G-DAP

PRECISION POINT DIAGNOSTICS

Gut and Detox Assessment Profile

MARKERS Level	Detox/ Ox Stress	Gut	Immune/Cancer Prevention	Cardio	Memory
Glutathione (% Reduced/Total) (Low)	Major Intercellular Antioxidant	Leaky Gut = Major cause ox stress	Reduces DNA mutations	Decrease glycocalyx	Antioxidant of brain
F2-Isoprostane (High)	Oxidized Lipids = Decrease Neuroprostane			Plaque former	Damaged Neuroprostanes
8-OHdG (High)	DNA damage		Increases Risk	Mitochondrial dysfunction	
OxLDL (High)	Increases secondary to toxins			Increase risk	Damage Neuroprostane, Decrease nerve conductio
LPS (High)	Leading cause of NAFLD	Damages tight junctions	Decrease immune reserves, decrease Treg cells	Oxidizes LDL, Damages Heart	Induces Depression/Anxiety
Zonulin (High)	Leaky Gut = Major cause of ox stress	Opens tight junctions			Leaky Blood Brain Barrier
DAO (Low)	Impairs detox	Atrophy of gut lining causing permability	Increases TH2 and TH17 branch		Anxiety, Decrease focus
Vitamin D (Low)		If low but patient is taking, possible leaky gut	Decreases risk	Decrease risk	Decrease risk
hsCRP (High)	Slow liver detox	IL-6 secondary to leaky gut can increase		Most predictive cardiac marker	Increase risk
HgA1C (High)	Impairs conjugation	Glycation damages tight junctions and negatively influences microbiome	Increased risk >5.7	Metablolic Syndrome	> 5.2 associated with diminishing brain size
Histamine	Congests the liver	Can be a result of leaky gut			Causes irritability and anxiety

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PRECISION POINT DIAGNOSTICS — Advanced Intestinal Barrier Assessment

A "Targeted" Approach to Wellness

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PATIENT INFO	
NAME:	John Doe
REQUISITION ID:	2104210007
DOB:	12/8/1991
SAMPLE DATE:	4/20/2021
RECEIVE DATE:	4/21/2021
DRAFT DATE:	4/29/2021

CLINIC INFO

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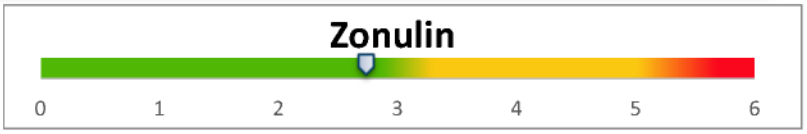
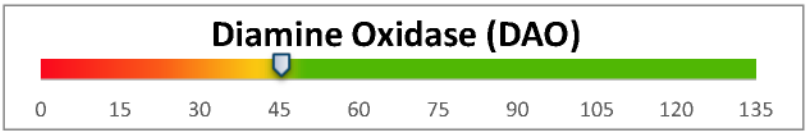
ADVANCED INTESTINAL BARRIER ASSESSMENT (PLASMA) | 1/2

2.74
Normal Range: < 3.19 ng/ml
NORMAL



45.40
Normal Range: > 42.9 ng/mL
NORMAL



2.34
Normal Range: < 1.2 ng/mL
HIGH

19.40
Normal Range: > 34.0 ng/mL
BORDERLINE LOW

A high DAO-to-Histamine ratio suggests that there is sufficient DAO present to degrade any free histamine.

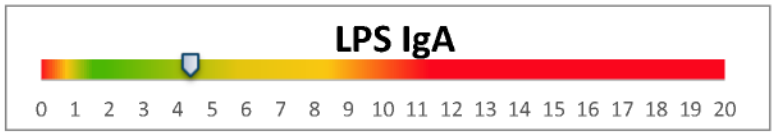
Conversely, a low DAO:Histamine ratio may be more indicative of histamine intolerance.

This test has been developed and its performance characteristics determined by Precision Point Diagnostics. It has not been cleared by the U.S. Food and Drug Administration.

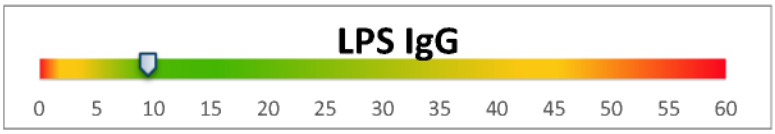
PATIENT NAME:	John Doe	REQUSITION ID:	2104210007	DRAFT DATE:	4/29/2021

ADVANCED INTESTINAL BARRIER ASSESSMENT (PLASMA) | 2/2

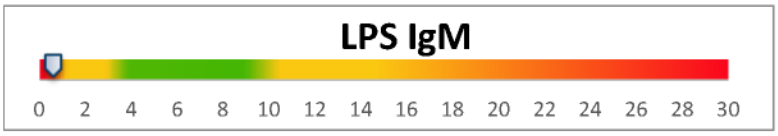
4.37
Normal RANGE: 0.83-4.47 µg/mL
NORMAL



9.51
Normal RANGE: 9.09-31.5 µg/mL
NORMAL



0.60
Normal RANGE: 2.5-9.4 µg/mL
LOW



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PRECISION POINT
DIAGNOSTICS

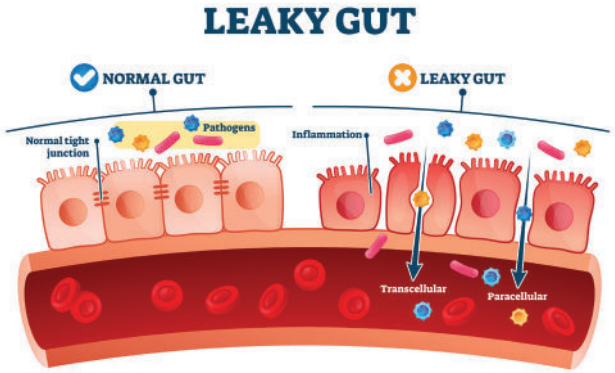
ADVANCED INTESTINAL BARRIER ASSESSMENT

Imbalances in Zonulin, Histamine, DAO and LPS are associated with intestinal permeability, often referred to as, "leaky gut."

When the gut barrier is weakened, a person is more vulnerable to food antigens, toxins, and unfriendly microbes. A leaky gut tears down the body's defenses and opens up the system to increased inflammation. There are many possible causes of damage to the GI lining and subsequent leaky gut.

Common causes of intestinal permeability are bacterial overgrowth, food sensitivities including gluten sensitivity, antibiotics, PPI inhibitors, stress, food additivies, NSAIDs, and alcohol consumption.

