrease or stop prescription drugs if a problem is encountered. This is exactly the opposite of what needs to be done with amino acid associated paradoxical reactions. When treating with amino acids, decreasing the dose and then bringing it up slowly will greatly prolong the time that patients experience an exacerbation of symptoms.

In general, paradoxical reactions occur in the first two weeks of treatment. However, in Parkinson patients, paradoxical reactions commonly occur after the patient has been treated for several weeks and dopamine is ready to inflect into the therapeutic range as the amino acid dose is increased. While this primarily happens with Parkinson patients, in rare cases, it can happen with other diseases. In these cases, the paradoxical reaction occurs when amino acids are adjusted and the patient is on the verge of relief of symptoms. Paradoxical reactions indicate a need to increase the amino acid dosing. If you spot a paradoxical reaction when obtaining a urinary neurotransmitter test, simply increase the amino acid dosing (prior to the laboratory results returning or call NeuroResearch at 877-626-2220 for a consult if you are awaiting lab results).
Managing Side Effects and Adverse Reactions

Heartburn
Heartburn that occurs after taking pills can easily be managed. The amino acid pills are large and can become stuck in the esophagus. When this occurs, it produces an irritation that leads to heartburn. To properly manage heartburn, have patients hold the pills in their mouths for 10 to 15 seconds with a small amount of water. As the surface of the pills start to dissolve, instruct the patient to swallow. This allows the pills to travel down the esophagus without getting stuck. Some patients may need to take one pill at a time in order to successfully move the pills down the esophagus. For patients where this is not effective, have them twist open the capsules and dissolve the powder in water or juice, and then drink the combination.

Dizziness
Patients complaining of dizziness need to have a history taken. Many patients complaining of dizziness have previously experienced dizziness when they missed a meal. Dizziness is often related to carbohydrate addiction. To properly manage dizziness, increase the patient to the next dosing protocol. The mechanism of action for improving the dizziness is complex and beyond the scope of this discussion.

Hypersomnolence
Patients that complain of extreme exhaustion when they start amino acid therapy need to have a sleep history taken. Many patients who complain of exhaustion have poor sleep histories. They will need to pay back the acquired “sleep debt” prior to sleeping normal. The easiest way to manage hypersomnolence is to have the patient start the amino acids on a Friday and spend the weekend sleeping. When trying to achieve normal sleep habits, changes in lifestyle and the new allocation of time may pose a challenge to patients.
Neurotransmitters found in the urine are not systemic neurotransmitters filtered by the kidneys. They are neurotransmitters that are synthesized by the kidneys. Therefore, urinary neurotransmitter levels have no correlation with systemic neurotransmitter levels. For most diseases, this excretion by the kidneys is inappropriate and neurotransmitters are excreted into the urine instead of being secreted into the system. This leads to low systemic neurotransmitter levels and high urinary neurotransmitter levels (in patients not under treatment with amino acids that have been diagnosed with a neurotransmitter dysfunction disease). **Urinary serotonin and dopamine levels in The Phase 3 Response™ and in the therapeutic range correlate with the resolution of disease symptoms and an optimal feeling of well-being.**

Urinary serotonin AND dopamine levels must be in the therapeutic range AND in The Phase 3 Response™ in order for the optimal relief of symptoms and an optimal feeling of well-being to be established. Every DBS Lab report outlines serotonin and dopamine phases. In addition, each lab report provides recommendations for amino acid treatment.
Baseline Neurotransmitter Testing Has No Value!

Baseline neurotransmitter testing prior to treatment with amino acids has no value. From a practical standpoint, urinary master neurotransmitters in a patient not taking supplemental amino acid precursors versus when the patient is taking supplemental amino acid precursors appears to be two distinctly different populations of neurotransmitters. Lab testing shows no correlation between the two populations. Testing measures the response of the kidneys to the amino acids administered. Patients that are not taking amino acids have random results relative to the results in patients taking amino acids (i.e., there is no correlation between the two).

Projects attempting to integrate baseline testing into patient treatment have been met with little or no success in optimally controlling disease symptoms in group treatment. Proper use of urinary neurotransmitter testing is an "amino acid challenge test." The patient is started on amino acid precursors of serotonin and dopamine. One week later a urinary neurotransmitter test is preformed.

After which, the amino acid dosing is changed and a second test is performed in one week. The results of the two tests are compared in order to determine the urinary phases of dopamine and serotonin. The dopamine and serotonin phases are used to formulate a plan for further adjusting amino acid dosing.

Baseline testing prior to starting amino acids has no value. Despite this fact, approximately 15% of the samples submitted to DBS Labs are from patients not taking amino acids. This is a waste of time and money. Baseline testing has no value in making proper treatment decisions. Baseline testing prior to starting amino acids has no place in medical treatment.

Baseline neurotransmitter levels do not correlate with neurotransmitter testing once the patient begins taking amino acids. Neurotransmitters found in the urine are not systemic neurotransmitters filtered by the kidneys then excreted into the urine. They are neurotransmitters that are synthesized by the kidneys, excreted into the urine or secreted into the system via the renal veins.
Urinary neurotransmitters are not neurotransmitters filtered by the kidneys, then excreted into the urine. They are neurotransmitters synthesized by the kidneys.

Serotonin in the urine is synthesized by the kidneys
These findings provide further evidence that increases in urine serotonin after administration of serotonin precursors is largely due to serotonin synthesized within the kidney.

Dopamine in the urine is synthesized by the kidneys
Plasma dopa is the main source of urinary dopamine.

Dopamine in the urine is synthesized by the kidneys
This data indicates that urinary free dopamine is mainly derived from plasma dopa, which is converted by dopa decarboxylase in the kidneys.

Norepinephrine in the urine is synthesized by the kidneys
Perfusion of L-dopa and free dopamine led to the generation of norepinephrine in the kidneys. Renal nerves were the main sites of the norepinephrine synthesis.

Epinephrine in the urine is synthesized by the kidneys
The kidneys are a likely source for some urinary epinephrine. Urinary epinephrine is derived from the kidneys.
Kidney Int. 1997 Jan;51(1):324-7

Urinary neurotransmitters levels DO NOT:
- Correlate with central nervous system levels
- Correlate with peripheral nervous system levels
- Correlate with neurotransmitter illnesses caused by low levels of neurotransmitters
- Have the ability to diagnose diseases associated with low levels of neurotransmitters
- Determine initial amino acid doses to be used in treatment

Urinary Serotonin and Dopamine:
- When in the therapeutic range AND The Phase 3 Response™ correlate with the resolution of disease symptoms.

Urinary neurotransmitters are not neurotransmitters filtered by the kidneys and excreted into the urine. They are neurotransmitters synthesized by the kidneys.

Enough is enough, the emperor has no clothes!
In the first half of 2006, we have hosted and talked at 9 seminars and conferences around the U.S. In the process, we have spoke in front of over 300 physicians.
At every talk, we have physicians telling us that they have been led to believe that urinary neurotransmitters are systemic neurotransmitters filtered by the kidneys and excreted into the urine. They have been told that urinary neurotransmitters correlate with peripheral systemic neurotransmitter levels. This could not be further from the truth. The fact is that the systemic neurotransmitters filtered by the kidneys are metabolized by the MAO and COMT enzymes in the kidneys. Very little of these neurotransmitters make it to the final concentrated urine.

