

ELITE GENE LABS

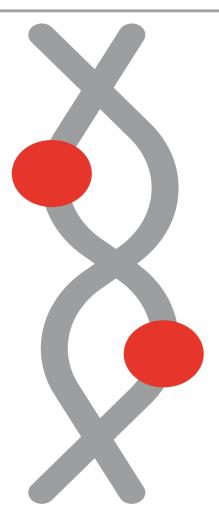
GENETIC POTENTIAL FOR OPTIMAL PERFORMANCE

Gene Comprehensive Nutrigenomic Report

Report Generated: August 12, 2025

Specimen Type: Buccal Swab Provider: ####### ###### Patient Name: ###### ######

Patient DOB: ##/##/###
Patient Gender: Female



Do not make any decisions about your health solely based on the information contained in this report. Always consult with a licensed and experienced health practitioner when you receive this report.



Elite Detox

Fagron Genomics US | 844-258-5564 | FagronGenomicsUS.com Lab | 807 Las Cimas Pkwy, Suite 145 | Austin, TX 78746 Laboratory Director: James W. Jacobson, Ph.D

- 49 - Female

(-/-) Normal Risk (-/+) Medium Risk (+/+) High Risk

rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
				DETOXIFIC	CATION		
rs6721961	NFE2L2	G/G (-/-)	Pterostilbene, Green Tea (Epigallocatechin Gallate), Tumeric, Sulforaphane, Endurance Exercise				
rs8190955	GSR	G/G (-/-)	Riboflavin, Reduced Glutathione				
rs13043752	AHCY	G/G (-/-)	N-Acetyl Cysteine (NAC), Glutathione				
rs234706	CBS	G/G (+/+)	Methyltetrahydrofolate, Methylcobalamin, Pyridoxal 5'- Phosphate (B6), Choline, Trimethylglycine, Serine, N- Acetyl Cysteine		Methylation Complete Pro if Homocyteine Levels Are High N-Acetyl Cysteine if Homocysteine Levels Are Low	Herbicide and Pesticide Avoidance Caution with Long Duration Anesthesia	Plasma Homocysteine
rs1021737	СТН	G/G (-/-)	N-Acetyl Cysteine (NAC), Glutathione				



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				DETOXIFIC	CATION		
rs17883901	GCLC	G/G (-/-)	Glutathione				
rs7483	GSTM3	C/C (-/-)	Glutathione	Liposomal G		Consider IV Glutathione Consider Pre-Anesthesia Glutathione	
rs1695	GSTP1	G/G (+/+)				Herbicide and Pesticide Avoidance Caution with Long Duration Anesthesia	
rs1050450	GPX1	G/G (-/-)	Glutathione, Selenium				



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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
				DETOXIFIC	CATION		
rs2070424	SOD1	A/A (+/+)					
rs4880	SOD2	A/A (+/+)	High Dose Antioxidants, Curcumin, Sulforaphane, Vitamin C	Intracellular Detox Complex	Consider High Dose Vitamin C Therapy	Consume Antioxidant Rich Diet	
rs2536512	SOD3	A/A (-/-)					
rs1801280	NAT2	T/T (-/-)	Silymarin, Alpha Lipoic Acid (ALA), P-5-P, Catechins				

Summary for Elite Detox

Highly Recommended Therapeutics

Provider Discretion: As Needed Formula Recommendations Lifestyle Recommendations

Laboratory Recommendations

DETOXIFICATION

- Herbicide and Pesticide Avoidance
- Caution with Long Duration Anesthesia
- Plasma Homocysteine

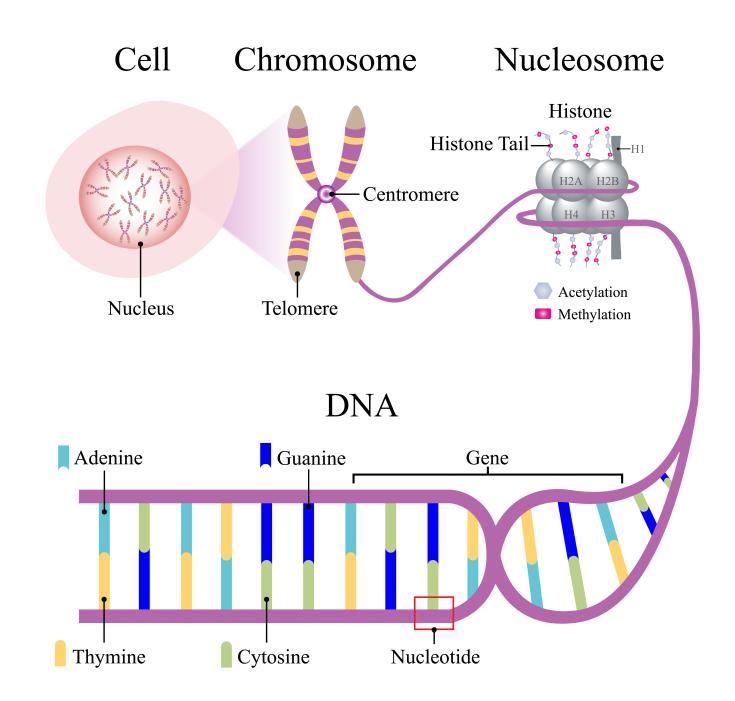
- Methylation Complete Pro if Homocyteine Levels Are High
- N-Acetyl Cysteine if Homocysteine Levels Are

