

Homocysteine Processor

Initially designed as a specialty formula for persons with high homocysteine levels, newer standards for homocysteine recognized by experts in prevention (below 6 UMOL/L) have moved this indispensable formula to the mainstream. An amino acid that accumulates when there are inadequate levels of the cofactors that process it (vitamins B6, pyridoxal-5-phosphate, B12, folic acid, 5-methyl tetrahydrofolate and trimethylglycine (TMG)), higher levels of homocysteine aggressively promote both atherosclerosis and dementia. Simply supplementing these cofactors to the diet may dramatically reduce circulating homocysteine. The trick is in achieving an adequate dose for the individual's physiology.

Designed to Combine The Homocysteine Processor, in conjunction with our Foundation Formula provides 9,600 mcg. folic acid, 800 of which is in the form of 5-methyl tetrahydrofolate, accommodating even those who have the SNP (genetic mutation) preventing them from converting folic acid into its active metabolite. The two formulas also supply 150 mg. of vitamin B6, **50% in its active form of pyridoxal-5-phosphate** for those who may not convert pyridoxine efficiently. Trimethylglycine, 1500 mg. is our additional methyl group donor, supporting the 600 mcg. of vitamin B12 (200 in the Foundation Formula and 400 in Homocysteine Processor) in converting homocysteine to methionine.

There is no formula on the market with as much processing power to lower dangerous homocysteine levels. The only remaining question is whether your patient will require one or two capsules per day. Simply follow the homocysteine level to determine the appropriate dose.

Related Abstracts

Serum vitamin B₁₂ levels are significantly low, and vitamin B₁₂ deficiency is significantly common in Alzheimer's disease patients.^{[28] [36] [37]} Supplementation of B₁₂ and/or folic acid may result in complete reversal in some patients, but generally there is little improvement in patients who have had Alzheimer's symptoms for greater than 6 months. It has been hypothesized that prolonged low levels of vitamin B₁₂ may lead to irreversible changes that will not respond to supplementation. **Pizzorno: Textbook of Natural Medicine, 2nd ed.**, Copyright © 1999 Churchill Livingstone, Inc. (Copied from MD Consult website www.MDconsult.com)

Arch Neurol. 1998 Nov;55(11):1449-55.

Folate, vitamin B12, and serum total homocysteine levels in confirmed Alzheimer disease.

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BACKGROUND: Recent studies suggest that vascular disease may contribute to the cause of Alzheimer disease (AD). Since elevated plasma total homocysteine (tHcy) level is a risk factor for vascular disease, it may also be relevant to AD. **OBJECTIVE:** To examine the association of AD with blood levels of tHcy, and its biological determinants folate and vitamin B12. **DESIGN:** Case-control study of 164 patients, aged 55 years or older, with a clinical diagnosis of dementia of Alzheimer type (DAT), including 76 patients with histologically confirmed AD and 108 control subjects. **SETTING:** Referral population to a hospital clinic between July 1988 and April 1996. **MAIN OUTCOME MEASURES:** Serum tHcy, folate, and vitamin B12 levels in patients and controls at entry; the odds ratio of DAT or confirmed AD with elevated tHcy or low vitamin levels; and the rate of disease progression in relation to tHcy levels at entry. **RESULTS:** Serum tHcy levels were significantly higher and serum folate and vitamin B12 levels were lower in patients with DAT and patients with histologically confirmed AD than in controls. The odds ratio of confirmed AD associated with a tHcy level in the top third (≥ 14 micromol/L) compared with the bottom third (≤ 11 micromol/L) of the control distribution was 4.5 (95% confidence interval, 2.2-9.2), after adjustment for age, sex, social class, cigarette smoking, and apolipoprotein E epsilon4. The corresponding odds ratio for the lower third compared with the upper third of serum folate distribution was 3.3 (95% confidence interval, 1.8-6.3) and of vitamin B12 distribution was 4.3 (95% confidence interval, 2.1-8.8). The mean tHcy levels were unaltered by duration of symptoms before enrollment and were stable for several years afterward. In a 3-year follow-up of patients with DAT, radiological evidence of disease progression was greater among those with higher tHcy levels at entry. **CONCLUSIONS:** Low blood levels of folate and vitamin B12, and elevated tHcy levels were associated with AD. The stability of tHcy levels over time and lack of relationship with duration of symptoms argue against these findings being a consequence of disease and warrant further studies to assess the clinical relevance of these associations for AD.

N Engl J Med. 2002 Feb 14;346(7):476-83.

Plasma homocysteine as a risk factor for dementia and Alzheimer's disease.

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BACKGROUND: In cross-sectional studies, elevated plasma homocysteine levels have been associated with poor cognition and dementia. Studies of newly diagnosed dementia are required in order to establish whether the elevated homocysteine levels precede the onset of dementia or result from dementia-related nutritional and vitamin deficiencies. **METHODS:** A total of 1092 subjects without dementia (667 women and 425 men; mean age, 76 years) from the Framingham Study constituted our study sample. We examined the relation of the plasma total homocysteine level measured at base line and that measured eight years earlier to the risk of newly diagnosed dementia on follow-up. We used multivariable proportional-hazards regression to adjust for age, sex, apolipoprotein E genotype, vascular risk factors other than homocysteine, and plasma levels of folate and vitamins B12 and B6. **RESULTS:** Over a median follow-up period of eight years,

dementia developed in 111 subjects, including 83 given a diagnosis of Alzheimer's disease. **The multivariable-adjusted relative risk of dementia was 1.4 (95 percent confidence interval, 1.1 to 1.9) for each increase of 1 SD in the log-transformed homocysteine value either at base line or eight years earlier. The relative risk of Alzheimer's disease was 1.8 (95 percent confidence interval, 1.3 to 2.5) per increase of 1 SD at base line and 1.6 (95 percent confidence interval, 1.2 to 2.1) per increase of 1 SD eight years before base line.** With a plasma homocysteine level greater than 14 micromol per liter, the risk of Alzheimer's disease nearly doubled. **CONCLUSIONS: An increased plasma homocysteine level is a strong, independent risk factor for the development of dementia and Alzheimer's disease.**

Stroke. 2002 Oct;33(10):2351-6.

Moderately elevated plasma homocysteine, methylenetetrahydrofolate reductase genotype, and risk for stroke, vascular dementia, and Alzheimer disease in Northern Ireland.

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BACKGROUND AND PURPOSE: Elevated plasma homocysteine level has been associated with increased risk for cardiovascular and cerebrovascular disease. Variation in the levels of this amino acid has been shown to be due to nutritional status and methylenetetrahydrofolate reductase (MTHFR) genotype. **METHODS: Under a case-control design we compared fasting levels of homocysteine and MTHFR genotypes in groups of subjects consisting of stroke, vascular dementia (VaD), and Alzheimer disease patients and normal controls from Northern Ireland.**

RESULTS: A significant increase in plasma homocysteine was observed in all 3 disease groups compared with controls. This remained significant after allowance for confounding factors (age, sex, hypertension, cholesterol, smoking, creatinine, and nutritional measures). MTHFR genotype was not found to influence homocysteine levels, although the T allele was found to increase risk for VaD and perhaps dementia after stroke. **CONCLUSIONS: We report that moderately high plasma levels of homocysteine are associated with stroke, VaD, and Alzheimer disease. This is not due to vascular risk factors, nutritional status, or MTHFR genotype.**