The urinary neurotransmitters serotonin, dopamine, norepinephrine and epinephrine are neurotransmitters that are synthesized by the kidneys, then excreted into the urine or secreted into the system via the renal veins. There is absolutely no correlation between urinary neurotransmitters and systemic neurotransmitter levels.
The Three Phases of Urinary Neurotransmitter Response

**PHASE 1**: is where urinary neurotransmitters synthesized by the kidneys are inappropriately excreted into the urine, when challenged with supplemental amino acid precursors, instead of being secreted into the system where neurotransmitter levels are low. This makes the cause of neurotransmitter diseases (low levels of neurotransmitters in the central and peripheral nervous system) worse. In phase 1, as the amino acid dosing is increased, the urinary neurotransmitter levels decrease. When patients are in phase 1, administering proper dosing levels of 5-HTP, in combination with tyrosine and/or L-dopa, reverses the inappropriate excretion of the master neurotransmitters by the kidneys. Patients suffering from master neurotransmitter dysfunction diseases will not be able to obtain enough of the master neurotransmitter precursors from foods (even foods high in the master neurotransmitter amino acid precursors) to reverse the inappropriate excretion.

**PHASE 2**: is where urinary neurotransmitters are no longer inappropriately excreted into the urine. In phase 2, the neurotransmitters synthesized by the kidneys are now primarily being secreted into the system where they are needed, but levels are still low. In phase 2, urinary neurotransmitter levels are low (less than therapeutic levels).

**PHASE 3**: is where, during the administration of serotonin and dopamine precursors, dosing reaches the point that urinary neurotransmitters synthesized by the kidneys (and neurotransmitter production in other parts of the body) are adequate. As a result, optimal relief of disease symptoms is obtained. In phase 3, systemic neurotransmitters have been restored to levels needed for the system to function properly. The excess synthesized neurotransmitters are now appropriately excreted into the urine. In phase 3, as the amino acid dosing is increased, the urinary neurotransmitter levels increase.
The Phase 3 Response™

- The Phase 3 Response™ applies only to urinary serotonin and dopamine levels.
- After urinary serotonin and dopamine levels are in the therapeutic range and in “The Phase 3 Response™”, it can take 2 to 6 weeks for norepinephrine to equilibrate and 3 to 6 months for epinephrine to equilibrate.
- Amino acid dosing should be adjusted so that urinary serotonin and dopamine levels move toward the therapeutic range in “The Phase 3 Response.” This allows optimal treatment results to be obtained.
- In rare instances, patients pass through phases 1 and 2 into “The Phase 3 Response™” on a low dose of amino acids.
- Two urinary neurotransmitter labs are needed, with patients taking two different amino acid doses, to determine with certainty the phases of urinary serotonin and dopamine.
- If urinary neurotransmitter levels are in the therapeutic range and in the phase 1 response, there may be little or no relief of symptoms.

When urinary serotonin and dopamine levels are in phase 1, urinary serotonin and dopamine levels will pass through phase 1 into phase 2, and then into phase 3, as the amino acid precursor dosing is increased.

- 86.6% of patients tested in the PM and 61% of patients tested in the AM (diagnosed by a licensed health care provider as having one or more neurotransmitter dysfunction diseases), who are not taking supplemental master neurotransmitter amino acid precursors have serotonin levels above the reference range reported by the lab. Testing in the AM may lead to missing the phase response in some patients.
- We have seen the rare patient who was taking:
  - 75 mg per day of 5-HTP with tyrosine - urinary serotonin was in phase 1.
  - 150 mg per day of 5-HTP with tyrosine - urinary serotonin was in phase 2.
  - 225 mg per day of 5-HTP with tyrosine - urinary serotonin was in phase 3.

Increasing the daily balanced amino acid dosing
Neurotransmitter Testing Overview

- Neurotransmitters found in the urine are not neurotransmitters from the system. They are neurotransmitters that are synthesized by the kidneys, and then excreted into the urine.
- All patients with illnesses related to master neurotransmitter dysfunction should be started on the same starting dose of amino acids (see page 9).
- Systemic neurotransmitters excreted into the dilute urine of the proximal tubules in the kidneys undergo uptake by the cation ports. They are metabolized. There are no significant amounts of systemic master neurotransmitters excreted into the urine.
- Urinary neurotransmitter testing to assist in the treatment of patients suffering from diseases caused by or associated with low levels of neurotransmitters is only of value when the patient is taking serotonin and dopamine amino acid precursors.

<table>
<thead>
<tr>
<th></th>
<th>Amino Acid Dosing Increases</th>
<th>Amino Acid Dosing Decreases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary neurotransmitter</td>
<td>Phase 3</td>
<td>Phase 1</td>
</tr>
<tr>
<td>levels increase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary neurotransmitter</td>
<td>Phase 2</td>
<td>Phase 2</td>
</tr>
<tr>
<td>levels are subtherapeutic (see below)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary neurotransmitter</td>
<td>Phase 1</td>
<td>Phase 3</td>
</tr>
<tr>
<td>levels decrease</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In phase 2, urinary serotonin levels are less than 800 micrograms of serotonin per gram of creatinine. Urinary dopamine levels are less than 300 micrograms of dopamine per gram of creatinine.

The Two Populations of Urinary Master Neurotransmitters

While testing of the master urinary neurotransmitters (serotonin, dopamine, norepinephrine, and epinephrine) is exactly the same from a laboratory standpoint. From a clinical and treatment standpoint, we have identified two distinct and separate populations of urinary master neurotransmitters.

1. The population of the urinary master neurotransmitters prior to taking amino acids.
2. The population of the urinary master neurotransmitters when taking amino acids.

Advanced statistical analyses coupled with clinical correlations have demonstrated that these two populations of neurotransmitters respond in completely different ways. There is no correlation between urinary neurotransmitter prior to starting amino acids versus after amino acids are started. Due to the existence of two distinct populations of urinary neurotransmitters, baseline testing prior to starting amino acids has no value unless your only goal is to prove that amino acids change urinary neurotransmitter levels. If the goal of treatment is the relief of disease symptoms, baseline testing has no value.
The Goal of Treatment
is to establish urinary serotonin and dopamine levels in “the phase 3 therapeutic range”. It should be noted that not all patients need to have both urinary serotonin and dopamine levels in the phase 3 therapeutic range for relief of symptoms (see page 43). In many cases, simply adjusting the acid dosing so the patient moves closer to the phase 3 therapeutic range for urinary serotonin and dopamine levels, induces relief of symptoms. Once relief of symptoms is attained, no further adjusting of amino acids or testing is required (unless disease symptoms return).

Therapeutic Ranges

Serotonin
- 800 to 2,400 micrograms of serotonin per gram creatinine.

Dopamine
- Most diseases
  - 300 to 600 micrograms of dopamine per gram creatinine.
- Restless leg syndrome
  - 1,800 to 3,000 micrograms of dopamine per gram creatinine.
- Parkinsonism
  - 6,000 to 8,000 micrograms of dopamine per gram creatinine.

