NUTRIGENOMI8™

EAT ACCORDING TO YOUR GENES

Personalized Nutrition Report

Sample ID: 15001112090986

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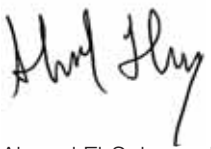
Introduction

Hello Susan:

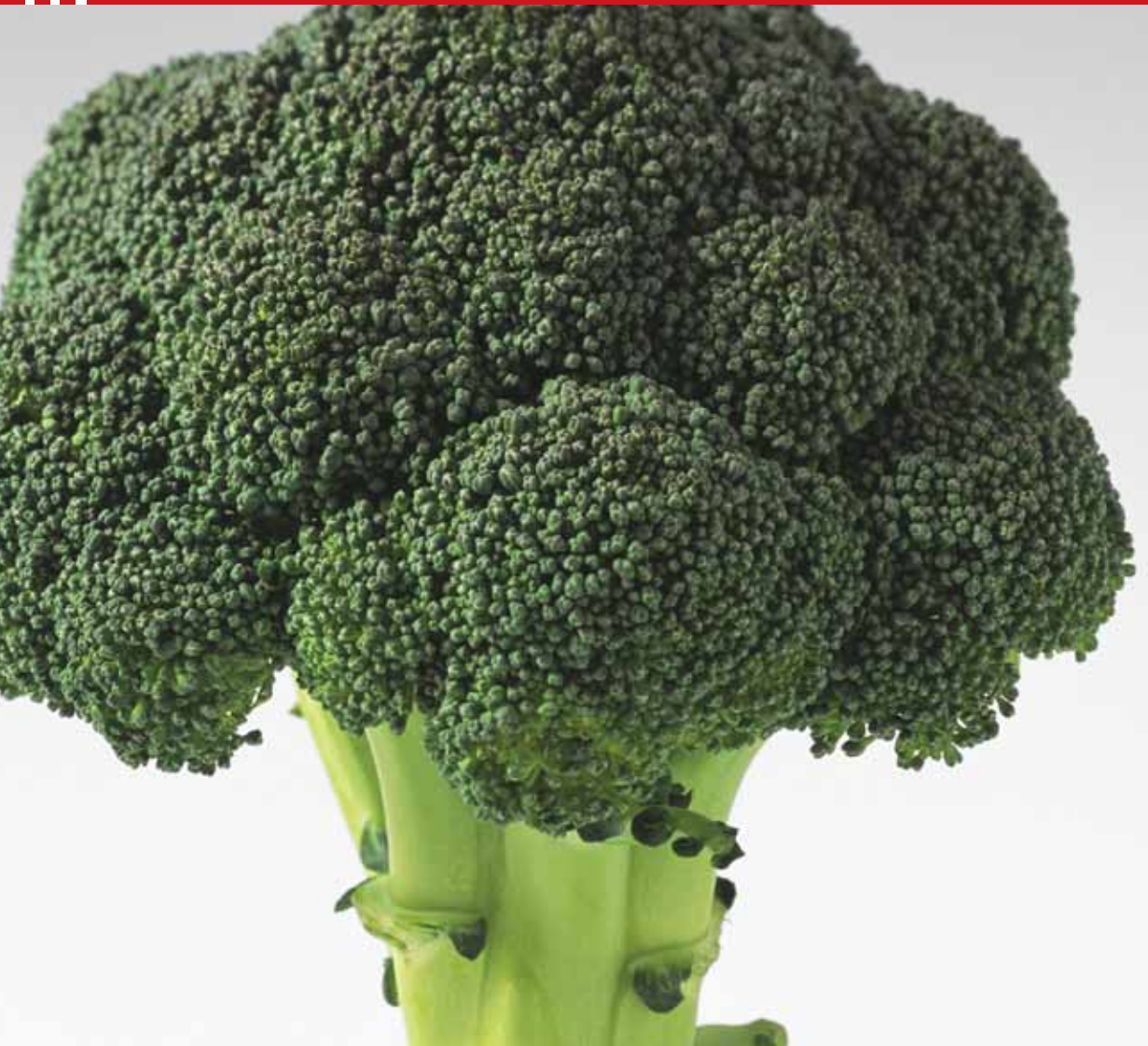
Nutrigenomix is pleased to provide you with your Personalized Nutrition Report based on your unique genetic profile. The recommendations provided are based on the cutting-edge research and comprehensive review of the scientific evidence by world-renowned experts in the field of nutrigenomics.

