Sample Reprort

FINAL REPORT

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Specimen Information (+ Redraw sample)

Sample Type	Collection Time	Received Time	Report	Final Report Date
Saliva	Local 2026-05-06 11:11 Local 2026-06-08 10:25 +	2026-05-15 02:05 (+04) 2026-06-13 00:41 (+04) +	Toxin Genetics - P2	2026-06-17 04:02 (+04)

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Toxin Genetics

INTRODUCTION

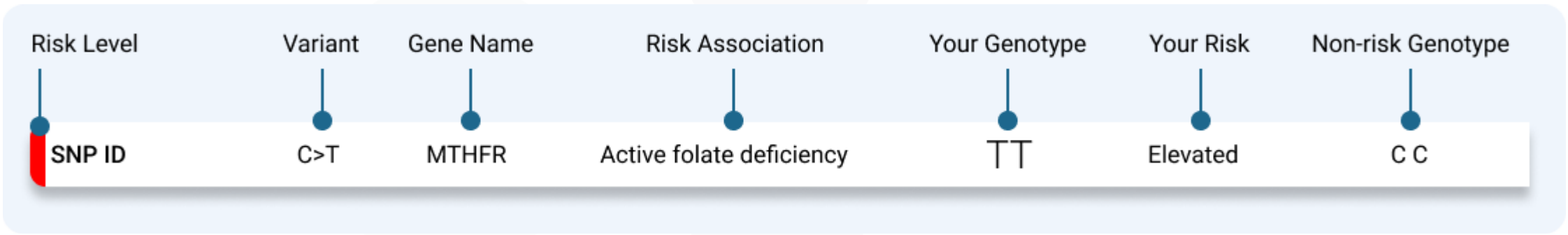
Sample Reprort Wellness is pleased to present Toxin Genetics to support healthy lifestyle choices in consultation with your healthcare provider. The report identifies genetic variants associated with detoxification pathways and is meant to be interpreted alongside complementary Sample Reprort Wellness Zoomer testing. This test is intended to be used by healthcare providers to guide personalized wellness strategies based on genetic predispositions that may influence current biological status.

Methodology

The Toxin 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 genetic variants in the report are organized as multiple tables under different subheadings for associated markers. The summary page lists the set of analytes with risk associated variants. Following this section is the complete list of all genetic markers measured in the panel. The results of the genetic testing will be listed in the format in the image below. The “Risk Level” is a visual representation of risk associated with the genotype result, where red indicates an “Elevated risk” genotype, and yellow indicates a “Partially Elevated” risk genotype. The “SNP ID” identifies a defined location in the DNA where genetic variation can occur between individuals. The “Variant” describes the genetic change at that location. For example, “C>T” means that at this location in the DNA, the nucleotide cytosine (C) may be replaced by thymine (T). The “Gene Name” refers to the gene impacted by or associated with that SNP ID. The “Risk Association” describes how genetic variation at that location may impact some biochemical pathway, physiological process, and/or health state. "Your Genotype” refers to the specific nucleotide combination identified to be carried by the patient being tested. “Your Risk” indicates whether the patient’s genotype is associated with an elevated, partially elevated, or normal risk for the defined Risk Association. The level of risk will vary based on the findings from respective scientific studies. The “Non-risk Genotype” refers to the nucleotide combination associated with normal risk.



Please note: It is important that you discuss any modifications to your diet, exercise, drug, and/or nutritional supplementation with your healthcare provider before making any changes. Not all recommended supplements are appropriate in all individual cases. Pediatric risk interpretation for the reported variants has not been established. For each SNP ID, this test detects only the specified nucleotides under Variant (ex. C or T) and does not test for other possible nucleotides at that location.