Reducing inflammation and healing the GI lining can help restore the GI barrier and normalize Zonulin, DAO, histamine, and LPS.



High Zonulin

High plasma zonulin is associated with intestinal permeability. Zonulin is a protein that leads to the break-down of tight gap junctions in the GI lining. These junctions are critical for a healthy barrier against the outside world.

When the gut barrier is weakened, a person is more vulnerable to food antigens, toxins, and unfriendly microbes. A leaky gut tears down the body's defenses and opens up the system to increased inflammation.

Increased levels of zonulin may be a contributing factor in the development of celiac disease, autoimmune disorders, insulin dependent diabetes, multiple sclerosis and rheumatoid arthritis. Higher zonulin levels have been reported in patients with active celiac disease compared to non-celiac patients.1-3 Zonulin levels elevate 2-5 years before diabetes, autoimmune conditions, and allergies. Zonulin may therefore be an early marker of disease processes.

Histamine Intolerance

Histamine Intolerance - Histamine intolerance can develop when a person has abnormal levels of histamine and the histamine degrading enzyme, diamine oxidase (DAO).

Typical symptoms of histamine intolerance are headache, diarrhea, migraine, general inflamed, circles under the eyes and runny nose.

Histamine intolerance might be more obvious with specific food triggers leading to asthma and arrhythmia, hypotension, urticaria, and dysmenorrhea. When DAO or histamine is imbalanced, the main focus of treatment is to increase DAO, reduce histamine, and heal the gut.

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PRECISION POINT
DIAGNOSTICS

High Histamine

Anaphylaxis	Dizziness	Affected locomotion	Sneezing
Painful menstruation	Congestion	Stomach Ache	Reproductive
Circadian rhythm	Runny nose	Itching	Issues
High blood pressure	Arrhythmia	Cramps	Diarrhea
Shortness of breath	Nausea, vomiting	Abnormal	Flush
Body temperature	Hives	heart rate	Gas
changes	Memory Loss	Headache	Low muscle tone

Histamine

Histamine balance is a critical factor in patients with allergic and gastrointestinal symptoms. Neither too high, nor too low of a level of Histamine is desirable. Histamine was first discovered in its role in anaphylactic allergy. A specific allergen can trigger the degranulation of mast cells, subsequently releasing histamine. This can lead to severe, life-threatening symptoms. When the gut barrier is weakened, a person is more vulnerable to food antigens, toxins, and unfriendly microbes. A leaky gut tears down the body's defenses and opens the system up to increased inflammation.

Classic symptoms of high histamine are tachycardia, headache, flushing, urticaria, pruritis, hypotension, bronchospasm, and cardiac arrest. However histamine can have far-reaching impacts and lead to many atypical symptoms because it binds cells throughout the body- in the gastrointestinal tract, respiratory tract, skin, cardiovascular system, and central nervous system, among others.

Gut permeability can also increase histamine. Leaky gut activates T cells and triggers degranulation of histamine-containing mast cells. In addition to histamine made in the body, we consume histamine in varying amounts in foods.

After extreme histamine exposure, as in anaphylactic shock, levels of both diamine oxidase and histamine will be elevated. Low histamine levels may cause fatigue or depression. Alterations of histamine have been noted in sleep-wake disorders such as narcolepsy, as well as other neurological and psychiatric diseases. Brain levels of histamine are decreased in Alzheimer's and low histamine has been seen in cases of convulsions and seizures.

High Histamine Foods

Very High: Aged or fermented foods: kimchi, syogurt or kefir, kombucha, aged cheese, alcohol of any kind, vinegar, and cured meat. Fish and seafood, especially canned or smoked fish.

Medium: Spinach, eggplant, mushrooms, tomatoes, canned vegetables, dried fruit, avocados, strawberries, papaya, pineapple, and leftovers.

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DAO

Diamine Oxidase (DAO) is histamine's vital counterpart and the primary enzyme responsible for keeping histamine levels in check. DAO degrades extracellular histamine and is mainly produced in the microvilli of the small intestine. When diamine oxidase is low it means the patient cannot properly break down Histamine. Histamine-N-methyltransferase (HNMT) is the secondary enzyme involved in Histamine break down.

Low Diamine Oxidase is associated with headaches, fatigue, hives, any allergy symptom, dysmenorrhea, estrogen dominance, arrhythmia, inflammation, arthritis, and certain neurologic conditions such as multiple sclerosis. Symptoms of low DAO are essentially identical to symptoms of Histamine excess because they are two sides of the same coin.

Low levels of DAO correlate with poor mucosal integrity and indicate poor gut function. Atrophy of the microvilli can cause low DAO. Patients suffering from diseases like urticaria, Crohn's, or celiac disease are reported to show low DAO activity in serum or plasma. Low DAO can also be a trigge for depression or anxiety. Low Diamine Oxidase in plasma can be used to diagnose Histamine intolerance. Individuals with an inability to break down Histamine may seem to "react to everything," or improve on anti-histamines. Those with anaphylactic reactions often have lower DAO activity. Following a Histamine-free diet can result in a significant reduction, or even disappearance, of symptoms within a few weeks.

Many medications inhibit DAO or damage the gut lining, reducing DAO production. Alcohol and its degradation product, acetaldehyde, are inhibitorsof DAO.

DAO: Histamine Ratio

The DAO: Histamine Ratio helps detect even (insert) subtle imbalances between Histamine and DAO levels. Even if the DAO enzyme level is normal, symptoms can occur when Histamine is high. A low ratio indicates that there may not be enough of the DAO enzyme relative to the amount of Histamine in the body.

Treatments to normalize DAO or Histamine will also improve this ratio.

High LPS

An elevated Lipopolysaccharide (LPS) reaction indicates intestinal permeability or "leaky gut". Lipopolysaccharide is the immunogenic portion, as well as the major constituent, of the outer cell membrane of gram-negative bacteria. LPS is a bacterial endotoxin made by bacteria in the body.

When Lipopolysaccharides are high in the blood, it means they are passing not only between intestinal cells, but also directly through the cells, potentially causing neuroinflammation and brain injury. When LPS is absorbed into the blood stream it can elicit a strong immune response.

Elevated levels may be associated with bacterial infection, food sensitivities, chronic inflammation, autoimmune conditions, digestive disorders, and neurological conditions.

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Low LPS

There is clinical importance to having a low immune reaction to LPS antibodies. Since there will always be some LPS present, there should be an immune response recorded. When a patient tests on the low end of the spectrum for an immune response for LPS IgG, LPS IgA and LPS IgM, this is a good indication that their immune system is not functioning as it should. When there is a low response this means immunoglobulin levels go down and bacterial levels stay up. Ongoing gut pain and flairs persist, as patients can no longer fight infections as they should and the higher level of bacteria in the gut causes irritation.