• Liposomal G

- Consider IV Glutathione
- Consider Pre-Anesthesia Glutathione

• Intracellular Detox Complex

- Consider High Dose Vitamin C Therapy
- Consume Antioxidant Rich Diet



DETOXIFICATION

GLUTATHIONE IN DETOXIFICATION

Relevant genes for production are AHCY, CTH, GSTP1, GSTM1, GSTM3, GSR, MTRR & MTR

WHY IS IT IMPORTANT?



Maintains health by protecting the body from toxins



Regulates cell production and programmed cell death



Critical role in chemical detoxification



Vital for proper mitochondrial function



WAYS TO INCREASE GLUTATHIONE

- · Limit alcohol intake
- N-acetyl-cysteine (NAC)
- · Glutathione therapies
- (ie. IV Glutatione, Glutatione suppository, Lipsomal Glutatione)
- · Include whey in diet, unless allergic or intolerant
- · Methylation Support if necessary

SUPEROXIDES & ANTIOXIDANTS

- SOD1, SOD2, SOD3 genes are important to transform superoxides to protect against mitochondrial damage
- Reactive Oxygen Species (ROS) can damage mitochondria and cause cell death.
- Antioxidants such as Vitamin A, Vitamin C and Vitamin E act as a defense against ROS

DEFICIENCY CAUSES

- · Auto-immune diseases
- · Cardiovascular diseases
- · Neurodegenerative diseases
- · Cell death
- · Poor mitochondrial function

HIGH ANTIOXIDANT DIET

HIGH

ANTIOXIDANT

DIET

BENEFITS



Protection from oxidative stress



Helps reduce risk of heart disease, cancers & diabetes



Helps maintain functions of the liver, kidney, brain and digestive system

FOODS HIGH IN ANTIOXIDANTS



Dark chocolate



Fruit (berries, red grapes, prunes, apples, cherries, black plums)



Vegetables (artichokes, beets, dark leafy greens)



Beans (pinto, red, kidney, black)



Spices/herbs (cinnamon, oregano, turmeric, cumin, sage, thyme)



Whole grains (unless gluten free)



Nuts (pecans, walnut, hazelnut, pistachios, almonds, cashews, macadamias)



Beverages: juices (apple, tomato, pomegranate, pink grapefruit juice), teas (green, black)

SELENIUM

WAYS TO INCREASE LEVELS



Brazil nuts



Low-fat milk products



Meats & seafood fish (tuna, halibut, sardines), ham, shrimp, beef, liver, chicken, turkey



Boiled eggs



Whole grains (unless gluten free)



Wheat germ, Brewer's yeast



Supplements





SELENOMETHIONINE & SELENOCYSTEINE **ACTIVE FORM**



FUNCTIONS



Role in proper thyroid function & thyroid hormone metabolism



Role in reproduction



Role in DNA synthesis



Protection from infection & oxidative damage

DEFICIENCY VS HIGH INTAKE

Deficiency

- Cardiovascular disorders
- Developmental issues
- · Thyroid disorders
- · Joint & bone issues
- · Infertility issues
- Cancers

High intake

- · Metallic taste in mouth
- · Garlic odor of breath
- · Hair and nail loss or brittleness
- Nervous system abnormalities
- Nausea
- Diarrhea
- · Skin rashes
- Fatigue
- · Irritability

Gene Information Key

rsID	Gene	"-" variant	"+" variant
rs13043752	AHCY	G	Α
rs234706	CBS	Α	G
rs1021737	СТН	G	Т
rs17883901	GCLC	G	Α
rs1050450	GPX1	G	Α
rs8190955	GSR	G	Α
rs7483	GSTM3	С	Т
rs1695	GSTP1	Α	G
rs1801280	NAT2	Т	С
rs6721961	NFE2L2	G	Т
rs2070424	SOD1	G	Α
rs4880	SOD2	G	Α
rs2536512	SOD3	Α	G

