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NUTRIGENOMIX®

PERSONALIZED NUTRITION & FITNESS REPORT

Hello Caroline:

Nutrigenomix is pleased to provide you with your Personalized Nutrition and Fitness Report based on your individual genetic profile. Your recommendations are based on the most current evidence-based scientific research that has been published in peer-reviewed journals and reviewed by our team of world-renowned experts in the field of nutrigenomics.

Our laboratory has used state-of-the-art genetic testing procedures to analyze your DNA sample. We examined your genetic code to determine how your genes can influence recommendations related to weight management, body composition, cardiometabolic health, food intolerances, eating habits, fitness performance and injury risk. Based on these results, we developed a series of nutrition and fitness recommendations that are aligned with your genetic profile and gathered additional genetic insights for you and your healthcare provider to consider. As new discoveries in the field of nutrigenomics are made, you will have the opportunity to access this information to further fine-tune your personalized nutrition and fitness plan.

You and your healthcare professional can now use the personalized recommendations contained in this report to help you achieve optimal nutritional status and fitness level. In this way, you can create a plan to maximize your genetic potential and overall health and start to *eat according to your genes!*

Ahmed El-Sohemy, PhD
Chief Scientific Officer

The Science Behind Nutrigenomix

One man's food is another man's poison – Lucretius

Nutrition is one of the most important lifestyle factors affecting your risk for developing certain diseases and has a significant impact on overall well-being. Over the past decade, there has been growing recognition of the importance of how genes influence our nutritional status, which directly impacts our health. The human genome consists of about 25,000 genes and virtually all can exist in different forms. The variations in our genes make us unique from one another. Genetic variation determines not only the color of our eyes and hair, but how we metabolize and utilize the foods, nutrients and supplements we ingest. Nutrigenomics is the science that applies genomic information and advanced technologies to uncover the relationship between genes, nutrition and human health. The term nutrigenomics refers to both the study of how the food, beverages and supplements we consume affects our genes and how our genes can influence our body's response to what we consume.

Different versions of a gene can make us respond differently to certain components in food such as the lactose in milk, the gluten in bread, the caffeine in coffee, along with carbohydrates, fats, proteins vitamins and minerals found in various foods. We are all familiar with people who are lactose intolerant or cannot eat gluten. These differences between individuals can be explained by gene variations within the population. Through science and research we have learned that genetic variations in the population and between individuals affect a wide variety of responses to key components of the human diet. For instance, some individuals may benefit from limiting their consumption of caffeine or increasing their intake of omega-3 fat, while others can follow the general recommendation for either or both. Your best diet depends on the specific variants you have for these nutrient-related genes. Understanding your genetic profile and its implications on your unique response to the foods, supplements and beverages you consume will provide you with the tools needed to make the best dietary choices.

The science of how specific genes change how we respond to dietary components enables us to use nutrition to its fullest potential to prevent, manage or improve various health issues. These personalized diets can optimize an individual's nutritional status and empower them to focus on preventing diet-related diseases or conditions. A healthy, balanced diet should provide enough energy and nutrients to support optimal health, reduce the risk of disease and maintain a healthy body weight. While general dietary recommendations might be prudent to follow, the one-size-fits-all approach to nutritional advice could limit some individuals from reaching their full potential for health and wellness. By tailoring one's nutritional needs to their genetic profile, the benefits of nutrition on health status can be maximized.

