Phenylethylamine: More Than Just A Pea-Sized Neurochemical

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Chronic stress has long been held responsible as a primary contributor to neurotransmitter imbalance. Stress, both physical and emotional can lead to increased neurotransmitter excretion by the neurons in order to help the body cope with the situation. Acute stress is generally well tolerated by the body and normally does not lead to significant neurotransmitter imbalances. In contrast however, chronic stress will tax the nervous system and over time deplete neurotransmitter supplies. Poor dietary habits may also contribute to neurotransmitter imbalance, particularly when coupled with stress. The production of these brain chemicals is dependant upon sufficient levels of amino acid precursors. Diets which are low in protein may limit the supply of these amino acids.

In the situation where the neurotransmitter concentrations are too low, they will be unable to simultaneously engage enough of the post synaptic receptor sites needed to continue a message. This disrupts signal transduction and important messages may not be sent. Another common situation is that the levels of certain neurotransmitters become too high and as such, the frequency of inappropriate signals that are relayed increases. This increased signaling can be described as “static” and make it difficult for the neurons to discern between incoming signals that are important against those background signals that should be ignored.

Why choose nutrients over drugs?

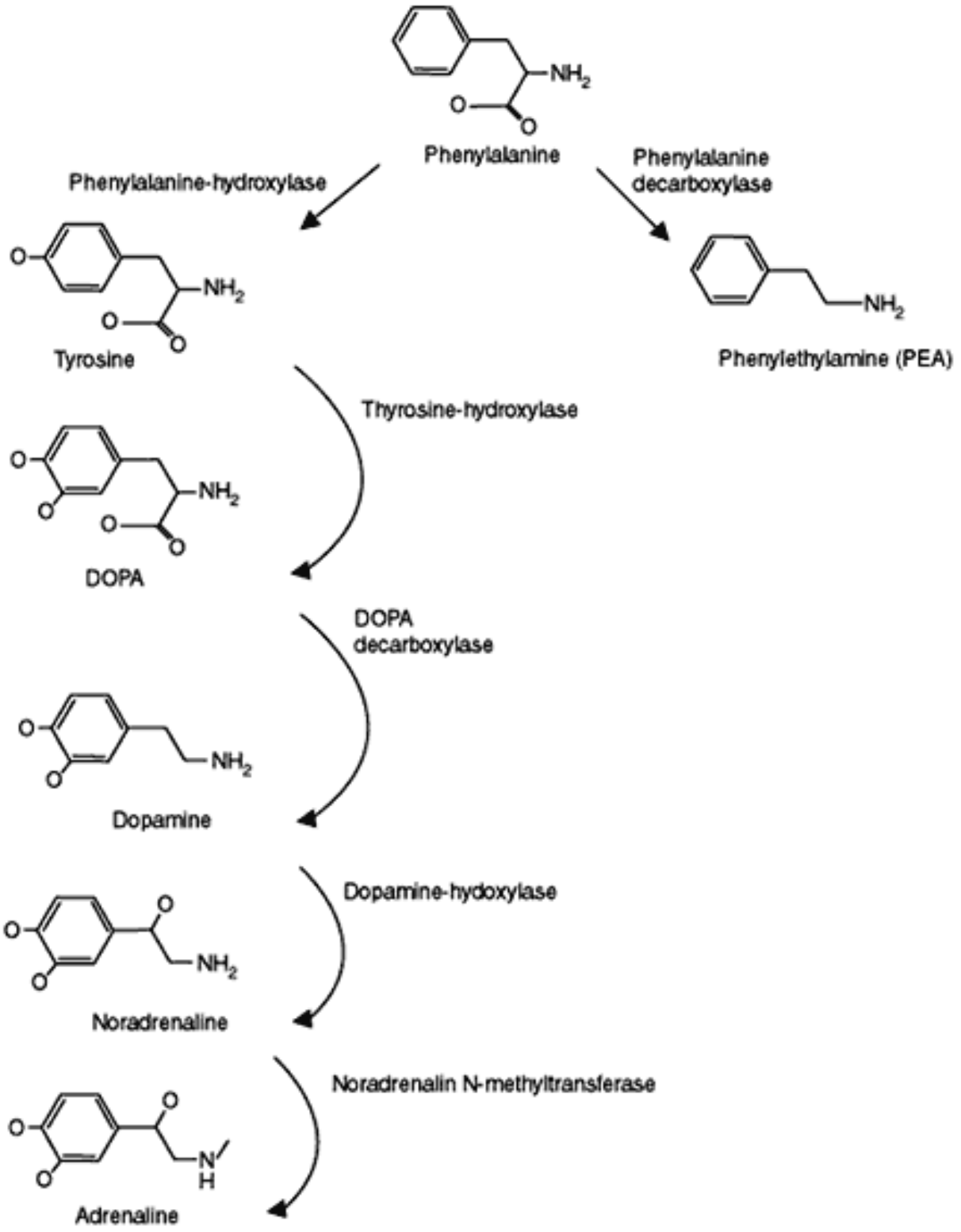
The pharmaceutical industry has developed hundreds of drugs designed to treat a whole host of mood disorders. The vast majority of these drugs work directly upon the function of neurotransmission. A major drawback of these drugs however, is that they only affect the transportation or release of existing pools of neurotransmitters in the body. If the diet does not provide sufficient amounts of neurotransmitter precursors, then there may not be enough neurotransmitters to properly relay signals within the nervous system, even if the drugs are used.