THERAPEUTIC RANGES
(Only applies when urinary serotonin and dopamine are in “The Phase 3 Response™”)

When treating most patients, urinary neurotransmitter testing is only required after the patient has been on the level 3 protocol (page 9) for one week and the patient’s symptoms have not resolved.

To treat pediatric patients with ADHD, start the patient on one-half the adult level 1 amino acid dosing (page 9). Obtain a urinary neurotransmitter test in one week. Follow the recommendations when the lab results are reported back.

To treat Parkinsonism and Restless Leg Syndrome, start the patient on level 1 of the 5-HTP/tyrosine/L-dopa protocol (page 9). In one week, when the patient returns, start 240 mg of L-dopa (in the form of Mucuna Pruriens) 3 times a day with a tyrosine base. One week later, obtain a urinary neurotransmitter test. Follow the recommendations when the lab results are reported back.
Patients May Not Need to be Fully in “The Phase 3 Therapeutic Response™” for Relief of Symptoms.

If amino acids are appropriately adjusted (up to level 3), only 20 to 30% of patients need neurotransmitter testing for relief of symptoms. Many patients find relief of symptoms in the first two weeks of starting the amino acid formulas.

At the start of treatment, patients need to be seen weekly. If a patient's symptoms are not under control after one week on the level 3 dosing, obtain a urinary neurotransmitter test. When the test results return, follow the amino acid dosing recommendations. The goal of testing is to establish urinary serotonin and dopamine levels in the therapeutic range and in The Phase 3 Response™. Testing and amino acid adjustments should continue until the patient's symptoms are under control OR until urinary serotonin and dopamine levels are in the therapeutic range and The Phase 3 Response™.

On average, when dosing protocols are followed properly, patients need 1.81 urinary neurotransmitter tests to gain control of their symptoms. Although, one patient needed 13 tests in order to obtain relief of their symptoms. This was an unusually large number of tests. But, it was the first time in many years that the patient was symptom-free.

During treatment, most patients do not need urinary serotonin and dopamine levels in the therapeutic range and The Phase 3 Response™ in order to obtain relief of symptoms. In the process of adjusting amino acids, which moves them toward the bull's eye of the phase 3 therapeutic response, most patients experience relief of their symptoms. On the other hand, there are patients whose symptoms will not be under control until you hit the bull's eye by obtaining urinary serotonin and dopamine levels in the therapeutic range and The Phase 3 Response™.

Beyond the resolution of disease symptoms, is an “optimal feeling of well-being.” This develops when the patient's urinary serotonin and dopamine levels reach the therapeutic range and The Phase 3 Response™. It requires an amino acid dosing that is distinctly different from the dosing needed to achieve a resolution of disease symptoms. This feeling of optimal well-being appears to be more pronounced in patients over 50.
Proper Timing of Urine Sample Collection

Systemic neurotransmitters, in patients with neurotransmitter dysfunction diseases, have a diurnal variation throughout the day. The diurnal variation of urinary neurotransmitters in patients with neurotransmitter dysfunction diseases is the exact opposite of the normal systemic diurnal variation in most patients.

As the day progresses, urinary neurotransmitter levels increase in patients with neurotransmitter dysfunction diseases. Obtaining urinary lab samples in the AM has been the standard in medicine for years. However, this leads to a situation where the inappropriate excretion of neurotransmitters (phase 1) is missed. The Phase 3 Response™ may not be identified for proper treatment.

If the goal of testing is to identify the urinary neurotransmitter phase of serotonin and dopamine, the proper time to obtain urine samples for neurotransmitter testing is 5 to 6 hours before bedtime. In most patients this is around 4 PM, just before the late afternoon amino acid dose.

—— = Urinary neurotransmitter variation during the day in patients with neurotransmitter dysfunction diseases.

—— = Systemic neurotransmitter levels during the day.

Urine samples for neurotransmitter testing should be obtained 5 to 6 hours before bedtime.
The following is the suggested protocol for use of amino acids with urinary neurotransmitter testing to achieve optimal results.

- Start all patients on the level 1 dosing protocol at the first visit. Formulas containing L-dopa are recommended only for treatment of obesity, Parkinsonism, and Restless Leg Syndrome.
- Patients should return in one week. At which time, the question to ask is, “How were things yesterday?” It takes 3 to 5 days for the full effects of starting or changing an amino acid dosing to be displayed. Asking, “How were things last week” is not an adequate indicator of the status of and changes in the patient’s system. If symptoms are not fully under control, adjust the patient to the level 2 dosing. The patient should return in one week.
- At the 3rd visit, if symptoms are not under control, increase to the level 3 dosing. The patient should return in one week if their dosing was increased. They should return in two weeks, if their dosing level was maintained.
- At the next visit, if symptoms are under control, continue level 3 dosing. If symptoms are not under control, continue the level 3 dosing and obtain a urinary neurotransmitter test. Follow the amino acid dosing recommendations when the test results are returned.
- When you obtain a neurotransmitter test, patients should return in one week to evaluate clinical progress, discuss lab results and prescribe any amino acid dosing changes that may be needed.
- When you change an amino acid dose, patients return in one week to evaluate the results. DO NOT let your patients suffer needlessly by allowing them to go unseen for several weeks, when their symptoms are not under control.
- Over 60% of patients tested receive only one neurotransmitter test. This is consistent with the complete resolution of symptoms after adjusting the amino acid dosing in accordance with the consultant recommendations provided with the lab results. It is important that you treat the patient, not the lab. When disease symptoms have resolved, but dopamine and serotonin levels are not in the therapeutic Phase 3 response™, you do not need to order further tests unless you are attempting to establish an optimal feeling of wellness.

The goal is urinary serotonin and dopamine levels in the therapeutic phase 3 response.
Urinary neurotransmitter testing prior to the administration of amino acid precursors has no value. The samples have no correlation with the phase responses seen when the patient is treated with amino acid precursors. Urinary neurotransmitter testing is an amino acid challenge test. Proper use of urinary neurotransmitter testing is performed when two urinary neurotransmitter tests are performed while the patient is taking amino acid precursors at different dosing levels. The differences observed for each test determine the urinary serotonin and dopamine phases.

Contrary to other approaches in neurotransmitter assays, the optimal time for the collection of urinary neurotransmitter samples is 4 to 6 hours prior to bedtime. In most patients, this is just prior to taking the 4PM amino acid dosing.

Urinary neurotransmitter testing prior to treatment with amino acids has no diagnostic value. These samples fail to be useful in establishing The Phase 3 Response™, which identifies the response of the kidneys when amino acid dosing levels are changed.

7% of the general population, not being treated with amino acids, have elevated urinary neurotransmitter levels (above the reference range reported by the lab).

Urinary neurotransmitter testing on patients diagnosed by a licensed health care provider as suffering from one or more neurotransmitter dysfunction diseases (see page 1), who were not under treatment with amino acids, showed the following data:

- 86.6% have urinary serotonin levels above the reference range reported by the lab, when testing is performed properly.
- 18.7% have urinary dopamine levels above the reference range reported by the lab, when testing is performed properly.
- 94.4% have one or more of the master neurotransmitters elevated in the urine above the reference range reported by the lab, when testing is performed properly.

