

OXIDATIVE STRESS PROFILE DEMO

FINAL REPORT

Accession ID: 2403266072

Name: OXIDATIVE STRESS
PROFILE DEMO
Date of Birth: 01-01-1111
Gender: Male
Age: 01
Height:
Weight:
Fasting: UNKNOWN

Telephone: 000-000-0000
Street Address:
Email:

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
Phlebotomist: 0

Telephone: 000-000-0000
Address: 3521 Leonard Ct, Santa
Clara, CA 95054

Report Information

Current Result Previous Result In Control Moderate Risk

Specimen Information

| Sample Type | Collection Time | Received Time | Report | Final Report Date |
|-------------|-----------------|---------------|--------|-------------------|
|-------------|-----------------|---------------|--------|-------------------|

SAMPLE



3521 Leonard Ct, Santa Clara, CA 95054
1-866-364-0963 | support@vibrant-america.com | www.vibrant-america.com

TNP Test not performed

R&L Refer to risks and limitations at the end of report

Notes Refer to Lab notes at the end of the table

INTRODUCTION

Vibrant Wellness is pleased to present to you, 'Oxidative Stress Profile', to help you make healthy lifestyle, dietary and treatment choices in consultation with your healthcare provider. It is intended to be used as a tool to encourage a general state of health and well-being. The Vibrant 'Oxidative Stress Profile' is a test to identify and quantify the level of a large set of oxidative damage markers and to identify antioxidant genetics variations. The panel is designed to evaluate oxidative stress by measuring the levels of damage caused by oxidative species resulting from the impact of ROS and RNS on lipids, DNA, RNA and proteins and to give a complete picture of genetic predispositions that code for enzymes and antioxidants which can significantly impact oxidative stress response.

Methodology:

The Vibrant Oxidative Damage Markers panel uses tandem mass spectrometry methodology (LC-MS/MS) for quantitative detection of damage markers in urine samples. Urine creatinine is measured using a kinetic colorimetric assay based on the Jaffé method. All damage markers are reported as the quantitative result normalized to urine creatinine to account for urine dilution variations.

The Vibrant Antioxidant Genetics panel uses real-time PCR methodology. DNA is extracted and purified from saliva samples and a SNP (single nucleotide polymorphism) genotyping assay is performed using real-time PCR to detect the specific allele targets of each assay performed.

Interpretation of Report:

The report begins with the summary page which displays a summary flowchart of all antioxidant genetic variations of the human body's defense against oxidative stress and indicates the areas of concern from the genetic results observed. The set of analytes with risk associated variants are also summarized. The summary also includes the damage markers whose levels are high or moderate based on the reference range. This is followed by a graphical representation of the overall oxidative damage score which is calculated using the results from all urine damage markers tested applied to a linear regression model and displayed with respect to your age group. The score in green represents a normal score based on 50th percentile population, the score in yellow represents a moderate score based on 90th percentile and the score in red represent a high score based on the relatively healthy population. Reference ranges were determined using urine samples from 1000 apparently healthy individuals. Additionally, the previous value is also indicated to help check for improvements every time the test is ordered.

Following this section is the complete list of the genetic markers measured in the panel. Elevated risk associated variants are indicated with red, partially elevated risk associated variants are indicated with yellow and alleles with no risk are indicated with green. This is followed by a list of all damage marker results and their absolute levels are normalized with respect to Creatinine in a histogram format to enable a full overview along with the reference ranges. The level of the analyte with reference range is shown with three shades of color – Green, Yellow and Red. The result in green corresponds to 0th to 75th percentile indicates mild detection of the analyte. The result in yellow corresponds to 75th to 95th percentile indicates moderate detection of the analyte whereas the result in red corresponding to greater than 95th percentile indicates high detection of the analyte.

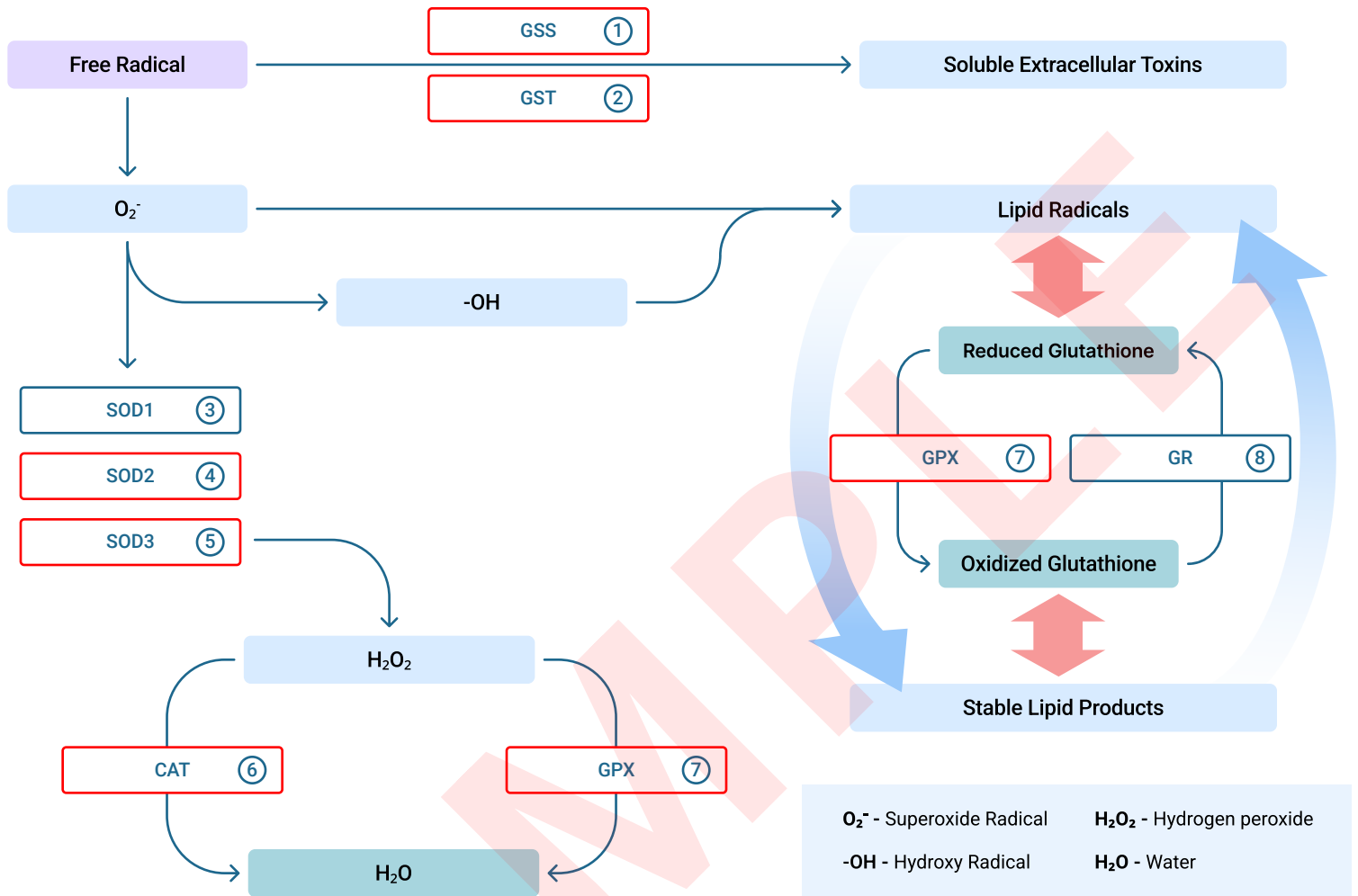
All contents provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information should be made in consultation with the healthcare provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Oxidative Stress panel is performed by Vibrant America, a CLIA certified lab CLIA#:05D2078809 and Vibrant Genomics, a CLIA certified lab CLIA#: 05D2098445. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to accept these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your healthcare provider for medication, treatment, or lifestyle management. This product is not intended to diagnose, treat, or cure any disease.