Definitions

NFE2L2 rs6721961	The NFE2L2 (NFE2 like bZIP transcription factor 2) gene encodes a transcription factor, known as NRF2, that has a crucial role in the regulation of a network of antioxidant genes. NRF2 activates expression of genes with a conserved promoter sequence called the antioxidant response elements (ARE). Genes with an ARE include superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX), etc. Therefore, NRF2 is a master regulator of oxidant and antioxidant balance. The polymorphism rs6721961 occurs in the promoter region of NFE2L2, and mechanistic studies have found that the variant encoded by the T allele has reduced promoter activity and mRNA levels. Consistent with these findings, carriers of the T allele have been shown to have lower total antioxidant capacity. T allele carriers had less SOD, CAT, GPX, and glutathione activity. Furthermore, T allele carriers are at increased risk for insulin resistance and vascular stiffness.
GSR rs8190955	The GSR (glutathione-disulfide reductase) gene encodes a riboflavin-dependent enzyme that reduces oxidized glutathione (GSSH) to its antioxidant form (GSH). Therefore, GSR is essential for maintaining adequate glutathione levels and cellular antioxidant capacity. The polymorphism rs8190955 results in a cysteine substitution for an arginine residue at position 153. Mechanistic studies have shown that the A allele, which encodes a cysteine residue, produces an enzyme that is less stable than the version produced by the G allele. Furthermore, the instability was predicted to reduce enzyme function, and the A allele has been associated with obstructive heart defects and hereditary anemia.
AHCY rs13043752	The AHCY (adenosylhomocysteinase) gene encodes an enzyme that catalyzes the reversible hydrolysis of S-adenosylhomocysteine (SAH) to adenosine and homocysteine. Therefore, this enzyme, sometimes referred to as S-adenosylhomocysteine hydrolase (SAHH) regulates intracellular SAH concentrations, which acts as an inhibitor of most transmethylation enzymes. Deficiency in this protein can lead to increased levels of methionine. The polymorphism rs13043752 results in a tryptophan substitution for an arginine residue at position 38 in the enzyme. Mechanistic studies have found that the variant produced by the A allele, which encodes a tryptophan residue, has decreased enzyme activity, suggesting the A allele may result in a reduced methylation index or less methylation capacity.
CBS rs234706	The CBS (cystathionine beta-synthase) gene encodes an enzyme that catalyzes the first step in the transsulfuration pathway. More specifically, CBS, a pyridoxal 5'-phosphate-dependent enzyme, consumes serine to convert homocysteine to cystathionine, which is further catabolized to generate substrate for glutathione synthesis. Therefore, homocysteine clearance and glutathione synthesis converge on the function of CBS. The polymorphism rs234706 results in a nucleotide substitution in exon 8. Carriers of the G allele have been found to have higher levels of homocysteine and lower levels of cystathionine and betaine, consistent with reduced CBS activity. Furthermore, individuals with the GG genotype had higher plasma homocysteine following the ingestion of a methionine load, and individuals with the GG genotype were less responsive to folate supplementation to lower homocysteine levels. Lastly, the GG genotype is associated with increased risk for coronary artery disease.
СТН	The CTH (cystathionine gamma-lyase) gene encodes an enzyme that catalyzes the last step in the transsulfuration of L-methionine to L-cysteine. More specifically, it converts cystathionine, derived from methionine, into cysteine, which is utilized in the liver to synthesis glutathione, a ubiquitous antioxidant. As a results, CTH has an important role in glutathione production. The polymorphism rs1021737 results in an isoleucine substitution for a serine residue in the enzyme at position 403. The T allele, which encodes the isoleucine variant, has been associated with an accumulation of homocysteine.
GCLC rs17883901	The GCLC (glutamate-cysteine ligase catalytic subunit) gene encodes the first and rate-limiting enzyme in glutathione biosynthesis. Glutathione is a potent, nonprotein antioxidant that has an important role in protecting cells from oxidative stress and xenobiotics. Therefore, a reduction in biosynthetic capacity through reduction of GCLC activity results in reduced antioxidant capacity and activity of enzyme that use glutathione as a cofactor, such as glutathione transferase and glutathione peroxidase. The polymorphism rs17883901 occurs in the promoter region of the gene, and mechanistic studies have found that the variant encoded by the A allele reduces promoter activity by as much as 60% in cell models. Furthermore, A allele carriers had significantly increased risk for coronary heart disease and heart attack. Lastly, plasma glutathione levels were reduced in individuals with the AA genotype.
GSTM3	Glutathione S-transferase mu 3 is an enzyme that detoxifies drugs, environmental toxins, and carcinogens by conjugating toxins to glutathione and subsequent excretion by the kidneys. Mutations in GSTM3 are associated with decreased clearance of toxins, anesthetics and drugs from the nervous system.
GSTP1	The GSTP1 (glutathione S?transferase pi 1) gene encodes a cytosolic enzyme that has a keystone role in cellular detoxification. It conjugates cytotoxic and carcinogenic substances to glutathione for elimination, thereby aiding in antioxidant defense and preserving DNA integrity. The polymorphism rs1695 results in a valine substitution for an isoleucine residue in the enzyme at position 105, which is a region of the protein that is known to undergo several post-translational modifications. Mechanistic studies have shown that the protein produced by the G allele, which encodes a valine residue, has reduced substrate binding capacity and enzymatic activity. Numerous clinical studies have shown that the GG genotype is a risk factor for asthma, especially when individuals are exposed to environmental toxins, such as cigarette smoke or traffic-related air pollution. Additionally, the G allele is associated with increased risk for heart failure, and the frequency of the G allele is decreased in populations of older, living adults, suggesting it does not confer increased longevity.
GPX1 rs1050450	The GPX1 (glutathione peroxidase 1) gene encodes an antioxidant enzyme that consumes glutathione to protect cells from hydrogen peroxides. GPX1 is the most abundant enzyme in the GPX family, and its antioxidant activity is dependent on selenium status. The polymorphism rs1050450 results in a leucine substitution for a proline residue at position 200. The A allele, which encodes a leucine residue, is associated with reduced enzyme activity, resulting in less protection against oxidative stress. Additionally, the enzyme encoded by the A allele is less responsive to selenium supplementation. Consistent with reduced antioxidant capacity, the A allele is associated with higher levels of lipoperoxides in LDL and increased risk for coronary artery disease. The A allele is also associated with a longer duration of migraine attacks, and individuals in the AA genotype may have increased risk for inflammatory bowel disease, which is coupled with high levels of oxidative stress.