The Phase 3 Response™ is the response of the kidneys to the administration of serotonin and dopamine amino acid precursors. Changes in amino acid precursor dosing, which move urinary serotonin and dopamine levels towards the therapeutic range with the phase 3 response, correlate highly with the resolution of symptoms and an optimal feeling of wellness.
Treat the Patient, Not the Lab

If your practice is on track, 70 to 80% of patients will find that dopamine and serotonin amino acid precursors, when used in proper combination and balance, provide relief of symptoms without the need for lab testing. For the remaining 20 to 30% who do not achieve relief of symptoms when moved to the level 3 amino acid dosing, urinary neurotransmitter testing is indicated. The following thoughts are inspired by technical support calls received at NeuroResearch.

It is important to remember that you are treating a patient, not the lab. So, what do we mean by this? When you order a urinary neurotransmitter test, an amino acid dosing recommendation is made based on the lab results. If you change the amino acid dosing and one week later the patient’s symptoms are fully under control, you do not need further testing unless disease symptoms return. While the goal of urinary neurotransmitter testing is to establish both urinary serotonin and dopamine levels in the therapeutic range AND in the phase 3 response, many patients do not actually have to arrive at the phase 3 therapeutic response for symptomatic relief to be obtained.

Pearls for Treatment

Adjusting amino acid doses with the guidance of urinary neurotransmitter testing is like a bull’s eye. The center of the bull’s-eye is the therapeutic phase 3 response of serotonin and dopamine (see page 43. Every time you order urinary neurotransmitter testing and make an amino acid dosing change based on the results, the patient moves closer to the bull’s-eye.

The fact is that many patients do not need to be fully driven into the bull’s-eye for full relief of symptoms. It would appear that for many patients, they merely have to get somewhere on the target for relief of symptoms to occur.

On the other side of the coin, there are indeed patients who need to arrive at the bull’s-eye (therapeutic phase 3 response) for serotonin and dopamine levels, prior to symptoms resolving. These represent less than 1% of the patients. They are very difficult patients to treat, but with proper management they too can find relief of symptoms.

Urinary Neurotransmitter Testing Stats

- 64.1% of patients receive only one test (consistent with the complete relief of symptoms after one test by following the recommended amino acid dosing change).
- At present, DBS Labs is performing over 20,000 neurotransmitter assays a year.
- Less than 2% of patient tests display an unusual pattern where the patient ultimately ends up on a low dose of 5-HTP (<150 mg per day) and a high dose of tyrosine (>6 grams per day). These patients typically need 4 to 6 urinary neurotransmitter tests for relief of symptoms. Over 95% carry a diagnosis of depression.
- In reviewing the lab testing of over 4,000 patients in the last year, the average number of urinary neurotransmitter tests per patient is 1.81 tests.
- The database record for “most tests ordered on one patient” is 21. In reviewing the data, virtually none of the NeuroResearch treatment suggestions were followed.
Urinary Neurotransmitter Testing, when used properly with amino acid therapy, is an “amino acid challenge test.”

Two urinary neurotransmitter tests are obtained. After the test results of the first test are reported, the patient’s amino acid dosing is changed to a different dosing level. A second urinary neurotransmitter test is obtained one week later. In the process, the phases of the urinary neurotransmitters are determined by comparing the first urinary neurotransmitter test with the second neurotransmitter test in correlation with the amino acid dosing change. The phases of dopamine and serotonin are independent of each other. For example, one can be in phase 1 while the other is in phase 2 or 3. Testing obtained prior to starting amino acids has no value in determining the urinary phases of serotonin and/or dopamine.

### Urinary Neurotransmitter Statistics

An overview of patients, not under treatment with amino acids, who have formally been diagnosed with a neurotransmitter dysfunction disease.

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Reference</th>
<th>% Above Reference</th>
<th>Range</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serotonin</td>
<td>0.98 - 173.341.94</td>
<td>1.479.28</td>
<td>13.395.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>1.54 - 7.956.1</td>
<td>200.2</td>
<td>702.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>0.39 - 945.79</td>
<td>45.87</td>
<td>110.60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.81 - 595.47</td>
<td>9.20</td>
<td>42.83</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total population N=710.

- **7% of the general population**, not under treatment with amino acids, has elevated urinary serotonin levels (above the reference range reported by the lab).
- **86.6% of patients** tested in the late afternoon and 61% of patients tested in the morning, who were diagnosed by a licensed health care provider as having one or more neurotransmitter dysfunction diseases and were not under treatment with amino acids, have elevated urinary serotonin levels (above the reference range reported by the lab) when testing is performed properly.
- **18.7% of patients** diagnosed by a licensed health care provider as having one or more neurotransmitter dysfunction diseases, who were not under treatment with amino acids, have elevated urinary dopamine levels (above the reference range reported by the lab) when testing is performed properly.
- **94.4% of patients** diagnosed by a licensed health care provider as having one or more neurotransmitter dysfunction diseases, who were not under treatment with amino acids, have one or more of the master neurotransmitters elevated in the urine (above the reference range reported by the lab) when testing is performed properly.
The Balancing Act
Why plain 5-HTP, plain Tyrosine, plain L-Dopa, or improperly balanced amino acids should not be used in treatment. Use of plain 5-HTP depletes dopamine. Use of plain tyrosine and/or L-dopa depletes serotonin.

Perspective
The two illustrations below are based on actual lab tests from patients under treatment with balanced tyrosine and 5-HTP. The numbers on the top of each set are actual test results prior to adding a plain and unbalanced amino acid precursor (5-HTP is on the top set. L-dopa is on the bottom set).

The numbers on the bottom of each set represent test results one week later. Note that in both cases, balanced amino acids were in place and the addition of plain 5-HTP or plain L-dopa caused urinary neurotransmitter levels to increase markedly. By adding plain 5-HTP to the top set, urinary serotonin levels went from 900 to 2,370 and urinary dopamine levels went from 350 to 1,450 in one week. By adding plain L-dopa to the bottom set, urinary serotonin levels went from 1,431 to 8,564 and urinary dopamine levels went from 110 to 575 in one week.

Using plain or improperly balanced amino acids will cause an increase in urinary excretion by the serotonin the catecholamine systems (see below).

Discussion
The Boxes
The boxes on the fulcrums represent systemic levels of neurotransmitters, while the numbers represent urinary neurotransmitter levels. Using plain 5-HTP, plain L-dopa, or improperly balanced amino acids will deplete the neurotransmitters in other systems. Plain 5-HTP depletes the catecholamines. Plain L-dopa depletes serotonin.

The Numbers
The numbers in the boxes represent urinary neurotransmitter levels. Use of plain 5-HTP or plain L-dopa will cause increases in the urinary excretion of the neurotransmitters of the catecholamine and the serotonin systems. Tyrosine has similar affects. N-acetyl Tyrosine and phenylalanine are inferior amino acids for the treatment of neurotransmitter dysfunction. They do not provide dramatic improvements.

