SUSTAINED RELEASE

MAG-AMIDE®

MAGTEIN® & NIACINAMIDE

Promotes brain health & cellular energy

endurance products COMPANY

Since 1978

WHAT IS IT?

MAG-AMIDE® Magtein® & Niacinamide is a sustained-release dietary supplement featuring Magtein magnesium L-threonate and niacinamide. The proprietary vegetable-based, wax-matrix tablet is formulated for slow, steady dissolution over 3 to 5 hours for optimal nutrient absorption and tissue retention.

HOW DOES IT WORK?

MAG-AMIDE delivers targeted brain health benefits by helping to maintain healthy neuron and cognitive function and fuel cellular energy production.

Magtein is a unique form of magnesium shown in pre-clinical studies to readily cross the blood-brain barrier and promote the synaptic plasticity neurons need to communicate. Inside neurons, magnesium plays a key role in cellular energy production.

Niacinamide, the amide form of niacin, also readily crosses the blood-brain barrier and is taken up by the brain. In neurons and other brain cells, niacinamide rapidly transforms into metabolically active cofactors that play a key role in cellular energy production, nerve function, and DNA repair.

WHO CAN BENEFIT?

For adults who need targeted nutritional support for optimal brain health, cognitive function and cellular energy production as they age.

PRODUCT AVAILABILITY

Bottle Size(s):
120 tablets

PRACTITIONER DISTRIBUTION

- WholeScript™ (www.wholescript.com)

Supplement Facts

Serving Size 2 Tablets
Servings Per Container 60

Amount Per 2 Tablets		% DV
Niacin (as niacinamide)	500 mg	3125%
Magnesium (from magnesium L-threonate)	84 mg	20%
Magnesium L-Threonate	1000 mg	*

* Daily Value (DV) not established.

Other Ingredients: Vegetable wax (rice bran and/or carnauba), stearic acid (vegetable), magnesium stearate (vegetable), and silica.

Suggested Use: Take two (2) tablets daily with a full glass of water, preferably with a meal, or as directed by your healthcare practitioner.

RESEARCH HIGHLIGHTS

As an NAD+ precursor, niacinamide helps offset the age-related decline in NAD+ production in cells and tissues

Nicotinamide adenine dinucleotide (NAD+) is an essential pyridine nucleotide present in all living cells. NAD+ acts as an important cofactor and substrate for a multitude of biological processes including energy production, DNA repair, gene expression, calcium-dependent secondary messenger signaling and immunoregulatory roles.

Emerging pre-clinical research indicates that NAD+ metabolism represents a promising therapeutic target for the treatment of metabolic and age-related disorders such as obesity, diabetes, cardiovascular and neurodegenerative diseases. Evidence suggests that raising NAD+ levels using niacinamide or other NAD+ precursors could slow down and reduce symptoms of metabolic stress and possibly treat age-related diseases.[1]

Researchers attribute the age-related depletion of NAD+ in aging cells to: (1) a need for a higher level of NAD+ to meet the metabolic demands of aging and age-related diseases; (2) the inability of cells to adequately synthesize enough NAD+ to meet metabolic demands; or, (3) a combination of both.[2]

Magtein promotes optimal brain health & cognitive function

One controlled clinical trial[3] indicates a potential role for Magtein magnesium L-threonate (1.5-2 g/day for 12 weeks) for treating cognitive impairment in older adults. This study involved 44 older adults, aged 50-70 years, with self-reported complaints of cognitive decline (memory and concentration), anxiety and poor sleep quality. Exclusion criteria included Alzheimer's disease or dementia, among other diseases.* Participants were given either Magtein (MMFS-01) or placebo for 12 weeks. Dosage was set to correspond to approximately 25 mg/kg/day. Subjects between 50-70 kg took 1.5 g/day of Magtein; subjects between 70-100 kg took 2 g/day. Compared to placebo, Magtein significantly ($P<.05$) improved overall cognitive ability as measured by a composite score of tests in four major cognitive domains (i.e., executive function, working memory, attention, and episodic memory). No significant between-group differences were reported for sleep and anxiety parameters.

* The complete exclusion criteria list includes active heart disease; uncontrolled high blood pressure; renal or hepatic impairment/disease; diabetes; bipolar disorder; Parkinson's disease; Alzheimer's disease or dementia.

Magtein supports learning and memory

Research in laboratory rats indicates the oral intake of Magtein magnesium L-threonate significantly increases brain magnesium and exerts a magnesium-mediated increase in synaptic plasticity and long-term potentiation.

- In one study,[4] Magtein significantly increased brain magnesium and enhanced learning abilities, working memory, and short- and long-term memory compared to no significant effect with common magnesium compounds. These findings indicate Magtein may help support learning and memory from a magnesium-mediated increase in synaptic plasticity and long-term potentiation.
- In one study,[5] Magtein increased brain magnesium levels and retention of the extinction of fear memory, without enhancing, impairing, or erasing the original fear memory. This action is attributed to an increase in NMDA receptor signaling, BDNF expression, density of presynaptic puncta, and synaptic plasticity in the prefrontal cortex. These findings suggest that elevation of brain magnesium might be a novel approach for enhancing synaptic plasticity in a region-specific manner leading to enhancing the efficacy of extinction without enhancing or impairing fear memory formation. These preliminary findings suggest a potential role in the treatment of fear-based anxiety and related behavioral disorders.
- In one study,[6] Magtein was found to enhance the consolidation/retention of a conditioned taste aversion, speed extinction, and inhibit spontaneous recovery of this learned aversion. These preliminary findings suggest a potential role in the treatment of fear-based anxiety and related behavioral disorders.

1. Braidy N, et al. *Exp Gerontol*. 2020;132:110831.
2. McReynolds MR, et al. *Exp Gerontol.* 2020;134:110888.
3. Liu G, et al. *J Alzheimers Dis.* 2016;49(4):971-90.
4. Slutsky I, et al. *Neuron.* 2010;65(2):165-77.
5. Abumaria N, et al. *J Neurosci.* 2011;31(42):14871-81.
6. Mickley GA, et al. *Pharmacol Biochem Behav.* 2013;106:16-26.