Conditions associated with low LPS antibodies are IBS, Crohns Disease and Colitis.

Conditions Associated with Elevated Levels of LPS

Shock	Type 2 Diabetes	Obesity
Multiple Organ Dysfunction	Alzheimer's	Mood and Appetite Disorders
	Autoimmunity	
Depression	Infertility	Cognitive Decline
Anxiety	Hypogonadism	Anorexia
Sepsis	Leptin Resistance	Parkinson's
Atherosclerosis	Chronic Constipation	Chronic Pain

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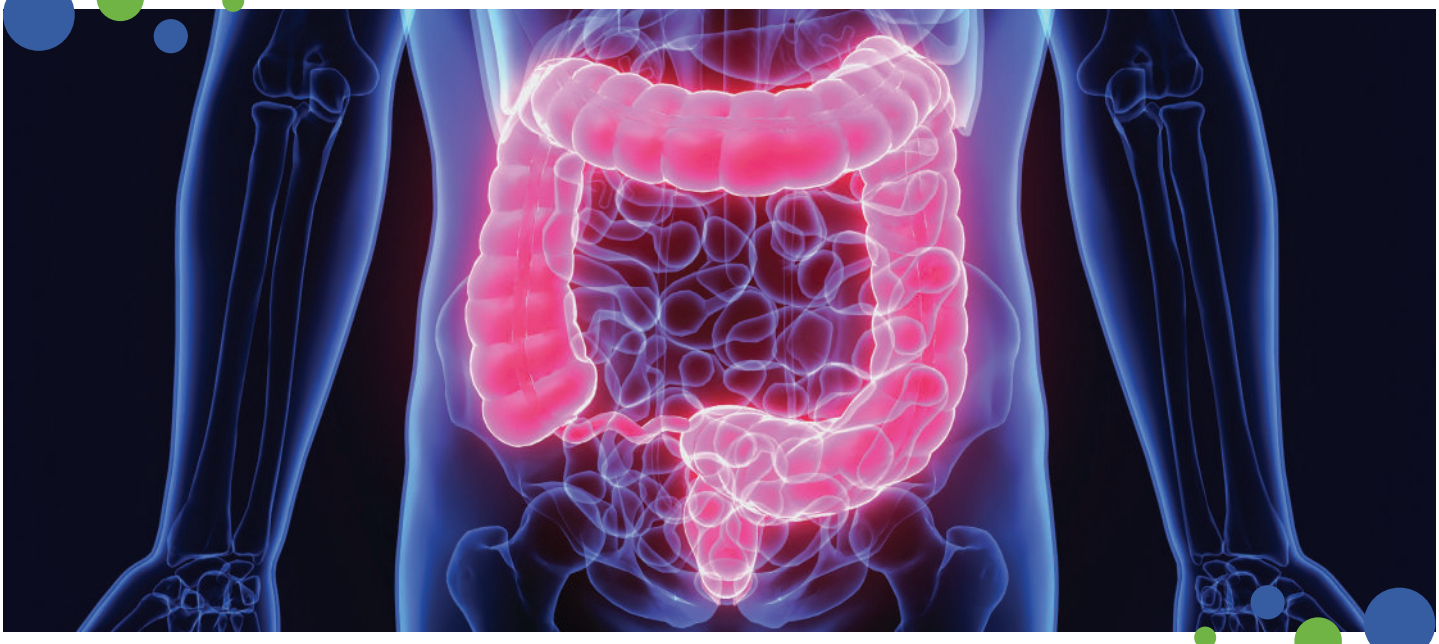
ANALYTES	LOW LEVELS
Zonulin	Low Zonulin is not clinically significant.
DAO	Low DAO is a result of atrophied microvilli demonstrating gut permeability. This will also result in an inability to degrade Histamine creating sensitivity and symptoms associated with histaminosis. See High Histamine for treatment.
Histamine	Low levels can be associated with fatigue, depression and certain types of schizophrenia. Histidine (sp) and accessory amino acids can be given to raise levels.
DAO: Histamine	A high ratio shows that the gut lining is in balance between its ability to make and degrade histamine.
LPS IgA, IgM, IgG	Low LPS Antibodies are associated with an immune system that is chronically worn down. IBS and IBD can both be a result of an infection that was chronic and that has resulted in little to no immune reserve. Immunoglobulins are an excellent intervention. Adequate Vitamin A and D as well as adequate protein can also help to increase levels. Assume there is a long-term infection that has decreased levels and consider antimicrobials such as Berberine and Garlic as well.



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ANALYTES	HIGH LEVELS
Zonulin	Possible bacteria, yeast, gluten. Treatment: • Treat dysbiosis with garlic, oregano, and with berberines from Goldenseal or Oregon grape. • Immunoglobulins sourced from colostrum, egg, or serum because immunoglobulins block Zonulin from binding to tight junctions • Remove wheat/gluten.
DAO	DAO will increase initially to compensate for higher levels of histamine from dysbiosis and/or immune dysregulation of foods. It is a compensatory response due to challenge of Histamine. Treat by lowering Histamine. Higher levels may also just be due to healthy microvilli and a robust production.
Histamine	Increased levels are secondary to antigens causing mast cell degranulation. Also, certain bacteria can create histamine and certain foods are higher in histamine. Treatments to increase DAO: Oral DAO, Omega 3 fatty acids, Vitamin C, Copper and b6 as cofactors, and Sacchromyces. Treatments to lower histamine: Oral DAO, SAMe to increase methylation of Histamine, or B5 to acetylate Histamine. Other therapies for degranulation of mast cells or histamine producing cells include: Quercetin, Vitamin C, and Omega 3 fatty acids. Other therapies to decrease degranulation of mast cells or histamine producing cells include: Quercetin, Vitamin C and Omega 3 fatty acids.
DAO: Histamine	When the ratio is low it means there is not enough Diamine Oxidase to degrade histamine. See Histamine section above for treatment.
LPS IgA, IgM, IgG	This indicates that the immune system is actively fighting bacterial overload. Treatments include antimicrobials to lower bacterial load. Berberine and Garlic are suggested as well as immunoglobulins to suppport the immune system.
LPS High IgM: Low IgG	A high IgM with a Low IgG means there was poor seroconversion to a matured response to LPS. Antimicrobial therapies and immunoglobulins will support improvement in these areas. Toxicity can block seroconversion from IgM to IgG. Detox may be warranted.