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Summary of Results

Nutrient Metabolism

Dietary Component	Gene, rs Number	Risk Variant	Your Variant	Your Risk	Recommendations
Vitamin A	BCMO1, rs11645428	GG	GG	Elevated	Focus on consuming preformed sources of vitamin A.
Vitamin B_{12}	FUT2, rs601338	GG or GA	GA	Elevated	Focus on consuming bioavailable sources of vitamin B12.
Vitamin C	GSTT1, rs2266633	Del	Ins	Typical	Meet the RDA for vitamin C daily.
Vitamin D	CYP2R1, rs10741657	Algorithm	GA	Elevated	Consume 1000 IU (25 mcg) vitamin D daily.
	GC, rs2282679		GG		
Vitamin E	COMT, rs4680	GG	GA	Typical	Meet the RDA for vitamin E daily from food sources rich in vitamin E.
Folate	MTHFR, rs1801133	CT or TT	TT	Elevated	Meet the RDA for folate daily.
Choline	MTHFD1, rs2236225	Algorithm	GG	Elevated	Meet the Adequate Intake (AI) level for choline daily.
	PEMT, rs12325817		CG		
Calcium	GC, rs7041	Algorithm	TG	Elevated	Consume 1200 mg of calcium daily.
	GC, rs4588		CA		
Iron Overload	SLC17A1, rs17342717	Algorithm	CC	Low	Follow the recommendations provided in the Low Iron Status section.
	HFE, rs1800562		GG		
	HFE, rs1799945		CC		
Low Iron Status	TMPRSS6, rs4820268	Algorithm	GA	Elevated	Meet the RDA for iron and consume sources of vitamin C with iron-rich foods.
	TFR2, rs7385804		CA		
	TF, rs3811647		AA		

Food Intolerances and Sensitivities

Dietary Component	Gene, rs Number	Risk Variant	Your Variant	Your Risk	Recommendations
Lactose	MCM6, rs4988235	CC or CT	CT	Slightly Elevated	Limit dairy intake if you experience gastrointestinal symptoms.
Gluten	HLA, rs2395182	Algorithm	GT	Medium	Medium risk for gluten intolerance.
	HLA, rs7775228		TT		
	HLA, rs2187668		CT		
	HLA, rs4639334		GG		
	HLA, rs7454108		TT		
	HLA, rs4713586		AA		
Caffeine	ADORA2A, rs5751876	TT	CT	Typical	Follow the recommendations provided by the CYP1A2 gene section of this report.

Cardiometabolic Health

Dietary Component	Gene, rs Number	Risk/ Response Variant	Your Variant	Your Risk/ Response	Recommendations
Caffeine	CYP1A2, rs2472300	GA or AA	AA	Elevated	Limit caffeine intake to 200 mg/day.
Whole Grains	TCF7L2, rs12255372	TT or GT	GT	Elevated	Consume most grain products as whole grains.
Sodium	ACE, rs4343	GA or AA	AA	Elevated	Limit sodium intake to the Adequate Intake level.
Omega-6 and Omega-3 Fat	FADS1, rs174547	CC or CT	TT	Typical	Meet the RDA for omega-6 LA fat and omega-3 ALA fat.
Physical Activity	LIPC, rs1800588	TT or CT	CT	Enhanced	Aim for 150 to 300 min/week of cardio and at least 2 days/week of muscle-strengthening activities.

1in5

People with Risk Variant

Your Results

Gene	Marker
GSTT1	Ins or Del

Risk Variant	Your Variant
Del	Ins

Your Risk

Typical

Recommendation

Since you possess the Ins variant of GSTT1, there is no increased risk of vitamin C deficiency. Therefore, following the RDA guidelines for vitamin C is sufficient for you. The RDA for vitamin C is 75 mg per day for women and 90 mg per day for men. Smokers require an additional 35 mg per day. Citrus fruits and juices, strawberries, tomatoes, red and green peppers, broccoli, potatoes, spinach, cauliflower and cabbage are examples of foods that are good sources of vitamin C.

Meet the RDA for vitamin C daily.

Vitamin C

Vitamin C is an essential nutrient and powerful antioxidant. Vitamin C also aids in the absorption of non-heme (plant) iron, and supports immune function and the formation of collagen, a protein used to make skin, connective tissue, and blood vessels, along with supporting bone and tissue repair. Low blood levels of vitamin C have been associated with an elevated risk of cardiovascular disease, type 2 diabetes and cancer. Research has shown that the amount of vitamin C absorbed into the blood can differ between people even when the same amount is consumed. Some people do not process vitamin C from the diet as efficiently as others and are at a greater risk of vitamin C deficiency. Studies have shown that the ability to process vitamin C efficiently depends on a gene called GSTT1.*

*Cahill LE et al. Functional genetic variants of glutathione S-transferase protect against serum ascorbic acid deficiency. American Journal of Clinical Nutrition. 2009;90:1411-7.
Horska A et al. Vitamin C levels in blood are influenced by polymorphisms in glutathione S-transferases. European Journal of Nutrition. 2011;50:437-46.

