

# CHK MEDICAL FOODS

## The standard medical approach used when reuptake inhibitors stop working can cause the disease to worsen!

### PROPER REUPTAKE INHIBITOR

## TACHYPHYLAXIS MANAGEMENT

Definition of tachyphylaxis: diminished response to later increments in a sequence of applications of a physiologically active substance

**Avoid the trap** of increasing the dosing value, adding a second drug or switching to a new drug when reuptake inhibitors stop working. All of these strategies may deplete neurotransmitters further, making the original cause of the disease worse.

Medical foods based on scientific principles found in peer-reviewed literature

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

### Reuptake inhibitors deplete neurotransmitters

As discussed on page 2, reuptake inhibitors may set up conditions that deplete neurotransmitters. When neurotransmitters become depleted enough, the drugs quit working and symptoms of disease return.

### The standard approach to tachyphylaxis

When reuptake inhibitors quit working and symptoms of disease, such as depression, return, the standard approach is to increase the dosing value of the drug, add a second drug or switch to a new drug. All three of these approaches (as discussed on page 2) only serve to deplete the neurotransmitters even further. This is a serious situation, without precedence in medicine, where the drugs used to treat disease are making the cause of the disease worse.

### Build neurotransmitters, don't deplete them

The monoamine neurotransmitters serotonin, dopamine, norepinephrine, and epinephrine do not cross the blood-brain barrier. Drugs do nothing to increase the total number of neurotransmitter molecules in the brain, they work by setting up conditions that move neurotransmitters from one place to another. The only way to increase monoamine neurotransmitter levels properly is through administration of both serotonin and dopamine amino acid precursors in proper balance. Administration of only serotonin or dopamine precursors or improperly balanced serotonin and dopamine precursors will deplete neurotransmitters.



