



YOUR Nutritional Needs genetic test Scientific Evidence Summary

MTHFR (+677):

Multiple published clinical studies^{1,2,3} indicate individuals with a suboptimal genotype (T,T) have a tendency to develop higher levels of the undesirable blood chemical homocysteine, and are at elevated risk for coronary heart disease, colon cancer, and neural tube defects in a setting of B-vitamin deficiency. Direct laboratory evidence has shown that the effects of this variation on enzyme efficiency can be reversed by vitamin B supplementation.⁴

TCN2 (+776):

One published study⁵ of vascular disease patients and healthy controls found an association between the suboptimal genotype and higher levels of the undesirable blood chemical homocysteine only among subjects with low vitamin B status. Another clinical study⁶ reported a significantly higher proportion of the TCN2 G,G (suboptimal) genotype among mothers of children born with neural tube defects.

SOD2 (+47):

In multiple published clinical studies^{7,8,9} individuals with the suboptimal genotype have been statistically shown to have an elevated risk for various types of cancer (Breast, Prostate, Ovarian); and clinical studies have shown that individuals with this genotype show reduced risks associated with higher antioxidant intake, which was not seen or not as strong in individuals with the opposing genotype. A large clinical study¹⁰ found that, among carriers of suboptimal genotype, higher antioxidant intake (i.e., Vit. C) resulted in significantly decreased risk of breast cancer. Another large clinical¹¹ study found significant interaction of antioxidant levels (selenium, Vit. E, lycopene) with reduced risk for prostate cancer only among men with the suboptimal C,C genotype.

Individuals with the suboptimal SOD2 genotype have been statistically shown to have an elevated risk for various types of cancer.

GSTM1:

Multiple published clinical studies^{12,13,14} have demonstrated that individuals with the suboptimal genotype have an elevated risk for various diseases (cancers, skin lesions, aplastic anemia) due to less efficient management of oxidative stress in their tissues. Clinical studies^{15,16} have shown that individuals with a null mutation especially benefit from higher intake of antioxidants (i.e., cruciferous vegetables).

Clinical studies show that individuals with the GSTM1 suboptimal genotype especially benefit from higher intake of antioxidants.

PON1 (+575):

Published clinical studies suggest that individuals with the suboptimal genetic variation have a slightly elevated risk for cardiovascular disease due to less efficient management of oxidative stress in their tissues. A large scientific analysis of several studies¹⁷ found a significant but modest increase in coronary heart disease risk for each copy of the G form of the variation. One clinical study¹⁸ demonstrated that individuals with the G,G result had a 3.6-fold elevated risk for ischemic stroke among young adults. A clinical study¹⁹ has demonstrated that a diet rich in the antioxidants in tomato juice resulted in a decline in blood markers of lipid (LDL) oxidation only in men who carried the suboptimal genotype.

XRCC1 (+26304):

In multiple published clinical studies,²⁰⁻²³ individuals suboptimal for this variation were demonstrated to maintain a lower risk or reduce their risk for certain diseases (i.e., breast cancer) when they increased their intake of dietary or supplemented antioxidants. In multiple published clinical studies,^{24,25} individuals with the suboptimal genotype were often associated with reduced risk for disease (i.e., cancers). Several published clinical studies,²¹⁻²³ which also monitored antioxidant intake or levels, demonstrated that the individuals with the C,T or T,T genotype only have reduced risk (for breast, lung cancer) with higher intake or levels of dietary or supplemented antioxidants (Vit. E, - carotene, Vit. C, fruits, vegetables).

Individuals with the G,G variation of PON1 had a 3.6-fold elevated risk for ischemic stroke among young adults.

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