| DEMO DEMO   FINAL REPORT   Accession ID: 2692051854 | Name: DEMO DEMO<br>Date of Birth: 05-29-1968<br>Biological Sex: Male<br>Age: 57<br>Height:<br>Weight:<br>Fasting: UNKNOWN | Telephone: 000-000-0000<br>Street Address:<br>Email:                          |
|---|---|---|
| Provider Information                                | Practice Name: DEMO CLIENT, MD<br>Provider Name: DEMO CLIENT, MD<br>Phlebotomist: 0                                       | Telephone: 000-000-0000<br>Address: 3521 Leonard Ct, Santa<br>Clara, CA 95054 |

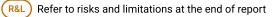
### **Report Information**

Current Result Previous Result In Control Moderate Risk

#### **Specimen Information**

| Sample Type      | Collection Time        | Received Time          | Report                        | Final Report Date      |
|------------------|------------------------|------------------------|-------------------------------|------------------------|
| Metal Free Urine | 2024-04-02 13:27 (UTC) | 2024-04-03 22:22 (UTC) | Oxidative Stress Profile - P2 | 2024-04-16 18:06 (UTC) |
| Saliva           | 2024-04-02 13:25 (UTC) | 2024-04-03 22:22 (UTC) | Oxidative Stress Profile - P2 | 2024-04-16 18:06 (UTC) |







## **Oxidative Stress Profile**

#### 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 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.

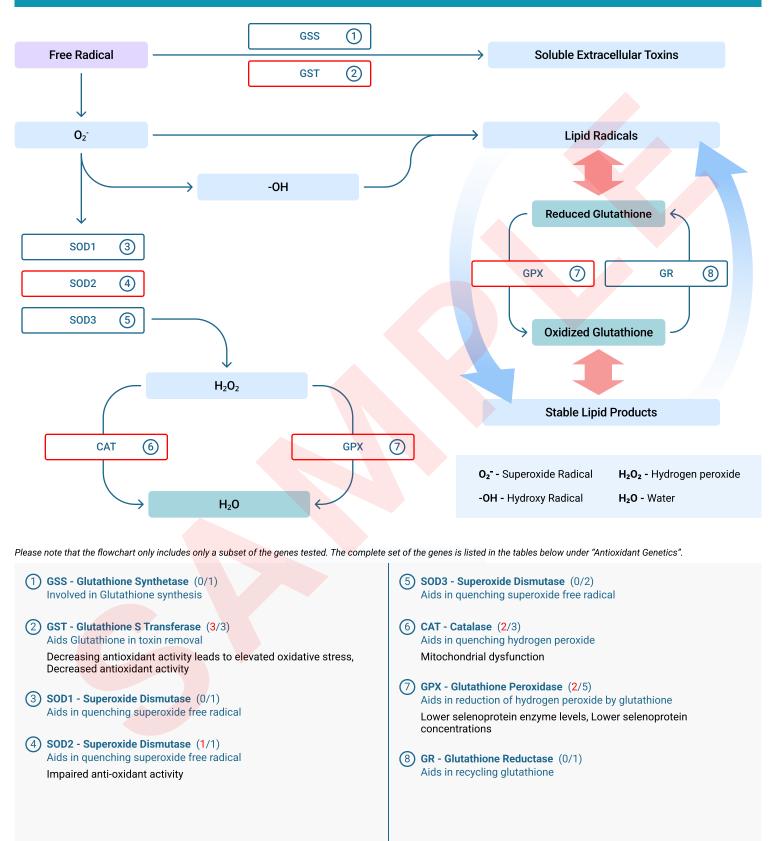
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 Accession ID:
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 Service Date:
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### Oxidative Stress Profile - Summary