Please note:

Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes.

Oxidative Stress Profile Summary



Please note that the flowchart only includes only a subset of the genes tested. The complete set of the genes is listed in the tables below under "Antioxidant Genetics".

- ① **GSS - Glutathione Synthetase (1/1)**
Involved in Glutathione synthesis
Lower glutathione levels
- ② **GST - Glutathione S Transferase (2/3)**
Aids Glutathione in toxin removal
Decreasing antioxidant activity leads to elevated oxidative stress,
Decreased antioxidant activity
- ③ **SOD1 - Superoxide Dismutase (0/1)**
Aids in quenching superoxide free radical
- ④ **SOD2 - Superoxide Dismutase (1/1)**
Aids in quenching superoxide free radical
Impaired anti-oxidant activity

- ⑤ **SOD3 - Superoxide Dismutase (1/2)**
Aids in quenching superoxide free radical
Disrupted EC-SOD activity
- ⑥ **CAT - Catalase (2/3)**
Aids in quenching hydrogen peroxide
Mitochondrial dysfunction
- ⑦ **GPX - Glutathione Peroxidase (1/5)**
Aids in reduction of hydrogen peroxide by glutathione
Elevated ROS production
- ⑧ **GR - Glutathione Reductase (0/1)**
Aids in recycling glutathione

Antioxidant Genetics

⊕⊕ Homozygous Mutant

⊕⊖ Heterozygous

⊖⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|--|-----------|---------------------------|---------------|-----------|-----------|
| rs8192287 | SOD3 | Disrupted EC-SOD activity | ⊕⊕ T/T | Elevated | G/G |
| <p>The SOD3 gene, also known as the superoxide dismutase 3 gene, is responsible for producing the extracellular superoxide dismutase (EC-SOD) enzyme. EC-SOD is an antioxidant enzyme that plays a critical role in protecting tissues and cells from the harmful effects of reactive oxygen species (ROS). EC-SOD is primarily found in the extracellular space, where it acts as a defense mechanism against oxidative stress by converting superoxide radicals into hydrogen peroxide and oxygen, which are less damaging to cells. Mutations in the SOD3 gene can disrupt the normal function of EC-SOD and impair its ability to protect against oxidative stress. Homozygous mutant (abnormal) individuals have disrupted EC-SOD function that impairs their ability to protect against oxidative stress. Homozygous mutant carriers are advised to follow a mediterranean diet and consume vegetables that has antioxidant properties.</p> | | | | | |
| rs4756146 | CAT | Mitochondrial dysfunction | ⊕⊕ T/T | Elevated | C/C |
| <p>The CAT gene encodes for the catalase enzyme, localized in mitochondria. Mitochondrial catalase was shown to protect cells from oxidative injury induced by hydrogen peroxide by degrading hydrogen peroxide generated by peroxisomal oxidases to water and oxygen, thereby protecting cells from the toxic effects of hydrogen peroxide. Thus, the enzyme participates in antioxidant functions in the body. Mutations in the gene lead to decreased catalase production resulting in excess ROS production. This induces mitochondrial dysfunction and elevated oxidative stress. Homozygous wild (abnormal) individuals have optimum catalase production and have reduced risk of oxidative stress. Susceptible individuals are recommended to consume carrots, spinach, kale, pappaya, banana, apple, orange, almonds, sunflower seeds, eggs and brazil nuts. Daily exercise is recommended.</p> | | | | | |
| rs7943316 | CAT | Mitochondrial dysfunction | ⊕⊕ T/T | Elevated | A/T, A/A |
| <p>The CAT gene encodes for the catalase enzyme, localized in mitochondria. Mitochondrial catalase was shown to protect cells from oxidative injury induced by hydrogen peroxide by degrading hydrogen peroxide generated by peroxisomal oxidases to water and oxygen, thereby protecting cells from the toxic effects of hydrogen peroxide. Thus, the enzyme participates in antioxidant functions in the body. Mutations in the gene lead to decreased catalase production resulting in excess ROS production. This induces mitochondrial dysfunction and elevated oxidative stress. Homozygous mutant (abnormal) individuals have reduced catalase production leading to ROS buildup and increased oxidative stress. Susceptible individuals are recommended to consume carrots, spinach, kale, pappaya, banana, apple, orange, almonds, sunflower seeds, eggs and brazil nuts. Daily exercise is recommended.</p> | | | | | |
| rs713041 | GPX4 | Elevated ROS production | ⊖⊖ C/C | Elevated | C/T, T/T |
| <p>The GPX4 gene encodes for glutathione peroxidase 4 which is an antioxidant selenoprotein. GPx4 is the only enzyme that reduces phospholipid hydroperoxides (reactive oxygen species which can give rise to oxidative stress). It protects cells against membrane lipid peroxidation (oxidative degradation of lipids). GPX4 modulates redox-dependent mitochondrial function where mitochondria generate reactive oxygen species (ROS) and respond to ROS-mediated changes in the cellular redox state. Mutations in the gene cause aberrant redox signaling and increase ROS leading to oxidative stress. Mutations in the gene lead to higher selenoprotein enzyme levels and reduced oxidative damage. Homozygous wild (abnormal) individuals experience oxidative stress due to reduced prostaglandin levels. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |

Antioxidant Genetics

⊕⊕ Homozygous Mutant

⊕⊖ Heterozygous

⊖⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|--|-----------|-------------------------------------|---------------|-----------|-----------|
| rs121909307 | GSS | Lower glutathione levels | ⊕⊕ C/C | Elevated | T/T |
| <p>GSS gene encodes for the glutathione synthetase enzyme, which plays a crucial role in the synthesis of glutathione. Glutathione is a tripeptide composed of three amino acids: glutamate, cysteine, and glycine. It serves as a powerful antioxidant within cells, helping to protect them from oxidative stress. The function of glutathione synthetase is to catalyze the final step in the biosynthesis of glutathione. This enzyme combines the three precursor amino acids mentioned above to form glutathione. The synthesized glutathione is then involved in various cellular processes, including detoxification of harmful substances, neutralization of reactive oxygen species (ROS), and maintenance of the cellular redox balance. Mutations in the GSS gene can lead to a decrease in the activity of the glutathione synthetase enzyme. This reduced enzymatic activity results in a diminished ability to produce glutathione, which, in turn, contributes to increased oxidative stress within cells. Homozygous mutant (abnormal) individuals with reduced glutathione synthetase activity experience decreased glutathione production, leading to heightened oxidative stress in cells. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |
| rs2071746 | HMOX1 | Decreased heme oxygenase 1 activity | ⊕⊕ T/T | Elevated | A/A |
| <p>HMOX1 gene encodes for the enzyme heme oxygenase 1. In the mitochondria, HMOX1 is anchored to the inner mitochondrial membrane, where it may detoxify mitochondrial heme. HMOX1 is capable of reducing oxidative stress because of the consumption of molecular oxygen in the heme oxygenase reaction pathway where it catalyzes the degradation of heme b to carbon monoxide, ferrous iron, and biliverdin. Polymorphisms in the HMOX1 gene can reduce enzyme activity which impairs the detoxification of mitochondrial heme and antioxidant activity and increases oxidative stress. Homozygous Wild (abnormal) individuals have impaired mitochondrial heme detoxification which might result in high oxidative stress. Susceptible individuals are recommended to consume cantaloupe, spinach, potato, mushroom, kidney beans, chickpeas, green peas, beef, tuna, turkey and cheese. Daily exercise is recommended.</p> | | | | | |
| rs2796498 | PRKAA2 | Impaired antioxidant activity | ⊕⊕ G/G | Elevated | A/A |
| <p>The PRKAA2 gene encodes for an enzyme AMP-activated protein kinase (AMPK). AMPK is an important energy-sensing enzyme that monitors cellular energy status. AMPK plays a role in cellular energy homeostasis, largely to activate glucose and fatty acid uptake and oxidation when cellular energy is low. AMPK is part of the antioxidant defense system and is needed to protect the cells from oxidative stress. AMPK promotes mitochondrial biogenesis (a process that occurs in response to increased energy expenditure to produce more ATP). Mutation reduces the expression of the PRKAA2 gene causing impaired AMPK synthesis and impaired antioxidant activity leading to oxidative stress. Homozygous Mutant (Abnormal) individuals impaired AMPK synthesis and impaired antioxidant activity leading to oxidative stress. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |
| rs1048943 | CYP1A1 | Elevated ROS production | ⊕⊕ A/A | Elevated | G/G |
| <p>The CYP1A1 gene encodes a member of the cytochrome P450 superfamily of enzymes. Cytochrome c (Cyt c) is located in the mitochondrial intermembrane spaces, where it functions as an electron shuttle in the respiratory chain. They participate in electron transport, inhibit reactive oxygen species (ROS) formation, and prevent oxidative stress. CYP1A1 metabolism serves a major role in detoxifying foreign chemicals and metabolic activation, which leads to oxidative damage. The mutation in the gene decreases the enzyme activity which impairs the detoxification of toxic compounds. This leads to a decrease in antioxidant activity and increased ROS production, thus contributing to oxidative stress. Homozygous mutant (abnormal) individuals who have decreased gene activity have decreased antioxidant activity and thus increased oxidative stress. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |

Antioxidant Genetics

⊕⊕ Homozygous Mutant

⊕⊖ Heterozygous

⊖⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|--|-----------|--|---------------|--------------------|-----------|
| rs4880 | SOD2 | Impaired anti-oxidant activity | ⊕⊖ C/T | Partially elevated | C/C |
| <p>The SOD2 encodes a mitochondrial protein, superoxide dismutase 2 that protects cells against mitochondrial superoxide. This protein binds to the superoxide byproducts of oxidative phosphorylation and converts them to hydrogen peroxide and diatomic oxygen. This function allows SOD2 to clear mitochondrial reactive oxygen species (ROS) and, as a result, confer protection against oxidative stress. It was shown, that the SOD2 T allele putatively reduces gene expression, mRNA stability, and enzymatic activity of the SOD, as well as impairs the import of this enzyme into the mitochondrion reducing antioxidant activity and leading to oxidative stress. Heterozygous (partially abnormal) individuals who have decreased gene expression have decreased antioxidant activity and increased oxidative stress. Homozygous mutant carriers are advised to follow a mediterranean diet and consume vegetables that has antioxidant properties.</p> | | | | | |
| rs10911021 | GLUL | Decreased levels of glutamine synthetase and glutathione | ⊕⊖ C/T | Partially elevated | C/C |
| <p>The GLUL gene encodes for glutamate ammonia ligase (glutamine synthetase) enzyme. Glutamine synthetase plays a role in maintaining cellular levels of glutamine, an amino acid with multiple functions, including antioxidant properties. Glutamine serves as a precursor for the synthesis of glutathione, a key antioxidant molecule. Glutathione protects the cell from oxidative stress, its availability in reduced form is mandatory to control the redox status of the cell. The mutation leads to the downregulation of the gene leading to enzyme inefficiency that may cause a deficiency of glutamine required for the synthesis of glutathione. Thus increasing the risk of oxidative stress.</p> | | | | | |
| rs3754446 | GSTM5 | Decreased antioxidant activity | ⊕⊖ G/T | Partially elevated | T/T |
| <p>The glutathione S transferase Muv 5 (GSTM5) gene belongs to the GST gene family. In addition to the cytoplasm, it is found in the mitochondria, lysosomes, and nuclear regions. Through the inhibition of cardiolipin (a lipid found in mitochondria) peroxidation and cytochrome c release, mitochondrial GSTP contributes to the protection of organelles from oxidative stress. The enzyme plays an important regulatory role in detoxification by catalyzing the modification of toxic compounds to glutathione, which is an antioxidant that helps combat Reactive oxygen species (ROS). The mutation leads to lead to significant damage in cells and mitochondrial function, which causes a build-up of ROS therefore, increasing the risk for oxidative stress. Heterozygous (partially abnormal) individuals who have impaired mitochondrial function with ROS build-up have increased oxidative stress. Individuals are advised to include at least 1 serving of allium vegetables, garlic, leeks, citrus foods, 2 to 5 cups of cruciferous vegetables as well as 5 to 10 g of anthocyanin-rich foods, such as blueberries, cherries and yams, per day.</p> | | | | | |
| rs4485648 | TrxR2 | Impaired mitochondrial redox balance | ⊕⊖ C/T | Partially elevated | T/T |
| <p>The TrxR2 gene encodes for the enzyme thioredoxin reductase 2. Thioredoxin reductases are a family of enzymes that maintain cellular redox balance and regulate various cellular processes. TrxR2 is primarily located in the mitochondria and plays a crucial role in maintaining the redox state of proteins and other molecules within the mitochondria. TrxR2 is responsible for reducing oxidized thioredoxin, an antioxidant protein, which allows thioredoxin to carry out its antioxidant and regulatory functions. Mutations or alterations in the TrxR2 gene can disrupt the normal functioning of the enzyme and impair mitochondrial redox balance, resulting in increased oxidative stress. Heterozygous (partially abnormal) individuals have reduced enzyme activity and impaired mitochondrial oxygen radical scavenging activity has higher oxidative stress. Susceptible individuals are recommended to consume vegetables and fruits as well as reduce the consumption of fat and meat. Daily exercise is recommended.</p> | | | | | |

Antioxidant Genetics

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|---|-----------|--|---------------|--------------------|-----------|
| rs4673 | CYBA | Elevated ROS production | ⊕⊖ C/T | Partially elevated | T/T |
| <p>The CYBA gene encodes the p22phox subunit of NADPH oxidase, an enzyme that plays an essential role in the immune system. Upon the detection of foreign invaders, phagocytes are stimulated, and NADPH oxidase is assembled. This enzyme catalyzes the conversion of oxygen to superoxide, a toxic molecule that is used to generate several other highly reactive and toxic substances collectively known as reactive oxygen species (ROS). Phagocytes use these ROS to kill foreign invaders, preventing them from reproducing in the body and causing illness. Mutations in the CYBA gene are associated with higher p22phox expression and increased levels of ROS. The accumulation of ROS can thus result in oxidative stress. Heterozygous (partially abnormal) individuals have higher p22phox expression and higher ROS levels, increasing their susceptibility to oxidative stress. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |
| rs206812 | XDH | Elevated ROS production | ⊕⊖ A/G | Partially elevated | G/G |
| <p>The XDH gene encodes the enzyme xanthine dehydrogenase, which is primarily involved in the metabolism of purine compounds. Xanthine dehydrogenase is responsible for the conversion of hypoxanthine to xanthine and xanthine to uric acid, which is antioxidant in nature. However, under certain conditions, xanthine dehydrogenase can undergo conversion to its other form called xanthine oxidase (XO). XO, the oxidized form of xanthine dehydrogenase, has the ability to produce superoxide radicals as a byproduct of its enzymatic activity. Superoxide radicals are reactive oxygen species (ROS) that can be generated during normal cellular processes. It is crucial for cells to efficiently break down these ROS to prevent cellular damage and oxidative stress. Mutations in the XDH gene can disrupt the normal regulation of xanthine dehydrogenase and promote the conversion to XO more readily. This increases the XO activity for higher production of ROS, including superoxide radicals. The accumulation of ROS can thus result in oxidative stress. Heterozygous (partially abnormal) individuals with heightened XO activity experience an elevation in ROS levels, increasing their susceptibility to oxidative stress. Susceptible individuals are recommended to consume a Mediterranean diet and the necessary supplements. Daily exercise is recommended.</p> | | | | | |
| rs1695 | GSTP1 | Decreasing antioxidant activity leads to elevated oxidative stress | ⊕⊖ A/G | Partially elevated | A/A |
| <p>Glutathione S-transferase P is an enzyme that in humans is encoded by the GSTP1 gene. In addition to the cytoplasm, it is found in the mitochondria, lysosomes, and nuclear regions. Through the inhibition of cardiolipin (a lipid found in mitochondria) peroxidation and cytochrome c release, mitochondrial GSTP contributes to the protection of organelles from oxidative stress. The enzyme plays an important regulatory role in detoxification by catalyzing the modification of toxic compounds to glutathione, which is an antioxidant that helps combat Reactive oxygen species (ROS). The mutation decreases enzyme activity and directly elicits mitochondrial dysfunction, resulting in the rapid generation of ROS, and thus leading to oxidative stress. Heterozygous (partially abnormal) individuals who have decreased gene activity have decreased antioxidant activity and thus increased oxidative stress. GG carriers are advised to adapt to a Mediterranean-style diet with a variety of antioxidant-rich foods (citrus foods and allium vegetables, garlic), heart healthy fats, and complex carbohydrates. Daily exercise is recommended.</p> | | | | | |
| rs3877899 | SELENOP | Impaired plasma selenium production leads to selenium deficiency | ⊕⊖ C/T | Partially elevated | C/C |
| <p>SELENOP encodes a protein Selenoprotein P that affects blood selenium or selenoprotein levels in response to supplementation. This selenoprotein accounts for most of the selenium in plasma. It has been implicated as an extracellular antioxidant, and in the transport of selenium to extra-hepatic tissues via apolipoprotein E receptor-2 (apoER2). Mutation in the gene reduces the gene activity and impairs plasma selenium production which leads to increased selenium deficiency, which has the potential of weakening an individual's capacity to respond to oxidative damage involved in the aging process and in most chronic diseases including cancer, cardiovascular disease, diabetes, and dementia. Heterozygous (partially abnormal) individuals who have gene deficiency have a slightly increased risk of selenium deficiency. Individuals with selenium deficiency are advised to consume brazil nuts, pork, beef, turkey, chicken, fish, shellfish, and eggs. A diet such as breads, grains, meat, poultry, fish, and eggs can increase selenium levels.</p> | | | | | |