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DIAGNOSTICS

REFERENCES

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2. Panula P, Karlstedt K, Sallmen T, et al. The histaminergic system in the brain: structural characteristics and changes in hibernation. Journal of chemical neuroanatomy. Feb 2000;18(1- 2):65-74.
3. Nuutinen S, Panula P. Histamine in neurotransmission and brain diseases. Advances in experimental medicine and biology. 2010;709:95-107.
4. Corazza GR, Falasca A, Strocchi A, Rossi CA, Gasbarrini G. Decreased plasma postheparin diamine oxidase levels in celiac disease. Digestive diseases and sciences. Aug 1988;33(8):956-961.
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Oxidized Low-Density Lipoprotein

A "Targeted" Approach to Wellness

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OXIDIZED LDL Profile

81.78

Normal Range: 11.6-90.5 U/L

NORMAL

Oxidized Low-Density Lipoprotein (Ox LDL)

Over the past few decades, evidence has accumulaterd that establishes oxidized low-density lipoprotien as a useful marker for cardiovascular diseases (CVD), with several research groups demonstrating that the plasma Ox LDL level in patients with CVDs is significantly higher that the levels measured in healthy subjects.[1]

Oxidized LDL can either be measured by itself in the Precision Point Diagnostics Oxidized LDL Profile PP10, or with a comprehensive cardiac provile (Oxidized LDL with Lipids: Profile PP11) that helps assess overall disease risk by including the assessment of lipids, and metabolic and cardiovascular markers related to nutritional and overall health.

1. Hiroyuki Itabe, Takashi Obama, and Rina Koto, "The Dynamics of Oxidized LDL during Atherogensis," Journal of Lipids, vol. 2011

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Advanced Oxidative Stress

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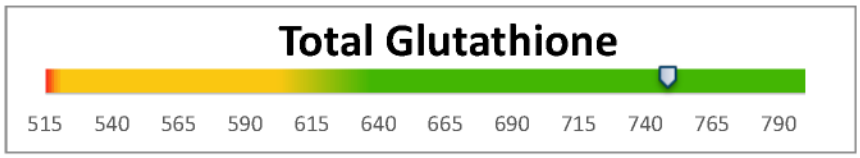
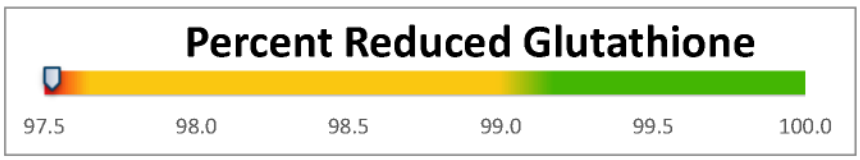
ADVANCED OXIDATIVE STRESS PROFILE

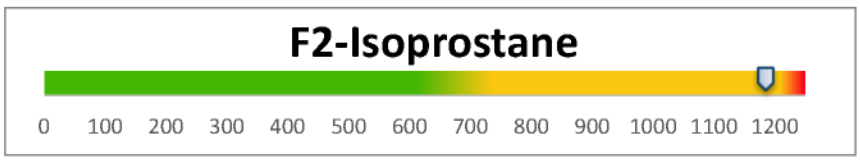
748.5
Normal Range: >613.9 µM
OPTIMAL



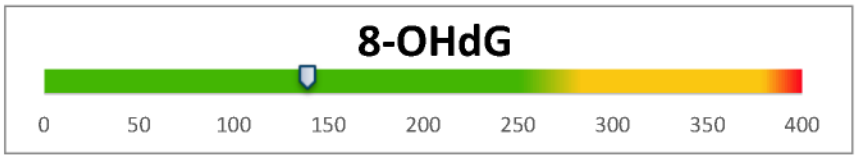
97.5
Normal Range: >99.1%
ABNORMALLY LOW



1185.6
Normal Range: <720.9 pg/mg*
BORDERLINE



139.0
Normal Range: <271.0 pg/mg*
OPTIMAL



100.4
m/dL

*pg/mg of Creatinine

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Lab Director: Steven Lobel, PhD
Analysis performed by:
Dunwoody Labs Inc. DBA, Precision Point Diagnostics

GA Clinical License: 044-160
CLIA ID: 11D1101209

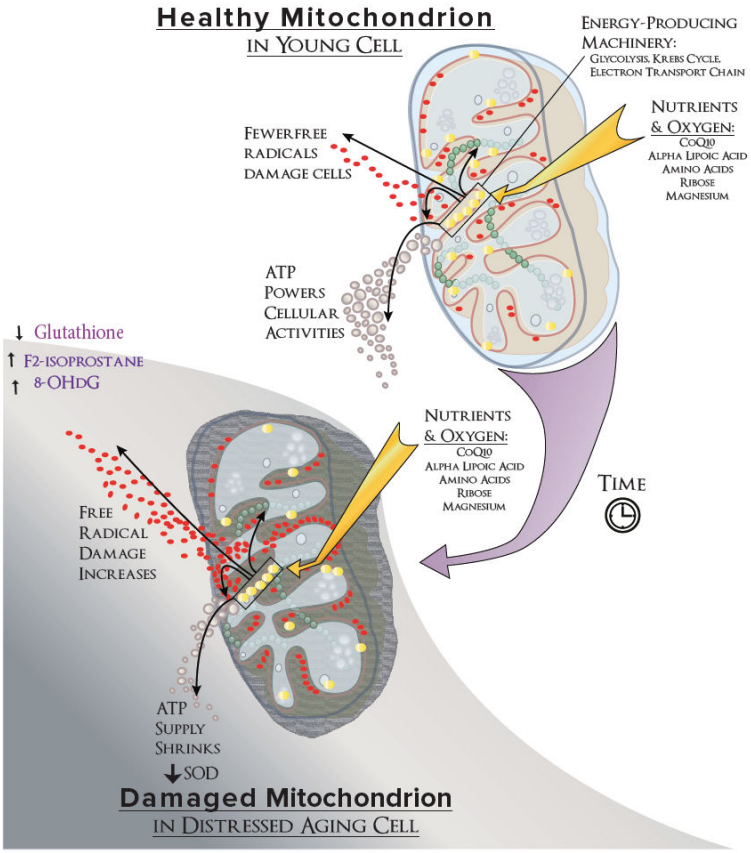
PRECISION POINT
DIAGNOSTICS

ADVANCED OXIDATIVE STRESS TREATMENT GUIDE

The Advanced Oxidative Stress test shows how much oxidative stress there is in the body and how effectively antioxidant enzymes remove it. Reactive oxygen species (ROS, also called free radicals) are produced by virtually every metabolic process in the body and are counterbalanced by the action of antioxidants.

