Amino Acid Team for the Human Psyche

We know the importance of amino acids in human physiology. We know that hundreds of grams of amino acids are recycled, synthesized, transported, degraded, regenerated, and rearranged on a daily basis to maintain normal body biochemistry in us. We know that these 20 standard amino acids make up the proteins our bodies use for a myriad of different functions. What many of us don’t know, is that when some of these little guys are supplemented in isolated form, some very interesting things happen.

DLPA

DLPA (dl-Phenylalanine) is an equimolar racemate of the two enantiomers of the amino acid phenylalanine. What this means is that this is a 50/50 mixture of the “almost” identical twins of phenylalanine, one being the mirror image of the other. The “l” enantiomer of phenylalanine is the natural form which gets incorporated into proteins. One of the pathways this amino acid goes down quite frequently is the catecholamine synthesis route. Catecholamines (figure 1) are the neurotransmitters synthesized by our adrenal glands that give us both the uplifting feeling and the “fight or flight” drive. In fact, the majority of our emotions are influenced by the types and amounts of these catecholamines in circulation. In patients starved of phenylalanine, there are marked decreases in dopamine (mood-enhancing / motor, cognitive function neurotransmitter) synthesis which result in EEG slowing and prolonged performance in neuropsychological tests (HC Lou, 1994) just to name a few, in which supplemental phenylalanine and tyrosine seemed to correct. This correction seemed to be a result in a Le Chatelier principle-like drive, forming product by adding the deficient substrate. Biochemical reaction kinetics has an interesting way of being manipulated by slight increases in these amino acids.

The “d” enantiomer, on the other hand, works its magic by a completely different mechanism. This “unnatural” form of phenylalanine is usually created by organic synthesis, although there are some natural sources. D-phenylalanine does its work by competitively inhibiting the enzyme Carboxypeptidase A (Christianson DW et al., 1989), the enzyme responsible for degrading enkephalins (oligopeptides involved in nociception via opiate receptor binding). What D-phenylalanine does by this action is actually increase the lifespan of these endorphin-like molecules, resulting in analgesic effects. There is no other natural supplement of its kind.
(with the exception of tyrosine) that has similar pain-killing mechanisms.

![L-Phenylalanine L-Tyrosine]

Figure 1. Structural similarities between catecholamines and their amino acid derivatives

GLUTAMINE

 Besides being one of the only amino acids that can actually cross the blood-brain barrier, this non-essential amino acid is the most abundant amino acid in the human body. Since the brain uses glucose and glutamine as its primary source of fuel, it’s pretty important that this amino acid gets to the brain. Since neurotransmitters involved in mood, memory, and mental balance are derived from amine sources, they use amino acids for their synthesis. One such amino acid that is used quite frequently for this purpose is L-glutamine. The therapeutic uses for glutamine include (but are not limited to): depression and various mood disorders, mental fatigue, memory, and stress tolerance (Ross J, 1999, Slagle P, 1992).

TYROSINE

 This non-essential amino acid is what results when phenylalanine gets hydroxylated (figure 1) in the stepwise pathway of making catecholamines. It is here where tyrosine meets the crossroads. It can either be made into thyroid hormones (T3 and T4), melanin, or into catecholamines, depending on what tissue is making the decision. In the process of making catecholamines in the adrenal glands, the enzyme tyrosine hydroxylase is the rate-limiting factor for the synthesizing of dopamine, norepinephrine, and epinephrine. A defect in this enzyme can be catastrophic to mood, appetite, metabolism, etc. Supplementation with L-Tyrosine has shown to yield exemplary health benefits. Again, due to an equilibrium shift in neurotransmitter biosynthesis, tyrosine has shown to increase plasma levels of some of our favorite catecholamines, like dopamine and norepinephrine (Rassmussen DD et al., 1983), which in turn take part in mood stabilization and cognitive endurance. In fact, tyrosine has shown incredible potential for improving cognitive motor performance under sleep deprivation and stress (Magill RA et al., 2003).

GABA

Γ-aminobutyric acid (GABA) is the commander in chief of the neuronal inhibitory network in higher mammals. Without this crucial amino acid trafficking neuronal spikes throughout the body, no one would have much control over their muscles or emotions when encountered with a few unkind words or threatening situations. People who have defects in either amounts of GABA synthesized or physiological factors leading to anxiety often take prescription medications which act like GABA, binding to its respective receptors (GABA_A,B,C receptor agonists). GABAergic receptors are the gateways in which certain ions flow in and out of neurons. Which ions and in what direction they flow determines neural membrane polarity (polarization / hyperpolarization) which in turn creates a neural spike and a resting period. GABA is the gate keeper to these ion channels, directing the traffic primarily of Cl^- or K^+ to achieve a non-exited neuron (hyperpolarization). Interestingly, babies use GABA primarily as an excitatory neurotransmitter and don’t do the transmembrane potential switch until early adolescence/adulthood (Li K et al., 2008). This is one of the reasons why we don’t give GABA supplements to infants.

References

HC Lou, Dopamine precursors and brain function in phenylalanine hydroxylase deficiency. John F Kennedy Institute, Gl. Landevej 7, DK-2600 Glostrup, Denmark; 1651-2227,1994


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