Our laboratory has used state-of-the-art genetic testing procedures to analyze the DNA from the saliva sample you provided. We analyzed your genetic code to determine how your body responds to vitamin C, folate, whole grains, omega-3 fats, saturated fat, sodium and caffeine. Finally, we developed a series of dietary recommendations based on your unique genetic profile and the best available scientific evidence. 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 dietary plan.

You and your registered dietitian can now use the dietary recommendations contained in this report to help you achieve optimal nutritional status. In this way, you can create a diet to maximize your genetic potential and overall health and start to eat according to your genes!



Ahmed El-Sohemy, PhD
Chief Science Officer



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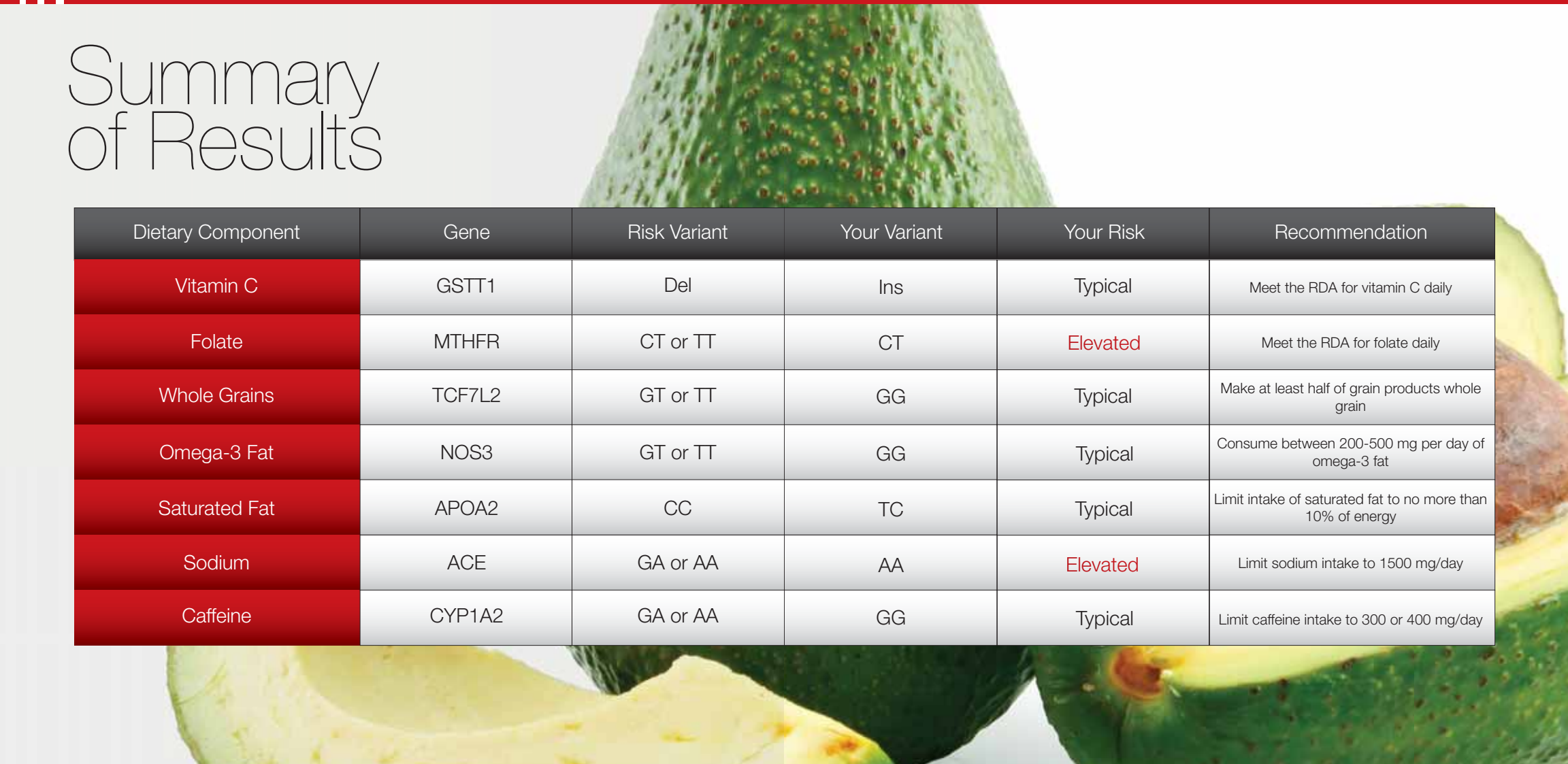