GSTT1

The GSTT1 gene produces a protein for the glutathione S-transferase enzyme family. These enzymes play a key role in the utilization of vitamin C. The GSTT1 gene can exist in one of two forms. The insertion ("Ins") form is considered functional while the deletion ("Del") form is not functional. The different versions of this gene influence the way vitamin C is utilized in the body. A deletion version of the gene results in a reduced ability to process vitamin C. This means that people who possess the deletion version (Del) will have lower blood levels of vitamin C at a given level of vitamin C intake compared to people who possess the insertion version (Ins) of the gene.

Sources of Vitamin C

	Amount (mg)
Red pepper (1 pepper)	216
Strawberries (1 cup)	96
Pineapple (1 cup)	92
Brussels sprouts (1 cup)	90
Orange juice (1 cup)	86
Broccoli (1 cup)	82
Grapefruit (1 fruit)	78
Mango (1 fruit)	75
Kiwi (1 fruit)	70

Source: TACO (UNICAMP), Canadian Nutrient File and USDA Nutrient Database

Vitamin D

Vitamin D is essential to calcium metabolism and promotes calcium absorption in the gut. Low levels of vitamin D are associated with decreased bone mineral density and an increased risk of fractures. Vitamin D also contributes to normal functions of most cells in the body. Vitamin D can be synthesized by the skin from UV light or it can be obtained from the diet. Low blood levels of vitamin D can result in weak, brittle bones, poor muscle function, and decreased immunity. Life-long vitamin D insufficiency has also been linked to accelerated cognitive decline, autoimmune disorders, neuro-degenerative diseases and cardiovascular disease. Vitamin D deficiency is diagnosed by measuring the most common form of vitamin D in the blood, which is 25-hydroxyvitamin D. Research shows that variations in the CYP2R1 and GC genes can affect your risk for low circulating 25-hydroxyvitamin D levels.*

*Slater NA et al. Genetic Variation in CYP2R1 and GC Genes Associated With Vitamin D Deficiency Status. Journal of Pharmacy Practice. 2015:1-6.
Wang TJ et al. Common genetic determinants of vitamin D insufficiency: a genome-wide association study. Lancet. 2010;376:180-88.

CYP2R1 & GC

Vitamin D 25-hydroxylase is the key enzyme that activates vitamin D from its pre-formed type, which is obtained through sun exposure and the diet. This enzyme is encoded by the CYP2R1 gene and a variant of this gene has been associated with an increased risk of low circulating levels of vitamin D. The GC gene encodes the vitamin D-binding protein, which binds vitamin D and transports it to tissues. A variant in this gene has also been associated with an increased risk of low circulating levels of vitamin D.

Sources of Vitamin D

	Amount (IU)
Sockeye salmon (75g)	680
Whitefish (75g)	448
Sardines, canned in oil (1/2 can)	254
Rainbow trout (75g)	192
Smoked salmon (40g)	168
Halibut (75g)	144
Fortified plant-based beverage (1 cup)	124
Arctic char (75g)	112
Milk (1 cup)	104
Orange juice, fortified with vitamin D (1/2 cup)	50

Source: Health Canada's Nutrient Value of Some Common Foods and Canadian Nutrient File

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People with Risk Variant(s)

Your Results

Genes	Markers
CYP2R1 GC	rs10741657 rs2282679

Risk Variant	Your Variants
Algorithm	GA GG

Your Risk

Elevated

only when vitamin D intake is low

Recommendation

Since you possess one or more elevated risk variants, you are at an increased risk for low circulating vitamin D levels, so getting enough vitamin D is important. Aim for 1000 IU (25 mcg) vitamin D per day. This can help to maintain and/or improve your bone health, muscle and brain function, immunity, and heart health. Since it may be challenging to get enough vitamin D in the diet, supplementation may be beneficial. Do not exceed 2000 IU (50 mcg) per day without first having your blood levels of vitamin D assessed and monitored by a healthcare professional.