Antioxidant Genetics

⊕⊕ Homozygous Mutant

⊕⊖ Heterozygous

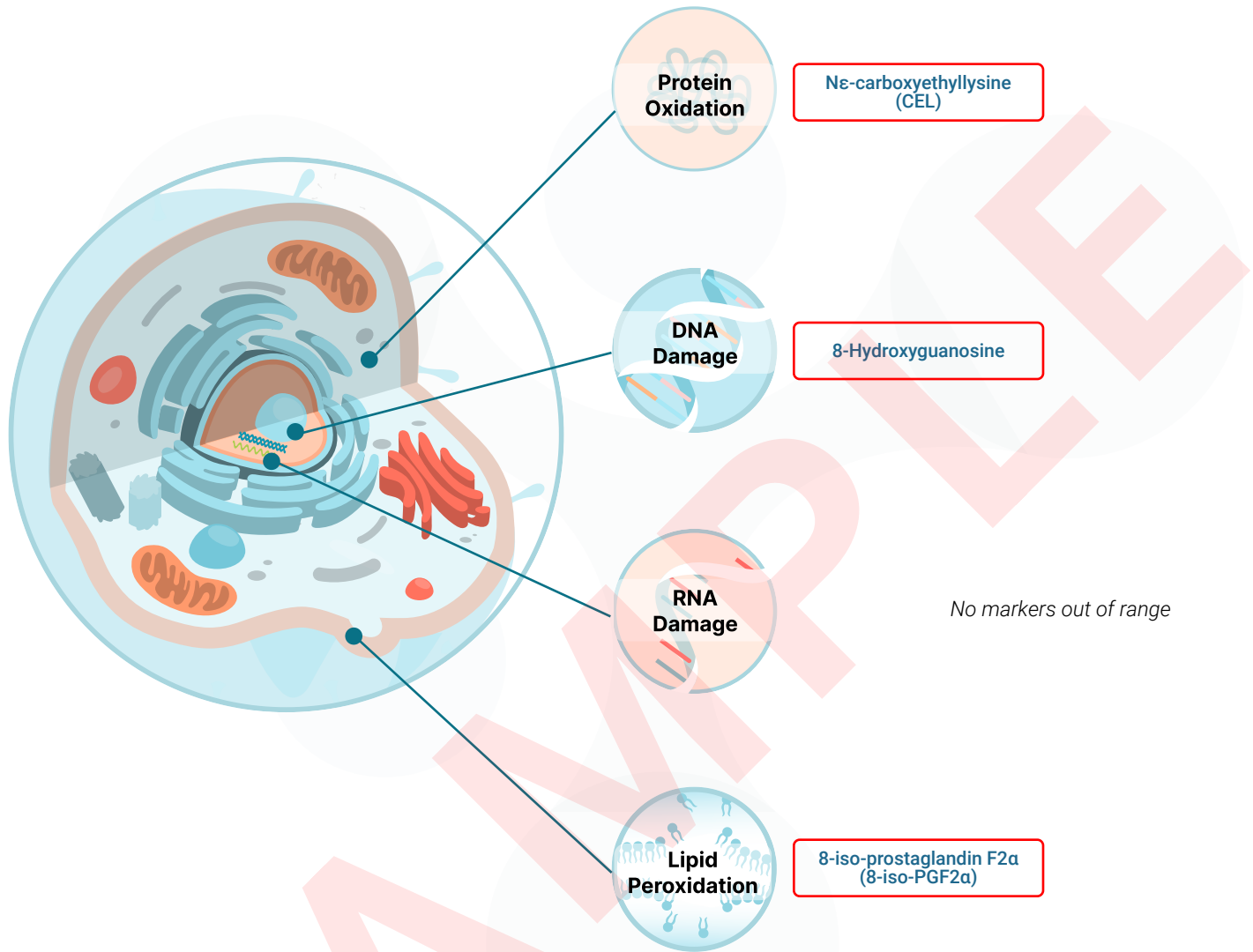
⊖⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|-----------|-----------|-------------------------|---------------|--------------------|-----------|
| rs916321 | CYB5R3 | Elevated ROS production | ⊕⊖ A/G | Partially elevated | G/G |

The CYB5R3 gene encodes for an enzyme called cytochrome b5 reductase 3, which is involved in the electron transport chain (ETC) in mitochondria. This enzyme plays an important role in the regeneration of the antioxidant coenzyme Q10 (CoQ10), which is an essential component of the ETC and a potent antioxidant. Cytochrome b5 reductase 3 exists in both membrane-bound and soluble forms. The soluble form in erythrocytes is responsible for reducing methemoglobin (MetHb) back into functional hemoglobin, allowing for the efficient transport of oxygen throughout the body. Accumulation of MetHb can lead to ROS production, causing oxidative damage. Enzymes like cytochrome b5 reductase 3 and methemoglobin reductase help reduce MetHb and prevent ROS production. Mutations in the CYB5R3 gene can result in reduced enzymatic activity of cytochrome b5 reductase 3, leading to decreased CoQ10 regeneration and impaired reduction of MetHb back into functional hemoglobin. This results in an elevated production of ROS, which consequently heightens susceptibility to oxidative stress.



Oxidative Stress Profile Summary



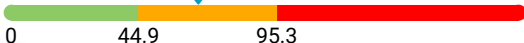
Oxidative Stress Biomarkers

| Lipid Peroxidation | Current | Previous | Result | Reference |
|--|---------|----------|-----------|-----------|
| | | | 75th 95th | |
| 8-iso-prostaglandin F2α (8-iso-PGF2α) (ug/g) | 0.12 | | 0.1 0.26 | ≤0.26 |

8-iso-prostaglandin F2α (8-iso-PGF2α) is an isoprostane generated through the non-enzymatic peroxidation of arachidonic acid in membrane phospholipids. It is found in human plasma and excreted in urine. This biomarker serves as an indicator of oxidative stress and can reliably reflect lipid peroxidation in chronic diseases. Elevated levels of 8-iso PGF2α can lead to DNA oxidation and subsequent structural DNA damage. 8-iso PGF2α is thereby, valuable in assessing oxidative damage to DNA and understanding its implications for cellular health and disease development. Studies have shown that increased levels of 8-iso PGF2α contribute to heightened oxidative stress associated with aging, hypertension, diabetes mellitus, hypercholesterolemia, smoking, and coronary artery disease.

