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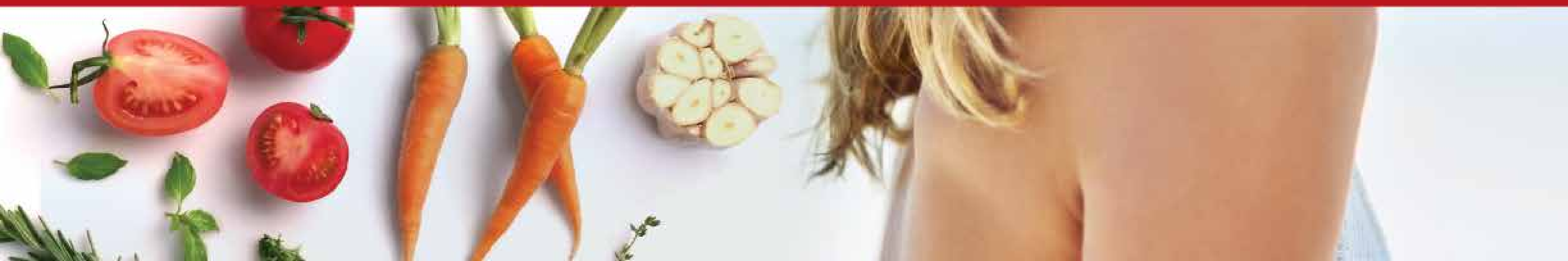
15151112090713

NUTRIGENOMIX®
EAT ACCORDING TO YOUR GENES

PERSONALIZED NUTRITION FOR SKIN HEALTH



Hello Caroline:

Nutrigenomix is pleased to provide you with your Personalized Skin Care Report based on your individual genetic profile. This report was developed based on scientific research published in peer-reviewed journals and reviewed by our team of experts in nutrigenomics.

Our laboratory has used state-of-the-art genetic testing procedures to analyse your DNA to determine how your genes can influence your skin's ability to combat the signs of aging, your eating habits, and how your body metabolizes nutrients that support skin health. Based on these results, we have determined your propensity to develop signs of skin aging and provided nutrition recommendations aligned with your genetic profile.

You and your healthcare provider can now use the information contained in this report to help you create a personalized skin care protocol. As new discoveries in the fields of nutrigenomics and dermatology are made, you will have the opportunity to access this information to further fine-tune your personalized skin health plan.