SOD1 rs2070424	The SOD1 (superoxide dismutase 1) gene encodes a copper and zinc-dependent enzyme that detoxifies superoxide radicals. The enzyme is soluble, and it primarily functions in the cytoplasm where it acts as a homodimer to convert naturally-occurring oxidants to molecular oxygen and hydrogen peroxide. Moreover, SOD enzymes participate in phase 1 detoxification to initiate the transformation of highly reactive oxygen species to a less reactive intermediary metabolite. Hydrogen peroxide, the intermediary metabolite produced by SOD activity, requires further detoxification by phase 2 enzymes, such as catalase or glutathione peroxidase. Nevertheless, SOD1 function is crucial to initiate the detoxification process of reactive oxygen species in the cytoplasm, aiding in protection against oxidative damage. The polymorphism rs2070424 occurs in the third intron, and carriers of the G allele were shown to have increased SOD1 activity in red blood cells. This increased activity was also associated with decreased mortality due to septic shock. Furthermore, the G allele was found to be protective against Alzheimer's disease and ulcerative colitis. Lastly, the A allele is associated with increased risk of type 2 diabetes and possibly PCOS, suggesting that the variant encoded by the A allele has decreased antioxidant function.
SOD2	The SOD2 (superoxide dismutase 2) gene encodes a mitochondrial matrix enzyme that uses iron and manganese to covert superoxide, a byproduct of the electron transport chain, to hydrogen peroxide and oxygen. Moreover, SOD2 participates in phase 1 detoxification to initiate the transformation of highly reactive oxygen species to a less reactive intermediary metabolite. Hydrogen peroxide, the intermediary metabolite produced by SOD2 activity, requires further detoxification by phase 2 enzymes, such as catalase or glutathione peroxidase. Nevertheless, SOD2 function is crucial to initiate the detoxification process of reactive oxygen species in the mitochondria, aiding in protection against oxidative damage. The polymorphism rs4880 results in a valine substitution for an alanine residue in the enzyme at amino acid position 16, which occurs in the mitochondrial targeting sequence. Mechanistic studies have shown that the A allele, which encodes a valine residue, has incomplete transfer to the mitochondria due to a conformational change in the targeting sequence. Furthermore, studies in animal models have shown that the enzyme encoded by the A allele has decreased formation of active enzyme in the mitochondrial matrix, and clinical studies have found individuals with the AA genotype to have less SOD2 activity. Consistently, individuals with the AA genotype have been shown to have decreased plasma total antioxidant status and increased markers of oxidative stress and lipid peroxidation. However, implementation of a healthy diet and exercise intervention has been shown to reduce markers of lipid peroxidation in those with the AA genotype. Lastly, the AA genotype was associated with increased risk for coronary heart disease.
SOD3 rs2536512	The SOD3 (superoxide dismutase 3) gene encodes a copper and zinc-dependent enzyme that detoxifies superoxide radicals. SOD3 is a soluble enzyme that primarily functions in extracellular spaces to covert naturally-occurring oxidants to molecular oxygen and hydrogen peroxide. Moreover, SOD enzymes participate in phase 1 detoxification to initiate the transformation of highly reactive oxygen species to a less reactive intermediary metabolite. Hydrogen peroxide, the intermediary metabolite produced by SOD activity, requires further detoxification by phase 2 enzymes, such as catalase or glutathione peroxidase. Nevertheless, SOD3 function is crucial to initiate the detoxification process of extracellular reactive oxygen species, aiding in protection against oxidative damage. The polymorphism rs2536512 results in a threonine substitution for an alanine residue at position 58. The A allele, which encodes a threonine residue, was associated with increased levels of protein, whereas individuals with the GG genotype were found to have reduced enzyme activity. Additionally, the GG genotype was associated with hypertension, and the G allele was associated with decreased lung function, suggesting that the G allele may be associated with decreased antioxidant function.
NAT2 rs1801280	The NAT2 (N-acetyltransferase 2) gene encodes an enzymes that catalyzes the transfer of an acetyl group from acetyl coenzyme A to compounds with aromatic amines, hetrocyclic amines, or hydrazine structures. Therefore, NAT2 has an important role in the metabolism and elimination of a large number of pharmaceutical drugs and environmental toxins. The polymorphism rs1801280 results in a threonine substitution for an isoleucine residue at position 114. The C allele, which encode a threonine residue, defines the NAT2*5B haplotype, which has been found to have slow acetylation activity. Consistent with this status, carriers of the C allele are at increased risk for various types of cancer and adverse drug reactions. Additionally, increased toxin exposure, such as frequent or intense smoking, can further increase these risks.