Mitochondria are the main producers of ROS and the main victims of ROS.[1] Two systems are important for proper removal of ROS. One is intracellular reduced glutathione, the primary antioxidant found within cells. The other is the superoxide dismutase (SOD) family, the main enzymes which remove ROS from the cell and mitochondria. When these antioxidants do not function normally, free radicals accumulate, which leads to cellular damage including DNA, protein and lipid membrane dysfunction.

Oxidative stress can increase a person's risk for heart disease,[2] neurologic conditions, [3-6] cancers,[7] or toxicity.[8,9] Oxidative damage to lipids occurs early in the development of atherosclerosis and can lead to cardiovascular and cerebrovascular disease.[10,11] Oxidative damage to mitochondria and DNA are hypothesized to be involved in the pathogenesis of Alzheimer's disease.[12] As seen in Table 1, oxidative stress is thought to contribute to Parkinson's disease, cancer,[13] and autoimmune thyroiditis.[14,15]



Oxidative damage in the central nervous system has been implicated in neuropathological changes. It has been estimated that 3-10% of the oxygen used by tissues is converted into reactive oxygen species, posing a threat to nearby cells and tissues.[16] The nervous system consumes a high amount of oxygen but does not have high antioxidant defenses, making it susceptible to damage by reactive oxygen species and reactive nitrogen species. Both oxidative and nitrosative stress have been implicated in neurodegenerative and neurological disorders[17] as well as traumatic brain injury.[18]

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Glutathione

Glutathione is the most abundant intracellular antioxidant[30] and is critical for defending the cell against oxidative stress. As the name suggests, gamma-L-glutamyl- L-cysteinylglycine (glutathione, GSH) is a tripeptide made from glutamate, cysteine, and glycine. Its sulfur (thiol) group is responsible for its biological activity.

GSH neutralizes free radicals and conjugates toxins for removal from the body, thereby protecting cells from oxidative stress and toxic xenobiotics.[31] GSH is important for mitochondrial function as a redox scavenger.[32] GSH is involved in cell proliferation, apoptosis, autophagy, and gene expression. GSH also is involved in regulation of Nrf2 (See "Nrf2 Activators" in the Treatment Options section).[30]

Cellular Antioxidant Defense Systems

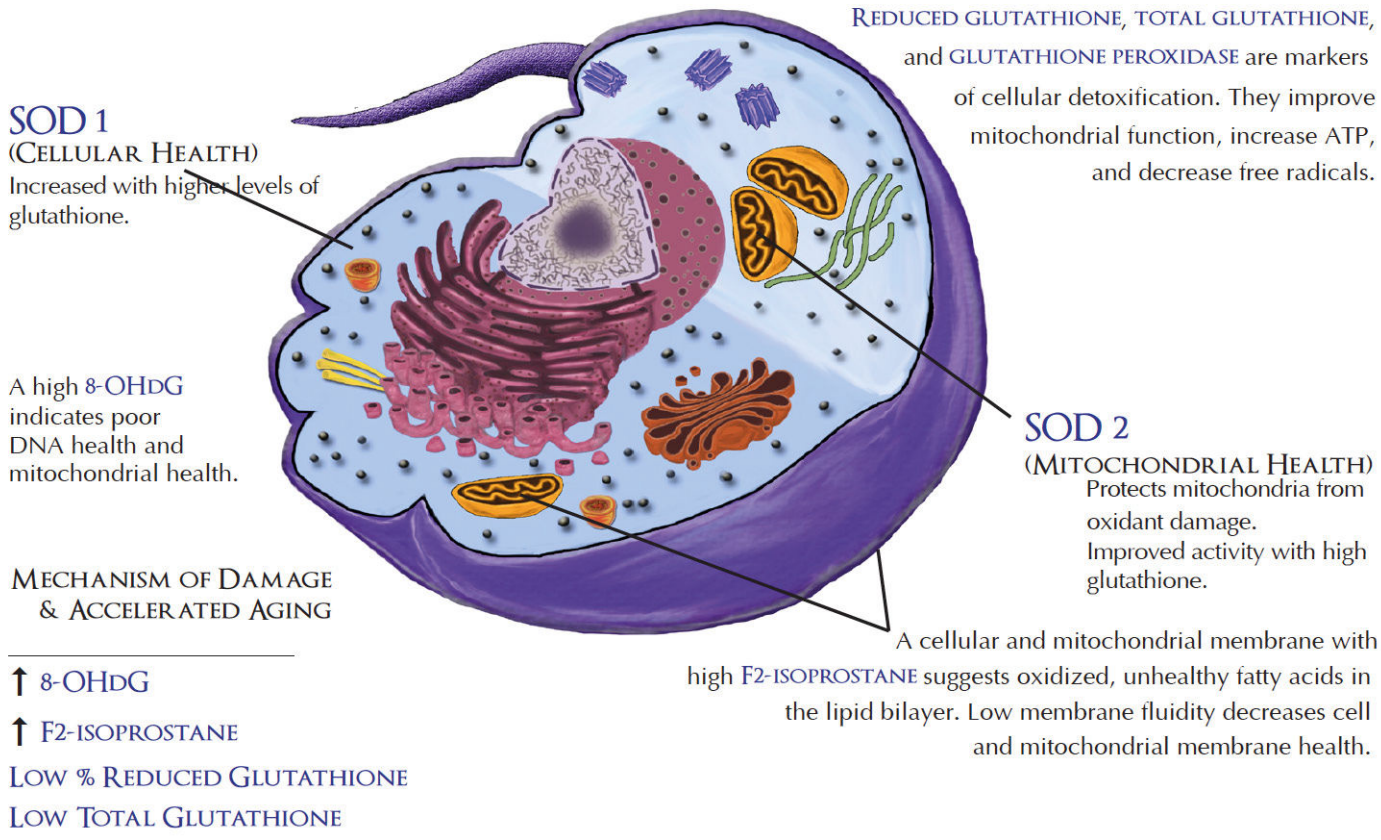

Figure 2. High levels of 8-OHdg, F2-isoprostane, with low glutathione and reduced glutathione indicate cell damage and free radical overload.