Balanced Amino Acids
What is balanced? At the heart of our work is the concept that using amino acids in properly balanced ratios will prevent amino acid precursors of one system from depleting the neurotransmitters of another system. The formulas that we have invented provide these proper ratios. We know of no other formulas as effective.
On March 28, 2005, the patient was on the level 3 dosing (see page 9) of amino acids. A serotonin level of 19,212 was reported (900 mg of 5-HTP with 5,000 mg of tyrosine). With the amino acid dosing decreased on April 27, 2005, the serotonin increased to 62,587. Clearly, the serotonin was in phase 1 on both tests. Interestingly, when the amino acid dosing decreased, dopamine increased. This indicates that it was going into phase 1 on April 27th. The recommendation was to increase to 1,200 mg of 5-HTP with 6,000 mg of tyrosine per day. A follow-up neurotransmitter test was recommended one week after the dosing change was in place.

By increasing the amino acid dosing, the serotonin dropped from 38,397 to 2,812. We know from experience that the serotonin on both tests is in phase 1. The dopamine remained relatively unchanged, indicating a phase 2 response. The recommendation was to increase to the level 3 dosing protocol, and then perform a follow-up neurotransmitter test one week after the dosing change was put in place. Another observation to be made here is that the patient was being treated for depression. The first and second tests were performed 4 months apart. When adjusting the amino acid dosing as guided by lab testing, follow-up testing should be done in one-to-two weeks. Allowing patients to suffer with symptoms, by allowing 4 months between tests, is bad medicine.

Serotonin and dopamine phases are independent of one another. In the example above on May 4, serotonin and dopamine are at or near the therapeutic range. But, serotonin is in phase 3 and dopamine is in phase 1. How do we know that dopamine is in phase 1? The answer is, “As the amino acid dosing was decreased, the dopamine increased.” The recommendation for the amino acid dosing is to continue at the present dosing (level 2) and start an additional 1,000 mg of tyrosine, three times a day. A follow-up neurotransmitter test was recommended in one week, if symptoms had not resolved.

The patient’s amino acid dosing was increased two dosing levels between tests (from level 2 to level 4). Serotonin dropped from 115,220 to 46,778. The first serotonin of 115,220 represents a phase 1 response. But, in looking at the April 19, 2006 test, we do not know if this is a phase 1 or phase 3 response due to the large change in amino acid dosing between tests. The recommendation was to decrease to the level 2 dosing (900 mg of 5-HTP per day with 5,000 mg of tyrosine per day), and then obtain a follow-up test one week after the dosing change was in place. Amino acid dosing changes need to be done in an orderly manner prior to each test. Extremely large dosing changes between tests may require dosing adjustments that are aimed solely at acquiring the data needed.

For amino acid dosing levels, refer to page 9.
The Downward Inflection of Phase 1 May Not Be the Same Slope as the Upward Inflection of Phase 3

This is a dramatic example of how as the amino acid dosing is increased, the slope of the downward inflection in phase 1 can be markedly different from the slope of upward inflection in phase 3. On tests 2 and 3, the serotonin changed very little with significant amino acid dosing changes. On test 4, there is a dramatic increase in serotonin as the inflection point of phase 3 is reached.

A Patient with Rare Amino Acid Dosing Needs

The above patient is being treated for obesity and depression. In studying the amino acid dosing level, it is apparent that in order to achieve the phase 3 therapeutic response, the patient’s 5-HTP dosing need is very low and the tyrosine dosing need is relatively high. This pattern of a low dose serotonin amino acid precursor being needed with a relatively high dose of dopamine amino acids most frequently occurs in patients who have a diagnosis of depression.

THERAPEUTIC NEUROTRANSMITTERS RANGES

In micrograms of neurotransmitter per gram of creatinine

Serotonin: 800 to 2,400
Dopamine (in general): 300 to 600
Dopamine (Restless Leg Syndrome): 1,800 to 2,400
Dopamine (Parkinsonism): 6,000 to 8,000

Therapeutic values are only valid if serotonin or dopamine levels are in The Phase 3 Response™.

A Typical Example of Fine-Tuning

All serotonin levels reported in the above example are in phase 3. Of interest is how dopamine moved from phase 2 to phase 1 after the second test, and then moved to phase 3, as verified by the third and fourth tests.

For amino acid dosing levels, refer to page 9.
Lab Study - Extra Catecholamine Precursors Needed

Between test 1 and test 2, the serotonin precursor 5-HTP was decreased and the dopamine precursor tyrosine was increased. With this change in amino acid dosing, the following phase results occurred. Serotonin is in phase 1 on both tests. Dopamine is in phase 2 (subtherapeutic) on both tests. The next step in treatment was to increase the daily 5-HTP dosing to 1,200 mg per day and increase the daily tyrosine dosing to 7,000 mg per day in equally divided doses. Test recommendations, after the first neurotransmitter test is submitted, are based on the statistical probability of the serotonin and dopamine phase. While most of the time these statistical probability recommendations are correct, in reviewing the above set of labs, it is obvious that the recommendation for the March 15th, 2005 was incorrect. The amino acid dosing needed to be increased, not decreased, as evidenced by the second test.

Patient lab number 1 was performed after the patient was on the level 3 amino acid dosing (900 mg of 5-HTP with 5,000 mg of tyrosine and cofactors) for one week. This test revealed a subtherapeutic (phase 2) urinary serotonin and dopamine. The dosing was increased to 1,200 mg of 5-HTP with 6,000 mg of tyrosine. One week later, a urine sample was obtained. The serotonin was high and in phase 3. Dopamine was in the therapeutic range (300 to 600) and in phase 3. 5-HTP was decreased back to the original levels of 900 mg. To this amino acid dosing change, an additional 3,000 mg of tyrosine per day (1,000 mg three times a day) was added. This provided a daily tyrosine dosing of 8,000 mg per day. On follow-up testing, urinary serotonin and dopamine were found to be in the phase 3 therapeutic range. The last dosing change and the subsequent lab analysis is a good example of how changing the amino acid precursor of one neurotransmitter affects both serotonin and dopamine levels.

This lab example shows a Phase 1 response for serotonin on both tests. It shows a Phase 2 response for dopamine on the second test. In adjusting amino acids, only the serotonin and dopamine are initially of importance. Once serotonin and dopamine are in the therapeutic range and in the Phase 3 Response™, norepinephrine will correct in 2 to 6 weeks and epinephrine will correct in 3 to 6 months. Of interest here is the fact that the caregiver allowed 2 months to lapse between tests. Once an amino acid dosing change is in place, a follow-up neurotransmitter test should be obtained in one week. Allowing a patient to go 2 months between tests only prolongs suffering with symptoms. So, what are the recommendations here? The answer is to increase to the level 3 dosing (900 mg of 5-HTP with 5,000 mg of tyrosine plus cofactors, divided in three equal daily doses). A follow-up neurotransmitter lab test should be obtained one week after the dosing change is in place.

The highest urinary serotonin reported by DBS Labs was 1,747,690 micrograms of serotonin per gram of creatinine.
THE TIP OF THE ICEBERG

When the master neurotransmitters (serotonin, dopamine, norepinephrine, and epinephrine) are not at optimal levels, it affects the entire body. The central nervous system displays diseases and the peripheral nervous system can display diseases. The organ systems innervated by the master neurotransmitters may not function optimally or may begin to experience problems as well.

