GLUTATHIONE S-TRANSFERASE T1
GSTT1

Cahill, L.E., Fontaine-Bisson, B., and El-Sohemy, A. (2009). Functional genetic variants of glutathione S-transferase protect against serum ascorbic acid deficiency. Am J Clin Nutr 90, 1411–1417.

GSTT1 genotype interacts with Vitamin C intake to influence serum ascorbic acid levels

Canadian researchers at the University of Toronto have conducted a novel study that links vitamin C consumption, serum ascorbic acid levels, and *GSTT1* / *GSTM1* genotype. Their findings add to one other study that associated low ascorbic acid concentrations with the *GSTT1* null genotype. Together, these studies indicate how individuals with nonfunctional copies of *GSTT1* and *GSTM1* can mitigate against the harmful health implications of vitamin C deficiency.

To date, vitamin C is known to inhibit oxidative damage. Serum ascorbic acid levels have also been shown to be inversely correlated with risk of cardiovascular disease, blood pressure, hsCRP, diabetes, BMI, waist-to-hip ratio, cancer, and all cause mortality. Despite the well-established benefit of optimal serum ascorbic acid levels, deficiencies are common: 11-17% of young adults in Canada are deficient.

While the current recommended daily amount of vitamin C is 75 mg for women and 90 mg for men, there are considerable differences in serum acid levels when the *same* amount of vitamin C is consumed by *different* people.

As ascorbic acid and glutathione have been shown to be interdependent, protecting each other from oxidation, and as serum ascorbic acid levels differ with different *GST* genotypes, researchers in Toronto attempted to determine the relationship between vitamin C consumption, serum ascorbic acid levels, and *GST* genotype.

They took 1090 Canadian men and women of different ethnicities aged between 20-29 years old. All were recruited from the Toronto Nutrigenomics and Heath Study.

KEY FINDINGS:

- 31% of individuals were deleted for *GSTT1*
- A significant diet-gene interaction between dietary vitamin C, *GSTT1* & *GSTM1*
- *GSTT1* deleted individuals who *did not meet the RDA for vitamin C* had a 12-fold risk of vitamin C deficiency
- This deficiency was present, though less significant, for *GSTM1* deleted genotypes
- Individuals who met the RDA for vitamin C *had adequate serum ascorbic levels regardless of genotype*

The researchers concluded that, "identifying and targeting young individuals at risk of serum ascorbic acid deficiency could have important public health implications."

The study has important and practical implications. Firstly, it could explain some of the inconsistencies in studies that have examined *GST*

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genotype, heart disease, and cancer. If serum ascorbic acid levels are hugely lower in *GSTT1* / *GSTM1* deleted genotypes who fail to consume the RDA of vitamin C, and as low serum ascorbic acid is factor in heart disease and cancer, then *GST* genotype coupled with vitamin C consumption explains these inconsistencies: inadequate dietary vitamin C plus deleted *GSTT1* / *GSTM1* equals higher risk of heart disease and cancer.

Additionally, *GST* genotype could explain other chronic health conditions associated with low serum ascorbic acid, including obesity, BMI, waist-to-hip ratio, and high hsCRP.

Practically, individuals who are *GSTT1* or *GSTM1* deleted should be advised to consume at least the recommended daily amount of vitamin C (75-90 mg) from dietary sources. Should this prove difficult then a very low dose vitamin C supplement could be given as an insurance. There is currently no evidence that high dosages of vitamin C interact with *GST* genotype to afford greater protection.

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