PRECISION POINT DIAGNOSTICS

Conditions Associated with Oxidative Stress

Alzheimer's disease[19]	Chronic inflammation[6,20]	Premature labor and stillbirth[20]
Atherosclerosis[6,20]	Diabetes[6,24]	
Autism[21]	Gastrointestinal disorders[25]	Rapid aging[6]
Autoimmune disorders[22]	Hormonal imbalance[26,27]	Schizophrenia[23]
Bipolar disorder[23]	Major depressive disorder[23,28]	Stroke[6,29]
Cancer[20]	Neurodegenerative disorders[6,20,29]	Thyroid dysfunction[14,15]

Glutathione

Total glutathione includes both reduced and oxidized glutathione levels. Glutathione is constantly undergoing oxidation and reduction and therefore exists in two forms, reduced and oxidized. The reduced form of glutathione is the radical scavenger (or antioxidant). Oxidized glutathione is the "used up" form of glutathione (Figure 3). Oxidized glutathione has contributed its antioxidant potential and then must be recycled to the reduced form of glutathione to be useful once again. Total glutathione levels can indicate the body's total reserves of glutathione for removing harmful free radicals and toxins. Total glutathione may be low due to genetic variation in enzymes involved in glutathione production, nutritional insufficiency, or exposure to reactive chemicals or medications.[13,33]

The Advanced Oxidative Stress Test measures these biomarkers from a whole blood and urine specimen:

- Total glutathione (whole blood)
- Percent reduced glutathione (whole blood)
- F2-isoprostane (urine)
- 8-hydroxy-2'-deoxyguanosine (8-OHdG, urine)

Disturbed glutathione levels have been seen in these conditions: 13,23,34

- Aging
- AIDS
- Bipolar disorder
- Cancer
- Cardiovascular disease
- Cystic fibrosis
- Diabetes
- Immune disease
- Inflammatory disease
- Liver disease
- Major depressive disorder
- Metabolic disease
- Neurodegenerative disease
- Schizophrenia

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Glutathione

Reduced glutathione is the active form of glutathione. It is the primary antioxidant needed to remove damaging free radicals from the brain, liver, and lungs. If the levels of reduced glutathione are low, it is difficult for the mitochondria to make cellular energy. Low reduced glutathione indicates poor antioxidant status. Aberrations in glutathione (more oxidized GSH) can lead to hypomethylation because methylation and transsulfuration pathways are interconnected.[35]

Treatments to support glutathione synthesis are amino acids (N-acetylcysteine (NAC), glycine, and glutamate) and selenium. Adjunctive treatments that help raise glutathione levels and support overall detoxification are: taurine, milk thistle, minerals, and the B vitamins (niacin, riboflavin, vitamins B12, B6, and folate). Selenium and alpha-lipoic acid can improve reduced glutathione levels. Remove toxic chemicals or medications that may deplete GSH.

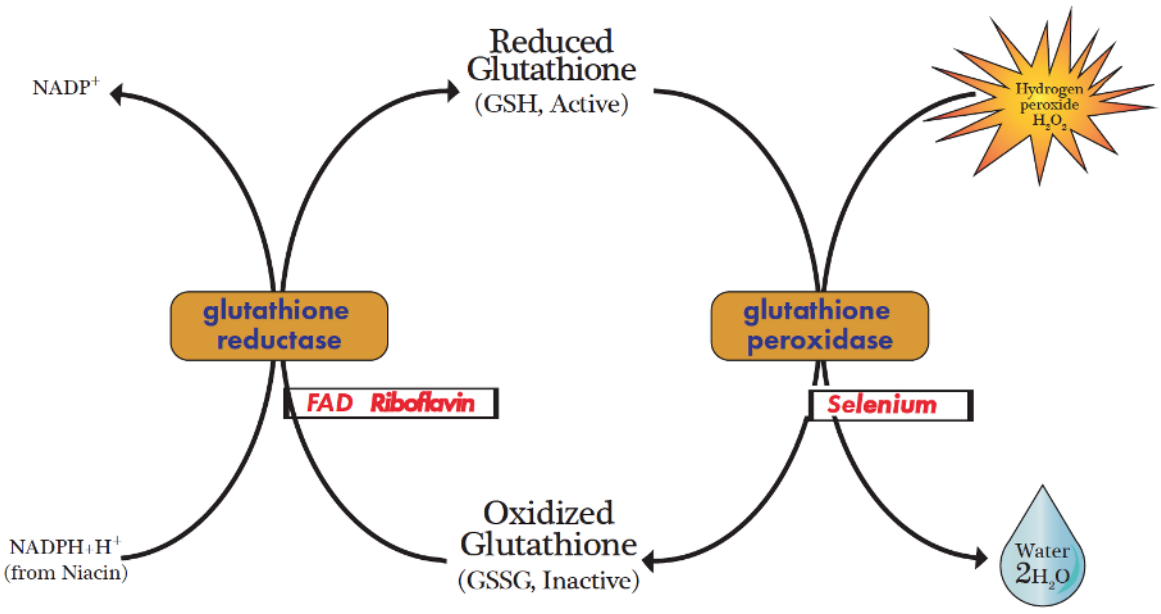

Figure 3. The Glutathione Oxidation-Reduction Cycle. Glutathione is constantly undergoing oxidation and reduction and therefore exists in two forms, reduced (GSH) and oxidized (GSSG). The reduced form of glutathione is the antioxidant. The oxidized form of glutathione is the "used up" form which cannot bind free radicals. Glutathione peroxidase uses the cofactor selenium to convert hydrogen peroxide to water. To recycle glutathione and produce the active, reduced form, glutathione reductase, riboflavin, and niacin are needed.

Low percent reduced glutathione may be associated with: [5,6]

- Anxiety
- Depression
- Fatigue
- Neurological conditions

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F2-Isoprostane

F2–isoprostane (F2-isoprostoglandin, 8-iso-prostoglandin F2α) is the oxidized degradation product of arachidonic acid, a fatty acid with significant inflammatory potential. F2-isoprostane is an oxidized lipid, pro-inflammatory, and a vasoconstrictor.[36] Urinary F2-isoprostane is the gold standard marker of lipid peroxidation in biological specimens[19,37] and has been described as a, "reliable approach to assess oxidative stress."[38]

F2-isoprostane can cause vasoconstriction in the kidneys, lungs, liver, bronchi, blood and lymph vessels, uterus and gastrointestinal tract. F2-isoprostanes are associated with increased perception of pain and are elevated in acutely hyperglycemic diabetics. They are also high in smokers and in autism.

F2-isoprostane has been suggested as an indicator of cardiovascular disease.[2] F2-isoprostanes were higher in patients with coronary artery disease[39] and the levels correlated with the number of lesions as well as the incidence of hypertension.[40] F2-isoprostanes are highly concentrated in atherosclerotic plaques.[41] Plasma F2-isoprostane correlated positively with systolic and diastolic blood pressure, supporting the role of oxidative stress in hypertension.[42]

High levels of F2-isoprostane may be found in:

- Atherosclerosis[41]
- Attention deficit disorders
- Cancer
- Cardiovascular disease[2]
- Depression[2]
- Generalized pain and inflammation
- Neurodegenerative diseases[3]
  - Alzheimer's disease [4,19]
  - Creutzfeldt-Jakob disease (mad cow)[4]
  - Huntington's disease[4]
  - Multiple sclerosis[4]

Treatments to lower F2-isoprostanes, the precursor, arachidonic acid, can be targeted. Clinicians may increase omega-3 fatty acids, decrease dietary arachidonic acid, and increase antioxidants. Lifestyle modifications aimed at decreasing body mass index and smoking cessation can reduce F2-isoprostane.[43] Oral antioxidants lowered isoprostanes in 45% in human intervention studies.[38] Therefore, dietary and supplemental antioxidants should help to lower oxidative stress markers like F2-isoprostane.