**Source of urinary neurotransmitters:**
Urinary neurotransmitters are not filtered by the kidneys and excreted into the urine. They are synthesized by the kidneys and excreted into the urine. From a physiological standpoint, an amazing thing takes place in the kidneys. The kidneys metabolize (breakdown) the systemic neurotransmitters that the kidneys filter out of the renal artery blood. Then, the kidneys synthesize new neurotransmitters from the amino acid precursors that were filtered along with the systemic neurotransmitters. Urinary neurotransmitters levels have nothing to do with systemic neurotransmitter levels. In patients suffering from diseases, associated with low levels of neurotransmitters in the system (such as depression), urinary neurotransmitters have no correlation with systemic neurotransmitters prior to treatment with amino acids.

**Urinary neurotransmitter testing** is a very sophisticated testing. CLIA classifies it as a “high complexity lab”. In general, it takes 2 days to perform this testing. On day 1, the samples are set up and incubated overnight. On the second day, the labs are run. DBS Lab results show that 86.6% of patients (N=710) who are not taking amino acid precursors and have been diagnosed with one or more neurotransmitter dysfunction diseases have elevated urinary serotonin levels prior to amino acid treatment.

**Urinary neurotransmitter testing is an “amino acid challenge test”.** The patient is given serotonin and dopamine amino acid precursors. One week later, a neurotransmitter test is obtained. After the first test is interpreted, the patient is placed on a second amino acid dosing of serotonin and dopamine precursors. A second urinary neurotransmitter test is obtained. Once the second test results are reported, the results of the serotonin and dopamine results of the first and second test are compared. A plan is laid out to adjust the amino acid dosing in order to move the urinary serotonin and dopamine levels into the “phase 3 therapeutic response,” which correlates highly with the relief of symptoms.
Correlations are important.
Urinary serotonin and dopamine levels prior to starting supplemental amino acid precursors do not correlate with results once patients are placed on serotonin and dopamine amino acid precursors. Furthermore, lab results obtained prior to starting amino acid precursors cannot be used to establish the phases of the urinary dopamine or serotonin, since we are gauging the response of the kidneys to amino acid doses at different levels.

Neurotransmitters are synthesized by the kidneys in the “proximal convoluted renal tubule cells” that line the proximal tubules.

Proximal renal tubule cells excrete the synthesized neurotransmitters into the urine or secrete them into the system.

Urinary phase response is identified when serotonin and dopamine amino acid precursors are administered at different dosing levels. The urinary neurotransmitter tests of each dosing are compared.

**In phase 1** - the proximal convoluted renal tubule cells are inappropriately excreting neurotransmitters into the urine instead of into the system (which has low levels in patients with neurotransmitter dysfunction diseases).

**In phase 2** - the problem of inappropriate neurotransmitter excretion has been corrected. Neurotransmitters are now appropriately going into the system which needs them. But, the levels are still low.

**Phase 3** - occurs as the amino acid dosing is increased. The best analogy is that as you increase the amino acid dosing, the system finally fills to proper levels. The excess neurotransmitters are now appropriately being excreted into the urine.

**The Tip of the Iceberg**
From clinical and laboratory observations, we believe the following is true: when a patient’s neurotransmitter levels are low, they are low everywhere in the body and other systems suffer.

Along with the response of the kidneys to amino acids, we have seen responses in the other systems dependent on these neurotransmitters. The central nervous system, the peripheral nervous system, the liver, the GI track, the lungs, the heart, hormone systems, as well as other systems, are all affected by neurotransmitter dysfunction.

We truly believe that identification of the three phases of amino acid response by the kidneys is just the tip of the iceberg in helping all systems function properly. The lab testing that we have brought in is the first objective measurement to show the effects of amino acid therapy. It is the first measurement that correlates with other systems improving. In the future, we have no doubt that other measurements of other systems will be found that will correlate in a similar manner as the 3 phases of kidney response to the administration of serotonin and dopamine precursors.
HOW DRUGS THAT WORK WITH NEUROTRANSMITTERS DEPLETE NEUROTRANSMITTERS.

Drugs that work with neurotransmitters do not work if there is not enough neurotransmitters with which to work.

What happens when prescription drugs deplete neurotransmitter levels during treatment?

- The drug looses effectiveness.
- Symptoms of the disease return.
- The real problem (low levels of neurotransmitters) becomes worse, which increases disease symptoms.

Neurotransmitters are metabolized by the COMT and MAO enzymes. When neurotransmitters are in the vesicles (store) of the axon (pre-synaptic neuron), they are safe from breakdown and metabolism. Once neurotransmitter molecules have been secreted into the synapse, this is no longer the case. They are now exposed to the enzymes which destroy them.

Reuptake inhibitor drugs, as well as any other drug whose mechanism of action is moving neurotransmitters from one place to another, do not increase the overall number of neurotransmitter molecules in the central nervous system. They merely work by moving neurotransmitters from the safety of the vesicles in the axon to the synapse. In the process, more neurotransmitter molecules come in contact with and are exposed to the enzymes that destroy them. Long-term use of prescription drugs depletes neurotransmitters. This places further stress on a system that is already suffering from lack of neurotransmitters.

The master neurotransmitters serotonin, dopamine, norepinephrine, and epinephrine do not cross the blood brain barrier. Prescription drugs do not increase the number of neurotransmitter molecules in the central nervous system. The only way to actually increase the number of neurotransmitter molecules in the central nervous system is to provide the amino acid precursors needed by the system to synthesize additional neurotransmitters.

Drugs that use the redistribution of neurotransmitters as their mechanism of action deplete neurotransmitters during long-term use. Proper levels of supplemental amino acid precursors with cofactors should be co-administered to combat this problem.
KEEPING DRUGS WORKING

In 1989, Prozac was the first prescription reuptake inhibitor (SSRI) on the market. Since then, many others have been released for use. Other reuptake inhibitors include, but are not limited to, Zoloft, Celexa, Lexapro, Luvox, and Paxil. After Prozac, came the SNRIs. Effexor and Meridia are examples of SNRIs.

Doctors have found that over time SSRI and SNRI become less effective or ineffective in most patients. At this point, disease symptoms return. Our research has found that these drugs further deplete neurotransmitter levels. So, what is happening here? Patients who have taken SSRIs and SNRIs drugs for a significant period of time have lower neurotransmitter levels than patients who have not. This finding has profound implications in medicine. We know of no other class of prescription drugs that are allowed to be used to treat symptoms that over time cause the real source of the problem to become worse.

Drugs that work with neurotransmitters do not work if there are not enough neurotransmitters with which to work. While the argument can be made that people suffering from neurotransmitter diseases may have lower levels of neurotransmitters than the general population, our findings indicate that the degree of neurotransmitter depletion is exacerbated by drugs that redistribute neurotransmitters.

So, what do you do with patients on neurotransmitter depleting prescription drugs? The recommendation is that all patients taking SSRIs, SNRIs, or other drugs that work by moving neurotransmitters be placed on the level 1 amino acid dosing (see page 9) to prevent further depletion of neurotransmitters.

We discuss this point because there are patients who need or want to continue taking their neurotransmitter depleting prescription drugs. When this is the case, start the patient on the level 1 amino acid dosing (as outlined on page 9) to prevent further depletion of neurotransmitters.