Toxin Genetics - Summary

Xenobiotics

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1056836	C>G	CYP1B1	Poor xenobiotic detoxification	C/G	Elevated	C/C

The CYP1B1 gene encodes a member of the cytochrome P450 superfamily, primarily located in the endoplasmic reticulum of liver tissue. It plays a key role in phase I metabolism, converting xenobiotics such as pesticides (Diazinon and Malathion) and persistent organic pollutants (POPs), including perfluorinated compounds (PFAS) and polychlorinated biphenyls (PCBs), from small, hydrophobic, lipophilic molecules into more water-soluble compounds for elimination. Mutations in the CYP1B1 gene can impair this metabolic process, reducing detoxification efficiency and increasing susceptibility to the toxic effects of pesticides and POPs. Individuals with CG genotype may experience impaired phase 1 metabolism, which can impact the body's detoxification of toxins, including pesticides and POPs. Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. These vegetables have been shown to enhance the activity of CYP1B1. Additionally, the inclusion of supplements such as indole-3-carbinol, black tea, soybeans, green tea, curcumin, resveratrol, garlic, fish oil, rosemary, and astaxanthin can contribute to an increase in CYP1B1 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs1871042	C>T	GSTP1	Poor xenobiotic detoxification	C/T	Elevated	C/C

The GSTP1 gene encodes glutathione S-transferase P (GSTP1), a cytosolic detoxification enzyme. GSTP1 plays a key role in phase II xenobiotic metabolism by conjugating glutathione with hydrophobic and electrophilic substrates. This process converts toxic compounds into more water-soluble forms, facilitating their excretion from the body. Mutations in the GSTP1 gene can reduce enzymatic activity, impairing phase II detoxification and leading to the accumulation of toxins in the body.Individuals with CT genotype impaired phase II xenobiotic detoxification and catalytic activity. This leads to the accumulation of toxins in the body.Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. Additionally, the inclusion of supplements such as isothiocyanates, allium, resveratrol, magnesium, selenium, and curcumin can contribute to an increase in GSTP1 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs713041	T>C	GPx4	Poor xenobiotic and heavy metal detoxification	C/T	Partially elevated	C/C

The GPX4 gene, encoding glutathione peroxidase 4, produces a key antioxidant enzyme that protects cells by reducing lipid hydroperoxides and maintaining redox homeostasis. Although GPX4 does not directly participate in xenobiotic detoxification via glutathione conjugation—a role primarily performed by glutathione S-transferases (GSTs)—it indirectly supports detoxification by preserving reduced glutathione (GSH), essential for other detoxifying enzymes. Variants in the GPX4 gene, such as rs713041, can reduce GPX4 expression and enzyme activity, impairing cellular antioxidant defenses, disrupting glutathione-dependent detoxification, and promoting the accumulation of toxicants, including cadmium, particularly under additional stressors like tobacco smoke exposure.Individuals with TC genotype and lower GPx enzyme activity have impaired xenobiotic detoxification. Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. Additionally, the inclusion of supplements such as oral magnesium, selenium, curcuminoids, silymarin, folic acid, and alpha lipoic acid can contribute to an increase in GPx4 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Toxin Genetics - Summary

Environmental Toxins

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1051741	C>T	EPHX1	Accumulation of benzene	C/T	Elevated	C/C

The EPHX1 gene encodes microsomal epoxide hydrolase (EH), an enzyme critical for detoxification processes. Epoxides, reactive intermediates in benzene metabolism, can bind to cellular macromolecules such as DNA and proteins, producing compounds that cause cellular toxicity or genetic alterations. EH catalyzes the hydrolysis of these reactive epoxides into more water-soluble derivatives, thereby reducing their toxicity. Consequently, EH plays a vital role in the detoxification of benzene metabolites. Mutations in the EPHX1 gene can impair EH enzymatic activity, disrupt benzene detoxification, promote benzene accumulation, and increase susceptibility to chronic benzene poisoning (CBP).Individuals with CT genotype experience disrupted benzene detoxification, leading to the accumulation of benzene in the body. This leads to an increased susceptibility to chronic benzene poisoning (CBP).Individuals who are more prone to susceptibility may find it advantageous to incorporating diet rich in omega 3 fatty acids. Additionally, the inclusion of milk fat globule membrane (MFGM) and ethanol extract of Panax ginseng C. A. Meyer supplements can contribute to an increase in EPHX1 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs1902023	A>C	UGT2B15	Higher urinary levels of parabens	A/C	Elevated	C/C