#### **Oxidative Stress Profile Summary**





| ate of Birth: 05-2<br>ervice Date: 202   | 29-1968 Accession<br>4-04-02 13:25 (UTC)   | ID: 2692051854 Oxida   | tive Stre   | ess Pro  | ofile - Summary   |
|--|--|--|---|--|---|
| Antioxidant  | Genetics   | ⊕ ⊕ Homo   | ozygous Mutant 🛛 🕂 🗲  | Heterozygous   | ⊖ ⊖ Homozygous Wild   |
| Test Name  | Gene Name  | <b>Risk Association</b>  | Your Mutation   | Your Risk  | Reference   |
| rs4756146  | CAT  | Mitochondrial dysfunction  | ⊕⊕T/T   | Elevated   | C/C, C/T  |
| oxidative injury<br>thereby protect<br>Mutations in the<br>dysfunction and<br>increased risk of                  | induced by hydroge<br>ing cells from the to<br>e gene lead to decre<br>d elevated oxidative<br>of oxidative stress. C                          | lase enzyme, localized in mitochon<br>n peroxide by degrading hydrogen p<br>xic effects of hydrogen peroxide. Th<br>ased catalase production resulting<br>stress.Individuals with TT genotype<br>consume antioxidant-rich foods like<br>sise is recommended.   | peroxide generated by p<br>hus, the enzyme particip<br>in excess ROS producti<br>es exhibit reduced catala  | eroxisomal oxid <mark>ase</mark><br>pates in antioxidant<br>on. This induces m<br>ase production lead                | s to water and oxygen,<br>functions in the body.<br>itochondrial<br>ling to ROS buildup and                     |
| rs7943316  | CAT  | Mitochondrial dysfunction  | ⊕⊕T/T   | Elevated   | А/Т, А/А  |
| oxidative injury<br>thereby protect<br>Mutations in the<br>dysfunction and<br>increased oxida                    | induced by hydroge<br>ing cells from the to<br>e gene lead to decre<br>d elevated oxidative  | lase enzyme, localized in mitochon<br>n peroxide by degrading hydrogen p<br>xic effects of hydrogen peroxide. Th<br>ased catalase production resulting<br>stress.Individuals with TT genotype<br>he antioxidant-rich foods like berries<br>mended.   | peroxide generated by p<br>hus, the enzyme particip<br>in excess ROS producti<br>es exhibit reduced catala  | eroxisomal oxidase<br>pates in antioxidant<br>on. This induces m<br>ase production lead                              | s to water and oxygen,<br>functions in the body.<br>itochondrial<br>ling to ROS buildup and                     |
| rs2071566  | GPX2 L   | ower selenoprotein enzyme levels   | <mark>⊝⊝G</mark> /G   | Elevated   | A/G, A/A  |
| The GPX2 enzy<br>radicals to alco<br>GPX2 modulate<br>ROS-mediated o<br>oxidative dama                           | me catalyzes the red<br>hols and oxygen thu<br>es redox-dependent<br>changes in the cellu<br>ge.Individuals with (                             | ependent antioxidant encoding for<br>duction of hydrogen peroxide to wa<br>is participating in the antioxidant de<br>mitochondrial function where mitoc<br>ar redox state. Mutations in the ger<br>GG genotypes have a reduced enzyr<br>nuts, and green tea while avoiding p   | ter and oxygen as well a<br>efense system by protec<br>chondria generate reacti<br>ne lead to higher seleno<br>me level and GPX activit                             | is catalyzing the red<br>ting cells against r<br>ve oxygen species<br>protein enzyme lev<br>y leading to oxidati     | duction of peroxide<br>eactive oxygen species.<br>(ROS) and respond to<br>els and reduced<br>ve stress. Consume |
| rs4902346  | GPX2   | Lower selenoprotein<br>concentrations  | ΘΘT/T   | Elevated   | C/T, C/C  |
| The GPX2 enzy<br>radicals to alco<br>GPX2 modulate<br>ROS-mediated o<br>oxidative dama                           | me catalyzes the red<br>hols and oxygen thu<br>s redox-dependent<br>changes in the cellu<br>ge.Individuals with                                | ependent antioxidant encoding for<br>duction of hydrogen peroxide to wa<br>s participating in the antioxidant de<br>mitochondrial function where mitoc<br>lar redox state. Mutations in the gen<br>T genotypes have a reduced enzyn<br>nuts, and green tea while avoiding p  | ter and oxygen as well a<br>efense system by protec<br>chondria generate reacti<br>ne cause higher selenop<br>ne level and GPX activity                             | as catalyzing the red<br>sting cells against r<br>ve oxygen species<br>protein enzyme leve<br>y leading to oxidation | duction of peroxide<br>eactive oxygen species.<br>(ROS) and respond to<br>Is and reduced<br>ve stress. Consume  |
| rs366631   | GSTM1  | Decreased antioxidant activity   | $\Theta\Theta$ T/T  | Elevated   | C/C   |
| lysosomes, and<br>release, mitoch<br>role in detoxific<br>Reactive oxygel<br>build-up of ROS<br>function with Re | I nuclear regions. Th<br>ondrial GST contribu<br>ation by catalyzing t<br>n species (ROS). The<br>therefore, increasin<br>OS build-up have inc | ne, glutathione S-transferase Muv 1<br>rough the inhibition of cardiolipin (a<br>utes to the protection of organelles<br>he modification of toxic compound<br>e mutation leads to lead to significa<br>g the risk for oxidative stress. Indivi<br>reased oxidative stress. Consume a<br>g in regular moderate exercise is re | a lipid found in mitocho<br>from oxidative stress. T<br>Is to glutathione, which<br>ant damage in cells and<br>iduals with TT genotype<br>antioxidant-rich foods li | ndria) peroxidation<br>he enzyme plays a<br>is an antioxidant th<br>mitochondrial func<br>s who have impaire         | and cytochrome c<br>n important regulatory<br>at helps combat<br>tion, which causes a<br>ed mitochondrial       |

