GABA

A Moderator for an Over-Excited Brain

- Promotes relaxation and enhances sleep
- Increases growth hormone levels
- Provides one of the highest dosing ranges available

Gluten Free  Vegan  Non-GMO  Brain Health  Sleep  Stress

AOR Code  Variant
AOR04313  60 VEGI-CAPS

Details
Neurotransmitters are messenger chemicals that communicate signals from the brain to the rest of the body. They control vital functions such as breathing, hunger and body temperature, but one of their most important roles is in regulating mood. Gamma aminobutyric acid, or GABA, is the most important inhibitory neurotransmitter in the brain. Inhibitory neurotransmitters protect against overstimulation or excitement of the brain, creating a calming effect which is important for mood, sleep, brain function and mental health. GABA is a natural remedy for improving and maintaining optimal mental health.

GABA has been studied for its effects in individuals experiencing chronic worry and insomnia as a result of not being able to “turn off” their thoughts at night. GABA is highly effective in reducing stress and anxiety, and promotes healthy sleep. It has been shown to affect the brain directly, increasing ?-brain waves (those associated with relaxation) and reducing ?-brain waves (those associated with worry and stress). During times of stress, GABA acts to regulate brain excitability and inhibits signals in the brain which cause mood imbalances and stress, effectively creating a ‘relaxing effect’. GABA also has a protective role in preventing cognitive disorders and may reduce the frequency and intensity of seizures.

GABA’s rapid action can increase the quality of life in those who suffer from worry and stress, those who have trouble sleeping such as older individuals, and may be a natural support for those with epilepsy.

Label Info
Discussion
GABA is a neurotransmitter that inhibits the activity of excitatory neuronal impulses to prevent the overstimulation of the brain. GABA has been referred to as the brain’s natural calming agent and helps to temporarily promote relaxation.

Product Variation

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<td>AOR04313</td>
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Supplements Facts

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<tr>
<th>Serving Size</th>
<th>Gamma-Aminobutyric acid</th>
<th>Amount</th>
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<tr>
<td>1 Capsule</td>
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<td>600 mg</td>
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Non-medical ingredients:

sodium stearyl fumarate. Capsule: hypromellose.

Guarantees

AOR™ guarantees that all ingredients have been declared on the label. Contains no wheat, gluten, corn, nuts, peanuts, sesame seeds, sulphites, mustard, soy, dairy, eggs, fish, shellfish or any animal byproduct.

Adult Dosage

Take 1-5 capsules daily without food, or as directed by a qualified health care practitioner.

Cautions

Consult a health care practitioner prior to use if you have epilepsy, if symptoms persist or worsen, or for use beyond 3 weeks. Consumption with alcohol, other medications or health products with sedative properties is not recommended. Do not use if pregnant or breastfeeding.

Source

Pharmaceutical synthesis

Main Application

Anxiety
Insomnia
Mood Disorders
Epilepsy

Disclaimer

The information and product descriptions appearing on this website are for information purposes only, and are not intended to provide or replace medical advice to individuals from a qualified health care professional. Consult with your physician if you have any health concerns, and before initiating any
new diet, exercise, supplement, or other lifestyle changes.

Research

Background

GABA, or Gamma-aminobutyric Acid, is a non-essential amino acid that is generally classified as a neurotransmitter. GABA is not found in significant amounts in food. Its status as an amino acid stems from the fact that it is a by-product of the decarboxylation of glutamic acid by vitamin B6. GABA’s neurotransmitter status has been sub-categorized to define it as an inhibitory neurotransmitter. The brain transfers signals by way of neurons sending impulses to one another through a network of junctions or gaps between nerve cells called synapses. GABA acts as an inhibitor of such impulses within the synapses of the human brain and spinal cord, effectively creating a ‘calming effect’ by preventing the overstimulation of the brain. The mechanism of action by which this is achieved depends on the binding of GABA to specific trans-membrane receptors within the plasma membrane of the neurons themselves, inhibiting both their pre- and post-synaptic impact.