The UGT2B15 gene encodes an enzyme UDP-glucuronosyltransferase 2B15 involved in the metabolism and elimination of xenobiotic toxins such as bisphenol A (BPA). The enzyme is responsible for glucuronidation, a phase II metabolic reaction that transforms small lipophilic molecules into water-soluble excretable metabolites. Mutation in the UGT2B15*2 liver enzyme can result in decreased enzymatic function, reduced drug metabolism, and unexpected drug serum levels due to lower clearance rates. This can result in increased BPA levels in the body and increased susceptibility to BPA toxicity. Individuals with AC genotype exhibit decreased systemic clearance of parabens leading to its higher urinary levels. This might increase the risk of parabens toxicity.Individuals who are more prone to susceptibility may find it advantageous to follow a basal diet supplemented with cruciferous vegetables (broccoli, cabbage, and daikon radish sprouts), soy foods (soy milk, veggies slices, tofu, and roasted soy nuts), and citrus fruits (grapefruit and orange juices, orange/grapefruit segments, and dried orange peel). Additionally, the inclusion of supplements such as curcumin and ellagic acid can contribute to an increase in UGT2B15 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs1695	A>G	GSTP1	Poor xenobiotic and heavy metal detoxification	A/G	Elevated	A/A

The GSTP1 gene encodes a cytosolic detoxifying enzyme known as glutathione S-transferase (GST). GST enzymes play a vital role in phase II metabolism of xenobiotics, including benzidine, styrene, arsenic, cadmium, mercury, and pesticides. They catalyze the conjugation of glutathione to electrophilic centers on toxic compounds, making them more water-soluble and facilitating their excretion from the body. Mutations in the GSTP1 gene can reduce GST enzyme activity, impairing phase II detoxification and catalytic efficiency. This may lead to the accumulation of toxins, increasing susceptibility to xenobiotic-induced toxicity.Individuals with AG genotype with lower GST enzyme activity have impaired phase II phase II detoxification of xenobiotics such as benzene, styrene, arsenic, cadmium, mercury, and pesticides. This leads to their accumulation in the body.Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. Additionally, the inclusion of supplements such as isothiocyanates, allium, resveratrol, magnesium, selenium, and curcumin can contribute to an increase in GSTP1 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Toxin Genetics - Summary

Heavy Metals

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1695	A>G	GSTP1	Poor xenobiotic and heavy metal detoxification	A/G	Elevated	A/A

The GSTP1 gene encodes a cytosolic detoxifying enzyme known as glutathione S-transferase (GST). GST enzymes play a vital role in phase II metabolism of xenobiotics, including benzidine, styrene, arsenic, cadmium, mercury, and pesticides. They catalyze the conjugation of glutathione to electrophilic centers on toxic compounds, making them more water-soluble and facilitating their excretion from the body. Mutations in the GSTP1 gene can reduce GST enzyme activity, impairing phase II detoxification and catalytic efficiency. This may lead to the accumulation of toxins, increasing susceptibility to xenobiotic-induced toxicity.Individuals with AG genotype with lower GST enzyme activity have impaired phase II phase II detoxification of xenobiotics such as benzene, styrene, arsenic, cadmium, mercury, and pesticides. This leads to their accumulation in the body.Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. Additionally, the inclusion of supplements such as isothiocyanates, allium, resveratrol, magnesium, selenium, and curcumin can contribute to an increase in GSTP1 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs713041	T>C	GPx4	Poor xenobiotic and heavy metal detoxification	C/T	Partially elevated	C/C

The GPX4 gene, encoding glutathione peroxidase 4, produces a key antioxidant enzyme that protects cells by reducing lipid hydroperoxides and maintaining redox homeostasis. Although GPX4 does not directly participate in xenobiotic detoxification via glutathione conjugation—a role primarily performed by glutathione S-transferases (GSTs)—it indirectly supports detoxification by preserving reduced glutathione (GSH), essential for other detoxifying enzymes. Variants in the GPX4 gene, such as rs713041, can reduce GPX4 expression and enzyme activity, impairing cellular antioxidant defenses, disrupting glutathione-dependent detoxification, and promoting the accumulation of toxicants, including cadmium, particularly under additional stressors like tobacco smoke exposure.Individuals with TC genotype and lower GPx enzyme activity have impaired xenobiotic detoxification. Individuals who are more prone to susceptibility may find it advantageous to incorporate cruciferous vegetables into their diet. Additionally, the inclusion of supplements such as oral magnesium, selenium, curcuminoids, silymarin, folic acid, and alpha lipoic acid can contribute to an increase in GPx4 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Mycotoxins