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### Oxidative Stress Profile - Summary

| Antioxidant Genetics |           |                         | nozygous Mutant     |             | 🗢 🗢 Homozygous Wild |
|----------------------|-----------|-------------------------|---------------------|-------------|---------------------|
| Test Name            | Gene Name | <b>Risk Association</b> | Your Mutatio        | n Your Risk | Reference           |
| rs9932581            | СҮВА      | Elevated ROS production | $\Theta \Theta G/0$ | B Elevated  | A/A, A/G            |

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.

| processed foods. Engaging in regular moderate exercise is recommended.   |  |  |   |  |  |  |  |
|--|--|--|---|--|--|--|--|
| rs2796498  | PRKAA2   | Impaired antioxidant activity  | ⊕⊕G/ <mark>G</mark>   | Elevated   | A/G, A/A   |  |  |
| monitors cellular<br>oxidation when ce<br>stress. AMPK pro<br>ATP). Mutation re<br>to oxidative stress   | energy status. A<br>ellular energy is<br>motes mitochor<br>duces the expre<br>s. Individuals wit<br>antioxidant-rich | n enzyme AMP-activated protein kinas<br>MPK plays a role in cellular energy ho<br>low. AMPK is part of the antioxidant of<br>ndrial biogenesis (a process that occu<br>ssion of the PRKAA2 gene causing in<br>th GG genotypes have impaired AMP<br>foods like berries, nuts, and green tea | omeostasis, largely to a<br>defense system and is n<br>urs in response to increa<br>npaired AMPK synthesis<br>K synthesis and impaire | ctivate glucose and fath<br>needed to protect the ce<br>ased energy expenditure<br>s and impaired antioxid<br>ed antioxidant activity le | y acid uptake and<br>Ils from oxidative<br>to produce more<br>ant activity leading<br>ading to oxidative |  |  |
| rs1548357  | TXNRD2   | Impaired mitochondrial oxygen radical scavenging activity  | ΘΘΤ/Τ   | Elevated   | C/T, C/C   |  |  |
| oxygen radical sca<br>oxidized cysteine<br>Mutation in the ge<br>resulting in increa<br>scavenging activit   | avenging. This p<br>in cellular prote<br>ene decreases e<br>sed oxidative st<br>ty leading to hig                    | mber of the thioredoxin (Trx) system,<br>protein plays a role in antioxidant defe<br>ins and scavenges peroxides by pero<br>nzyme activity which can affect mitoo<br>ress. Individuals with TT genotypes h<br>her oxidative stress. Consume antiox<br>jular moderate exercise is recommend | enses by reducing thiore<br>xiredoxins (PRDX), thus<br>chondrial oxygen radica<br>nave reduced enzyme ac<br>idant-rich foods like ber | edoxin 2 (TXN2), which i<br>protecting cells agains<br>Il scavenging and antiox<br>ctivity and mitochondria                              | n turn reduces<br>t oxidative stress.<br>xidant functions<br>l oxygen radical                            |  |  |
| rs4880   | SOD2   | Impaired anti-oxidant activity   | ⊕⊝C/T   | Partially elevated   | C/C  |  |  |
| 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.  |  |  |   |  |  |  |  |
| rs10911021   | GLUL   | Decreased levels of glutamine<br>synthetase and glutathione  | ⊕⊝C/T   | Partially elevated   | T/T  |  |  |
| 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. |  |  |   |  |  |  |  |



