

DEMO DEMO

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Weight:
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FINAL REPORT

Accession ID: 2450591967

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
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Report Information

Current Result Previous Result In Control Moderate Risk

Specimen Information

| Sample Type | Collection Time | Received Time | Report | Final Report Date |
|-------------|------------------------|------------------------|-------------------------------|------------------------|
| Saliva | 2024-04-02 13:25 (UTC) | 2024-04-03 22:22 (UTC) | Oxidative Stress Profile - P2 | 2024-04-09 16:15 (UTC) |



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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 'Antioxidant Genetics', 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 Antioxidant Genetics Panel is a test to measure levels of various genetic mutations present in an individual's body. The panel is designed to give a complete picture of these predispositions that code for enzymes and antioxidants which can significantly impact oxidative stress response.

Methodology:

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:

Antioxidant Genetics panel summary page provides a flowchart of the human body's defense against oxidative stress and indicates the areas of concern from the genetic results observed. The summary page also lists the set of analytes with risk associated variants. The set of analytes with risk associated variants are also summarized. 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.

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 provided should be made in consultation with the clinical provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Antioxidant Genetics panel is performed by 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 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, diet, exercise, or lifestyle management as appropriate. This product is not intended to diagnose, treat, or cure any disease or condition.

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.

The diagram illustrates the antioxidant defense system, showing the conversion of Free Radicals into various products through a series of enzymatic reactions. The system is organized into a flowchart with numbered steps (1-8) and color-coded boxes (purple, blue, red, green, and yellow).

Key Components and Reactions:

- Free Radical (Purple Box):** The starting point of the defense system.
- Soluble Extracellular Toxins (Blue Box):** Produced from Free Radicals via **GSS (1)** (blue box) and **GST (2)** (red box).
- O₂⁻ (Blue Box):** A superoxide radical that can be converted to Soluble Extracellular Toxins or Lipid Radicals.
- Lipid Radicals (Blue Box):** Produced from O₂⁻ and -OH. It is in a dynamic equilibrium with Stable Lipid Products (blue box) via a red double-headed arrow.
- OH (Blue Box):** A hydroxy radical that can be converted to Lipid Radicals.
- SOD1 (3) (Blue Box), SOD2 (4) (Red Box), and SOD3 (5) (Blue Box):** Enzymes that convert O₂⁻ into H₂O₂ (Hydrogen peroxide, blue box).
- H₂O₂ (Blue Box):** Hydrogen peroxide, which is converted into H₂O (Water, green box) by **CAT (6) (Red Box)** and **GPX (7) (Red Box)**.
- GPX (7) (Red Box):** A key enzyme involved in the conversion of H₂O₂ to H₂O and in the conversion of Lipid Radicals to Stable Lipid Products.
- GR (8) (Blue Box):** A redox enzyme that converts Oxidized Glutathione (green box) back to Reduced Glutathione (green box).
- Reduced Glutathione (Green Box) and Oxidized Glutathione (Green Box):** A cycle where Reduced Glutathione is oxidized to Oxidized Glutathione, which is then reduced back to Reduced Glutathione by GR.
- Stable Lipid Products (Blue Box):** The final product of the lipid radical pathway, formed from Lipid Radicals via GPX (7).

Legend:

- O₂⁻ - Superoxide Radical
- H₂O₂ - Hydrogen peroxide
- OH - Hydroxy Radical
- H₂O - Water

- ① **GSS - Glutathione Synthetase** (0/1)
Involved in Glutathione synthesis
- ② **GST - Glutathione S Transferase** (3/3)
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** (0/2)
Aids in quenching superoxide free radical
- ⑥ **CAT - Catalase** (2/3)
Aids in quenching hydrogen peroxide
Mitochondrial dysfunction
- ⑦ **GPX - Glutathione Peroxidase** (2/5)
Aids in reduction of hydrogen peroxide by glutathione
Lower selenoprotein enzyme levels, Lower selenoprotein concentrations
- ⑧ **GR - Glutathione Reductase** (0/1)
Aids in recycling glutathione