Research
Research with GABA supplementation has focused on addressing conditions of anxiety, growth hormone deficiency, mood disorders, epilepsy, and various other disorders of the central nervous system. In the early 1980’s, researchers believed that manipulating GABA receptors could alleviate the symptoms of anxiety, and supplementation with GABA itself was one such form of manipulation. This conclusion has since been strengthened by numerous studies confirming a direct correlation between more serious mood disorders and significantly decreased GABA concentrations in the occipital cortex of MDD subjects. This well-established correlation has led to the use of supplemental GABA as a means of addressing not only the symptoms of anxiety, but also of poor mood, premenstrual dysphoric disorder and manic-depressive (bipolar affective) disorder. The strategy is relatively straightforward; GABA inhibits the production of excitatory impulses from reaching the brain, including those that enhance panic, alarm, and/or fear. Anti-anxiety drugs such as benzodiazepine that are normally prescribed for such conditions can become addictive, a risk that is non-existent with GABA supplementation. Indeed, some research even supports using GABA to facilitate withdrawal from benzodiazepine medications. In addition to mood disorders, GABA research has also been applied to the study of epilepsy, particularly in regard to how GABA supplementation can reduce the frequency and intensity of seizures. GABA is the major inhibitory neurotransmitter in the brain, and anticonvulsant drugs for the treatment of epileptic seizures are designed to enhance endogenous GABA levels (i.e. Vigabatrin) or mimic its effects (i.e. Gabapentin). This emphasis on maintaining high levels of GABA has once again led to the formalized justification of GABA supplementation, this time to offset epileptic seizures. Finally, researchers have long believed that GABA can alleviate the symptoms of insomnia due to its ability to generate calm (via its inhibition of excitatory neural impulses) and thus induce sleep. The increase of plasma growth hormone (which also rises naturally during sleep) is yet another capability that has been attributed to exogenous GABA supplementation. This capability has made GABA a relative staple of the life-extensionist movement, which is always concerned with halting and/or reversing the inverse relationship between age and growth hormone levels. Research to support this exists in both human and animal studies, with one trial showing that a single 5-gram oral dose can raise growth hormone levels by as much as 550% within 90 minutes of ingestion. Elevated growth hormone levels play an important role in the prevention of a multitude of age-related conditions, including sarcopenia, metabolic syndrome, osteoporosis, and an overall impaired quality of life.

Market Trends

It is a common occurrence that people turn to medications to manage their anxiety and sleep problems. Unfortunately these products can carry the risk of side effect and may not be effective over a longer period of usage time.

AOR Advantage

GABA is a natural neurotransmitter which is present in the body and offers a safe alternative to medications that are used to treat anxiety disorders. Anti-anxiety drugs such as benzodiazepines that are normally prescribed for such conditions can become addictive, a risk that is non-existent with GABA supplementation.

References


Abstract

Role of Gamma-aminobutyric Acid in Anxiety.


Enna SJ.

The development of anxioselective agents has made it possible to examine the biochemical basis of anxiety. Electrophysiological analysis revealed that benzodiazepines selectively enhance gamma-aminobutyric acid (GABA) neurotransmission. Subsequent work demonstrated the presence of a specific population of benzodiazepine binding sites on neuronal membranes. These sites appear to be linked to certain GABA receptors such that occupation of the benzodiazepine component reveals a group of GABA recognition sites that may be more sensitive to the neurotransmitter. These data, coupled with the findings that barbiturates may act, at least in part, by interacting with the GABA receptor-coupled chloride channel, suggest that pharmacological manipulations of the GABA system can alleviate the symptoms of anxiety. The anxioselectivity of the benzodiazepines may be related to the fact that they activate only a certain population of GABA receptors, whereas barbiturates can potentiate the majority of these sites. These discoveries point to the possibility that alterations in the GABA system may partially explain the neurochemical basis of anxiety.

Effect of Acute and Repeated Administration of Gamma aminobutyric Acid (GABA) on Growth Hormone and Prolactin Secretion in Man.


Cavagnini F, Invitti C, Pinto M, Maraschini C, Di Landro A, Dubini A, Marelli A

A single oral dose of 5 g gamma aminobutyric acid (GABA) was given to 19 subjects and serial
venous blood samples were obtained before and 3 h after drug administration. A placebo was administered to 18 subjects who served as controls. GABA caused a significant elevation of plasma growth hormone levels (P less than 0.001), but did not consistently alter plasma prolactin concentration since only 5 out of 15 subjects showed an increase of the hormone. Eight additional subjects were submitted to an insulin tolerance test before and after per os administration of 18 g GABA daily for 4 days. Protracted GABA treatment significantly blunted the response of growth hormone and enhanced that of prolactin to insulin hypoglycaemia (P less than 0.01). These results indicate that pharmacological doses of GABA affect growth hormone and prolactin secretion in man. The precise nature of GABA’s effects as well as its mechanism of action remains to be clarified.

GABA(A) Receptors in the Lateral Hypothalamus as mediators of satiety and body weight regulation

Brain Res. 2009 Jan 20.

Turenius CI, Htut MM, Prodon DA, Ebersole PL, Ngo PT, Lara RN, Wilczynski JL, Stanley BG.

In the lateral hypothalamus (LH), the inhibitory amino acid neurotransmitter, GABA, has had a long-standing presumptive role as an inhibitor of food intake. However, minimal investigation has been focused on GABA, especially as compared to the attention received by many peptide transmitters. To begin to address this deficiency in the understanding of the role of GABA in the LH and feeding, we report that antagonism of GABA(A) receptors in the rat LH elicits feeding, consistent with previous findings, and provide evidence for the behavioral selectivity of this effect. We extend previous findings that activation of LH GABA(A) receptors suppresses feeding, in particular by showing that nighttime and deprivation-induced eating are dramatically suppressed. Finally, we show that chronic activation, but not blockade, of the LH GABA(A) receptors leads to a reduction in 24h food intake with concomitant body weight loss. These data collectively suggest that activation of GABA(A) receptors plays a fundamental role in controlling food intake and body weight, a role that has previously been somewhat underestimated.