The Nutrigenomix Team

Sample ID: 15151112090713

Date of Report: July 28, 2020

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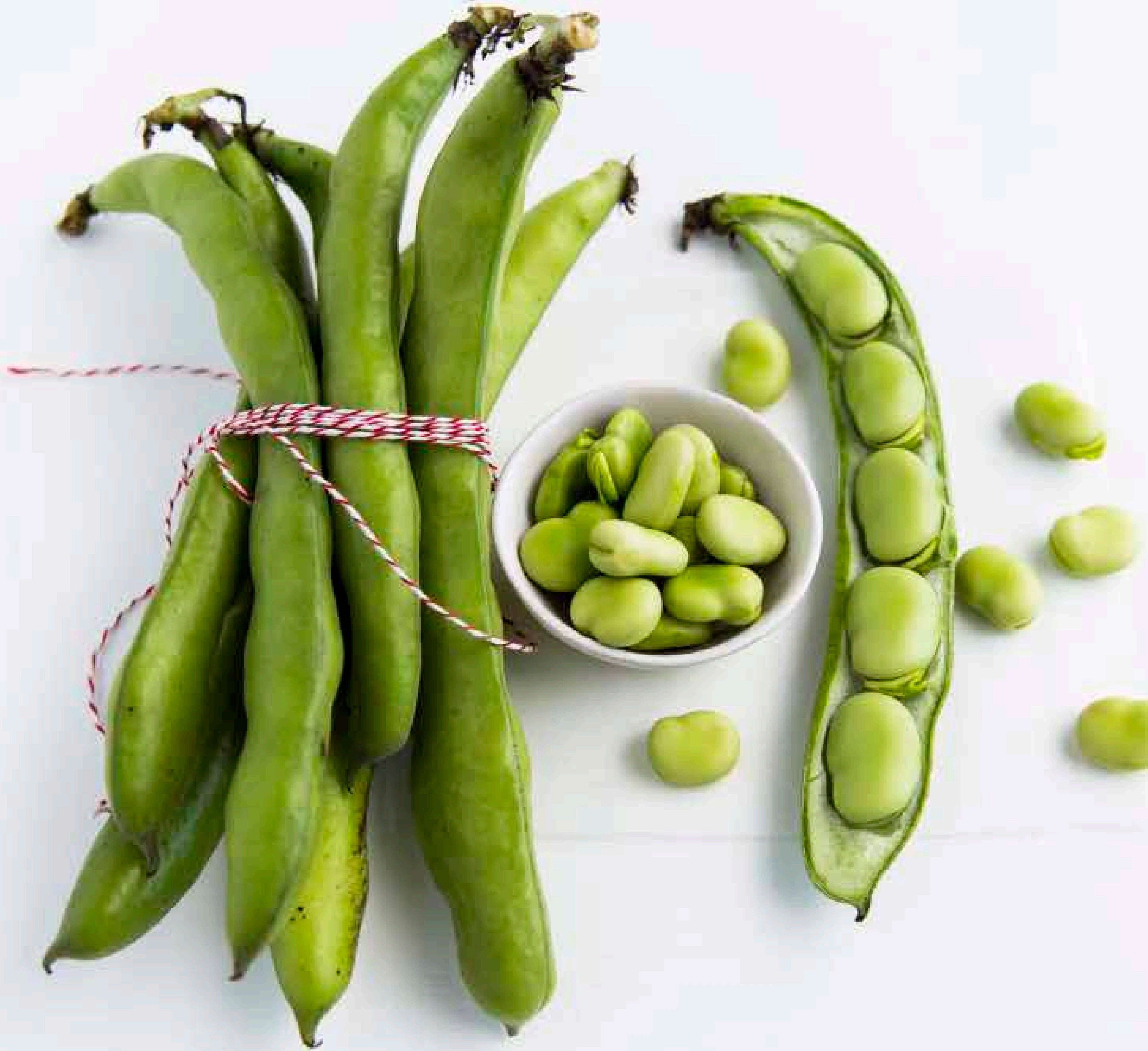