Disclaimers

TESTING:

Testing Performed By: AC

METHODOLOGY AND LIMITATIONS DISCLAIMER:

Testing for genetic variation/mutation on listed genes was performed using ProFlex PCR and Real-Time PCR with TaqMan® allele-specific probes on the QuantStudio 12K Flex. All genetic testing is performed by GX Sciences, LLC d/b/a Fagron Genomics US ("Fagron Genomics US") (807 Las Cimas Pkwy, Suite 145, Austin, TX. 78746). This test will not detect all the known alleles that result in altered or inactive tested genes. This test does not account for all individual variations in the individual tested. Test results do not rule out the possibility that this individual could be a carrier of other mutations/variations not detected by this gene mutation/variation panel. Rare mutations surrounding these alleles may also affect our detection of genetic variations. Thus, the interpretation is given as a probability. Therefore, this genetic information shall be interpreted in conjunction with other clinical findings and familial history for the administration of specific nutrients. Patients should receive appropriate genetic counseling to explain the implications of these test results. Details of assay performance and algorithms leading to clinical recommendations are available upon request. The analytical and performance characteristics of this laboratory developed test (LDT) were determined by Fagron Genomics US's laboratory (Laboratory Director: James Jacobson, PhD) pursuant to Clinical Laboratory Improvement Amendments (CLIA) requirements (CLIA #: 45D2144988).

MEDICAL DISCLAIMER:

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Fagron Genomics US SNP References

NFE2L2 rs6721961

• Marzec, J. M., Christie, J. D., Reddy, S. P., Jedlicka, A. E., Vuong, H., Lanken, P. N., Aplenc, R., Yamamoto, T., Yamamoto, M., Cho, H.-Y., & Kleeberger, S. R. (2007). Functional polymorphisms in the transcription factor NRF2 in humans increase the risk of acute lung injury. FASEB Journal: Official Publication of the Federation of American Societies For Experimental Biology, 21(9), 2237–2246. https://doi.org/10.1096/fj.06-7759com • Reuland, D. J., McCord, J. M., & Hamilton, K. L. (2013). The role of Nrf2 in the attenuation of cardiovascular disease. Exercise and Sport Sciences Reviews, 41(3), 162–168. https://doi.org/10.1097/j.S., Sbinizry, S. J. (2017). The Role of Nrf2 in Cardiovascular Function and Cellular Longevity, 2017, 9237263 * Shimizry, S., Mimmar, J., Hassegawa, M., White, S. J. (2017). The Role of Nrf2 in Cardiovascular Function and Cellular Longevity, 2017, 9237263 * https://doi.org/10.1371/journa, H., Tomita, H., Yamamoto, M., Nakaji, S., & Itoh, K. (2020). Association of single nucleotide polymorphisms in the NRF2 promoter with vascular stiffness with aging, 10. 1007/journal.pone.0236834 * Wang, X., Chen, H., Liu, J., Ouyang, Y., Wang, D., Bao, W., & Liu, L. (2015). Association between the NF-E2 Related Factor 2 Gene Polymorphism and Oxidative Stress, Anti-Oxidative Status, and Newly-Diagnosed Type 2 Diabetes Mellitus in a Chinese Population. International Journal of Molecular Sciences, 16(7), 16483–16496. https://doi.org/10.3390/ijms160716483

GSR rs8190955

• Krishnamurthy, H. K., Rajavelu, I., Pereira, M., Jayaraman, V., Krishna, K., Wang, T., Bei, K., & Rajasekaran, J. J. (2024). Inside the genome: Understanding genetic influences on oxidative stress. Frontiers in Genetics, 15, 1397352. https://doi.org/10.3389/fgene.2024.1397352 • Vyas, B., Bhowmik, R., Akhter, M., & Ahmad, F. J. (2022). Identification, analysis of deleterious SNPs of the human GSR gene and their effects on the structure and functions of associated proteins and other diseases. Scientific Reports, 12(1), 5474. https://doi.org/10.1038/s41598-022-09295-6

AHCY rs13043752

• Feng, Q., Keshtgarpour, M., Pelleymounter, L. L., Moon, I., Kalari, K. R., Eckloff, B. W., Wieben, E. D., & Weinshilboum, R. M. (2009). Human S-adenosylhomocysteine hydrolase: Common gene sequence variation and functional genomic characterization. Journal of Neurochemistry, 110(6), 1806–1817. https://doi.org/10.1111/j.1471-4159.2009.06276.x • Fumi?, K., Beluzi?, R., Cuk, M., Paykov, T., Kloor, D., Bari?, I., Miii?, I., & Vuorek, O. (2007). Functional analysis of human S-adenosylhomocysteine hydrolase isoforms SAHH-2 and SAHH-3. European Journal of Human Genetics: EJHG, 15(3), 347–351, https://doi.org/10.1038/si.eiho.5201757