Dietary Component	Gene	Risk Variant	Your Variant	Your Risk	Recommendation
Vitamin C	GSTT1	Del	Ins	Typical	Meet the RDA for vitamin C daily
Folate	MTHFR	CT or TT	CT	Elevated	Meet the RDA for folate daily
Whole Grains	TCF7L2	GT or TT	GG	Typical	Make at least half of grain products whole grain
Omega-3 Fat	NOS3	GT or TT	GG	Typical	Consume between 200-500 mg per day of omega-3 fat
Saturated Fat	APOA2	CC	TC	Typical	Limit intake of saturated fat to no more than 10% of energy
Sodium	ACE	GA or AA	AA	Elevated	Limit sodium intake to 1500 mg/day
Caffeine	CYP1A2	GA or AA	GG	Typical	Limit caffeine intake to 300 or 400 mg/day

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 colour of our eyes and hair, but how we metabolize and utilize the nutrients 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 eat.

Different versions of a gene can have different responses to certain components of food. 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. We are now learning 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. Understanding your genetic profile and its implications on your unique response to the food and beverages you consume will provide you with the tools needed to make the best dietary choices.

Recent scientific discoveries relating specific gene variants to dietary response enable us to use nutrition to its fullest potential to address various health issues. These personalized diets can optimize an individual's nutritional status and specifically focus on preventing diet-related diseases. 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 your genetic profile, the benefits of nutrition on health status can be maximized.

Vitamin C

Increased risk of vitamin C deficiency with low vitamin C intake and Del variant

150%

Vitamin C is an essential nutrient that must be obtained from dietary sources. 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 of vitamin C 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. Two recent 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 from 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 interact to 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 intake than people who possess the insertion version (Ins) of the gene.

Food sources of Vitamin C	Amount (mg)
Red peppers (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: Canadian Nutrient File and USDA Nutrient Database

1 in 5

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
GSTT1	Ins or Del	Del	Ins

Your Risk
Typical

Recommendation

Meet the RDA for vitamin C daily

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. Vitamin C can also be consumed in supplement form, either alone or as a multivitamin.

Folate

Increased risk of folate deficiency with low folate intake and CT or TT variant

180%

Folate is a water-soluble B vitamin that is necessary for cell growth and development. Low blood levels of folate have been associated with increased risk of heart disease and stroke. Research has shown that the amount of folate absorbed into the blood can differ between individuals even when the same amount of folate is consumed. Some people do not utilize dietary folate as efficiently as others and are consequently at a greater risk of folate deficiency. Two studies* have shown that an individual's ability to process dietary folate efficiently depends on a gene called MTHFR.

* Solis C et al. Folate Intake at RDA Levels Is Inadequate for Mexican American Men with the Methylenetetrahydrofolate Reductase 677TT Genotype. J Nutr. 2008 ;138 :67-72. Guinotte CL et al. Methylenetetrahydrofolate Reductase 677C T Variant Modulates Folate Status Response to Controlled Folate Intakes in Young Women. J Nutr. 2003 ;133 :1272-1280.

MTHFR

The MTHFR gene produces methylenetetrahydrofolate reductase (MTHFR), which is a vital enzyme for folate usage in the body. MTHFR converts folate obtained from the diet to an active form of the nutrient that can be used by the body at the cellular level. Variations in the MTHFR gene determine the way individuals can utilize dietary folate. Those people who have the CT or TT variant of the gene have reduced MTHFR enzyme activity and are at greater risk of folate deficiency when folate intake is low, compared to those with the CC variant.

Food sources of Folate	Amount (mcg)
Chicken liver (75g)	420
Edamame (soybeans) (1/2 cup)	382
Lentils, cooked (3/4 cup)	265
Spinach, cooked (1/2 cup)	130
Asparagus (6 spears)	128
Chickpeas (3/4 cup)	119
Black beans (3/4 cup)	108
Avocado (1/2 fruit)	81
Sunflower seeds (1/4 cup)	77

Source: Canadian Nutrient File and USDA Nutrient Database

2 in 3

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
MTHFR	rs1801133	CT or TT	CT

Your Risk

Elevated

only when folate intake is low

Recommendation

Meet the RDA for folate daily

Since you possess the CT variant of the MTHFR gene, there is a greater risk of folate deficiency if the RDA is not met on a daily basis. Ensure that folate intake is at least 400 mcg per day in order to reduce the risk of deficiency. Foods that are naturally high in folate include lentils, romano beans, black beans, white beans, okra, asparagus, spinach, and other leafy greens. Enriched ready-to-eat cereals, bread, and bread products are also good sources of folate. Folate can also be consumed in supplement form.