Oxidative Stress Profile - Summary

| Antioxidant Genetics | | | | | |
|--|-----------|------------------------------------|---------------|-----------|-----------|
| ⊕ ⊕ Homozygous Mutant ⊕ ⊖ Heterozygous ⊖ ⊖ Homozygous Wild | | | | | |
| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
| rs4756146 | CAT | Mitochondrial dysfunction | ⊕ ⊕ T/T | Elevated | C/C, C/T |
| <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. Individuals with TT genotypes exhibit reduced catalase production leading to ROS buildup and increased risk of oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate 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. Individuals with TT genotypes exhibit reduced catalase production leading to ROS buildup and increased oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs2071566 | GPX2 | Lower selenoprotein enzyme levels | ⊖ ⊖ G/G | Elevated | A/G, A/A |
| <p>GPx2 is an endogenous selenium-dependent antioxidant encoding for the major antioxidant enzyme called Glutathione peroxidase 2. The GPX2 enzyme catalyzes the reduction of hydrogen peroxide to water and oxygen as well as catalyzing the reduction of peroxide radicals to alcohols and oxygen thus participating in the antioxidant defense system by protecting cells against reactive oxygen species. GPX2 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 lead to higher selenoprotein enzyme levels and reduced oxidative damage. Individuals with GG genotypes have a reduced enzyme level and GPX activity leading to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs4902346 | GPX2 | Lower selenoprotein concentrations | ⊖ ⊖ T/T | Elevated | C/T, C/C |
| <p>GPx2 is an endogenous selenium-dependent antioxidant encoding for the major antioxidant enzyme called Glutathione peroxidase 2. The GPX2 enzyme catalyzes the reduction of hydrogen peroxide to water and oxygen as well as catalyzing the reduction of peroxide radicals to alcohols and oxygen thus participating in the antioxidant defense system by protecting cells against reactive oxygen species. GPX2 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 higher selenoprotein enzyme levels and reduced oxidative damage. Individuals with TT genotypes have a reduced enzyme level and GPX activity leading to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs366631 | GSTM1 | Decreased antioxidant activity | ⊖ ⊖ T/T | Elevated | C/C |
| <p>GSTM1 gene encodes for an enzyme, glutathione S-transferase Muv 1. 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 GST 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. Individuals with TT genotypes who have impaired mitochondrial function with ROS build-up have increased oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |

Oxidative Stress Profile - Summary

| Antioxidant Genetics | | | | | |
|---|-----------|---|---------------|--------------------|-----------|
| ⊕ ⊕ Homozygous Mutant ⊕ ⊖ Heterozygous ⊖ ⊖ Homozygous Wild | | | | | |
| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
| rs9932581 | CYBA | Elevated ROS production | ⊖ ⊖ G/G | Elevated | A/A, A/G |
| <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. Individuals with GG genotype have higher p22phox expression and higher ROS levels, increasing their susceptibility to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs2796498 | PRKAA2 | Impaired antioxidant activity | ⊕ ⊕ G/G | Elevated | A/G, 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. Individuals with GG genotypes have impaired AMPK synthesis and impaired antioxidant activity leading to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs1548357 | TXNRD2 | Impaired mitochondrial oxygen radical scavenging activity | ⊖ ⊖ T/T | Elevated | C/T, C/C |
| <p>The TXNRD2 gene encodes a member of the thioredoxin (Trx) system, a selenocysteine-containing enzyme essential for mitochondrial oxygen radical scavenging. This protein plays a role in antioxidant defenses by reducing thioredoxin 2 (TXN2), which in turn reduces oxidized cysteine in cellular proteins and scavenges peroxides by peroxiredoxins (PRDX), thus protecting cells against oxidative stress. Mutation in the gene decreases enzyme activity which can affect mitochondrial oxygen radical scavenging and antioxidant functions resulting in increased oxidative stress. Individuals with TT genotypes have reduced enzyme activity and mitochondrial oxygen radical scavenging activity leading to higher oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs4880 | SOD2 | Impaired anti-oxidant activity | ⊕ ⊖ C/T | Partially elevated | C/C |
| <p>The SOD2 gene encodes the mitochondrial enzyme superoxide dismutase 2 (MnSOD), which protects cells from mitochondrial superoxide by converting it to hydrogen peroxide and molecular oxygen. This function allows SOD2 to clear mitochondrial reactive oxygen species (ROS), thereby protecting against oxidative stress. Individuals with CT genotypes exhibit moderate MnSOD activity, with partially impaired mitochondrial import, resulting in intermediate antioxidant defense and moderate oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs10911021 | GLUL | Decreased levels of glutamine synthetase and glutathione | ⊕ ⊖ C/T | Partially elevated | T/T |
| <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. Individuals with CT genotype exhibit reduced levels of glutamine synthetase enzyme and glutathione, leading to increased oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |

Oxidative Stress Profile - Summary

| Antioxidant Genetics | | | | | |
|--|-----------|--------------------------------------|---------------|--------------------|-----------|
| ⊕ ⊕ Homozygous Mutant ⊕ ⊖ Heterozygous ⊖ ⊖ Homozygous Wild | | | | | |
| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
| 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. Individuals with GT genotypes exhibit impaired mitochondrial function with ROS build-up and have increased oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs4485648 | TrxR2 | Impaired mitochondrial redox balance | ⊕ ⊖ C/T | Partially elevated | C/C |
| <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. Individuals with CT genotypes have reduced enzyme activity and impaired mitochondrial oxygen radical scavenging activity leading to higher oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |
| rs4673 | CYBA | Elevated ROS production | ⊕ ⊖ C/T | Partially elevated | C/C |
| <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. When present alongside other functional SNPs such as -930 A/G, this variant has been shown to synergistically enhance oxidative stress, as evidenced by increased malondialdehyde (MDA) levels. Individuals with the CT genotype have higher p22phox expression and higher ROS levels, increasing their susceptibility to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate 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. Individuals with the AG genotype exhibit heightened XO activity and experience an elevation in ROS levels, increasing their susceptibility to oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.</p> | | | | | |

Oxidative Stress Profile - Summary

Antioxidant Genetics

⊕ ⊕ Homozygous Mutant ⊕ ⊖ Heterozygous ⊖ ⊖ Homozygous Wild

| Test Name | Gene Name | Risk Association | Your Mutation | Your Risk | Reference |
|-----------|-----------|------------------|---------------|-----------|-----------|
|-----------|-----------|------------------|---------------|-----------|-----------|

| | | | | | |
|--------|-------|--|---------|--------------------|-----|
| rs1695 | GSTP1 | Decreasing antioxidant activity leads to elevated oxidative stress | ⊕ ⊖ A/G | Partially elevated | A/A |
|--------|-------|--|---------|--------------------|-----|

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. Individuals with AG genotypes who have decreased gene activity have decreased antioxidant activity and thus increased oxidative stress. Consume antioxidant-rich foods like berries, nuts, and green tea while avoiding processed foods. Engaging in regular moderate exercise is recommended.

| | | | | | |
|-----------|---------|-------------------------------------|---------|--------------------|-----|
| rs3877899 | SELENOP | Impaired plasma selenium production | ⊕ ⊖ C/T | Partially elevated | C/C |
|-----------|---------|-------------------------------------|---------|--------------------|-----|

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. Individuals with CT genotypes who have gene deficiency have a slightly increased risk of selenium deficiency and oxidative stress. 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.

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 enhances GPX2 activity by donating electrons to reduce glutathione (GSH), which is then used by GPX2 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. |
|-----------|-----------|--|