CBS rs234706

• Aras, O., Hanson, N. Q., Yang, F., & Tsai, M. Y. (2000). Influence of 699C-->T and 1080C-->T polymorphisms of the cystathionine beta-synthase gene on plasma homocysteine levels. Clinical Genetics, 58(6), 455–459. https://doi.org/10.1034/j.1399-0004.2000.580605.x • De Stefano, V., Dekou, V., Nicaud, V., Chasse, J. F., London, J., Stansbie, D., Humphries, S. E., & Gudnason, V. (1998). Linkage disequilibrium at the cystathionine beta synthase (CBS) locus and plasma levels of homocysteine. The Ears II Group. European Atherosclerosis Research Study. Annals of Human Genetics, 62(Pt 6), 481–490. https://doi.org/10.1046/j.1469-1809.1998.6260481.x • Fredriksen, A., Meyer, K., Ueland, P. M., Vollset, S. E., Grotmol, T., & Schneede, J. (2007). Large-scale population-based metabolic phenotyping of thirteen genetic polymorphisms related to one-carbon metabolism. Human Mutation, 28(9), 856–865. https://doi.org/10.1002/humu.20522 • Kruger, W. D., Evans, A. A., Wang, L., Malinow, M. R., Duell, P. B., Anderson, P. H., Block, P. C., Hess, D. L., Graf, E. E., & Upson, B. (2000). Polymorphisms in the CBS gene associated with decreased risk of coronary artery disease and increased responsiveness to total homocysteine lowering by folic acid. Molecular Genetics and Metabolism. 70(1), 53–60. https://doi.org/10.106/mgme.2000.2993

CTH

• Wenstrom, K. D., Johanning, G. L., Owen, J., Johnston, K. E., Acton, S., & Tamura, T. (2000). Role of amniotic fluid homocysteine level and of fetal 5, 10-methylenetetrahydrafolate reductase genotype in the etiology of neural tube defects. American Journal of Medical Genetics, 90(1), 12–16. https://doi.org/10.1002/(sici)1096-8628(20000103)90:112::aid-aimq3>3.0.co:2-h

GCLC rs17883901

• Azarova, I., Klyosova, E., Lazarenko, V., Konoplya, A., & Polonikov, A. (2020). Genetic variants in glutamate cysteine ligase confer protection against type 2 diabetes. Molecular Biology Reports, 47(8), 5793–5805. https://doi.org/10.1007/s11033-020-05647-5 • Koide, S., Kugiyama, K., Sugiyama, S., Nakamura, S., Fukushima, H., Honda, O., Yoshimura, M., & Ogawa, H. (2003). Association of polymorphism in glutamate-cysteine ligase catalytic subunit gene with coronary vasomotor dysfunction and myocardial infarction. Journal of the American College of Cardiology, 41(4), 539–545. https://doi.org/10.1016/s0735-1097(02)02866-8 • Zuo, H., Xu, W., Luo, M., Zhu, Z., & Zhu, G. (2007). [The glutamate-cysteine ligase catalytic subunit gene C-129T and modifier subunit gene G-23T polymorphisms and risk for coronary diseases]. Zhonghua Xin Xue Guan Bing Za Zhi, 35(7), 637–640.

GSTM3

• Patskovsky, Y. V, Huang, M. Q., Takayama, T., Listowsky, I. & Pearson, W. R. Distinctive structure of the human GSTM3 gene-inverted orientation relative to the mu class glutathione transferase gene cluster. Arch. Biochem. Biophys. (1999). doi:10.1006/abbi.1998.0964 • Maes, O. C., Schipper, H. M., Chong, G., Chertkow, H. M. & Wang, E. A GSTM3 polymorphism associated with an etiopathogenetic mechanism in Alzheimer disease. Neurobiol. Aging (2010). doi:10.1016/j.neurobiolaging.2008.03.007