Increased risk of diabetes with low amounts of whole grains and GT or TT variant

36%

Whole grains

Whole grains are a low glycemic index carbohydrate that have more fiber than refined grains. They also contain more essential micronutrients such as folic acid, magnesium and vitamin E. Years of research have shown that whole grains may help to reduce the risk of several diseases, in particular, type 2 diabetes. Scientists have also demonstrated that the TCF7L2 gene is strongly associated with developing type 2 diabetes. Research now shows that some individuals might benefit more from increasing their whole grain consumption*.

* Cornelis MC et al. TCF7L2, dietary carbohydrate, and risk of type 2 diabetes in US women. American Journal of Clinical Nutrition. 2009;89:1256-62.

TCF7L2

The TCF7L2 gene produces a protein called transcription factor-7 like 2 (TCF7L2). This protein, in turn, affects how the body turns on or off a number of other genes. The interaction of these proteins and genes is complex, and not yet fully understood. However, the TCF7L2 gene is one of the most consistant predictors of the likelihood of developing type 2 diabetes. People who possess the high risk GT or TT variant of the gene are at greater risk of developing type 2 diabetes. Yet, recent studies have shown that consuming whole grain foods can reduce the risk of type 2 diabetes in individuals who carry the GT or TT variant of the TCF7L2 gene.

Replace these foods...	With these foods...
White bread, bagels, pitas	100% Whole grain bread, bagles,and pitas
White rice	Brown rice, wild rice, or quinoa
White pasta	100% Whole wheat pasta or brown rice pasta
High sugar cold cereals	Cooked oatmeal or 100% whole grain cold cereal
White flour baked goods	100% Whole wheat flour baked goods

1 in 2

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
TCF7L2	rs12255372	GT or TT	GG

Your Risk
Typical

Recommendation

Make at least half of grain products whole grain

Since you possess the GG genotype, there is no increased risk of developing type 2 diabetes. Follow Canada's Food Guide general recommendation to make half of all daily grain products whole grain. To identify these foods, look for the words "whole grain" on the label and in the ingredient list. Cereal and grains that can be found whole include wheat, rice, oats, barley, corn, wild rice, rye, quinoa and buckwheat. Products labeled with the words "multigrain" and "organic" are not necessarily whole grain.

Omega-3 Fat

Increase in triglycerides with low omega-3 and GT or TT variant

25%

Omega-3 fats, such as those found in fatty fish, have been associated with a reduced risk of heart disease. This is likely due, in part, to their ability to lower blood levels of triglyceride that impair blood circulation. Previous studies have produced mixed results relating to the effects of omega-3 fat on triglyceride levels between individuals. Some people experience a significant reduction in triglyceride levels in response to increasing omega-3 fat intake, whereas others experience little benefit. The reasons for these differences have been unclear until a recent breakthrough study* showed that the effect of omega-3 fat on triglyceride levels depends on variations in a gene called NOS3.

* Ferguson J et al. NOS3 gene polymorphisms are associated with risk markers of cardiovascular disease, and interact with omega-3 polyunsaturated fatty acids. Atherosclerosis. 2010;211:539-544.

NOS3

The NOS3 gene directs the production of an enzyme called nitric oxide synthase. This enzyme is responsible for making nitric oxide, which plays an important role in the function of cells that line our blood vessels. New research has shown that variations in the NOS3 gene interact with omega-3 fat in different ways to impact how the body processes triglycerides. Those who have the GT or TT variant of the gene are at greater risk of elevated triglyceride levels when consuming a diet low in omega-3 fats, compared to those who have the GG variant.

Food sources of Omega-3 fat*	Amount (g)
Salmon, (75g)	1.6
Herring (75g)	1.5
Anchovy (75g)	1.3
Mackerel (75g)	0.9
Trout (75g)	0.7
Tuna, white (75g)	0.6
Lobster (75g)	0.4
Crab (75g)	0.3
Tuna, light (75g)	0.2

* Long chain omega-3s EPA + DHA.