Consume 1000 IU (25 mcg) vitamin D daily.

1in5

People with Risk Variant

Your Results

Gene	Marker
ADORA2A	rs5751876

Risk Variant	Your Variant
TT	CT

Your Risk

Typical

Recommendation

Since you possess the CT or CC variant of the ADORA2A gene, you have a typical risk for an increase in feelings of anxiety after caffeine consumption. Aim to follow your DNA-based caffeine intake recommendations for the CYP1A2 gene included in your report.

Follow the recommendations provided by the CYP1A2 gene section of this report.

Caffeine

Anxiety

Many commonly consumed foods and beverages, such as coffee, tea, soft drinks and chocolate, as well as functional beverages such as energy drinks, contain caffeine. There are also hidden sources of caffeine found in pain medications, weight loss supplements, as well as chocolate or coffee-flavored beverages and food products. Caffeine is widely used to promote wakefulness and vigilance, reduce sleepiness and mitigate fatigue related to various shift-work occupations or travel across time zones. In the brain, the effects of caffeine are primarily due to its blocking action of adenosine, a neuromodulator that increases drowsiness and builds up over the day as bedtime approaches. Despite its widespread use, caffeine may cause anxiety in some people. A common variation in the ADORA2A gene contributes to the differences in subjective feelings of anxiety after caffeine ingestion,* especially in those who are habitually low caffeine consumers.**

*Childs E eta al. Association between ADORA2A and DRD2 polymorphisms and caffeine-induced anxiety. Neuropsychopharmacology. 2008 Nov;33(12):2791-800
Alsene K et al. Association between A2a receptor gene polymorphisms and caffeine-induced anxiety. Neuropsychopharmacology. 2003 Sep;28(9):1694-702.
**Rogers PJ, et al. Association of the anxiogenic and alerting effects of caffeine with ADORA2A and ADORA1 polymorphisms and habitual level of caffeine consumption. Neuropsychopharmacology. 2010. (9):1973–1983.

ADORA2A

The ADORA2A (adenosine A2A receptor) gene encodes one of the main receptors for adenosine. Adenosine has many functions in the body, including promoting sleep and calmness and suppressing arousal. Caffeine blocks adenosine receptors, resulting in the stimulating effects of coffee, tea, chocolate and other caffeinated food products and supplements. Individuals who possess the TT variant of the ADORA2A gene are more sensitive to the stimulating effects of caffeine and experience greater increases in feelings of anxiety after caffeine intake than do individuals with either the CT or CC variant.

Cardiometabolic Health

Caffeine is the most widely consumed stimulant in the world and coffee is the most significant source of caffeine, with tea, soda and chocolate also contributing to intakes. Research has shown that caffeine can influence cardiovascular health. However, the reported effects of coffee on the cardiovascular system have been inconsistent and at times have appeared contradictory. Some studies reported a link between high coffee consumption and an elevated risk of high blood pressure and heart disease, while other studies have shown no effect or even a protective effect with moderate intake. Two landmark studies* have now shown that the effect of coffee on cardiovascular disease depends on a variation in a gene called CYP1A2.

*Cornelis et al. Coffee, CYP1A2 genotype, and risk of myocardial infarction. Journal of the American Medical Association. 2006;295:1135-41.
Palatini P et al. CYP1A2 genotype modifies the association between coffee intake and the risk of hypertension. Journal of Hypertension. 2009;27:1594-1601.

CYP1A2

The CYP1A2 gene produces an enzyme called cytochrome P450 1A2 (CYP1A2), which is the main enzyme responsible for breaking down caffeine in the body. Variations in the CYP1A2 gene affect the rate at which caffeine is broken down, which determines the impact of caffeine on heart health. Individuals who possess the GA or AA variant of CYP1A2 break down caffeine more slowly and are at greater risk of high blood pressure and heart attack when caffeine intake is high. Those who have the GG variant actually have a lower risk of heart disease with moderate coffee consumption than those who consume no coffee at all.

