**DRUG-INDUCED**

Monoamine related relative nutritional deficiency™

A relative nutritional deficiency occurs when an optimal diet does not meet the needs of the system.*

When there is not enough (low, inadequate, depleted, deficient, or suboptimal)™ serotonin or dopamine concentrations on an optimal diet, a relative nutritional deficiency™ is always present.*

The mechanism of action whereby reuptake inhibitor drugs deplete dopamine, norepinephrine, and epinephrine is illustrated in Figures 1-3. When reuptake inhibitor-induced depletion™ occurs on an optimal diet, a dopamine related relative nutritional deficiency™ (RND™) is always present. 

Access a YouTube video on this topic.
CLICK ON: >>>> [https://youtu.be/mIgsk86k9fE](https://youtu.be/mIgsk86k9fE)

**Figure 1:** Low synaptic serotonin or dopamine levels, on an optimal diet, this represents a relative nutritional deficiency™ of serotonin or dopamine precursors and cofactors. 11 *

**Figure 2:** Reuptake inhibition (yellow airplane) is blocking reuptake causing increase of serotonin and dopamine to move from the pre-synaptic neuron into the synapse. 11 *

**Figure 3:** The serotonin and dopamine molecules, while in a pre-synaptic neuron, is safe from enzymatic metabolism by the MAO and COMT systems. With redistribution into the synapse, there is increased metabolism (depletion). On an optimal diet, this depletion of serotonin or dopamine represents a drug-induced relative nutritional deficiency.™

When serotonin or dopamine depletion (RND™) is great enough, a relative nutritional deficiency™ of serotonin or dopamine precursors or cofactors may cause the patient to become suicidal, drugs may stop working, the patient might not be able to stop the drug due to feeling worse, and disease or disease-like symptoms may return. 11 *

**Management** of reuptake inhibitor-induced serotonin or dopamine related relative nutritional deficiency™ requires properly administered nutrients under the serotonin or dopamine protocol,™ respectively.™ Mastery of amino acid administration and amino acid side effects should be in place prior to starting the amino acids. 11 *

*Low or depleted serotonin on an optimal diet, use the serotonin protocol.*

*THIS APPROACH:* When suboptimal serotonin or dopamine concentrations exist on an optimal diet, a relative nutritional deficiency of the naturally occurring aromatic amino acids or cofactors is always present™.
REUPTAKE INHIBITORS DEPLETE
Serotonin, dopamine, and norepinephrine

The white areas on this page are screen shots from the peer-reviewed papers referenced.

Depletion of monoamine neurotransmitters is known in the literature to be associated with administration of reuptake inhibitors. Reuptake inhibitors are not just prescription drugs used for treatment of depression and attention-deficit disorder, but are also available as street drugs, such as amphetamines, “Ecstasy,” and methamphetamine.

International Journal of General Medicine 2012:5 413–430

The SSRI's block this reuptake leading to a depletion of serotonin after several weeks of treatment [16].

Canad. M. A. J. Feb. 1, 1959. vol. 80 186-190

The SSRI's in therapeutic doses consistently deplete serotonin after several weeks of treatment [33,34]

Journal of Experimental Medicine, 1954 615-628

The white areas on this page are screen shots from the peer-reviewed papers referenced.

Depletion of serotonin, dopamine, or norepinephrine by reuptake inhibitors, on an optimal diet, may display symptoms of depression-like drug-induced relative nutritional deficiency™ whose symptoms may be identical to worsening depression. Severe display of these depression-like symptoms may be associated with suicidal ideation.

Neuropsychiatric Disease and Treatment 2016:12 763–775

Platelets contain 90% of the releasable serotonin stores. Reuptake inhibitor studies demonstrate that 90% of platelet serotonin is depleted in about 3 weeks. Over 80% of the total releasable serotonin is depleted within 3 weeks after starting reuptake inhibitors. The mechanism of platelet depletion is attributed to blocking reuptake transport of serotonin into platelets while allowing for transport out of the platelets. Once outside the platelets, the serotonin is exposed to the monoamine oxidase enzymes that affect metabolism at a higher rate leading to depletion. This is the same mechanism of action observed with monoamine transport in and out of the presynaptic neurons of the CNS, as illustrated in Figures Neuropsychiatric Disease and Treatment 2016:12 763–775

Platelet 3H-imipramine binding, serotonin (5-HT) uptake and 5-HT concentrations were studied in 14 hospitalized patients with depressive disorder following 6 weeks of treatment with a selective 5-HT uptake blocker, fluoxetine. After 3 weeks of treatment there was a significant decrease in Bmax of 3H-imipramine binding and a significant increase in Kd. A highly significant decrease in Vmax of 5-HT uptake was seen after 3 weeks of treatment which was accompanied by a slight increase in Km. At the same time the platelet 5-HT content was significantly reduced by about 90% of its original level.