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### **Oxidative Stress Profile** - Summary

| Antioxidant Genetics |           | 🕀 🕀 Homo                       | ozygous Mutant 🛛 🕂 | 🗩 🗕 Heterozygous   | 🗢 🗢 Homozygous Wild |
|----------------------|-----------|--------------------------------|--------------------|--------------------|---------------------|
| Test Name            | Gene Name | <b>Risk Association</b>        | Your Mutation      | Your Risk          | Reference           |
| rs3754446            | GSTM5     | Decreased antioxidant activity | ⊕⊝G/T              | Partially elevated | d T/T               |

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.

| rs4485648 | TrxR2 | Impaired mitochondrial redox<br>balance | ⊕⊝C/T | Partially elevated | C/C |  |
|-----------|-------|---|-------|--------------------|-----|--|
|           |       |   |       |                    |     |  |

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.

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rs4673
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CYBA

XDH

Elevated ROS production

 $\oplus \bigcirc C/T$ 

Partially elevated

C/C

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.

rs206812

Elevated ROS production



G/G

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.

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### Oxidative Stress Profile - Summary

| Antioxidant Genetics |           |  | zygous Mutant | 🛨 🗢 Heterozygous    | 🗢 🗢 Homozygous Wild |
|----------------------|-----------|--|---------------|---------------------|---------------------|
| Test Name            | Gene Name | <b>Risk Association</b>  | Your Mutatior | n Your Risk         | Reference           |
| rs1695               | GSTP1     | Decreasing antioxidant activity leads to elevated oxidative stress | €⊖A/G         | B Partially elevate | ed 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.

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.



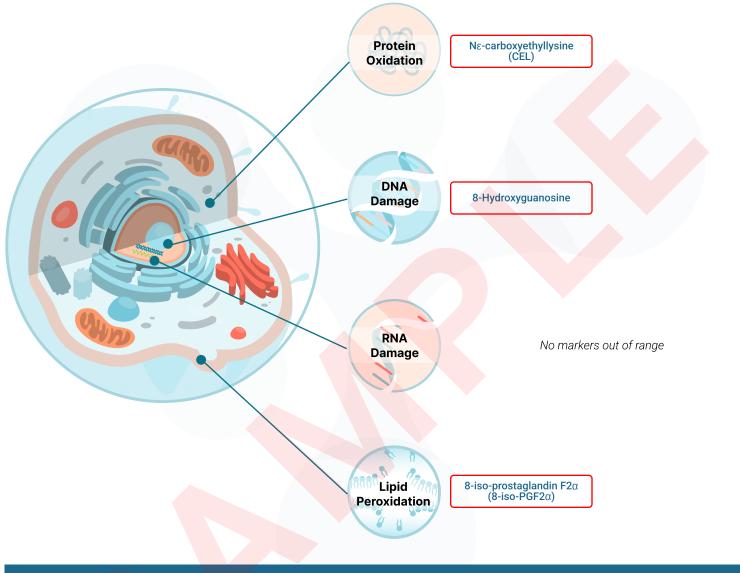
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## Oxidative Stress Profile - Summary

#### **Oxidative Stress Profile Summary**



#### **Oxidative Stress Biomarkers**

| Lipid Peroxidation  | Current | Previous | Result<br><sup>75th</sup> 95th | Reference |
|---|---------|----------|--------------------------------|-----------|
| 8-iso-pros <mark>taglandin F2</mark> α (8-iso-<br>PGF2α) (ug/g) | 0.12    |          | 0.1 0.26                       | ≤0.26     |

8-iso-prostaglandin F2 $\alpha$  (8-iso-PGF2 $\alpha$ ) 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 PGF2 $\alpha$  can lead to DNA oxidation and subsequent structural DNA damage. 8-iso PGF2 $\alpha$  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 PGF2 $\alpha$  contribute to heightened oxidative stress associated with aging, hypertension, diabetes mellitus, hypercholesterolemia, smoking, and coronary artery disease.