Sources of Caffeine

	Amount (mg)
Coffee (1 cup)	100
Energy drinks (1 cup)	80
Espresso (1 shot)	85
Black tea (1 cup)	50
Green tea (1 cup)	45
Cola (1 can)	26
Chocolate, dark (40g)	27
Decaf coffee, espresso, tea (1 cup)	0-15
Herbal tea (1 cup)	0

Source: Canadian Nutrient File and USDA Nutrient Database

1in2

People with Risk Variant

Your Results

Gene	Marker
CYP1A2	rs2472300

Risk Variant	Your Variant
GA or AA	AA

Your Risk

Elevated

only when caffeine intake is high

Recommendation

Since you possess the AA or GA variant of the CYP1A2 gene, there is an increased risk of high blood pressure and heart attack if consuming more than 200 mg of caffeine daily, which is approximately 2 small cups of coffee. Limit caffeine consumption to no more than 200 mg per day in order to reduce this risk. Caffeine occurs naturally in coffee, tea, cocoa, kola and guarana. It is also manufactured synthetically and added to cola, energy drinks, and certain over the counter cold remedies.

Limit caffeine intake to 200 mg/day.

International Science Advisory Board

Ahmed El-Sohemy, PhD

Dr. Ahmed El-Sohemy is a Professor and Associate Chair and held a Canada Research Chair in Nutrigenomics at the University of Toronto. He is also the founder of Nutrigenomix Inc., serves as the company's Chief Science Officer and is Chair of the company's International Science Advisory Board. Dr. El-Sohemy obtained his PhD from the University of Toronto and completed a postdoctoral fellowship at Harvard. He has published in the top scientific and medical journals with almost 200 peer reviewed publications and has given more than 300 invited talks around the world. He is currently Editor-in-Chief of the journal Genes & Nutrition, serves on the editorial board of 10 other journals, and has served as an expert reviewer for more than 30 different scientific and medical journals and 12 research granting agencies. He has been a member of international expert advisory panels and scientific advisory boards of several organizations. Dr. El-Sohemy is the recipient of several awards for excellence in research by the American College of Nutrition, the Canadian Society for Nutrition and the American Nutrition Association.

Sara Mahdavi, RD, MSc, PhD

Dr. Sara Mahdavi is a clinical scientist and holds a clinical instructor and research appointment with the Department of Community and Family Medicine at the University of Toronto. Dr. Mahdavi received her doctorate from the Faculty of Medicine at the University of Toronto in the field of gene-environment interactions and cardiometabolic disease. She has been practicing clinical dietetics over the last decade at several hospitals as well as private practices. Dr. Mahdavi has been an invited speaker at medical conferences and for government agencies. She has published over a dozen original scientific articles in top medical journals, has been an invited reviewer for several clinical journals and serves on the editorial board of the Canadian Journal of Kidney Health and Disease. Dr. Mahdavi's clinical research and practice have varied from early insulin sensitivity to kidney disease, rare genetic disorders, and innovative dermatological interventions.

Lynnette R Ferguson, D.Phil. (Oxon.), DSc

Dr. Lynn Ferguson is Program Leader of Nutrigenomics New Zealand. She obtained her D.Phil. from Oxford University working on DNA damage and repair. After her return to New Zealand, she began working as part of the Auckland Cancer Society Research Centre, using mutagenicity testing as a predictor of carcinogenesis. In 2000, she took on a 50% role as Head of a new Discipline of Nutrition at The University of Auckland. She has recently been investigating the interplay between genes and diet in the development of chronic disease, with particular focus on Inflammatory Bowel Disease. As Program Leader of Nutrigenomics New Zealand she is working with a range of others to bring nutrigenomics tools to the New Zealand science scene. She has supervised more than 30 students and has more than 300 peer reviewed publications. Dr. Ferguson serves as one of the managing Editors for Mutation Research: Fundamental and Molecular Mechanisms of Mutation, as well as on the Editorial Boards of several other major journals.