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs3734091	A>C	XRCC4	Aflatoxin B1-mediated DNA damage	C/C	Elevated	A/A

The XRCC4 gene encodes a protein essential for DNA ligation and the repair of DNA double-strand breaks (DSBs). Polymorphisms in XRCC4 can lead to dysfunction of the protein, impairing its DNA repair capabilities. This results in the accumulation of DNA damage and, in severe cases, increases the risk of tumor development. Aflatoxin B1 (AFB1) is a carcinogen that induces various types of DNA damage, including double-strand breaks, base modifications, and oxidative lesions. XRCC4 polymorphisms or dysfunction compromise DNA repair, allowing DNA lesions to accumulate and heightening susceptibility to AFB1-mediated genotoxic effects.Individuals with CC genotype have down-regulated XRCC4 expression leading to the increased risk of AFB1-mediated DNA damage.Individuals who are more prone to susceptibility may find it advantageous to incorporate supplements such as ginger, turmeric, curcumin, thyme oil, clove, quercetin, garlic, lycopene, and cyanidin can contribute to an increase in XRCC4 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Toxin Genetics - Summary

Mycotoxins

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs861539	G>A	XRCC3	Aflatoxin B1-mediated DNA damage	A/G	Partially elevated	G/G

The XRCC3 gene encodes a protein that helps maintain chromosome stability and repair DNA damage. Polymorphisms in XRCC3 can lead to dysfunction of the protein, impairing its DNA repair capabilities. This results in the accumulation of DNA damage and, in severe cases, increases the risk of tumor development. Aflatoxin B1 (AFB1) is a carcinogen that can induce various types of DNA damage, including double-strand breaks, base modifications, and oxidative lesions. XRCC3 polymorphisms or dysfunction compromise DNA repair, allowing DNA lesions to accumulate and heightening susceptibility to AFB1-mediated genotoxic effects.Individuals with GA genotype have down-regulated XRCC3 expression leading to the increased risk of AFB1-mediated DNA damage. Individuals who are more prone to susceptibility may find it advantageous to incorporate supplements such as ginger, turmeric, curcumin, thyme oil, clove, quercetin, garlic, lycopene, and cyanidin can contribute to an increase in XRCC3 activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs13181	T>G	XPD	Aflatoxin B1-mediated DNA damage	G/T	Partially elevated	T/T

The XPD gene encodes a protein involved in gene transcription and the repair of damaged DNA. Polymorphisms in this gene may lead to XPD dysfunction, compromising its DNA repair capabilities. This can result in DNA damage and, in severe cases, an increased risk of tumor development. Aflatoxin B1 (AFB1) is a carcinogen that can induce various types of DNA damage, including double-strand breaks (DSBs), base damage, and oxidative lesions. XPD polymorphisms or dysfunction can impair DNA repair, leading to the accumulation of DNA lesions and elevating the risk of AFB1-mediated DNA damage.Individuals with TG genotype have down-regulated XPD expression leading to the increased risk of AFB1-mediated DNA damage. Individuals who are more prone to susceptibility may find it advantageous to incorporate supplements such as ginger, turmeric, curcumin, thyme oil, clove, quercetin, garlic, lycopene, and cyanidin can contribute to an increase in XPD activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