### Medical Foods



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

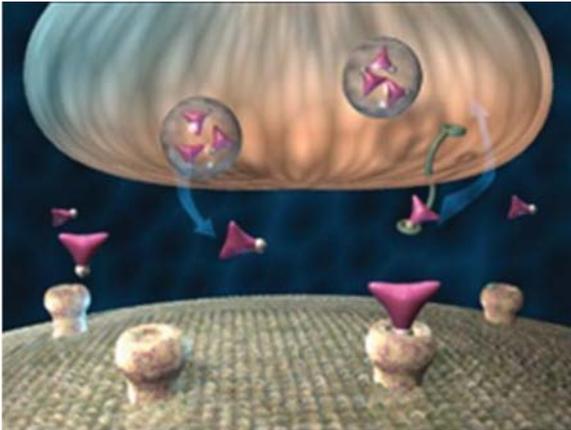
# THE MECHANISM OF ACTION

## HOW REUPTAKE INHIBITORS DEplete NEUROTRANSMITTERS



Illustrations courtesy of the National Institute on Drug Abuse

### Pre-treatment state

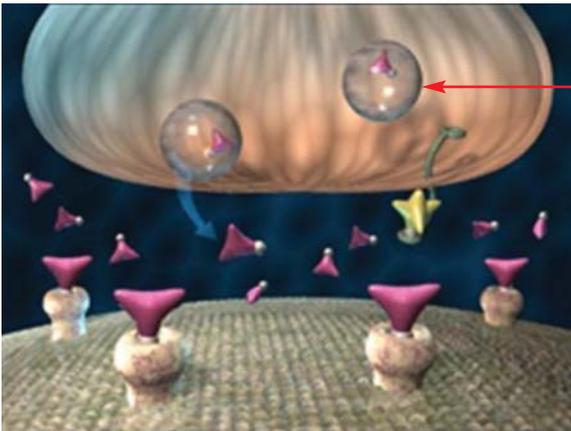


#### WHAT DOES NEUROTRANSMITTER DEPLETION DUE TO REUPTAKE INHIBITORS LOOK LIKE IN THE CLINIC?

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

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

### Early treatment with reuptake inhibitors



**Vesicle**

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

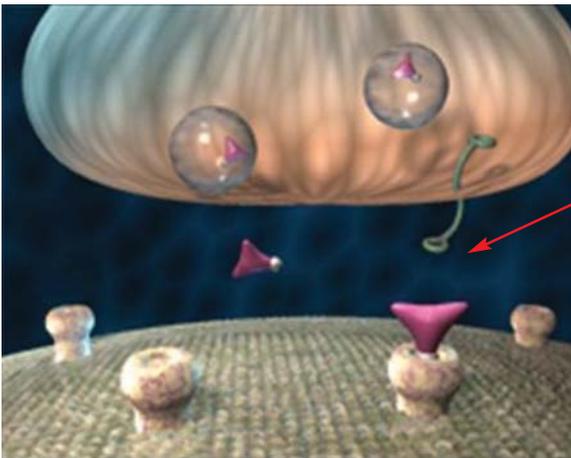
Drugs do nothing to increase the total number of neurotransmitter molecules in the brain.<sup>™</sup>

Drugs work by moving neurotransmitters from one place to another.<sup>™</sup>

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

Administration of improperly balanced serotonin and dopamine amino acid precursors will deplete neurotransmitters.<sup>™</sup>

### Depletion from reuptake inhibitors



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

When neurotransmitters become depleted, enough drugs that work with neurotransmitters stop working<sup>™</sup>

# MEDICAL FOODS

## PROPERLY ADDRESSING THE PROBLEMS WITHOUT MAKING THE CAUSE OF DISEASE WORSE

Reuptake inhibitors that do not work from the start of treatment due to extremely low levels of neurotransmitters.

**THE PROBLEMS**

Reuptake inhibitors stop working during treatment, and symptoms of disease return.

**PROPER TREATMENT**  
balanced amino acids



### The NeuroReplete Protocol

- 4 pills NeuroReplete in the AM and at 4 PM
  - 2 pills of CysReplete at noon, 4 PM, and bedtime
- It can take 3 to 5 days for maximum effects of any dosing value change to be seen.



### PATIENT ORIENTATION:

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

### GI UPSET → ON START UP

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

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

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

### PRESCRIPTION DRUG SIDE EFFECTS

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

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

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



### Amino acid only side effect profile

- Dry mouth . . . . . 2.1%
  - Insomnia . . . . . 0.9%
  - Headache . . . . . 0.7%
  - Nausea . . . . . 0.6%
  - Dizziness . . . . . 0.4%
  - Constipation . . . 0.4%
- All other side effects occur at a rate less than 1 in 500 visits



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# REUPTAKE INHIBITORS CAN MAKE THE CAUSE OF THE DISEASE THEY ARE TREATING WORSE BY DEPLETING NEUROTRANSMITTER LEVELS.



## TAKING RESPONSIBILITY

The only defense raised by the reuptake inhibitor industry for its drugs making the cause of disease worse is asserting that the placebo effect in treatment with reuptake inhibitors is strong, leading to the argument, "How does one know when the treatment response is from the drug or placebo?" Therefore, when symptoms of disease return, not all episodes represent reuptake inhibitor tachyphylaxis resulting from depletion of neurotransmitters by the drugs. Some are due to a placebo event that no longer is displaying benefit.

This argument is ridiculous and only serves as a distraction from the real problem. Reuptake inhibitors can and do deplete neurotransmitters making the very cause of the disease worse — inadequate levels of neurotransmitters. Any attempt to throw up a smoke screen and assert that return of symptoms may be due to failure of the placebo effect is merely a deflection of responsibility.

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

**Medical Consult: 877-626-2220**

## The following peer-reviewed scientific articles are found on the web site [www.HinzMD.com](http://www.HinzMD.com)

MEDICAL FOODS based on scientific writings found in the following peer-reviewed scientific research:

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