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### Oxidative Stress Profile - Summary

| Oxidative Stress Biomarkers |         |          |           |           |  |  |
|-----------------------------|---------|----------|-----------|-----------|--|--|
| DNA Damage                  | Current | Previous | Result    | Reference |  |  |
| 8-Hydroxyguanosine (ug/g)   | 65.23   | 0        | 44.0 05.3 | ≤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)<br>(ug/g) | 28.61   |          | 0 19.5 91 | ≤91       |

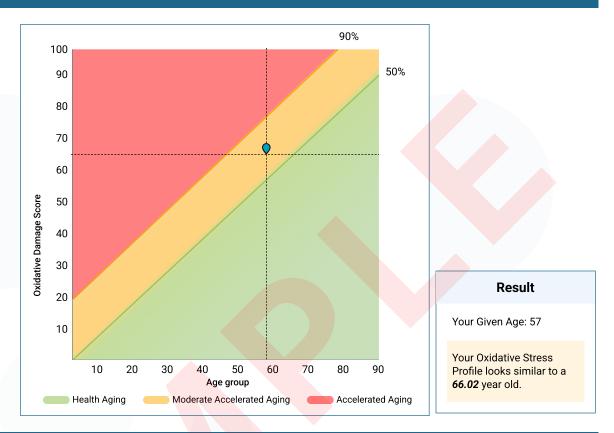
Glycation is a spontaneous non-enzymatic reaction wherein free reducing sugars bind to free amino groups of proteins, DNA, and lipids. This results in the formation of advanced glycation end-products (AGE). Glycation and oxidative stress are closely linked, and they are together referred to as "glycoxidation". 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, N $\epsilon$  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 0.24 | 2.16   | 0.25-2.16 |
|                          |         |          |        |        |           |

### Oxidative Stress Profile - Summary

#### **Oxidative Damage Score**

O Current Result O Previous Result



#### 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. Vitamin C supplementation decreases 8-iso-prostaglandin F2 $\alpha$ (8-iso-PGF2 $\alpha$ ) levels by acting as a powerful antioxidant, scavenging free radicals and inhibiting lipid peroxidation, thereby reducing oxidative stress. |

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### Oxidative Stress Profile - Summary

#### **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. Selenium reduces 8-iso-prostaglandin F2 $\alpha$ (8-iso-PGF2 $\alpha$ ) levels by acting as a cofactor for the enzyme glutathione peroxidase, which helps neutralize reactive oxygen species responsible for the formation of 8-iso-PGF2 $\alpha$ . |
| Quercetin | 500 mg/day | Quercetin suppresses Xanthine Dehydrogenase (XDH) activity by<br>inhibiting its conversion to Xanthine Oxidase (XO), thus reducing the<br>production of reactive oxygen species and preventing oxidative<br>stress. Quercetin supplements downregulate p22phox expression by<br>inhibiting NF-KB activation, thereby reducing oxidative stress through<br>decreased NADPH oxidase activity. Quercetin supplements may<br>increase thioredoxin 2 (Trx2) levels by acting as an antioxidant,<br>scavenging reactive oxygen species (ROS) and reducing oxidative<br>stress, which in turn upregulates Trx2 expression through redox-<br>sensitive pathways.   |
| 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 F2 $\alpha$ (8-iso-PGF2 $\alpha$ ) levels by acting as an antioxidant, neutralizing reactive oxygen species that would otherwise promote the formation of 8-iso-PGF2 $\alpha$ .   |
| Vitamin D | 600 IU/day | Vitamin D increases GPX1 activity by binding to vitamin D receptors<br>(VDRs) in cells, which in turn promotes the transcription of GPX1<br>gene, leading to higher GPX1 enzyme levels and enhanced<br>antioxidant defense. Vitamin D increases GPX2 activity by binding to<br>vitamin D receptors (VDRs) in cells, which in turn promotes the<br>transcription of GPX2 gene, leading to higher GPX2 enzyme levels<br>and enhanced antioxidant defense.  |