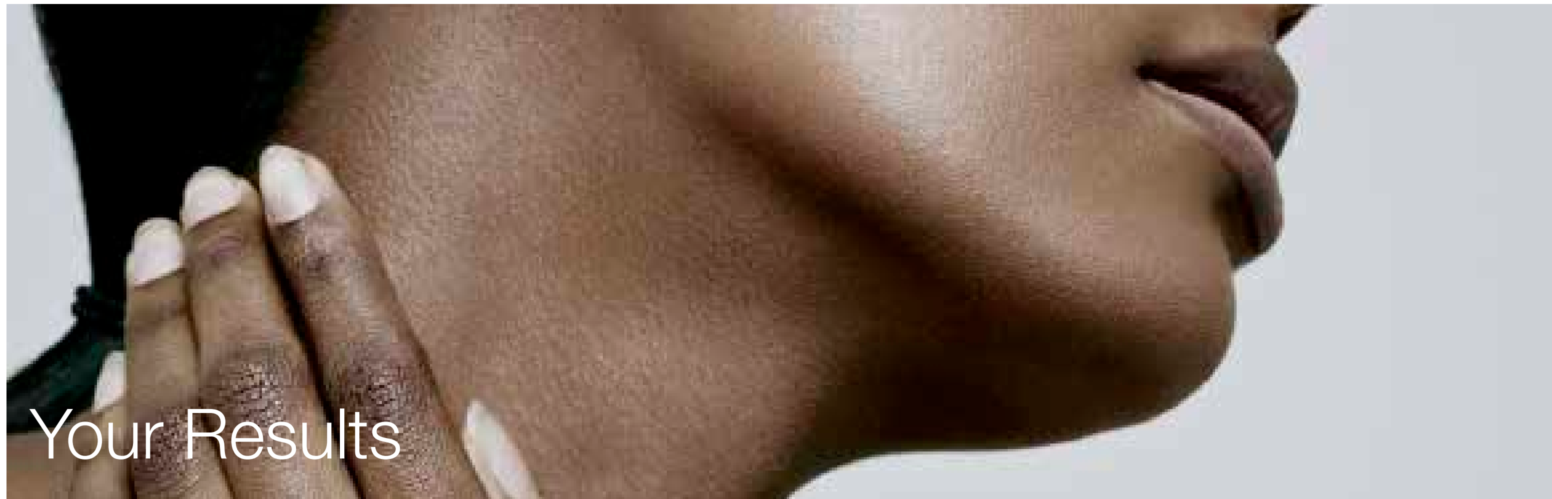
Summary of Results

Skin Aging

Skin Trait	Gene rs Number	Risk Variant	Your Variant	Your Risk	Implications
Facial Pigmented Spots	IRF4, rs12203592	TC or TT	CC	Typical	Typical risk of age-related facial pigmented spots.
Antioxidant Capacity	SOD2, rs4880 NQO1, rs1800566	Algorithm	CT CT	Diminished	Diminished antioxidant activity.
Loss of Elasticity	MMP1, rs1799750	GG	GG	Elevated	Elevated collagen breakdown.
Advanced Glycation End Products (AGE)	GLO1, rs1130534 GLO1, rs1049346	Algorithm	AT CT	Slightly Diminished	Slightly diminished AGE-neutralizing enzymatic activity.

Eating Habits and Nutrient Metabolism

Dietary Component	Gene rs Number	Risk Variant	Your Variant	Your Risk	Recommendations
Sugar Preference	GLUT2, rs5400	CT or TT	CT	Elevated	You have a high preference for sugar.
Vitamin A	BCMO1, rs11645428	GG	GG	Elevated	Focus on consuming pre-formed sources of vitamin A.
Vitamin C	GSTT1, rs2266633	Del	Ins	Typical	Meet the RDA for vitamin C daily.
Vitamin D	CYP2R1, rs10741657 GC, rs2282679	Algorithm	GA GG	Elevated	Consume 1000 IU (25 mcg) vitamin D daily.
Vitamin E	APOA5, rs12272004	CC	CA	Typical	Meet the AI for vitamin E daily.
Zinc	SLC30A3, rs11126936	CC	CC	Elevated	Focus on consuming bioavailable sources of zinc.



Your Results

Gene	Marker	Risk Variant	Your Variant
IRF4	rs12203592	TC or TT	CC

Your Risk: **Typical**

Recommendation: Since you possess the CC variant of the IRF4 gene, you have a typical risk of developing facial pigmented spots. The best way to prevent the formation of pigmented spots is to practice sun safety by minimizing excessive sun and UV ray exposure and using sunscreen.

1 in 10 — *Africans with Risk Variant*

1 in 7 — *Hispanics with Risk Variant*

1 in 4 — *Europeans with Risk Variant*

Facial Pigmented Spots

Dark pigmented spots on the face and other skin areas are a common sign of aging. Pigmented spots, also known as solar lentigines, result from cumulative skin exposure to UV rays. Cells within the skin produce melanin, a pigment that acts as a natural sunscreen upon UV ray exposure. Over time, melanin build-up within skin cells can result in the dark pigmented spots characteristic of aging. Research shows that variation in the Interferon Regulatory Factor 4 (IRF4) gene is associated with facial pigmented spots. Individuals who carry the T variant of the IRF4 gene have a greater percentage of their facial skin covered by pigmented spots than those who do not carry this genetic variant*.

* Jacobs LC et al. A Genome-Wide Association Study Identifies the Skin Color Genes IRF4, MC1R, ASIP, and BNC2 Influencing Facial Pigmented Spots. Journal of Investigative Dermatology. 2015;135:1735–1742.

IRF4

The IRF4 gene affects the activity of an enzyme involved in melanin synthesis. Variation within this gene has been linked to skin, hair aand eye colour, and even hair graying*. A large study showed that individuals who carry the T variant of the IRF4 gene have a greater percentage of their facial skin covered by pigmented spots than those who do not. This effect is observed consistently, regardless of the person's skin tone**.

* Adhikari K et al. Genome-wide association scan in admixed Latin Americans identifies loci influencing facial and scalp hair features. Nature Communications. 2016; 7:10815.