GSTP1

* Dai, X., Bui, D. S., & Lodge, C. (2021). Glutathione S-Transferase Gene Associations and Gene-Environment Interactions for Asthma. Current Allergy and Asthma Reports, 21(5), 31. https://doi.org/10.1007/s11882-021-01005-y- do Nascimento, M. R., Silva de Souza, R. O., Silva, A. L., Lima, E. S., Gonçalves, M. S., & de Moura Neto, J. P. (2021). GSTP1 rs1695 and rs1871042, and SOD2 rs4880 as molecular markers of lipid peroxidation in blood storage. Blood Transfusion on Ed Sangue, 19(4), 309–316. https://doi.org/10.2450/2020.0065-20 - Hollman, A. L., Tchounwou, P. B., & Huang, H.-C. (2016). R. A. L., Tchounwou, P. B., & Huang, H.-C. (2016). Superial mily with SNP Variants. International Journal of Environmental Research and Public Health, 13(4), 379. https://doi.org/10.1039/jen/103040379 - Katsarou, M.-S., Giakoumaki, M., Papadimitriou, A., Demertzis, N., Androuve, P. B., & Drakoulis, N. (2018). Genetically driven antioxidant capacity in a Caucasian Southeastern European population. Mechanisms of Ageing and Development, 172, 1–5. https://doi.org/10.1016/j.mad.2017.08.010 - Melén, E., Nyberg, F., Lindgren, C. M., Berglind, N., Zucchellii, M., Nordling, E., Hallberg, J., Svartengren, M., Moyre, M., Servar, J., Bellander, T., Wickman, M., & Pershagen, G. (2008). Interactions between glutathione S-transferases P1, tumor necrosis factor, and traffic-related air pollution for development of childhood allergic diseases. Environmental Health Perspectives, 116(8), 1077–1084. https://doi.org/10.1288/ehp.110.1288/ehp.1110.1288/ehp.1110.1288/ehp.1110.1288/ehp.1110.1288/ehp.

GPX1 rs1050450

*Costa Pereira, C., Durães, C., Coelho, R., Grácio, D., Silva, M., Peixoto, A., Lago, P., Pereira, M., Catarino, T., Pinho, S., Teixeira, J. P., Macedo, G., Annese, V., & Magro, F. (2017). Association between Polymorphisms in Antioxidant Genes and Inflammatory Bowel Disease. PloS One, 12(1), e0169102. https://doi.org/10.1371/journal.pone.0169102 bottom. In selenoprotein genes modulate biomarkers of selenium derraschien in selenoprotein in selenoprotein in selenoprotein (Einburgh, Scotland), 38(2), 539–548. https://doi.org/10.1016/j.chu.2018.pd. Corello, L. L., Goraga, B. M. de S., Bonecini-Almeida, M. de S., Goracini-Almeida, M. de S., Goracini-In-Almeida, M. de S., Goracin

E. V., Raptis, A., Kalliantasi, A., Dardiotis, E., & Drakoulis, N. (2023). Variability in oxidative stress-related genes (SOD2, CAT, GPX1, GSTP1, NOS3, NFE2L2, and UCP2) and susceptibility to migraine clinical phenotypes and features. Frontiers in Neurology, 13, 1054333. https://doi.org/10.3389/fneur.2022.1054333

SOD1 rs2070424

• Bizor, A., Tchórz, A., Madej, P., Le?niewski, M., Wójtowicz, M., Piwowar, A., & Franik, G. (2022), The Activity of Superoxide Dismutase, Its Relationship with the Concentration of Zinc and Copper and the Prevalence of rs2070424 Superoxide Dismutase Gene in Women with Polycystic Ovary Syndrome-Preliminary Study, Journal of Clinical Medicine, 11(9), 2548. https://doi.org/10.3390/jcm11092548 • Costa, N. A., Cunha, N. B., Gut, A. L., Azevedo, P. S., Polegato, B. F., Zornoff, L. A. M., de Paiva, S. A. R., Reis, B. Z., Fernandes, A. A. H., Rogero, M. M., & Minicucci, M. F. (2018). Erythrocyte SODI activity, but not SOD1 polymorphisms of A2516 SOD1 activity, but not SODI polymorphisms of A2516 SOD1 and C-262T Daylorphisms of A2516 SOD1 and C-262T CAT. Journal of Medical Biochemistry, 35(3), 333–336. https://doi.org/10.1515/jomb-2016-0002 • Spisak, K., Klimkowicz-Mrowiec, A., Pera, J., Dziedzic, T., Aleksandra, G., & Slowik, A. (2014). Rs2070424 of the SOD1 gene is associated with risk of Alzheimer's disease. Neurologia I Neurochirurgia Polska, 48(5), 342–345. https://doi.org/10.1016/j.pinns.2014.09.002 • Yin, J., Wang, X., Li, S., Zhu, Y., Chen, S., Li, P., Luo, C., Huang, Y., Li, X., Hu, X., Yang, W., Bao, W., Shan, Z., & Liu, L. (2019). Interactions between plasma copper concentrations and SOD1 gene polymorphism for impaired glucose regulation and type 2 diabetes. Redox Biology, 24, 101172. https://doi.org/10.1016/j.redox.2019.101172