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8-OHdG

8-OHdG (8-hydroxy-2'-deoxyguanosine) is an oxidized base that has been identified and removed from the DNA, and excreted in the urine. It is the most studied marker of oxidative damage to DNA. DNA damage is a normal part of life, but chronic elevation can be a concern because it signals increased oxidative damage. While 8-OHdG is not a diagnostic marker of cancer, it has been widely used as a marker of oxidative stress, degenerative diseases, and is considered a risk factor for certain cancers.[7] 8-OHdG is of special biological interest because it induces a transversion of DNA bases guanine to thymine, which is a mutation frequently found in cancers.[44]

Oxidative stress as evidenced by high 8-OHdG has been found in depression, fatigue, diabetes, extreme exercise, neurodegenerative disease, toxicity, and other conditions. High urinary 8-OHdG was found in patients with major depression and chronic fatigue syndrome. There was a correlation between 8-OHdG levels and scores for sadness and flulike malaise.[45]

Extreme sports, medications, and environmental toxins, such as arsenic and mercury, can increase 8-OHdG. After four days of antibiotic therapy, urinary 8-OHdG dramatically increased the following: ciprofloxan (100%), tetracycline (230%), kanamycin (400%), and ampicillin (720%).[46] Antibiotic therapy also increased other markers of oxidative damage and decreased ATP production. In-house clinical observations show that chemotherapy increases 8-OHdG as a normal consequence of treatment.

DNA damage has been shown to be involved in the pathogenesis of Alzheimer's disease.[47,48] High 8-OHdG was detected in the cerebrospinal fluid of patients with Alzheimer's and is correlated with the duration of illness.[12] In Alzheimer's patients, astrocytes from the hippocampus and cerebral cortex had increased levels of double-stranded DNA breaks and there was increased 8-OHdG within cerebral tissue.[49,50]

Elevations of urinary 8-OHdG have been seen in:[8]

- Alzheimer's Disease
- Arsenic Poisoning
- Bladder Cancer
- Cardiomyopathy
- Cigarette Smoking
- Chemical Exposure
- Chronic Obstrutive
- Depression
- Diabetes
- Parkinson's Disease
- Diabetic Nephropathy
- Exposure to Ultraviolet Light
- Extreme Exercise
- Fatigue
- Hemodialysis
- Insomnia
- Neurodegenerative Diseases
- Pulmonary Disease

Treatments to decrease urinary 8-OHdG include water-soluble antioxidants, as DNA resides in a water soluble compartment of the cell. Vitamin C and plant-derived antioxidants, such as flavonoids and polyphenols, help to lower 8-OHdG. Other treatments to reduce oxidant stress, avoid toxins and optimize liver function can also decrease 8-OHdG.

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Oxidant Stress Treatment Options

Oxidative stress can cause high or low levels of an antioxidant enzyme. At the initial onset of oxidative stress, the body produces more of the enzyme to cope with the free radicals. However, with chronic oxidative stress, enzyme levels are depleted and cannot respond properly to the oxidant challenge. Toxins can also deplete antioxidant enzymes. Therefore, treatment options are the same for both high and low levels of GSH. In contrast, F2-isoprostanes and 8-OHdg are elevated in cases of oxidative stress. Generally, treatments include nutritional support for detoxification, stimulation of Nrf2-ARE pathway, antioxidants, minerals, adjunctive nutritional treatments, and elimination of toxins, infection, and environmental sources of oxidative stress. Females may have higher needs for antioxidants.[27]

Biomarker	Increase Antioxidant Defenses	Reduce Oxidative Stressors
Total GSH	Se, B vitamins, NAC, ALA, amino acids, taurine, GSH, SAG, broccoli seed extract or glucaphoranin, turmeric, green tea, pterostilbene, black pepper, resveratrol	Avoid toxins, infections, and unnecessary medications
Reduced GSH	ALA, B vitamins, Se, Nrf2 activators, glucaphoranin, SAG, vitamins A, E, D and CoQ10	Avoid toxins, infections, and unnecessary medications
F2-isoprostane	Omega-3 fatty acids, SAG, vitamins A, E, D, CoQ10, dietary antioxidants, reduce BMI	Reduce arachidonic acid, stop smoking
8-OHdG	Vitamin C, flavonoids, water-soluble phytonutrients, green tea, broccoli seed extract or glucaphoranin, turmeric, pterostilbene, black pepper, resveratrol, quercetin	Avoid toxins, infections, and unnecessary medications

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Sulfur amino acids and nutritional support for detoxification

Amino acids are necessary for enzyme synthesis, glutathione synthesis and phase II detoxification. Free form amino acids such as cysteine, glycine, and taurine increase glutathione and aid in detoxification. Taurine also aids in detoxification.

S-acetyl glutathione (SAG) can be used to increase glutathione levels (200 to 400 mg, once or twice daily).

N-acety-L-cysteine (NAC) is a derivative of the dietary amino acid, L-cysteine. It is a powerful free radical scavenger and thus supports the body's natural defense system. NAC provides cysteine, the major precursor in the biosynthesis of glutathione.

NAC has the ability to chelate heavy metals[87] and has shown efficacy in treating psychiatric disorders.[88] Two to four grams of NAC each day can provide the building blocks for glutathione.

Medical foods for detoxification, together with a modified elimination diet, help to remove toxins and quench oxidative stress. These typically consist of 21 grams of protein, 2.5 grams of immunoglobulins, 1 gram of arabinogalactan (a prebiotic), and 30 mg of broccoli seed extract (as glucoraphanin) per day.