While most patients can be successfully treated with only amino acids, in patients with severe symptoms it is recommended that the patient be treated with prescription drugs and amino acids. Examples include suicidal patients and patients with depression that affects their ability to participate in day-to-day activities.

FUNCTIONS OF AMINO ACIDS

Maintains the effectiveness of prescription drugs

Serves as a stand-alone treatment modality
N-Acetyl Tyrosine

N-Acetyl Tyrosine is a side chain reaction of tyrosine. N-Acetyl Tyrosine has to be converted into tyrosine, in order to be effective in neurotransmitter treatment as a precursor of the catecholamines.

Perspective
Delivery of N-Acetyl Tyrosine (NAT) through parenteral routes (other than through the GI tract) via IV have shown no increase in systemic tyrosine levels. If N-Acetyl Tyrosine is not suitable for parenteral nutrition, it is certainly not suitable as an oral neurotransmitter precursor.

N-Acetyl Tyrosine IS NOT tyrosine
It cannot do the job of tyrosine

The Hoax on the Market
We are aware of some medical practices that thought they were using 5-HTP with L-tyrosine when in fact they were using N-acetyl-tyrosine, not L-tyrosine. Use of N-acetyl-tyrosine is not recommended. It leads to results that are markedly less effective than L-tyrosine when treating patients.

Medical Literature Article One
Kidney Int Suppl. 1989 Nov;27:S282-6
Phenylalanine and tyrosine metabolism in renal failure: dipeptides as tyrosine source.
Druml W, Roth E, Lenz K, Lochs H, Kopsa H.

...After (parenteral) infusion of N-Acetyl Tyrosine, no increase in plasma tyrosine is seen...

Medical Literature Article Two
Hepatology. 1995 Apr;21(4):923-8
Utilization of tyrosine-containing dipeptides and N-Acetyl Tyrosine in hepatic failure.
Druml W, Hubl W, Roth E, Lochs H.

...N-Acetyl Tyrosine was not grossly affected by hepatic failure, but neither in healthy nor in hepatic failure patients was an increase in tyrosine seen. Both dipeptides but not N-Acetyl Tyrosine may serve as a tyrosine source in parenteral nutrition...
The Sulfur Amino Acid Cycle

N-acetyl Cysteine (NAC) does not directly contribute sulfur to the sulfur amino acid cycle.

In recent years, use of N-acetyl Cysteine (NAC) and glutathione have come into vogue. NAC (brand name, Mucomist) is used in emergency rooms for certain overdose situations. NAC is readily metabolized into the body’s most powerful detoxifying agent, glutathione. NAC is a sulfur-based amino acid that is synthesized from cysteine.

If amino acids are not properly balanced, amino acid therapy of the serotonin and the catecholamine (dopamine, norepinephrine, and epinephrine) systems may deplete the sulfur amino acid system. In general, the use of tyrosine and/or L-dopa will deplete the sulfur amino acid system if proper supplementation with sulfur amino acids is not provided. Cysteine, methionine, S-adenosyl methionine and several other intermediates directly participate in the sulfur cycle. Properly balanced amino acids can maintain proper sulfur levels, which will prevent the overall depletion of the sulfur amino acid cycle by tyrosine and/or L-dopa.

NAC does not have the ability to contribute sulfur to the sulfur amino acid cycle. NAC is a side-chain reaction of the sulfur cycle. Administering NAC is like plugging one hole in a leaky bucket with many holes. NAC prevents depletion of the sulfur cycle at one point in the cycle, but it does not prevent depletion of sulfur at other points. Whereas, using cysteine maintains and prevents the depletion of sulfur throughout the cycle by directly increasing sulfur amino acid levels.

Use of NAC does not prevent the depletion of the sulfur amino acid cycle during amino acid treatment. Therefore, it is not recommended as part of amino acid therapy.
Beta-Phenylethylamine (PEA)

PEA regulates the release of dopamine when dopamine levels are low. When dopamine levels are properly addressed with amino acids, PEA no longer is a consideration in treatment. Testing for PEA, in patients properly treated with amino acids, is a waste of time and money.

We are aware of PEA testing being used as a part of treatment. Testing PEA is a waste of time and money, if the master neurotransmitter amino acid precursors are used as outlined in this book.

We have seen gross inaccuracies regarding the clinical and medical applications of PEA. For example, there are claims that PEA is a neurotransmitter. It is not. PEA is a neurotransmitter modulator, which regulates the excretion of dopamine when dopamine levels are low (see abstract on this page). It is not a factor when the master neurotransmitters have been optimized.

Virtually all published works in the past relating to PEA have been studies on 24 hour urines. There are virtually no published studies using spot urine tests with regards to PEA. PEA appears to be active in only one of many dopamine pathways in the body, “the nigrostriatal pathway.” From a professional standpoint, PEA is not a consideration during amino acid therapy of the serotonin and the catecholamine systems. This is especially true when there are ample levels of dopamine, such as occurs during properly implemented amino acid therapy, since PEA functions only as a neurotransmitter modulator when dopamine levels are low.

PEA regulates the release of dopamine when the system is depleted. When the precursors and cofactors of the catecholamine and the serotonin systems are in proper balance and adequate dopamine levels are in place, PEA does not regulate dopamine release. The research conducted by NeuroResearch has shown that proper use of amino acids will regulate dopamine levels by correcting the overall system. Thus, PEA as a means of regulating dopamine release will become insignificant to the system. Correcting and restoring dopamine levels with the master neurotransmitter precursors will render PEA considerations meaningless. Therefore, testing PEA levels as part of master neurotransmitter amino acid therapy is a waste of time and money.
GABA In Clinical Practice

Over the years, NeuroResearch has refused to promote any product or form of treatment that we do not have firsthand clinical experience and data showing function, efficacy and safety. Currently, we lack the proper data to endorse testing and treatment with GABA. Therefore, NeuroResearch does not offer any GABA amino acid formulas and DBS Labs does not provide lab testing of GABA.

As we move forward in our research, we have found ourselves asking, what would constitute clinical proof? We would expect to find that increasing synaptic GABA levels of the system would result in decreased incidences of anxiety and panic attacks. Currently, our research and data has found that treatment with GABA and testing of GABA are not as effective as optimizing the master neurotransmitters in the treatment of anxiety. As a result, we continue to promote the use of serotonin and catecholamine precursors, which optimize the system, as the primary means of treating anxiety and panic attacks.

We would also expect that if treating the GABA directly with GABA precursors is effective, there would be a decrease in the incidences of epileptic seizures with a mechanism of action similar to GABA transaminase inhibitor drugs, such as Valproic Acid. Valproic Acid elevates GABA levels by blocking the metabolism of GABA. In the process it significantly increases synaptic GABA levels, resulting in antiepileptic effects. Again, we have found no evidence that using GABA, GABA precursors, or GABA testing will produce antiepileptic effects.