Your Results

Genes	Markers	Risk Variant	Your Variants
SOD2 / NQ01	rs4880 / rs1800566	Algorithm	CT / CT

Your Risk: Diminished

Recommendation: Since you possess one or more diminished risk variants, your skin's antioxidant capacity may be diminished. To minimize the skin-aging effects of oxidative stress, reduce your exposure to UV rays by wearing sunscreen or long-sleeved clothing and a hat while out in the sun, and avoiding tanning beds. If you smoke, consult your healthcare professional about ways to quit. Prevent exposure to second-hand smoke and heavy pollution.

1 in 3 — *Other ethnicities with Risk Variant*

2 in 3 — *East Asians with Risk Variant*

Antioxidant Capacity

The skin is the body's first line of protection against environmental damage from UV rays, pollution and other oxidative stressors. As such, skin possesses a complex antioxidant defence mechanism involving several endogenous enzymes and free radical-quenching molecules. Over time, oxidative damage promotes aging-associated changes in the skin, such as wrinkles, reduced elasticity, and dryness. The capacity of the skin to counter oxidative damage, or its antioxidant capacity, may affect the aging process. Research shows that genetic variation in superoxide dismutase 2 (SOD2) and NADPH quinone oxidoreductase 1 (NQO1), two enzymes involved in the body's antioxidant defence cascade, is associated with reduced enzymatic activity. Individuals who carry risk variants in these genes may be less efficient at fighting oxidative stress, which could result in older looking skin*.

* Naval J et al. Genetic polymorphisms and skin aging: the identification of population genotypic groups holds potential for personalized treatments. Clinical, Cosmetic and Investigational Dermatology. 2014;7:207–14.

SOD2 and NQO1

The SOD2 gene encodes an enzyme found throughout cells in the body, including the skin. SOD2 is a potent free radical scavenger. Individuals with two copies of the T variant have reduced SOD2 enzyme activity, compared to those with two copies of the C variant*. The NQO1 gene encodes an enzyme that restores the antioxidant ability of coenzyme Q10. Coenzyme Q10 is a vitamin-like substance that our body produces naturally, although it can also be obtained from the diet and supplements. It is an important endogenous antioxidant that scavenges free radicals, protects skin cells against UV damage and reduces inflammation**. Carrying the T variant of the NQO1 gene results in a less active enzyme, which is less able to recycle coenzyme Q10 into its active form. Individuals with the risk variants of SOD2 or NQO1 may be more susceptible to oxidative stress, and their skin may be more vulnerable to the aging effects of UV rays, tobacco smoke, and other environmental pollutants.

* Flekac M et al. Gene polymorphisms of superoxide dismutases and catalase in diabetes mellitus. BMC Medical Genetics. 2008;9:30.
** Vollmer DL et al. Enhancing Skin Health: By Oral Administration of Natural Compounds and Minerals with Implications to the Dermal Microbiome. International Journal of Molecular Sciences. 2018;19:3059-93.

Your Results

Gene	Marker	Risk Varaiant	Your Variant
MMPa1	rs1799750	GG	GG

Your Risk: Elevated

Recommendation: Since you possess two copies of the MMP1 G variant, you produce more MMP1 enzyme and your rate of collagen breakdown is higher than average. Therefore, you are at a higher risk of premature loss of skin elasticity, especially if you are exposed to UV rays regularly. To slow down this loss of elasticity and better manage wrinkles and fine lines, reduce your exposure to UV rays and practice sun safety by wearing sunscreen or protective clothing, and avoiding tanning beds.

1 in 10 East Asians with Risk Variant

1 in 6 Hispanics with Risk Variant

1 in 4 Africans and Europeans with Risk Variant

Loss of Elasticity

As skin ages, it loses its inherent elasticity. The resulting stiffness affects the skin's architecture, contributing to the appearance of fine lines and wrinkles. Collagen is a protein produced by skin cells which plays a role in the elasticity characteristic of youthful skin, but its production and turnover decreases as we age. Furthermore, damage from exposure to UV rays also decreases elasticity. This is partly mediated by enzymes called matrix metalloproteinases (MMPs) that break down and alter the structure of collagen and other connective tissue molecules. The presence of MMPs increases after UV ray exposure. Research shows that genetic variation in MMP1 is associated with increased enzyme activity, which results in more collagen breakdown*. This may lead to premature loss of skin elasticity and more wrinkling.