Source: Canadian Nutrient File and USDA Nutrient Database

1 in 2

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
NOS3	rs1799983	GT or TT	GG

Your Risk
Typical

Recommendation

Consume between 200-500 mg per day of omega-3 fat

Since you possess the GG genotype, there is no benefit to increasing omega-3 intake in order to lower serum triglyceride levels. You should, therefore, follow the recommendation to consume 200-500 mg daily in order to lower general cardiovascular disease risk.

Increased risk of obesity with high saturated fat and CC variant

67%

Saturated Fat

Saturated fats, such as those found in red meat, have long been associated with health conditions like diabetes, cardiovascular disease and obesity. However, the connection between saturated fats and obesity, until recently, has been poorly understood. Scientists could not explain why certain people seemed prone to obesity when consuming a diet high in saturated fats, but others were less susceptible. A number of studies* have now shown that the effect of saturated fat on obesity can be influenced by variations in a gene called APOA2.

* Corella et al. (2009) APOA2, dietary fat, and body mass index: replication of a gene-diet interaction in 3 independent populations. Arch. Intern. Med. 169(20):1897-906.

APOA2

The APOA2 gene directs the body to produce a specific protein called apolipoprotein A-II, which plays an important role in the body's ability to utilize different kinds of fat. Scientists now understand that there are different variations in the APOA2 gene present in the human population and that these different versions of the gene interact with saturated fat in unique ways to influence energy balance and ultimately the risk of obesity. Those people who have the CC variant of the gene are at a higher risk of developing obesity when consuming a diet high in saturated fats than those possessing the TT or TC variant of the gene.

Food sources of saturated fat	Amount (g)
Short ribs (75g)	11
Cheddar cheese (50 g)	10
Ice cream, premium (1/2 cup)	11
Butter (1 tbsp)	8
Salami (75g)	8
Regular ground beef, cooked (75g)	7
Cheeseburger (single patty)	6
Muffin (1 small)	5
French fries (20-25 fries)	5
Coffee cream, 18% MF (1 tbsp)	2

Source: Canadian Nutrient File and USDA Nutrient Database

1 in 7

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
APOA2	rs5082	CC	TC

Your Risk
Typical

Recommendation

Limit intake of saturated fat to no more than 10% of energy

Since you possess the TC genotype, there is no increased risk of high BMI and obesity with a diet high in saturated fat. However, you should still limit saturated fat intake to less than 10% of total energy intake, as recommended, in order to reduce the general risk of other associated health issues such as cardiovascular disease. Foods high in saturated fat include coconut and palm oils, fatty meats (lamb, pork and beef), butter, cheese, fried foods and baked goods. Suitable alternatives low in saturated fat include olive and vegetable oils, lean meats, low-fat dairy products, fish, and plant protein sources such as beans, nuts or tofu.

Increased risk of high blood pressure with high sodium intake and GA or AA variant

230%

Sodium

Sodium is an essential micronutrient that regulates blood pressure and blood volume. Most people consume more sodium than the body requires. The major adverse effect of excess sodium intake is elevated blood pressure, which predisposes to hypertension and heart disease. However, some individuals do not experience as great an increase in blood pressure in response to excess sodium intake as others. Research* now shows that the effect of sodium intake on blood pressure is influenced by variations in a gene called ACE.

* Poch E et al. Molecular basis of salt sensitivity in human hypertension: Evaluation of renin-angiotensin-aldosterone system gene polymorphisms. Hypertension. 2001;38:1204-9.

ACE

The ACE gene directs the body to produce the angiotensin-converting enzyme (ACE), which is known to play a role in regulating the response of blood pressure to sodium intake. It is now known that a person's specific blood pressure response to excess sodium intake is dependent on which variant of the ACE gene they possess. Those who have the GA or AA variant of the ACE gene are at a greater risk of experiencing elevated blood pressure when higher amounts of sodium are consumed than those possessing the GG variant of the gene.