More than just a PEA-sized neurochemical

In many previous Nutritional News articles, we have addressed some of the major players involved in mood disorders– such as serotonin, dopamine, glutamate, gaba, norepinephrine and epinephrine. In this issue, we would like to focus on phenylethylamine (PEA) which is a stimulatory neurotransmitter that increases mental activity and alertness. PEA is classified as a minor neurotransmitter, this means that there is less of it; however it does not mean that it has any less significance than some of the other more well known and studied neurotransmitters. Early findings originally suggested that PEA was only a neuromodulator in that it altered the function or release of the major neurotransmitters, however in 2001 scientists discovered a receptor specific for PEA. [1] This then meant that classification for PEA changed from a neuromodulator to neurotransmitter as PEA has the potential to relay messages on its own.

Where does it come from?

PEA is synthesized from the amino acid phenylalanine via aromatic amino acid decarboxylase. It can be seen in the diagram below that phenylalanine, tyrosine and L-DOPA are all amino acids which can be used to support catecholamine synthesis. Phenylalanine however, will also increase PEA levels while the other amino acids further downstream, will not affect PEA. Understanding of this biochemical pathway becomes very important when choosing appropriate nutritional interventional therapies.



What role does PEA play in specific clinical conditions?

Depression

In some studies, patients with depression have exhibited decreased PEA levels, whilst some schizophrenic patients and other psychopathic subjects were associated with increased levels. In other research, administration of the precursor amino acid phenylalanine has demonstrated the ability to improve depression on its own and even the therapeutic outcome when used as an adjunct to some antidepressants. PEA has also been associated with the antidepressant effects after exercise. This observation determined that the excretion of a PEA metabolite increased by 77% during a 24 hour period which included 30 minutes of moderate exercise compared to the previous 24 hour period in which subjects abstained from physical activity. [2] Biochemically, PEA acts as an excitatory neurotransmitter and modulates neuron potentials to favour glutamate activity and neurotransmitter firing. As such, patients who have low PEA and related symptoms such as depression and fatigue are most

likely to have the best outcome when their therapies include products like Orthoplex Inkephalin which contains 400mg of phenylalanine. In saying this, products which contain phenylalanine, may not be suitable for patients who present with anxiety or insomnia and potentially have elevated PEA levels.

Schizophrenia
Excess PEA has been invoked particularly in paranoid schizophrenia, in which it is thought to act as an endogenous amphetamine and therefore, would be antagonized by neuroleptics. [3], [4]

Attention Deficit Hyperactive Disorder
Numerous studies have examined the levels of PEA in children with attention deficit hyperactivity disorder (ADHD) with results indicating urinary output is much lower in this population. [5], [6] One study assessed the urinary output of specific neurotransmitter metabolites, one of which being PEA in order to determine the neurochemical mechanism responsible in ADHD. It was found that PEA levels were significantly lower in ADHD individuals than in controls. The subjects with ADHD were then treated with methylphenidate and then further divided into methylphenidate responders and non-responders. It was shown that PEA levels were significantly increased after methylphenidate therapy in the responders, whereas the levels did not increase in non-responders. [7]

Other drugs and PEA

Methylphenidate (Ritalin) and dexamphetamine are two types of stimulant drugs commonly used to treat ADD/ADHD symptoms in children. Both of these drugs have been shown to dramatically increase PEA levels. However, these drugs also demonstrate some undesired side effects such as dependence, insomnia, gastrointestinal upset, over-stimulation, headache, tremor, anorexia, psychosis, mania and tachycardia to name but a few.

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