MMP1

MMP1 encodes an enzyme that carries out a significant proportion of skin collagen degradation upon exposure to UV rays. Some individuals carry two extra copies of the G variant, which leads to increased production of the resulting MMP1 enzyme. This, in turn, results in a higher level of collagen breakdown**. Individuals who carry two G variants may experience a greater loss of elasticity over time, and they may be particularly vulnerable to the skin-aging effects of UV rays.

* Quan T et al. Matrix-degrading metalloproteinases in photoaging. Journal of Investigative Dermatology Symposium Proceedings. 2009;14(1):20-4.
** Rutter JT et al. A single nucleotide polymorphism in the matrix metalloproteinase-1 promoter creates an Ets binding site and augments transcription. Cancer Res. 1998;58(23):5321–5325.

Your Results

Genes	Markers	Risk Variant	Your Variants
GL01	rs1130534 / rs1049346	Algorithm	AT / CT

Your Risk: Slightly Diminished

Recommendation: Since you possess this specific combination of risk variants, you have a partially diminished AGE-neutralizing GLO1 enzymatic activity. As such, your skin may be more susceptible to glycation damage. To better manage the fine lines, wrinkles and saggy skin associated with AGEs, you should minimize excessive UV ray exposure. Consuming less sugar will also result in fewer AGEs. Furthermore, eating plenty of antioxidant-rich fruits and vegetables and limiting sugary foods and beverages will benefit your general health.