Neuropsychiatric Disease and Treatment 2016:12 763–775 5-HT; serotonin

Low or depleted serotonin on an optimal diet,™ use the serotonin protocol.

The Food and Drug Administration (FDA) has not evaluated these statements. These nutrients are not intended to diagnose, treat, cure, or prevent any disease.
As depletion of serotonin, dopamine, or norepinephrine by reuptake inhibitors on an optimal diet may display symptoms of depression-like drug-induced relative nutritional deficiency whose symptoms may be identical to worsening depression. Severe display of these depression-like symptoms may be associated with suicidal ideation.

Amitriptyline is an effective antidepressant. It is known that TCA potentiate the effects of amines on the CNS by blocking the re-uptake of norepinephrine at the nerve terminals. This action has also been used to explain the etiology of hypotension. After an initial hypertensive effect due to the blockage of norepinephrine uptake by the sympathetic nerve terminals, there is a gradual depletion of norepinephrine.

Current Pharmaceutical Design, 2006, 12, 47-57

As serotonin levels are known to be present in suicidal patients, this initial robust 5-HT1a dampening of serotonin neuronal activity may add to a low serotonin state, making a patient’s depressive state temporarily worse and causing suicidal thoughts and behaviors emerge acutely.


Low serotonin concentrations have been observed in depression and suicide.


WARNING: SUICIDAL THOUGHTS AND BEHAVIORS

See full prescribing information for complete boxed warning.

• Increased risk of suicidal thinking and behavior in children, adolescents, and young adults taking antidepressants (5.1).
• Monitor for worsening and emergence of suicidal thoughts and behaviors (5.1).

When using PROZAC and olanzapine in combination, also refer to Boxed Warning section of the package insert for Symbbyax.

Suicidality and Antidepressant Drugs
Antidepressant medicines may increase suicidal thoughts or actions in some children, teenagers, and young adults especially within the first few months of treatment. Depression and certain other serious mental illnesses are important causes of suicidal thoughts and actions. Patients of all ages who are started on antidepressant therapy should be monitored appropriately and observed closely for clinical worsening, suicidality, or unusual changes in behavior. Anyone considering the use of ZOLOFT or any other antidepressant in a child, adolescent, or young adult must balance this risk with the clinical need. ZOLOFT is not approved for use in pediatric patients except for patients with Obsessive-Compulsive Disorder (OCD).

Low or depleted dopamine or norepinephrine on an optimal diet,™ use the dopamine protocol.

Low or depleted serotonin on an optimal diet,™ use the serotonin protocol.
While only 7% to 13% of patients treated for depression with these drugs get results better than a sugar pill (placebo), 100% are exposed to this:

**FDA required prescribing information**

The Food and Drug Administration (FDA) has not evaluated these statements. These nutrients are not intended to diagnose, treat, cure, or prevent any disease.
ANTIDEPRESSANT DRUGS: The risks of depression getting worse and suicide.

Depression is caused by neurotransmitter (serotonin, dopamine, norepinephrine and epinephrine) levels that are not high enough.

The drugs listed on the front cover can set up conditions that deplete these neurotransmitters.

When drug-induced neurotransmitter depletion is great enough:
- The patient may become suicidal.
- The drugs may stop working.
- The patient may not be able to stop the drug secondary to feeling worse.
- Symptoms of disease may return.
- Nutritional deficiency may develop.

From the prescribing information for each drug:
Prozac: “There has been a long-standing concern, however, that antidepressants may have a role in inducing worsening of depression…”
Zoloft: “Patients with major depressive disorder (MDD), both adult and pediatric, may experience worsening of their depression…”
Celexa: “All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening…”
Paxil: “Patients of all ages who are started on antidepressant therapy should be monitored appropriately and observed closely for clinical worsening…”

What are the symptoms of drug-induced worsening?
Clinical Worsening and Suicide Risk (the following is listed by each drug):
- Worsening of depression
- Suicidal ideation
- Anxiety
- Agitation
- Panic attacks
- Insomnia
- Irritability
- Hostility
- Aggressiveness
- Impulsivity
- Akathisia (psychomotor restlessness)
- Hypomania
- Mania
- Other unusual changes in behavior

The drugs are associated with suicidal ideation.
Multiple studies found reuptake inhibitors are a drug class that offers only 7%–13% of patients relief of depression symptoms that is greater than that provided by placebo (a sugar pill).
They may make the disease cause worse by depleting the centrally acting monoamine neurotransmitters in all patients who do not ingest adequate amounts of serotonin and dopamine amino acid precursors, which leads to a relative nutritional deficiency.
They also expose 100% of patients to drug side effects and the economic costs of the drugs.