rs2228001	G>T	XPC	Aflatoxin B1-mediated DNA damage	G/T	Partially elevated	T/T

The XPC gene encodes a protein that is essential for DNA damage recognition and binding, playing a critical role in DNA repair mechanisms. Polymorphisms in this gene may result in XPC dysfunction, impairing its DNA repair capabilities. This can lead to DNA damage and, in severe cases, an increased risk of tumor development. Aflatoxin B1 (AFB1) is a carcinogen that can induce various types of DNA damage, including double-strand breaks (DSBs), base damage, and oxidative lesions. XPC polymorphisms or dysfunction can compromise DNA repair, causing the accumulation of DNA lesions and increasing the risk of AFB1-mediated DNA damage.Individuals with TG genotype have down-regulated XPC expression leading to the increased risk of AFB1-mediated DNA damage. Individuals who are more prone to susceptibility may find it advantageous to incorporate supplements such as ginger, turmeric, curcumin, thyme oil, clove, quercetin, garlic, lycopene, and cyanidin can contribute to an increase in XPC activity, aiding in the detoxification of toxins. It is also advisable to engage in fasting and regular exercise as part of a recommended regimen for enhancing detoxification processes.

Patient Name:Sample Reprort
Date of Birth: Sample ReprortAccession ID: 2605066012
Service Date: 2026-05-06 22:11 (+04)

Toxin Genetics -All Markers

Xenobiotics

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1042157	C>T	SULT1A1	Poor xenobiotic detoxification	C/C	Normal	C/C
rs762551	C>A	CYP1A2	Poor xenobiotic detoxification	A/C	Normal	C/C, A/C
rs1048943	A>G	CYP1A1	Poor xenobiotic detoxification	A/A	Normal	A/A
rs1056836	C>G	CYP1B1	Poor xenobiotic detoxification	C/G	Elevated	C/C
rs1871042	C>T	GSTP1	Poor xenobiotic detoxification	C/T	Elevated	C/C
rs713041	T>C	GPx4	Poor xenobiotic and heavy metal detoxification	C/T	Partially elevated	C/C
rs4680	G>A	COMT	Poor xenobiotic detoxification	G/G	Normal	G/G

Environmental Toxins

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2234922	A>G	EPHX1	Accumulation of benzene	A/G	Normal	G/G, A/G
rs1051741	C>T	EPHX1	Accumulation of benzene	C/T	Elevated	C/C
rs751141	G>A	EPHX2	Accumulation of benzene	G/G	Normal	G/G
rs1902023	A>C	UGT2B15	Higher urinary levels of parabens	A/C	Elevated	C/C
rs1695	A>G	GSTP1	Poor xenobiotic and heavy metal detoxification	A/G	Elevated	A/A
rs1138272	C>T	GSTP1	Poor xenobiotic and heavy metal detoxification	C/C	Normal	C/C

Heavy Metals

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs11076161	A>G	MT1A	Lower Cd levels in blood	G/G	Normal	G/G, A/G
rs1695	A>G	GSTP1	Poor xenobiotic and heavy metal detoxification	A/G	Elevated	A/A
rs1138272	C>T	GSTP1	Poor xenobiotic and heavy metal detoxification	C/C	Normal	C/C
rs713041	T>C	GPx4	Poor xenobiotic and heavy metal detoxification	C/T	Partially elevated	C/C
rs1050450	C>T	GPx1	Poor xenobiotic detoxification	C/C	Normal	C/C

Toxin Genetics -All Markers

Mycotoxins

Test Name	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2056131	A>G	ITGB3	Increased mold spore-triggered allergic reactions	A/G	Normal	A/A, A/G
rs28383151	G>A	XRCC4	Aflatoxin B1-mediated DNA damage	G/G	Normal	G/G
rs3734091	A>C	XRCC4	Aflatoxin B1-mediated DNA damage	C/C	Elevated	A/A
rs25487	T>C	XRCC1	Aflatoxin B1-mediated DNA damage	C/C	Normal	C/C
rs861539	G>A	XRCC3	Aflatoxin B1-mediated DNA damage	A/G	Partially elevated	G/G
rs7003908	C>A	XRCC7	Aflatoxin B1-mediated DNA damage	A/A	Normal	A/A
rs13181	T>G	XPD	Aflatoxin B1-mediated DNA damage	G/T	Partially elevated	T/T
rs2228001	G>T	XPC	Aflatoxin B1-mediated DNA damage	G/T	Partially elevated	T/T

Risk and Limitations

Test results reflect biological and analytical findings at the time of specimen collection and may vary between individuals. Reference ranges for most of laboratory-developed tests (LDT) were established using a healthy adult population and may not be representative of other specific populations (e.g. pediatric, pregnant, individuals with chronic conditions or from all ethnic backgrounds). They do not provide absolute levels at which the symptoms may occur and hence clinical correlation by the provider is recommended.