1 in 3 Europeans or Hispanics with Risk Variant

1 in 2 East Asians or Africans with Risk Variant

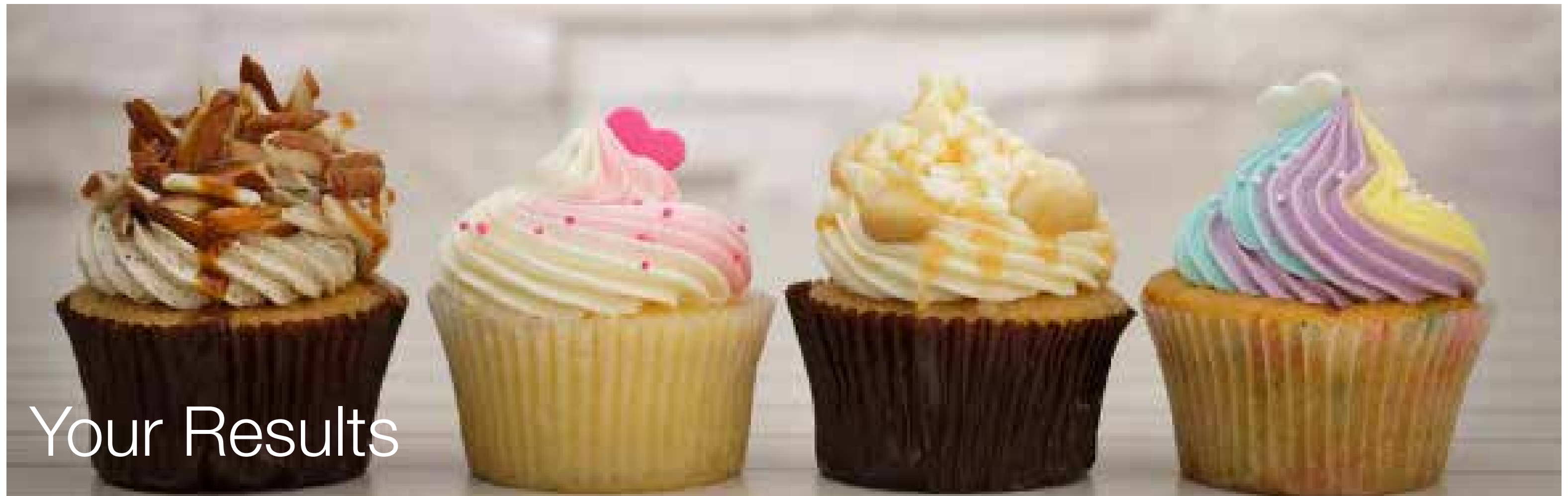
Advanced Glycation End Products

As we age, sugar molecules from the foods we eat build up inside our body and stick to proteins and lipids, affecting their function. These sugar-bound complexes are called advanced glycation end products (AGEs). In the skin, sugars bind to collagen and elastin, which are important dermal structural components. This makes both collagen and elastin brittle and prone to breaking, which leads to a more wrinkled, saggy skin appearance. While this process occurs naturally over time, eating too much sugar, as well as exposure to UV rays, may also accelerate skin glycation*. Other sources of AGEs are foods browned or prepared at high temperatures, such as donuts, barbequed meats and caramel-coloured soft drinks. These AGEs promote oxidation and inflammation, and they damage not only the skin but tissues throughout the body. The enzyme glyoxalase 1 (GLO1) is involved in the body's defence mechanism against AGEs. Research shows that variation in the GLO1 gene affects the enzyme's activity, which may make some individuals more susceptible to the skin-aging effects of AGEs**.

* Draelos ZD. Aging skin: The role of diet: Facts and controversies. Clinics in Dermatology. 2013;31:701–706.
** Peculis R et al. Identification of glyoxalase 1 polymorphisms associated with enzyme activity. Gene. 2013;515:140–143.

GLO1

The GLO1 gene encodes an enzyme that plays a central role in the body's defence mechanism against AGEs. GLO1 neutralizes a highly reactive AGE called methylglyoxal. Research on GLO1 has found that two variants within this gene affect the resulting enzyme's activity in the blood. Individuals can carry 0 to 4 copies of the risk variants, and GLO1 activity decreases proportionally with each additional risk variant. Individuals who carry more risk variants may be less efficient at neutralising AGEs, and the resulting glycation damage could lead to fine lines, wrinkles and sagging skin.



Your Results

Gene	Marker	Risk Variant	Your Variant
GLUT2	rs5400	CT or TT	CT

Your Risk: Elevated

Recommendation: Since you possess the CT or TT variant of the GLUT2 gene, you are at an increased risk of over-consuming sugar. This means you may be more likely to enjoy sweet foods and beverages. Be mindful of this craving and aim to keep your intake of added sugar below 5% of your total daily energy intake. A high intake of added sugar is linked to overweight & obesity and cardiometabolic disease.

1 in 3 Hispanics with Risk Variant

1 in 4 Europeans with Risk Variant

3 in 4 Africans with Risk Variant

Sugar Preference

Consuming a diet high in sugar can lead to the excessive formation of AGEs, which bind to collagen and elastin and make them prone to breakage. This results in a more wrinkled, saggy skin appearance*. Sugar intake is partly determined by our sweet taste preference and cravings for certain foods and beverages. There is considerable variability in individuals' preferences and cravings for sweet foods and beverages. There are many factors that may impact your preference for sugary foods, including the age that you are first introduced to sweets, and psychological associations between consuming these foods and certain life experiences or emotions. In the brain, there are even 'pleasure-generating' signals given off in response to eating or drinking something sweet. Research has shown that your intake of sweet foods can also be determined by your genes**.

* Draelos ZD. Aging skin: The role of diet: Facts and controversies. Clinics in Dermavtology. 2013;31:701–706.
** Eny KM et al. Genetic variant in the glucose transporter type 2 is associated with higher intakes of sugars in two distinct populations. Physiol Genomics. 2008;33:355-360.

GLUT2

Glucose transporter type 2 (GLUT2) is involved in regulating glucose (sugar) in the body. The expression of this gene has been found in areas of the brain that are involved in controlling food intake. Individuals who possess the TT or TC variant of this gene seem to have a greater preference for sweet foods and beverages and are more likely to over-consume sugar.

	Amount (g)
Soft drink, cola (1 can)	36
Lollies (40g)	26
Caramels (40g)	26
Maple syrup (2 Tbsp)	24
Milk chocolate (50g)	22
Jellybeans (10 beans)	20
Flourless chocolate cake (1 slice)	20
Banana bread (1 slice)	18
Icy pole (1)	10
Jam (1 Tbsp)	10

Source: Health Canada's Nutrient Value of Some Common Foods and Nutrient Tables for Use in Australia (NUTTAB2010)

Your Results

Gene	Markers	Risk Variant	Your Variants
BCM01	rs11645428	GG	GG

Your Risk: Elevated

Recommendation: Since you possess the GG variant of the BCMO1 gene, it is important for you to meet the RDA for vitamin A. Consuming foods that are higher in pre-formed vitamin A can help you to meet your needs more easily. These foods include fish, liver, eggs, and dairy products. Meeting your recommendations for vitamin A will help to support healthy immunity, vision, and reproductive health. It will also act as an antioxidant when consumed in the form of beta-carotene (plant sources). Women should aim for 700 mcg RAE/day and men should aim for 900 mcg RAE/day.