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 supplements increase GPX2 activity by incorporating selenium atoms into the GPX2 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. |
| Quercetin | 500 mg/day | Quercetin suppresses Xanthine Dehydrogenase (XDH) activity by inhibiting its conversion to Xanthine Oxidase (XO), thus reducing the production of reactive oxygen species and preventing oxidative stress. Quercetin supplements downregulate p22phox expression by inhibiting NF-κB activation, thereby reducing oxidative stress through decreased NADPH oxidase activity. Quercetin supplements may increase thioredoxin 2 (Trx2) levels by acting as an antioxidant, scavenging reactive oxygen species (ROS) and reducing oxidative stress, which in turn upregulates Trx2 expression through redox-sensitive pathways. |
| Vitamin D | 600 IU/day | Vitamin D increases GPX1 activity by binding to vitamin D receptors (VDRs) in cells, which in turn promotes the transcription of GPX1 gene, leading to higher GPX1 enzyme levels and enhanced antioxidant defense. Vitamin D increases GPX2 activity by binding to vitamin D receptors (VDRs) in cells, which in turn promotes the transcription of GPX2 gene, leading to higher GPX2 enzyme levels and enhanced antioxidant defense. |
| Lutein | 10mg/day | Lutein supplements increase GPX1 activity by enhancing the antioxidant defense system through their ability to scavenge free radicals, reducing oxidative stress and thereby promoting GPX1 enzyme function. Lutein supplements increase GPX2 activity by enhancing the antioxidant defense system through their ability to scavenge free radicals, reducing oxidative stress and thereby promoting GPX2 enzyme function. |
| Selenomethionine | 40 mcg/day | Selenomethionine supplements incorporate selenium into selenoproteins, including Thioredoxin 2, by replacing methionine during protein synthesis, enhancing Thioredoxin 2's antioxidant activity and supporting various cellular functions. Selenomethionine supplements provide an exogenous source of selenium, which might increase SELENOP activity and supporting its antioxidant and selenium transport functions in the body. |

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 | Increased risk of impaired EC-SOD tissue protection | ⊖ ⊖ C/C | Normal | C/G, C/C |
| rs8192287 | SOD3 | Disrupted EC-SOD activity | ⊕ ⊕ T/T | Normal | T/T, G/T |
| rs1001179 | CAT | Mitochondrial dysfunction | ⊖ ⊖ C/C | Normal | C/C |
| rs4756146 | CAT | Mitochondrial dysfunction | ⊕ ⊕ T/T | Elevated | C/C, C/T |
| rs7943316 | CAT | Mitochondrial dysfunction | ⊕ ⊕ T/T | Elevated | A/T, A/A |
| rs10911021 | GLUL | Decreased levels of glutamine synthetase and glutathione | ⊕ ⊖ C/T | Partially elevated | T/T |
| 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 | Lower selenoprotein enzyme levels | ⊖ ⊖ G/G | Elevated | A/G, A/A |
| rs4902346 | GPX2 | Lower selenoprotein concentrations | ⊖ ⊖ T/T | Elevated | C/T, C/C |
| rs713041 | GPX4 | Lower selenoprotein concentrations | ⊖ ⊖ C/C | Normal | C/T, C/C |
| rs121909307 | GSS | Lower glutathione levels | ⊕ ⊕ C/C | Normal | C/C |
| rs2071746 | HMOX1 | Decreased heme oxygenase 1 activity | ⊕ ⊕ T/T | Normal | T/T |
| rs366631 | GSTM1 | Decreased antioxidant activity | ⊖ ⊖ T/T | Elevated | C/C |
| rs3754446 | GSTM5 | Decreased antioxidant activity | ⊕ ⊖ G/T | Partially elevated | T/T |
| rs4485648 | TrxR2 | Impaired mitochondrial redox balance | ⊕ ⊖ C/T | Partially elevated | C/C |
| rs4673 | CYBA | Elevated ROS production | ⊕ ⊖ C/T | Partially elevated | C/C |
| rs9932581 | CYBA | Elevated ROS production | ⊖ ⊖ G/G | Elevated | A/A, A/G |
| rs10789038 | PRKAA2 | Impaired antioxidant activity | ⊖ ⊖ A/A | Normal | A/A |
| rs2796498 | PRKAA2 | Impaired antioxidant activity | ⊕ ⊕ G/G | Elevated | A/G, 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 | Normal | A/A |
| rs1548357 | TXNRD2 | Impaired mitochondrial oxygen radical scavenging activity | ⊖ ⊖ T/T | Elevated | C/T, C/C |
| 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 | ⊕ ⊖ 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 | Normal | G/G, A/G |

Risk and Limitations

This test has been developed and its performance characteristics determined and validated by Vibrant Genomics LLC., a CLIA certified lab. 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 Antioxidant Genetics panel 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.

Antioxidant Genetics testing is performed at Vibrant Genomics, a CLIA certified laboratory. Vibrant Genomics 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.