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### Oxidative Stress Profile - Summary

#### **Supplementation Suggestions**

| Nutrients | Dosage   | Purpose   |
|-----------|----------|---|
| Leutin    | 10mg/day | Lutein supplements increase GPX1 activity by enhancing the<br>antioxidant defense system through their ability to scavenge free<br>radicals, reducing oxidative stress and thereby promoting GPX1<br>enzyme function. Lutein supplements increase GPX2 activity by<br>enhancing the antioxidant defense system through their ability to<br>scavenge free radicals, reducing oxidative stress and thereby<br>promoting GPX2 enzyme function. |



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# **Oxidative Stress Profile**

| Antioxidant             | Genetics  | ↔ ↔ Homo:  | zygous Mutant          | 🕈 🗢 Heterozygous        | 🗢 🖨 Homozygous Wild |
|-------------------------|-----------|--|------------------------|-------------------------|---------------------|
| Test Name               | Gene Name | <b>Risk Association</b>  | Your Mutatior          | n Your Risk             | Reference           |
| rs2234694               | SOD1      | Increased superoxide levels  | $\Theta \Theta A/A$    | Normal                  | A/A                 |
| rs4880                  | SOD2      | Impaired anti-oxidant activity   | ⊕⊝C/T                  | - Partially elevate     | d C/C               |
| rs1799895               | SOD3      | Increased risk of impaired EC-SOD<br>tissue protection                   | ⊖⊝C/C                  | ) Normal                | C/G, C/C            |
| rs8192287               | SOD3      | Disrupted EC-SOD activity  | $\oplus \oplus T/T$    | -<br>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              | Α/Τ, Α/Α            |
| rs10911021              | GLUL      | Decreased levels of glutamine synthetase and glutathione                 | ⊕⊖C/1                  | -<br>Partially elevated | d T/T               |
| rs1050450               | GPX1      | Aberrant redox signaling   | ⊖⊝C/C                  | Normal                  | C/C                 |
| rs1987628               | GPX1      | Reduced antioxidant enzyme leads<br>to selenium defic <mark>iency</mark> | ⊕⊕C/C                  | Normal                  | C/C                 |
| rs2071566               | GPX2      | Lower selenoprotein enzyme levels  | $\Theta \Theta G/G$    | Elevated                | A/G, A/A            |
| rs4902346               | GPX2      | Lower selenoprotein concentrations                                       | ΘΘΤ/Τ                  | -<br>Elevated           | C/T, C/C            |
| rs713041                | GPX4      | Lower selenoprotein concentrations                                       | $\Theta \Theta C/C$    | Normal                  | C/T, C/C            |
| rs121909307             | GSS       | Lower glutathione levels   |                        | Normal                  | C/C                 |
| rs2071746               | HMOX1     | Decreased heme oxygenase 1<br>activity                                   | $\oplus \oplus \top/1$ | -<br>Normal             | T/T                 |
| rs366631                | GSTM1     | Decreased antioxidant activity   | $\Theta\Theta T/T$     | -<br>Elevated           | C/C                 |
| rs3754446               | GSTM5     | Decreased antioxidant activity   | ⊕⊝G/1                  | -<br>Partially elevate  | d T/T               |
| rs448564 <mark>8</mark> | TrxR2     | Impaired mitochondrial redox<br>balance                                  | €⊝C/T                  | -<br>Partially elevate  | d C/C               |
| rs4673                  | СУВА      | Elevated ROS production  | ⊕⊝C/T                  | -<br>Partially elevated | d C/C               |
| rs9932581               | СҮВА      | Elevated ROS production  | ⊖⊝G/G                  | Elevated                | A/A, A/G            |
| rs10789038              | PRKAA2    | Impaired antioxidant activity  | $\Theta \Theta A/A$    | Normal                  | A/A                 |
| rs2796498               | PRKAA2    | Impaired antioxidant activity  | ⊕⊕G/G                  | Elevated                | A/G, A/A            |
| rs206812                | XDH       | Elevated ROS production  | ⊕⊝A/C                  | Partially elevate       | d G/G               |