J. Bruce German, PhD

Bruce German is the Director of the Foods for Health Institute at the University of California Davis, and is Professor of Food Science and Technology (http://ffhi.ucdavis.edu/). Dr German received his PhD from Cornell University and joined the faculty at the University of California (Davis) in 1988. In 1997, he was named the first John E. Kinsella Endowed Chair in Food, Nutrition and Health. His research interests in personalized nutrition include the structure and function of dietary lipids, the role of milk components in food and health and the application of metabolic assessment to personalizing diet and health. Dr German has published more than 350 papers and holds a number of patents related to various technologies and applications of bioactive food components. The research articles from his lab rank in the top 5 most cited in the field.

David Jenkins, MD, DSc, PhD

Dr. Jenkins earned his MD and PhD at Oxford University, and is currently a Professor in both the Departments of Medicine and Nutritional Sciences at the University of Toronto. He is also a staff physician in the Division of Endocrinology and Metabolism and the Director of the Clinical Nutrition and Risk Factor Modification Center, St. Michael's Hospital. Dr Jenkins has published over 300 peer reviewed articles and given hundreds of invited talks around the world. He has served on numerous international committees to set guidelines for the treatment of diabetes and most recently on the new joint United States-Canada DRI system (RDAs) of the National Academy of Sciences. His team was the first to define and explore the concept of the glycemic index of foods and demonstrate the breadth of metabolic effects of viscous soluble fibre. He has received many national and International awards in recognition of his contribution to nutrition research. Dr Jenkins currently holds a Canada Research Chair in Nutrition and Metabolism.

Jose Ordovas, PhD

Jose M. Ordovas is Professor of Nutrition and Director of the Nutrigenomics Laboratory at the United States Department of Agriculture, Human Nutrition Research Center on Aging at Tufts University in Boston. After obtaining his PhD from the University of Zaragoza, Spain, he completed postdoctoral work at Harvard, MIT and Tufts University. Dr Ordovas' major research interests focus on the genetic factors predisposing to cardiovascular disease and their interaction with environmental factors. Dr Ordovas has published ~700 articles in peer reviewed journals, and written numerous reviews and edited 5 books on nutrigenomics. He has been an invited speaker at hundreds of International meetings all over the world and is currently a member of the Institute of Medicine's Food and Nutrition Board (National Academies). He serves as Editor for Current Opinion in Lipidology (Genetics Section), and on the Editorial Board of numerous journals. Dr. Ordovas is a Member of Honor of the Spanish Society of Atherosclerosis and has received other awards for his contributions to the field of nutrigenomics.

Ben van Ommen, PhD

Dr. Ben van Ommen is Director of the Nutrigenomics Organization (NuGO) and Principal Scientist at TNO, one of the largest independent research organizations in the area of nutrition world-wide. He is also Director of the TNO systems biology program and leading the activities on nutrigenomics, nutritional systems biology, personalized health and personalized medicine. His research applies systems biology to metabolic health and metabolic disease, focusing on understanding all relevant processes involved in maintaining optimal health and causing specific disease sub-phenotypes, developing new biomarkers and treatment strategies.

Nanci S. Guest, PhD, RD, CSCS

Dr. Nanci Guest is a registered dietitian (sport specialty), certified personal trainer and a certified strength and conditioning specialist, and she has been working in private practice in this field for two decades. She completed her doctoral degree in the area of nutrigenomics and athletic performance at the University of Toronto. She obtained her BSc in agriculture and dietetics, and her MSc in nutritional sciences with a sport focus at the University of British Columbia. Dr. Guest has published her research in top journals, presented at international conferences and has given dozens of invited talks around the world. She also teaches advanced sport nutrition courses at the college level. Dr. Guest is a global consultant to professional and amateur athletes and teams, and she was also involved in creating past athlete nutrition guidelines for the International Olympic Committee. She was the Head Dietitian at both the Vancouver 2010 Olympics and the Toronto 2015 Pan Am games and served as a consultant to a variety of international athletes in preparation for the past four London, Sochi, Rio and PyeongChang Olympics.