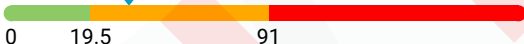
Oxidative Stress Biomarkers

| DNA Damage | Current | Previous | Result | Reference |
|------------|---------|----------|--------|-----------|
|------------|---------|----------|--------|-----------|

| | | | | |
|---------------------------|-------|--|--|-------|
| 8-Hydroxyguanosine (ug/g) | 65.23 | |  | ≤95.3 |
|---------------------------|-------|--|--|-------|

Free radicals produced either endogenously or exogenously can attack nucleic acid in living cells. Reactions of reactive oxygen species (ROS) and reactive nitrative species (RNS) with RNA yield 8-hydroxyguanosine (8-HdG). Among the known oxidative lesions in nucleic acids, 8-HdG is abundant and appears to be most deleterious due to its high mutagenic potential. This implies that 8-HdG is capable of inducing genetic mutation. RNA dysfunction caused by oxidative damage may contribute to the development of various degenerative diseases. Urinary levels of 8-HdG have risen as indicators of oxidative damage of RNA by ROS.

| Advanced Glycation Products | Current | Previous | Result | Reference |
|-----------------------------|---------|----------|--------|-----------|
|-----------------------------|---------|----------|--------|-----------|

| | | | | |
|------------------------------------|-------|--|--|-----|
| Nε-carboxyethyllysine (CEL) (ug/g) | 28.61 | |  | ≤91 |
|------------------------------------|-------|--|--|-----|

Glycation is a spontaneous non-enzymatic reaction wherein free reducing sugars bind to free amino groups of proteins, DNA, and lipids. This results in the formation of advanced glycation end-products (AGE). Glycation and oxidative stress are closely linked, and they are together referred to as "glycoxidation". All steps of glycoxidation generate free radicals, some of them being common with the lipid peroxidation pathway. Owing to this, AGE has been considered a urinary biomarker of oxidative stress. The AGE product, Nε-carboxyethyllysine (CEL) is formed when methylglyoxal (formed from the oxidation of lipids and sugars) reacts with lysine. CEL interacts with AGE receptors (RAGEs) which may give rise to oxidative stress. This may even induce cellular dysfunction. Urinary levels of CEL can be used to monitor the degree of oxidative stress in the body system.

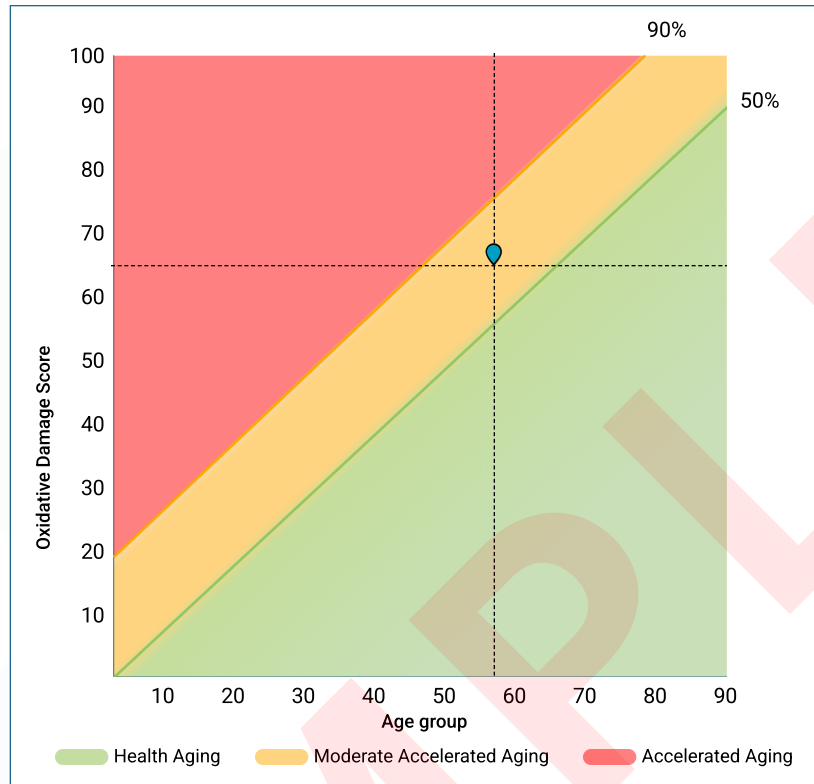
Creatinine

| Test Name | Current | Previous | Result | Reference |
|-----------|---------|----------|--------|-----------|
|-----------|---------|----------|--------|-----------|

| | | | | |
|--------------------------|------|--|--|-----------|
| Urine Creatinine (mg/ml) | 1.38 | |  | 0.25-2.16 |
|--------------------------|------|--|--|-----------|

Oxidative Damage Score

📍 Current Result 📍 Previous Result



Result

Your Given Age: 56

Your Oxidative Stress Profile looks similar to a **66.02** year old.

Supplementation Suggestions

| Nutrients | Dosage | Purpose |
|-----------|------------|--|
| Selenium | 55 mcg/day | Selenium supplements increase GPX1 activity by incorporating selenium atoms into the GPX1 enzyme's active site, enhancing its ability to catalyze the reduction of harmful reactive oxygen species. Selenium, when incorporated into selenoproteins, enhances the activity of catalase by serving as a cofactor, facilitating the breakdown of hydrogen peroxide into water and oxygen, thus increasing catalase's antioxidant function. Selenium supplements enhance the synthesis of selenoproteins, including selenium-dependent superoxide dismutase (SOD), which, in turn, increases SOD activity and levels, boosting cellular antioxidant defenses against superoxide radicals. Selenium supplements increase GPX4 activity by incorporating selenium atoms into the GPX4 enzyme's active site, enhancing its ability to catalyze the reduction of harmful reactive oxygen species. Selenium reduces 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as a cofactor for the enzyme glutathione peroxidase, which helps neutralize reactive oxygen species responsible for the formation of 8-iso-PGF2α. |