SOD2

• Becer, E., & Ç?rako?lu, A. (2015). Association of the Ala16Val MnSOD gene polymorphism with plasma leptin levels and oxidative stress biomarkers in obese patients. Gene, 568(1), 35–39. https://doi.org/10.1016/j.(inbiochem.2015.05.009 • Bresciani, G., González-Gallego, J., da Cruz, I. B., de Paz, J. A., & Cuevas, M. J. (2013). The Ala16Val MnSOD gene polymorphism modulates oxidative response to exercise. Clinical Biochemistry, 46(4-6-), 335–340. https://doi.org/10.1016/j.(cliniochem.2012.11.020 • Broz, M., Eura, U. (2022). The Effect of the Ala16Val Mutation on the Secondary Interview of the Manganeses Superoxide Dismutase Mitochondrial Targeting Sequence. Antioxidants, 11(12), 248. https://doi.org/10.309/antiox1122348 • Ftp., A, S.-P., C., S., D., D., G., & J., D., G., & J., M. (2013). Participation of 47C-T SNP (Ala-9Val polymorphism) of the SOD2 gene in the intracellular environment of human period mononuclear desired in the intracellular environment of human period of the SoD2 gene in the intracellular environment of human period of the SoD2 gene in the polymorphism of the SoD2 gene in the production of intracellular reactive species in peripheral blood mononuclear cells with and without lipopolysaccharides induction. Free Radical Research, 48(2), 190–199. https://doi.org/10.1016/j.diorg/10.116/j.0179. The Ala16Val Mutation on the Secondary in the intracellular environment of the National Academy of Solicas Solicas of the National Academy of Sciences of the United States of America, 39(9), 4471–4473. * Sutton, A., Khony, H., Prip'-Bus, C., Cepsoul, F. (2003). The Ala16Val Mutation on the Secondary in the Intracellular reactive species in peripheral blood mononuclear cells with and without incorpolysearcharides in the mitocharia of the National Academy of the Nat

Pharmacogenetics, 13(3), 145-157. https://doi.org/10.1097/01.fpc.0000054067.64000.8f

SOD3 rs2536512

• Decharatchakul, N., Settasatian, N., Komanasin, N., Kukongviriyapan, U., Intharapetch, P., Senthong, V., & Sawanyawisuth, K. (2020). Association of combined genetic variations in SOD3, GPX3, PON1, and GSTT1 with hypertension and severity of coronary artery disease. Heart and Vessels, 35(7), 918–929. https://doi.org/10.1007/s00380-020-01564-6 • Dong, X., Li, D., Liu, H., & Zhao, Y. (2014). SOD3 and eNOS genotypes are associated with SOD activity and NOx. Experimental and Therapeutic Medicine, 8(1), 328–334. https://doi.org/10.1392/etm.2014.1720 • Ganguly, K., Depner, M., Fattman, C., Bein, K., Oury, T. D., Wesselkamper, S. C., Borchers, M. T., Schreiber, M., Gao, F., von Mutius, E., Kabesch, M., Leikauf, G. D., & Schulz, H. (2009). Superoxide dismutase 3, extracellular (SOD3) variants and lung function. Physiological Genomics, 37(3), 260–267. https://doi.org/10.1152/physiologenomics.90363.2008 • Pietzner, M., Wheeler, E., Carrasco-Zanini, J., Cortes, A., Koprulu, M., Wörheide, M. A., Oerton, E., Cook, J., Stewart, I. D., Kerrison, N. D., Luan, J., Raffler, J., Arnold, M., Arlt, W., O'Rahilly, S., Kastenmüller, G., Gamazon, E. R., Hingorani, A. D., Scott, R. A., ... Langenberg, C. (2021). Mapping the proteo-genomic convergence of human diseases. Science (New York, N.Y.), 374(6569), eabj1541. https://doi.org/10.1126/science.abj1541

NAT2 rs1801280

• García-Closas, M., Malats, N., Silverman, D., Dosemeci, M., Kogevinas, M., Hein, D. W., Tardón, A., Serra, C., Carrato, A., García-Closas, R., Lloreta, J., Castaño-Vinyals, G., Yeager, M., Welch, R., Chanock, S., Chatterjee, N., Wacholder, S., Samanic, C., Torà, M., Rothman, N. (2005). NAT2 slow acetylation, GSTM1 null genotype, and risk of bladder cancer: Results from the Spanish Bladder Cancer Study and meta-analyses. Lancet (London, England), 366(9486), 649–659. https://doi.org/10.1016/S0027-5107(02)0015-7 • Magalon, H., Patin, E., Austerlitz, F., Hegay, T., Aldashev, A., Quintana-Murci, L., & Heyer, E. (2008). Population genetic curversity of the NAT2 gene supports a role of acetylation in human daptation to farming in Central Asia. European Journal of Human Genetics: EJHG, 16(2), 243–251. https://doi.org/10.1038/sj.ejiqs.5201963 • Martinez-Gonzalez, L. J., Antúnez-Rodríguez, A., Vazquez-Alonso, F., Hernandez, A. F., & Alvarez-Cubero, M. J. (2020). Genetic science of the Total Environment, 730, 138314 • Moore, L. E., Barris, D. J. D., García-Closas, M., Karagas, M. R., Schwenn, M. F., Libin, J. H., Hein, D. W., Dansyall, C. L., Color, Johnson, A. T., Libin, J. H., Hein, D. W., Dansyall, C. L., Color, Johnson, A. T., Libin, J. H., Hein, D. W., Dansyall - Hin, D. W., Dansyall - Hi