Food sources of sodium	Amount (mg)
Ramen noodles, with flavour (1 package)	1760
Breakfast bagel with ham, egg and cheese	1260
Canned soup (1 cup)	1130
Ham (75g)	1040
Pickle (1 medium)	830
Tomato sauce, canned (1/2 cup)	650
Feta cheese (50g)	560
Potato chips (1 small bag)	390
Bread (1 slice)	230
Cold cereal (1 cup)	350

Source: Canadian Nutrient File and USDA Nutrient Database

7 in 10

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
ACE	rs4343	GA or AA	AA

Your Risk

Elevated

only when sodium intake is high

Recommendation

Limit sodium intake to 1500 mg/day

Since you possess the AA variant of the ACE gene, there is an increased risk of elevated blood pressure when sodium intake is high. Limiting sodium consumption to the Adequate Intake (AI) level of 1500 mg per day should help to reduce the risk. The AI is equivalent to ¾ teaspoon of salt per day, which includes sodium that is found naturally in food as well as salt that is added during processing and preparation. Foods that are high in sodium include canned soups and canned vegetables, potato chips, processed meats, soy sauce, ketchup and processed cheeses.

Increased risk of a heart attack with high caffeine consumption and GA or AA variant

53%

Caffeine

Caffeine is the most widely consumed stimulant in the world and coffee is the most significant source of caffeine. Research has shown that caffeinated coffee intake can have a significant influence on 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. We now know that variations in the CYP1A2 gene affect the rate at which caffeine is broken down. It is the rate at which caffeine is broken down that determines whether consumption of caffeine-containing products, such as coffee, is harmful to 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 (40 g)	27
Decaf coffee, espresso, tea (1 cup)	0-15
Herbal tea (1 cup)	0

Source: Canadian Nutrient File and USDA Nutrient Database

1 in 2

People with Risk variant

Your Results

Gene	Marker	Risk variant	Your variant
CYP1A2	rs2472300	GA or AA	GG

Your Risk
Typical

Recommendation

Limit caffeine intake to 300 or 400 mg/day

Since you possess the GG genotype, there is no increased risk of hypertension or heart attack if consuming more than 200 mg of caffeine daily. Therefore, following Health Canada's recommendation to consume at most 300 mg per day for women of child-bearing age and at most 400 mg per day for other adults is appropriate. 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.

Nutrigenomix International Science Advisory Board

Ahmed El-Sohemy, PhD
Chair

Dr. Ahmed El-Sohemy is the Founder of Nutrigenomix Inc. and serves as the President and Chief Science Officer. He also serves as Chair of Nutrigenomix's International Science Advisory Board (SAB), which consists of key opinion leaders in the field of nutrigenomics. Dr. El-Sohemy obtained his PhD from the University of Toronto and completed a postdoctoral fellowship at the Harvard School of Public Health. He currently holds a Canada Research Chair in Nutrigenomics at the University of Toronto and serves on Health Canada's Science Advisory Board. Dr. El-Sohemy has published in the top scientific and medical journals with more than 80 peer-reviewed publications and has given more than 100 invited talks around the world. He is on the editorial board of 8 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.

David Castle, PhD

David Castle is Professor and Chair of Innovation in the Life Sciences at the University of Edinburgh. His research focuses on social aspects of life science innovation including democratic engagement, regulation and governance, and intellectual property and knowledge management. Prof. Castle is a world-renowned expert on the social, ethical and legal aspects of nutrigenomics. He is author of a book entitled Science, Society, and the Supermarket: The Opportunities and Challenges of Nutrigenomics, and has published extensively on the social dimensions of science, technology and innovation. Prof. Castle has held several major research awards and has considerable experience leading strategic research initiatives and research project management. Prof. Castle has consulted widely to government and industry on issues such as the impact of national technology transfer policies and programs, intellectual property and knowledge management strategies, and the role of non-scientific considerations in the regulation of science and technology.

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.

Bénédicte Fontaine-Bisson, RD, PhD

Dr. Fontaine-Bisson is an Assistant Professor in the Nutrition Sciences Program at the University of Ottawa and a Registered Dietitian (RD) with the College of Dietitians of Ontario. She received her BSc from Laval University, PhD from the University of Toronto and postdoctoral training at the Institut National de la Santé et de la Recherche Médicale (INSERM) in Paris, France. Dr. Fontaine-Bisson is one of the first RDs in Canada to obtain a PhD in nutrigenomics. She uses both epidemiological and clinical approaches to explore the complex interplay between nutrients and the human genome. The goal of her research program in nutrigenomics is to elucidate how genetic variation affecting inflammation or specific micronutrient pathways modify the effect of dietary components on the development of chronic diseases such as cardiovascular disease and type 2 diabetes.

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.

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 Organisation (NuGO) and Principal Scientist at TNO, one of the largest independent research organisations 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.

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