Supplementation Suggestions

| Nutrients | Dosage | Purpose |
|--------------|----------------|---|
| Vitamin C | 90 mg/day | Vitamin C enhances GPX1 activity by donating electrons to reduce glutathione (GSH), which is then used by GPX1 to neutralize harmful reactive oxygen species (ROS), thereby protecting cells from oxidative damage. Vitamin C supplements enhance AMPK activity by promoting the phosphorylation of AMPK through activation of the upstream kinase, LKB1, leading to increased cellular energy sensing and metabolic regulation. Vitamin C supplements enhance catalase activity by donating electrons to the enzyme's active site, increasing its ability to break down hydrogen peroxide into water and oxygen, thus bolstering the antioxidant defense system. Vitamin C supplements enhance the activity of superoxide dismutase (SOD) enzymes by providing electrons necessary for SOD's antioxidant function, thereby increasing SOD activity and reducing oxidative stress markers. Vitamin C supplementation enhances CoQ10 regeneration by acting as a reducing agent, donating electrons to CoQ10 radicals, stabilizing it, and allowing it to continue its role in cellular energy production. This helps maintain optimal cellular energy levels and overall health. Vitamin C supplementation decreases 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as a powerful antioxidant, scavenging free radicals and inhibiting lipid peroxidation, thereby reducing oxidative stress. |
| Vitamin D3 | 4000 IU/day | Vitamin D3 supplementation upregulates catalase expression by activating the vitamin D receptor (VDR) in cells, leading to increased transcription of catalase genes, thus enhancing antioxidant defenses against oxidative stress. Vitamin D3 supplementation enhances the expression of superoxide dismutase (SOD) genes by binding to vitamin D receptors (VDRs) in cells, which leads to increased transcription of SOD genes and subsequently elevates SOD antioxidant markers to counteract oxidative stress. |
| Vitamin E | 22 IU/day | Vitamin E supplements enhance cellular antioxidant defenses by reducing lipid peroxidation, indirectly leading to increased catalase enzyme activity, which helps neutralize harmful reactive oxygen species (ROS). Vitamin E supplements enhance the activity of superoxide dismutase (SOD) enzymes by reducing lipid peroxidation and protecting cellular membranes, which indirectly increases SOD antioxidant markers, helping to neutralize harmful superoxide radicals. Vitamin E supplements reduce 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as an antioxidant, neutralizing reactive oxygen species that would otherwise promote the formation of 8-iso-PGF2α. |
| Coenzyme Q10 | 100-200 mg/day | Coenzyme Q10 (CoQ10) supplements enhance mitochondrial function, increasing cellular energy production and aiding catalase enzyme activity, which in turn boosts the breakdown of hydrogen peroxide, reducing oxidative stress and elevating catalase antioxidant markers. Coenzyme Q10 (CoQ10) supplementation enhances mitochondrial function, promoting efficient electron transport in the respiratory chain, which, in turn, reduces oxidative stress, increases cellular ATP production, and stimulates the expression and activity of superoxide dismutase (SOD) enzymes, leading to higher SOD antioxidant marker levels. |
| Manganese | 2.3 mg/day | Manganese supplements facilitate catalase enzyme activation by acting as a cofactor, promoting the breakdown of hydrogen peroxide into water and oxygen, thereby increasing catalase activity and antioxidant defense in cells. Manganese supplements support the activity of superoxide dismutase (SOD) enzymes by acting as a cofactor, enhancing their ability to convert harmful superoxide radicals into less damaging molecules, thus increasing SOD antioxidant markers. |

Oxidative Stress Profile

| Antioxidant Genetics | | | | | |
|--|-----------|--|---------------|--------------------|-----------|
| ⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild | | | | | |
| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
| rs2234694 | SOD1 | Increased superoxide levels | ⊖⊖ A/A | Normal | A/A |
| rs4880 | SOD2 | Impaired anti-oxidant activity | ⊕⊖ C/T | Partially elevated | C/C |
| rs1799895 | SOD3 | Elevated ROS production | ⊖⊖ C/C | Normal | C/C |
| rs8192287 | SOD3 | Disrupted EC-SOD activity | ⊕⊕ T/T | Elevated | G/G |
| rs1001179 | CAT | Mitochondrial dysfunction | ⊖⊖ C/C | Normal | C/C |
| rs4756146 | CAT | Mitochondrial dysfunction | ⊕⊕ T/T | Elevated | C/C |
| rs7943316 | CAT | Mitochondrial dysfunction | ⊕⊕ T/T | Elevated | A/T, A/A |
| rs10911021 | GLUL | Decreased levels of glutamine synthetase and glutathione | ⊕⊖ C/T | Partially elevated | C/C |
| rs1050450 | GPX1 | Aberrant redox signaling | ⊖⊖ C/C | Normal | C/C |
| rs1987628 | GPX1 | Reduced antioxidant enzyme leads to selenium deficiency | ⊕⊕ C/C | Normal | C/C |
| rs2071566 | GPX2 | Higher selenoprotein concentrations | ⊖⊖ G/G | Normal | G/G |
| rs4902346 | GPX2 | Higher selenoprotein concentrations | ⊖⊖ T/T | Normal | T/T |
| rs713041 | GPX4 | Elevated ROS production | ⊖⊖ C/C | Elevated | C/T, T/T |
| rs121909307 | GSS | Lower glutathione levels | ⊕⊕ C/C | Elevated | T/T |
| rs2071746 | HMOX1 | Decreased heme oxygenase 1 activity | ⊕⊕ T/T | Elevated | A/A |
| rs366631 | GSTM1 | Decreased antioxidant activity | ⊖⊖ T/T | Normal | T/T |
| rs3754446 | GSTM5 | Decreased antioxidant activity | ⊕⊖ G/T | Partially elevated | T/T |
| rs4485648 | TrxR2 | Impaired mitochondrial redox balance | ⊕⊖ C/T | Partially elevated | T/T |
| rs4673 | CYBA | Elevated ROS production | ⊕⊖ C/T | Partially elevated | T/T |
| rs9932581 | CYBA | Elevated ROS production | ⊖⊖ G/G | Normal | G/G |
| rs10789038 | PRKAA2 | Impaired antioxidant activity | ⊖⊖ A/A | Normal | A/A |
| rs2796498 | PRKAA2 | Impaired antioxidant activity | ⊕⊕ G/G | Elevated | A/A |
| rs206812 | XDH | Elevated ROS production | ⊕⊖ A/G | Partially elevated | G/G |