Due to the inconsistent clinical outcomes of using GABA when compared to using master neurotransmitters precursors, we do not recommended treatment using GABA until the master neurotransmitters are optimized. Currently, our position is that treatment involving GABA is not as effective as properly using our research defined master neurotransmitter amino acid therapy protocols, which are based on statistical analyses and direct observations in the care of thousands of patient during clinic visits.
Properly Prioritizing Medical Care

The master neurotransmitters are serotonin, dopamine, norepinephrine, and epinephrine. Master neurotransmitters control the function of numerous systems in the brain and body. The box on the right illustrates a small fraction of the biological factors that are controlled by the master neurotransmitters.

Changes in the master neurotransmitters will cause changes in all of the factors listed to the right. Any condition that is actively under treatment at the start of amino acid therapy must be rechecked once neurotransmitters are optimized.

Doing Things Properly

Over the years, the clinical work of NeuroResearch has proven that manipulation of the master neurotransmitters with amino acids will cause profound changes in hormones and other biological functions. Treating or testing any factor controlled by the master neurotransmitters before the master neurotransmitters are in the therapeutic range and in The Phase 3 Response™ will prove to be a waste of time, money and is unneeded (from a medical standpoint).

Any diseases or conditions that are related to neurotransmitter dysfunction must be treated in an orderly manner. To properly treat neurotransmitter dysfunction diseases and conditions, the master neurotransmitters need to have levels established in the therapeutic range and in The Phase 3 Response™, as verified by laboratory testing. Secondary diseases and conditions should be addressed once the master neurotransmitters are properly balanced.

Treating all of the patient’s conditions at once will result in unpredictable and uncontrollable responses by many different systems. For example, hormones, melatonin, and cortisol are partly controlled by norepinephrine. Norepinephrine takes 2 to 6 weeks to stabilize, once serotonin and dopamine levels are optimized, as verified by laboratory testing. Treatment of a patient’s hormone, melatonin or cortisol levels will be greatly affected by changes in norepinephrine levels, as well as by any treatment that occurs at the same time. This will likely cause the patient to have unpredictable changes in the levels of these secondary conditions, as well as cause the patient to endure prolonged suffering of their symptoms. Many of the patients with hormone imbalances and most of the patients with improper levels of melatonin or cortisol do not need treatment once master neurotransmitters have been properly balanced.
As with other functions controlled by the master neurotransmitters, baseline testing of cortisol at the start of neurotransmitter treatment will not provide diagnostically useful information. Our research has shown that changes in the master neurotransmitters will cause dramatic changes in cortisol levels, if manipulation of the master neurotransmitters with amino acid precursors takes place after baseline testing of cortisol is performed.

As occurs in many areas of medicine, the medical care plan has to be prioritized. The proper time to test cortisol levels is after the master neurotransmitters have been optimized, as verified by lab testing. Testing cortisol levels before the master neurotransmitters are optimized is a waste of time and money.

79% of patients, not being treated by amino acids, have low epinephrine levels.

It is recommended that when there is a need to start patients on DHEA, patients should also be started on the level 1 amino acid dosing, as outlined on page 9.
Hormones are Controlled by Neurotransmitters

Optimized neurotransmitters have profound effects on hormones. Neurotransmitters regulate and control hormone synthesis and function. Years of clinical research and statistical analysis by NeuroResearch have shown a need to prioritize care when treating hormone dysfunction.

The first step to optimizing master neurotransmitters when treating neurotransmitter dysfunction disease in adults is to start patients on the level 1 treatment protocol (see page 9). Once the neurotransmitters are optimized for 6 weeks, obtain a baseline hormone test.

Tests that are conducted after the master neurotransmitters have been optimized will be able to accurately guide hormone treatment. Properly Prioritize Care by First Treating the Master Neurotransmitters, and then Treating Hormones. Testing hormones prior to optimizing the master neurotransmitters will not provide diagnostically useful information, due to the impact that changes in neurotransmitter levels have on hormone levels.
Melatonin

Serotonin is a precursor of melatonin. Melatonin synthesis is regulated by norepinephrine. **THE PROPER TIME TO TEST MELATONIN IS AFTER THE MASTER NEUROTRANSMITTERS HAVE BEEN OPTIMIZED.** Testing melatonin at the start of amino acid therapy is a waste of time and money if manipulation of the master neurotransmitters serotonin, dopamine, norepinephrine, and epinephrine which control the minor neurotransmitters is being done. It does not provide diagnostically useful information if manipulations of the neurotransmitters that control melatonin synthesis have not been controlled.

The Minor Neurotransmitters

The minor neurotransmitters of the brain are controlled by or greatly affected by the manipulation of the master neurotransmitters. **THE PROPER TIME TO TEST THE MINOR NEUROTRANSMITTERS IS AFTER THE MASTER NEUROTRANSMITTERS HAVE BEEN OPTIMIZED.** If manipulation of the master neurotransmitters (serotonin, dopamine, norepinephrine, and epinephrine), which control the minor neurotransmitters is not complete, testing the minor neurotransmitters will not provide diagnostically useful or meaningful information.
Properly balanced amino acids will produce effective results in the treatment of obesity. However, NeuroResearch has added this page of treatment alternatives that use prescription drugs with amino acids to provide caregivers with additional options. NeuroResearch’s early research focused on using amino acids to keep prescription drugs working for the treatment of obesity.

This early research was databased and statistically analyzed. This led to NeuroResearch’s first patents on the use of amino acids in order to keep prescription drugs effectively working in the treatment of obesity. Many of the treatments on this page are covered under NeuroResearch’s patents. The treatment modalities listed at the right are covered under patents and patents pending of NeuroResearch. For more information on their use, call NeuroResearch at 877-626-2220.

In 1997, fenfluramine (Pondimin and Redux) was pulled from the market due to concerns of heart valve problems. Shortly thereafter, phentermine obtained the warning below. This warning is viewed as a liability management tool by the companies marketing phentermine. There are no known problems from using phentermine with SSRI medications. To this end, NeuroResearch obtained letters from the SSRI manufacturing companies, which state that there are no known problems with the use of phentermine with their medications. The warning below is misleading.

**A Misleading Warning for Phentermine (See above):**
Capsules are indicated only as a short-term monotherapy for the management of exogenous obesity. The safety and efficacy of combination therapy with phentermine and any other drug products for weight loss, including selective serotonin reuptake inhibitors (e.g., fluoxetine, sertraline, fluvoxamine, paroxetine), have not been established. Therefore, the coadministration of these drug products for weight loss is not recommended.


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Prescription Drugs Versus Amino Acids

The New Patient Information Brochure Is Ready

The updated reprint of the original NeuroResearch brochure, “Prescription Drugs Versus Amino Acids,” is now available. This brochure is intended to be given by caregivers to patients who are looking for basic information on amino acid therapy. These brochures are sent free of charge to healthcare providers in needed quantities requested.

To obtain FREE BROCHUREs for your patients, call: NeuroResearch 877-626-2220
Neurotransmitter testing
DBS Labs: 877-476-7229
www.LabDBS.com
E-mail: DBS1@LabDBS.com
8723 Falcon St.
Duluth, MN 55808

Neurotransmitter technical support
NeuroResearch Clinics, Inc.
877-626-2220
www.NeuroAssist.com
E-mail: info@NeuroAssist.com
1150 88th Ave. W.
Duluth, MN 55808