Milk thistle extract or silymarin from milk thistle (260 – 525 mg) and alpha-lipoic acid (200 to 400 mg) help with liver detoxification. Nutrients to support phase I and phase II detoxification can be taken twice daily: pomegranate extract (75 mg), green tea extract (95 mg), watercress powder (250 mg), methylsulfonylmethane (MSM, 50 mg), and calcium D-glucarate (200 mg/day, especially to detoxify hormones).[89]

Nrf2 Activators

Nrf2 has been called the "master regulator" of the antioxidant response.[90] Nrf2 is a transcription factor that regulates the expression of antioxidants and anti-inflammatory genes through the human Antioxidant Response Element (ARE). The Nrf2-ARE signaling pathway is the cell's defense against oxidative stress and it is triggered when free radicals increase and/or when there is reduced antioxidant capacity.[91] Activation of the Nrf2-ARE pathway initiates the expression of genes that make proteins involved in glutathione synthesis, Phase II detoxification, drug transport, removal of ROS, reduction of inflammation, and protection from ischemia/reperfusion injury.[90]

The Nrf2-ARE signaling pathway upregulates SOD, GSH, and GPx, among others.[92] For these reasons, targeting the Nrf2- ARE pathway with nutrients is a popular strategy to promote a person's antioxidant defenses. Sulforaphane and curcumin can activate the Nrf2-ARE pathway[90] while sulforaphane increases SOD, GSH, and GPx.[93] Broccoli extract is a source of sulforaphane. Broccoli seed extract or glucoraphanin (also known as sulforaphane glucosinolate or SGS) can increase glutathione levels.

Green tea extract, trans-pterostilbene (the primary antioxidant in blueberries), and black pepper extract work together with broccoli extract and turmeric to activate the Nrf2 genetic pathway. These ingredients also activate glutathione- S transferase and down-regulate inflammatory factors such as nuclear factor kappa B (NF-kB). Typical daily doses are: 60 to 120 mg of broccoli extract, 400 to 800 mg of turmeric extract, 400 to 800 mg of green tea extract, 100 to 200 mg of transpterostilbene, and 4 to 8 mg of black pepper extract.

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Antioxidants

Water-soluble and fat-soluble antioxidants can target both compartments of the cell. Water-soluble antioxidants include vitamin C (1-4 g/d), green tea extract,[94] quercetin (250 to 500 mg/d) and resveratrol (75 to 150 mg/d). A diet high in colorful, organic fruits and vegetables is high in antioxidants, polyphenols and flavonoids. Green tea treatment of diabetic rats with nephropathy reduced oxidative stress markers, including 8-OHdG.[94] Resveratrol, quercetin, and pterostilbene work synergistically to reduce toxicity or infection that elevates MnSOD (SOD2).

ALA helps to neutralize free radicals in both the water-based and lipid-based portion of cells, synthesize glutathione, and recharge important antioxidants. It decreases oxidative stress and increases MnSOD activity and glutathione activity in mitochondria.[95] The commonly given dose of ALA is 300 mg to 600 mg to support antioxidant activity and liver function. Also consider vitamins A, E, D and CoQ10 to deliver antioxidants to the fat-soluble portion of the cell.

Omega-3 Fatty Acids

Healthy fats are needed when F2-isoprostanes are elevated. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), taken with vitamin E as mixed tocopherols, reduce the rate of lipid peroxide formation in cells. One to two grams of EPA, 600 to 1500 mg of DHA, and 20 – 40 IU of mixed tocopherol vitamin E can be taken one to three times daily for cardiovascular health, nervous system function, and joint tissue health.

Minerals

Selenium, copper, zinc, manganese, iodine, and iron are the cofactors for many antioxidant enzymes. Selenium protects the thyroid from radical damage, presumably through its role in GPx.63 Copper, manganese, and zinc are important cofactors for SOD enzymes. Selenium is a cofactor for GPx. Chronic use of proton pump inhibitors or a nutritionally poor diet will decrease mineral status. Typical dosages are: selenium (100-200 mcg/d), copper (2-10 mg/d), zinc (15-50 mg/d with food), manganese (5-13 mg/d), iodine (150 mcg-6 mg/d), and iron (15-50 mg/d).

Lifestyle Modifications

Environmental stresses, such as chemical toxins, endocrine disruptors, heavy metals, and infectious agents can cause oxidative damage. Detoxification of the body is additionally supported with an anti-inflammatory diet free of allergens such as gluten, dairy, eggs, caffeine, nicotine, artificial sugars and flavors, and stimulants. In addition, it is important to foster good mental hygiene with healthy sleeping habits, moderate exercise, and stress reduction techniques to minimize mental and emotional stressors.

Other testing to consider when a patient shows high oxidative stress:

- Urinary or plasma amino acids: Amino acids are necessary to produce glutathione and are important for phase II detoxification.
- Red cell or urinary minerals: Minerals are important cofactors in antioxidant enzymes.
- Plasma neurotransmitters: Oxidative stress can lead to neurotransmitter imbalances, receptor damage, and ultimately cause neurological disorders.
- Gastrointestinal tract (intestinal permeability, stool testing, food sensitivities): In a patient with chronic oxidative stress, gastrointestinal infection or inflammation may be the underlying cause.
- Serum CoQ10 and serum vitamins A, E, D: CoQ10 and vitamin E are important fat-soluble antioxidants.
- Methylation: Imbalances in glutathione may interfere with methylation pathways.

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FINAL REPORT

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RESEARCH & DEVELOPMENT 9 Dunwoody park Dunwoody, ga 30338 **Acct:** 2724 **Phone:** (123) 123-1234 **Phys:**	**DOE, JOHN** **DOB:** 12/08/1991 **Age:** 29 **Sex:** M **ID#:** **Address:** 9 DUNWOODY PARK DUNWOODY, GA 30338 **Phone:** (678) 736-6374	**Accession #: 2104210008** **Specimen ID:** **Date Collected:** 04/21/21 11:27 **Date Received:** 04/21/21 11:27 **Date of Report:** 04/29/21 **First Reported:** 04/22/21

CLINICAL REPORT

Comments:

Fasting: NOT PROVIDED

Test	Result	Abnormal	Reference	Units	Previous Result	Date	
			reported: 04/22/21 08:13				
Hemoglobin A1c	5.3		4.8 - 5.6	%			*1

.

Prediabetes: 5.7 - 6.4
Diabetes: >6.4
Glycemic control for adults with diabetes: <7.0

			reported: 04/22/21 08:13				
Vitamin D, 25-Hydroxy	29.3 L		30.0 - 100.0	ng/mL			*1

Vitamin D deficiency has been defined by the Institute of Medicine and an Endocrine Society practice guideline as a level of serum 25-OH vitamin D less than 20 ng/mL (1,2). The Endocrine Society went on to further define vitamin D insufficiency as a level between 21 and 29 ng/mL (2).

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			reported: 04/22/21 08:13				
C-Reactive Protein, Cardiac	1.16		0.00 - 3.00	mg/L			*1

Relative Risk for Future Cardiovascular Event

Low	<1.00
Average	1.00 - 3.00
High	>3.00

END OF REPORT

1) Unless otherwise noted, Tests Performed at :
Labcorp, Birmingham, AL 35233 CLIA# 01D0301471

Laboratory Director: Dr. Steven Lobell