Homocysteine Metabolite Formula

(Hypoallergenic)

Item # 72580
90 Vegetarian Capsules

The Possible Benefits of Homocysteine Metabolite Formula, a Food Supplement

- Provides nutrients involved with regulation of homocysteine within normal levels
- Nutritionally supports liver detoxification pathways

Description

Homocysteine Metabolite Formula supports healthy homocysteine levels by providing the necessary nutrients to facilitate the breakdown of homocysteine into methionine and other metabolites. Folic acid, vitamin B12 (cyanocobalamin), vitamin B6, and the active form of B6, pyridoxal 5'-phosphate (P5P) play significant roles in homocysteine metabolism. Trimethylglycine (TMG) and serine are also involved in homocysteine pathways.

Homocysteine is metabolised from the essential amino acid methionine, and is then degraded through two pathways of liver detoxification, transsulfuration and re-methylation. The transsulfuration pathway requires both vitamin B6 and P5P, and involves interaction of homocysteine with serine to form cystathionine, that is subsequently converted to cysteine and then taurine. The re-methylation pathway has two intersecting biochemical pathways, each resulting in the formation of methionine. Re-methylation can occur via either the enzyme betaine homocysteine methyltransferase, to form methionine and dimethylglycine, or via the enzyme N-5-methyltetrahydrofolate homocysteine methyltransferase, to produce methionine and tetrahydrofolate. The process transfers a methyl group to homocysteine from methylcobalamin, which receives its methyl group from either S-adenosylmethionine (SAMe), from the active form of folic acid (5-methyltetrahydrofolate), or from TMG (betaine). The methionine formed from either reduction can be used in protein synthesis or converted to SAMe, which may be further used for polyamine synthesis or for re-synthesis of homocysteine via S-adenosyl-homocysteine. Homocysteine may affect all other methyl and sulphur group metabolism processes either directly or indirectly.

Normal homocysteine levels have been associated with cardiovascular health, normal metabolism and detoxification. One controlled human study looked at the status of vitamin B12, B6 and folate in a group of males with mild hyperhomocysteinemia, defined as plasma homocysteine concentrations in excess of 16.3 μmol/liter. These men had statistically significant lower mean plasma levels of all three nutrients compared to control subjects. When they took the nutrients for 6 weeks, their mean plasma homocysteine levels were significantly lowered. Because a weak negative correlation between folate and plasma homocysteine levels was found and no significant correlations were found for cobalamin or P5P, the possibility that a combined deficiency may be responsible for high homocysteine levels was considered. Another study of homocysteine metabolism found significantly lower plasma homocysteine levels in individuals who had a history of good heart health, and that folate and B12, but not B6, correlated inversely with plasma homocysteine levels. The authors concluded that impairment of the re-methylation pathway (dependent predominantly on folate and B12), rather than transsulfuration (dependent on B6), was the major determinant of increased homocysteine levels here. An analysis of elderly subjects from the original Framingham Heart Study group further implicated nutritional factors in elevated homocysteine plasma levels. In this group, plasma homocysteine levels were inversely correlated with plasma folate, and to a lesser extent, with B12 and B6. Inverse correlations were also found between plasma homocysteine levels and dietary intake of folate and B6, but not B12.

Elevated plasma homocysteine levels associated with vitamin deficiencies of folate and B12 can be rapidly normalised by correction of the

deficiency. Vitamin B6 deficiency alone does not appear to result in elevated homocysteine levels. Folic acid is able to lower homocysteine levels even in the absence of folate deficiency, probably as a result of conversion of excess folic acid to methyltetrahydrofolate, which increases the homocysteine re-methylation rate. Excess vitamin B6 and B12 do not have the same effect because, unlike methyl-tetrahydrofolate, they serve as cofactors rather than as cosubstrates. Hyperhomocysteinemia due to vitamin B12 deficiency does not respond to folate therapy alone because of the dependence on vitamin B12 for N-5-methyltetrahydrofolate homocysteine methyltransferase activity and remethylation. Whereas high basal homocysteine levels are believed to be due to impaired remethylation pathways, impaired P5P dependent transsulfuration is believed to be responsible for abnormally high homocysteine levels observed in methionine loading tests. This reasoning suggests pyridoxine in combination with folic acid is likely to be more effective in lowering hyperhomocysteinemia due to contributions from both pathways.

TMG has been shown to be helpful when other nutrients did not improve elevated homocysteine levels. TMG can stimulate remethylation of homocysteine to dimethylglycine in humans with pyridoxine-resistant homocysteinuria and hyperhomocysteinemia due to cystathione b-synthase deficiency. Additionally, TMG's methyl donating activity is crucial to liver function and detoxification, and TMG may protect against chemical damage to the liver.

While we may not yet know all the factors that contribute to regulation of homocysteine metabolism, folate, vitamin B12, vitamin B6, serine and trimethylglycine undoubtedly play significant roles.

Serving size: 1 Capsule
Servings per container: 90

Amount per serving:

Vitamin B6 (83% as Pyridoxine Hydrochloride & 17% as Pyridoxal-5'-Phosphate)	30	mg
Folic Acid	400	µg
Vitamin B12 (as Cyanocobalamin)	400	µg
Trimethylglycine	500	mg
L-Serine	100	mg

Other ingredients: Hydroxypropyl methylcellulose, silicon dioxide, L-leucine.

Suggested Use: As a food supplement, 1 or 2 capsules daily with meals, or as directed by a healthcare practitioner.

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