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# **Oxidative Stress Profile**

| Antioxidant Genetics |           | ⊕ ⊕ Homo   | ozygous Mutant     | 🕈 🗢 Heterozygous    | ⊖ ⊖ Homozygous Wild |
|----------------------|-----------|--|--------------------|---------------------|---------------------|
| Test Name            | Gene Name | <b>Risk Association</b>  | Your Mutation      | n Your Risk         | Reference           |
| rs2073316            | XDH       | Elevated ROS production  | €⊝C/1              | –<br>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<br>radical scavenging activity       | $\Theta\Theta T/T$ | - Elevated          | С/Т, С/С            |
| rs1695               | GSTP1     | Decreasing antioxidant activity leads to elevated oxidative stress | ⊕⊝A/G              | B Partially elevate | ed A/A              |
| rs20417              | COX-2     | Elevated ROS production  | ⊕⊖C/C              | S Normal            | C/C, C/G            |
| rs3877899            | SELENOP   | Impaired plasma selenium production                                | ⊕⊝C/1              | - Partially elevate | ed C/C              |
| rs8190955            | GSR       | Increased oxidative stress in red<br>blood cells                   | ⊕⊕C/C              | ) Normal            | C/C                 |
| rs916321             | CYB5R3    | Elevated ROS production  | ⊕⊝A/G              | S Normal            | G/G, A/G            |

#### **Oxidative Stress Biomarkers**

| Lipid Peroxidation                               | Current             | Previous | 75th 95th    | Reference |
|--|---------------------|----------|--------------|-----------|
| 11-β-Prostaglandin F2α (ug/g)                    | 0.11                |          | 0.11 0.4     | ≤0.4      |
| 15(R)-Prostaglandin F2 $\alpha$ (ug/g)           | <0.05               |          | 0.07 0.22    | ≤0.22     |
| 8-iso-prostaglandin F2α (8-iso-<br>PGF2α) (ug/g) | 0.12                |          | 0.1 0.26     | ≤0.26     |
| Glutathione 4-hydroxynonenal<br>(GS-HNE) (ug/g)  | 0.23                |          | 0.3 2.5      | ≤2.5      |
| Malondialdehyde (ug/g)                           | <mark>60</mark> .15 |          | 72.87 163.53 | ≤163.53   |
| DNA Damage                                       | Current             | Previous | Result       | Reference |
| 8-Hydroxy-2-deoxyguanosine<br>(ug/g)             | 0.73                |          | 0 1.14 4     | ≤4        |
| 8-Hydroxyguanine (ug/g)                          | 10.87               |          | 0 16 49.4    | ≤49.4     |
| 8-Hydroxyguanosine (ug/g)                        | 65.23               |          | 0 44.9 95.3  | ≤95.3     |
| RNA Damage                                       | Current             | Previous | Result       | Reference |
| 8-Nitroguanine (ug/g)                            | 22.01               |          | 0 33.4 107   | ≤107.47   |
| 8-Nitroguanosine (ug/g)                          | 454.21              |          | 0 778 2608   | ≤2608.9   |



Vibrant America Clinical Laboratory Laboratory Director: Dr. Claude O. Burdick, M.D. CLIA: 05D2078809 1-866-364-0963 | Support@vibrant-america.com | www.vibrant-america.com Patient Name:DEMO DEMODate of Birth:05-29-1968Accession ID:2692051854Service Date:2024-04-02 13:25 (UTC)

# **Oxidative Stress Profile**

#### **Oxidative Stress Biomarkers**

| Oxidative Stress Diomarke                | 515     |          |        |                |           |
|--|---------|----------|--------|----------------|-----------|
| Protein Oxidation Products               | Current | Previous | 75th   | Result<br>95th | Reference |
| 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)<br>(ug/g) | 15.31   |          | 0 15.8 | 70.3           | ≤70.3     |
| Nε-carboxyethyllysine (CEL)<br>(ug/g)    | 28.61   |          | 0 19.5 | 91             | ≤91       |
|  |         |          |        |                |           |

## **Oxidative Stress Profile**

#### **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.