Results may be affected by pre-analytical variables related to specimen collection, handling, transport, storage, and inherent biological variability. Specimens including urine, saliva, stool, and blood-based samples (serum, plasma, EDTA whole blood, TES, and dried blood spots) may be impacted by improper collection technique, contamination, insufficient sample volume, delayed shipment or processing, temperature excursions, or improper storage conditions. Additional factors such as hemolysis; anticoagulant effects; clotting, centrifugation, or mixing parameters; incomplete mixing with transport media; and variability in dried blood spot application or saturation may further affect analyte stability or result accuracy. Specimen-specific factors, including urine dilution or concentration, variability in saliva composition or flow rate, and intermittent microbial shedding in stool, may also contribute to result variability. These factors may impact result accuracy and, in some cases, lead to a Test Not Performed (TNP). When clinically appropriate, repeat testing may be recommended; however, repeat testing may still fail to produce a reportable result if the underlying limitations persist.

All laboratory testing methodologies are subject to inherent analytical limitations related to instrument performance, assay design, methodological variability, and the specifications of FDA-approved and laboratory-developed analytes included in a test panel. As with all clinical laboratory testing, there is a small possibility of incorrect results due to technical errors, sample misidentification, contamination, rare genetic variants, or software-related issues.

Genetic testing is helpful in analyzing risks to various diseases. However, it is important to note that genetic risk determinants are neither necessary nor sufficient for the development of disease. Environmental and lifestyle risk factors could also affect the risk of disease development. Genetic risk does not indicate how common a health condition or variant is within the population; a risk-associated variant may be common or uncommon. Interpretation of genetic results should consider individual health context, as population-based reference frameworks may not fully represent all age groups, ethnic backgrounds, or health profiles. Genetic testing evaluates only the genotypes indicated and does not assess other genetic abnormalities found elsewhere in the genome. Different laboratories may test different variants when evaluating genetic risk for a given condition; therefore, genetic risk results may not be directly comparable between laboratories.

Some individuals may experience anxiety related to their genetic test results. Sample Reprort encourages any concerned individual to consult with a qualified healthcare professional prior to sample collection for a genetic test. Users of the test are encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional. In some cases, the identification of risk-associated genetic variants may prompt discussion with a healthcare provider about additional testing or follow-up.

The reported analytes, SNPs, and associated informational content are informed by scientific knowledge at the time of reporting, including peer-reviewed scientific publications, publicly available research, and guidance from recognized scientific and public health organizations. Interpretive content may be updated as scientific knowledge continues to evolve. The informational content included in this report is derived from publicly available scientific literature and is provided for educational and informational purposes only. This content does not replace medical advice from a qualified healthcare professional. Any wellness, nutritional, or dietary recommendations, diagnoses of medical conditions, or treatment decisions based on these results are made at the discretion and responsibility of the ordering healthcare professional.

Sample Reprort does not diagnose, treat, or cure medical conditions and does not replace the care of a licensed medical practitioner or counselor, nor does Sample Reprort recommend self-diagnosis or self-medication. Depending on the nature of testing, individuals who receive moderate- or high-risk results may be advised to pursue confirmatory testing and appropriate medical follow-up. Sample Reprort assumes no liability for any loss, injury, or damages arising from the procurement, compilation, interpretation, delivery, or reporting of information contained in this report, nor from any decisions made or actions taken based on these results.

The supplement recommendations and dosage guidelines provided are intended for general informational purposes only and should not replace professional medical advice; final dosage decisions must be made in consultation with your healthcare provider. Sample Reprort disclaims any liability for adverse effects, outcomes, or consequences arising from the use of these suggestions.