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ORIGINAL RESEARCH

Relative nutritional deficiencies associated with centrally acting monoamines

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Background: Two primary categories of nutritional deficiency exist. An absolute nutritional deficiency occurs when nutrient intake is not sufficient to meet the normal needs of the system, and a relative nutritional deficiency exists when nutrient intake and systemic levels of nutrients are normal, while a change occurs in the system that induces a nutrient intake requirement that cannot be supplied from diet alone. The purpose of this paper is to demonstrate that the primary component of chronic centrally acting monoamine (serotonin, dopamine, norepinephrine, and epinephrine) disease is a relative nutritional deficiency induced by postsynaptic neuron damage.

Materials and methods: Monoamine transporter optimization results were investigated, re-evaluated, and correlated with previous publications by the authors under the relative nutritional deficiency hypothesis. Most of those previous publications did not discuss the concept of a relative nutritional deficiency. It is the purpose of this paper to redefine the etiology expressed in these previous writings into the realm of relative nutritional deficiency, as demonstrated by monoamine transporter optimization. The novel and broad range of amino acid precursor dosing values required to address centrally acting monoamine relative nutritional deficiency properly is also discussed.

Results: Four primary etiologies are described for postsynaptic neuron damage leading to a centrally acting monoamine relative nutritional deficiency, all of which require monoamine transporter optimization to define the proper amino acid dosing values of serotonin and dopamine precursors.

Conclusion: Humans suffering from chronic centrally acting monoamine-related disease are not suffering from a drug deficiency; they are suffering from a relative nutritional deficiency involving serotonin and dopamine amino acid precursors. Whenever low or inadequate levels of monoamine neurotransmitters exist, a relative nutritional deficiency is present. These precursors must be administered simultaneously under the guidance of monoamine transporter optimization in order to achieve optimal relative nutritional deficiency management. Improper administration of these precursors can exacerbate and/or facilitate new onset of centrally acting monoamine-related relative nutritional deficiencies.

Keywords: nutritional deficiency, serotonin, dopamine, monoamine

Introduction

It is much more desirable to identify, address, and eliminate the cause of a disease than to treat its symptoms. Until this research project defined the relative nutritional deficiencies associated with disease and dysfunction of the centrally acting monoamines due to low or inadequate levels of neurotransmitters, there was no awareness of these nutritional deficiencies and no ability to address them properly and optimally.[1] The authors of this paper have published extensively on the topic of monoamine amino acid precursor

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management relating to various diseases and dysfunctions. Further research in the areas covered in the previous writings has revealed a relative nutritional deficiency (RND) etiology not previously recognized or reported. The novel concept of a monoamine-related RND is developed in this paper.[1–13]

Serotonin, dopamine, norepinephrine, and epinephrine are "centrally acting monoamines" (herein referred to as monoamine[s]), and are also involved in the control and regulation of peripheral functions.

This novel concept hypothesizes the etiology of chronic disease and/or regulatory dysfunctional symptoms to be inadequate levels of monoamines as opposed to low levels of synaptic monoamines. The RND described herein are the most prevalent type of nutritional deficiency afflicting humans. An extensive list of diseases, conditions, and dysfunctions has been identified in which synaptic monoamine RND are recognized (see Appendix A and Appendix B).[1–13] It is postulated that over 80% of humans suffer from symptoms relating to a serotonin and/or catecholamine RND. Monoamine-related RND was unrecognized prior to this research due to the inability to manage and verify results of monoamine transporter manipulation objectively. The organic cation transporters (OCT) are the primary determinants of intercellular and extracellular monoamine concentrations throughout the body.

Absolute nutritional deficiency versus RND

Two primary categories of nutritional deficiency exist, ie, absolute nutritional deficiency and RND.[1] Insufficient dietary nutrient intake causes absolute nutritional deficiencies. An absolute nutritional deficiency can be corrected by optimizing nutrient intake in the diet. Management of the problem is often enhanced by administration of nutritional supplements, but they are not required.[1]

When an RND exists, nutritional intake and systemic nutrient levels are normal. However, systemic needs are increased above normal by outside forces and cannot be achieved by dietary modification alone. Burns and postsurgical patients are examples where an RND may develop.[1]

In this paper, the authors discuss the novel finding that an RND is the primary etiology whenever there is a chronic disease or dysfunction relating to a compromise in the flow of electricity through the presynaptic neurons (axons) across the synapses then through the postsynaptic neurons (dendrites). An extensive list of diseases, conditions, and dysfunctions has been identified in which synaptic monoamine RND are recognized (see Appendix A and Appendix B).[1–13]

The monoamine-associated RND is by far the most prevalent constellation of nutritional deficiencies found in humans (see Appendix A and Appendix B). It is postulated that over 80% of humans suffer from symptoms relating to a serotonin and/or catecholamine RND. Conditions prior to in situ monoamine transporter optimization (MTO, referred to in some previous papers as OCT functional status optimization) made it impossible to achieve consistent results with the administration of monoamine amino acid precursors. With the invention and refinement of MTO, the ability to study, manipulate, and optimally manage monoamine-related RND became clinically possible.[1–13]

Four primary classes of monoamine-associated RND have been identified by this research project:

- RND associated with monoamine disease or dysfunction
- RND induced by inappropriate administration of amino acids
- RND induced iatrogenically or by the administration of certain drug classes
- RND associated with genetic defects or predisposition.

Endogenous versus competitive inhibition state

Serotonin and dopamine, and their precursors, exist in one of two distinctly unique and physiologically divergent states, ie, the endogenous state and the competitive inhibition state.[1–8,11–13] The monoamine hypothesis advocates that the etiology of disease symptoms and/or regulatory dysfunction in the endogenous state is low synaptic monoamine concentrations which induce trans-synaptic electrical defects. Under this model, an absolute nutritional deficiency type of approach is advocated, where simply returning synaptic monoamine levels to normal corrects the electrical problem, leading to relief of disease symptoms. None of this is true. There is no documentation illustrating that merely establishing normal synaptic neurotransmitter levels is effective in correcting an electrical defect.[1]

Subjects in the endogenous state, with and without monoamine-related disease, if not suffering from a monoamine-secreting tumor, cannot be differentiated by laboratory monoamine assays including MTO. Statistical distribution of monoamine levels are the same in subjects with and without disease.[1,5,7,11]

The term "competitive inhibition" refers primarily to interaction between monoamines and their amino acid precursors in synthesis, metabolism, and transport. The competitive inhibition state occurs when significant amounts of serotonin and dopamine amino acid precursors are administered simultaneously.