Oxidative Stress Profile

| Antioxidant Genetics | | | | | |
|---|-----------|--|---------------|--------------------|-----------|
| ⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild | | | | | |
| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
| rs2073316 | XDH | Elevated ROS production | ⊕⊖ C/T | Normal | C/T, T/T |
| rs7310505 | TXNRD1 | Poor antioxidant activity | ⊕⊖ A/C | Normal | C/C, A/C |
| rs1048943 | CYP1A1 | Elevated ROS production | ⊕⊕ A/A | Elevated | G/G |
| rs1548357 | TXNRD2 | Impaired mitochondrial oxygen radical scavenging activity | ⊖⊖ T/T | Normal | T/T |
| rs1695 | GSTP1 | Decreasing antioxidant activity leads to elevated oxidative stress | ⊕⊖ A/G | Partially elevated | A/A |
| rs20417 | COX-2 | Elevated ROS production | ⊕⊖ C/G | Normal | C/C, C/G |
| rs3877899 | SELENOP | Impaired plasma selenium production leads to selenium deficiency | ⊕⊖ C/T | Partially elevated | C/C |
| rs8190955 | GSR | Increased oxidative stress in red blood cells | ⊕⊕ C/C | Normal | C/C |
| rs916321 | CYB5R3 | Elevated ROS production | ⊕⊖ A/G | Partially elevated | G/G |

| Oxidative Stress Biomarkers | | | | | |
|--|---------|----------|-----------|-----------|--|
| Lipid Peroxidation | Current | Previous | Result | Reference | |
| | | | 75th 95th | | |
| 11-β-Prostaglandin F2α (ug/g) | 0.11 | | | ≤0.4 | |
| 15(R)-Prostaglandin F2α (ug/g) | <0.05 | | | ≤0.22 | |
| 8-iso-prostaglandin F2α (8-iso-PGF2α) (ug/g) | 0.12 | | | ≤0.26 | |
| Glutathione 4-hydroxynonenal (GS-HNE) (ug/g) | 0.23 | | | ≤2.5 | |
| Malondialdehyde (ug/g) | 60.15 | | | ≤163.53 | |
| DNA Damage | Current | Previous | Result | Reference | |
| 8-Hydroxy-2-deoxyguanosine (ug/g) | 0.73 | | | ≤4 | |
| 8-Hydroxyguanine (ug/g) | 10.87 | | | ≤49.4 | |
| 8-Hydroxyguanosine (ug/g) | 65.23 | | | ≤95.3 | |
| RNA Damage | Current | Previous | Result | Reference | |
| 8-Nitroguanine (ug/g) | 22.01 | | | ≤107.47 | |
| 8-Nitroguanosine (ug/g) | 454.21 | | | ≤2608.9 | |

Oxidative Stress Profile

Oxidative Stress Biomarkers

| Protein Oxidation Products | Current | Previous | Result | | Reference | |
|---------------------------------------|---------|----------|--------|--------|-----------|-------|
| | | | 75th | 95th | | |
| 3-Bromotyrosine (ug/g) | 12.69 | | 167.53 | 349.6 | ≤349.6 | |
| 3-Chlorotyrosine (ug/g) | 3.10 | | 3.43 | 9.92 | ≤9.92 | |
| Dityrosine (ug/g) | 1.15 | | 1.31 | 5 | ≤5 | |
| Nitrotyrosine (ug/g) | 29.99 | | 91.32 | 285.69 | ≤285.69 | |
| Advanced Glycation Products | Current | Previous | Result | | Reference | |
| Nε-(carboxymethyl)lysine (CML) (ug/g) | 15.31 | | 0 | 15.8 | 70.3 | ≤70.3 |
| Nε-carboxyethyllysine (CEL) (ug/g) | 28.61 | | 0 | 19.5 | 91 | ≤91 |

SAMPLE

Risk and Limitations

This test has been developed and its performance characteristics determined and validated by Vibrant America and Vibrant Genomics LLC., CLIA certified laboratories. These assays have not been cleared or approved by the U.S. Food and Drug Administration. Vibrant Wellness provides additional contextual information on these tests and provides the report in a more descriptive fashion.

The Vibrant Oxidative Stress Profile does not demonstrate absolute positive and negative predictive values for any condition. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a healthcare provider's clinical assessment.

Vibrant has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific test due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

Genetic testing is helpful in analyzing risk to various diseases. However, it is important to note that Genetic risk determinants are neither necessary nor sufficient for the development of diseases. Environmental and lifestyle risk factors could also affect the risk of disease development. Results from genetic analysis should always be interpreted along with clinical findings on the individual. Genetic testing evaluates only for the particular genotypes indicated; it does not test for other genetic abnormalities found elsewhere in the genome. Different genetic variants can be tested by different genetic labs to evaluate the risk for a particular disease, depending on what is tested, genetic risk may not be comparable between labs. It should be realized that there are possible sources of error similar to any lab testing which include sample misidentification, trace contamination of PCR reactions, technical errors and rare genetic variants that may interfere with analysis.

Some individuals may feel anxious about getting their genetic test health results. If the potential user feels very anxious, such user should speak to his or her doctor or other health care professional prior to collection of a sample for testing. Users should consult with their doctor or other health care professional if they have any questions or concerns about the results of their test or their current state of health. Users of the test are also encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions. Tested individuals may find their experience is not consistent with Vibrant's selected peer reviewed scientific research findings of relative improvement for study groups. The science in this area is still developing and many personal health factors affect diet and health. Since subjects in the scientific studies referenced in this report may have had personal health and other factors different from those of tested individuals, results from these studies may not be representative of the results experienced by tested individuals. Further, some recommendations may or may not be attainable, depending on the tested individual's physical ability or other personal health factors. A limitation of this testing is that many of these scientific studies may have been performed in selected populations only. The interpretations and recommendations are done in the context of these studies, but the results may or may not be relevant to tested individuals of different or mixed ethnicities.

Vibrant Wellness makes no claims as to the diagnostic or therapeutic use of its tests or other informational materials. Vibrant Wellness reports and other information do not constitute medical advice and are not a substitute for professional medical advice. Please consult your healthcare practitioner for questions regarding test results, or before beginning any course of medication, supplementation, or dietary changes.