Inadequate levels of neurotransmitters in the brain are the leading cause of depression.
These drugs deplete neurotransmitters leading to depression getting worse and suicide-associated events.

Neurotransmitter

Drug blocking reuptake

Neurotransmitter

Receptor

With depression, there are not enough neurotransmitters conducting electricity between the nerve cells of the brain.

The MAO and COMT enzymes break down neurotransmitters outside the nerve cells.
These drugs set up conditions which move neurotransmitters from inside the nerve cells of the brain, where they are safe, to the space between nerve cells where they come in contact with the enzymes that break them down.

Low or depleted serotonin on an optimal diet, ™ use the serotonin protocol.
Role of norepinephrine in depression. AUTHORS: Delgado PL; Moreno FA, Department of Psychiatry, University of Arizona, J Clin Psychiatry 2000;61 Suppl 1:5-12

NE-selective antidepressant drugs appear to be primarily dependent on the availability of NE for their effects. Likewise, 5-HT-selective antidepressants appear to be primarily dependent on the availability of 5-HT for their effects.

**Phentermine** (A norepinephrine reuptake inhibitor) Reuptake inhibitors for weight loss lose effectiveness. Average weight loss:
- 12 pounds the first month.
- 12.5 pounds the first year

Data from over 2,000 patients Los Angeles clinic. Phentermine depletes norepinephrine and the drug quits working due to a **relative nutritional deficiency** of dopamine precursors and cofactors.

When phentermine quits working doctors increase the dose, this further depletes the norepinephrine.

**FOOD FOR THOUGHT**

Antidepressant Use During Pregnancy and the Risk of Autism Spectrum Disorder in Children
Taloua Boukhers, MsC; Odile Sheehy, MsC; Laurent Mottron, MD, PhD; Ani Bédard, PhD
JAMA Pediatrics
December 14, 2015

87% INCREASED RISK OF AUTISM
DRUG-INDUCED MONOAMINE DEPLETION

There is only one place that neurotransmitters come from: The nutrients that we take in are metabolized by enzymes into neurotransmitters.

Whenever inadequate concentrations of serotonin, catecholamines, and/or thiols exists on an optimal diet: A relative nutritional deficiency of their precursors and/or cofactors is always present.

When inadequate serotonin, dopamine, norepinephrine, epinephrine concentrations exists on an optimal diet, medicine turns a blind eye to the associated relative nutritional deficiencies that are the real cause of the problems.
ALCOHOL-INDUCED dopamine related relative nutritional deficiency™

A relative nutritional deficiency occurs when an optimal diet does not meet the needs of the system.*

Depletion of dopamine, norepinephrine, and epinephrine by alcohol occurs by a mechanism of action that is like depletion by reuptake inhibitor drugs. When alcohol-induced depletion occurs on an optimal diet, a dopamine related relative nutritional deficiency™ (RND™) is always present.*

Access a YouTube video on this topic. CLICK ON: >>>> https://youtu.be/mlgsk86k9fE

Figure 1: A normal synapse with normal dopamine concentrations.

Figure 2: Alcohol (ETOH) is causing increased excretion of dopamine (DA) from the pre-synaptic neuron into the synapse. 26

Figure 3: The dopamine molecules, while in a pre-synaptic neuron, are safe from enzymatic metabolism by the MAO and COMT systems. With the increased movement of dopamine molecules into the synapse caused by alcohol, there is an accelerated metabolism (depletion) of dopamine molecules. On an optimal diet, this depletion of dopamine represents a dopamine related relative nutritional deficiency.™ When alcohol-induced dopamine depletion™ (dopamine related RND™) becomes severe enough, daily alcohol intake is required to achieve functional levels of synaptic dopamine. 26 *

Management of dopamine related relative nutritional deficiency™ is under the dopamine protocol.™ As with any of the relative nutritional deficiencies™ complete resolution of alcohol induced relative nutritional deficiency symptoms may occur in one week or require many weeks of weekly nutrient adjustments. 26 *

Cerebral dopamine depletion is the hallmark of Parkinson disease. Nature Neuroscience Vol 7 No 7 Jul 2004 726-736