1 in 5 People with Risk Variant across ethnicities

Vitamin A (Beta-Carotene)

Vitamin A is a fat-soluble vitamin important for eye and skin health. Indeed, vitamin A receptors are found in the skin, highlighting the role of this nutrient in skin-related processes*. Beta-carotene, a precursor of active vitamin A, is an antioxidant found in certain fruits and vegetables that are orange-red in colour, and it has been shown to protect against sun damage*. Beta-carotene can be converted to pre-formed vitamin A (retinol) in the body to exert its biological functions. Research shows that individuals with the GG version of the BCMO1 gene are inefficient at converting beta-carotene to active vitamin A**. These individuals are considered low responders to dietary beta-carotene, so consuming enough active vitamin A can help ensure circulating levels of active vitamin A are adequate to support vision and skin health.

* Stahl W et al. Carotenoids and carotenoids plus vitamin E protect against ultraviolet light-induced erythema in humans. American Journal of Clinical Nutrition. 2000;71:795-8.
Pappas A et al. Nutrition and skin. Reviews in Endocrine and Metabolic Disorders. 2016;17:443–448.

BCMO1

Beta-carotene mono-oxygenase 1 (BCMO1) is an enzyme that plays a key role in the conversion of beta-carotene into the active form of vitamin A. Beta-carotene is a plant form of vitamin A. Individuals who possess the GG version of the BCMO1 gene are inefficient at converting beta-carotene into the active form of vitamin A. These individuals need to ensure they are consuming adequate amounts of vitamin A, particularly pre-formed vitamin A.

	High in Preformed Vitamin A	Amount (mcg RAE)
Pumpkin, canned (1/2 cup)		1010
Carrots, cooked (1/2 cup)		650
Sweet potato, boiled without skin (1/2 medium)		600
Bluefin tuna (75g)	✓	530
Spinach, boiled (1/2 cup)		500
Butternut pumpkin (1/2 cup)		340
Goat cheese, hard (50g)	✓	240
Eggs (2 large)	✓	220
Mackerel (75g)	✓	190

Source: Health Canada's Nutrient Value of Some Common Foods and Nutrient Tables for Use in Australia (NUTTAB2010)

Your Results

Gene	Marker	Risk Variant	Your Variant
GSTT1	Ins or Del	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. Vitamin C can also be consumed in supplement form, either alone or as a multivitamin.

1 in 2 East Asians with Risk Variant

1 in 4 Other Ethnicities with Risk Variant

Vitamin C

Vitamin C is an essential nutrient and powerful antioxidant. Vitamin C circulating in the bloodstream aids in the absorption of non-heme (plant) iron, and supports the formation of collagen, a protein used to make skin, connective tissue, and blood vessels, along with playing a key role in wound healing. Vitamin C also helps restore the antioxidant properties of vitamin E, which protects the skin against free radical damage. Lower dietary intakes of vitamin C have been associated with a wrinkled appearance and skin dryness *. 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, which may contribute to a more aged skin appearance. Studies** have shown that the ability to process vitamin C efficiently depends on a gene called GSTT1.

* Cosgrove MC et al. Dietary nutrient intakes and skin-aging appearance among middleaged American women. American Journal of Clinical Nutrition. 2007;86:1225–31.
** 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 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.

Sources of Vitamin C	Amount (mg)
Red pepper (1)	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

Your Results

Gene	Markers	Risk Variant	Your Variants
CYP2R1 GC	rs10741657 / rs2282679	Algorithm	GA / GG

Your Risk: Elevated

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.

1 in 2 People with Risk Variant(s) across ethnicities

Vitamin D

Vitamin D is essential to calcium metabolism and increasing calcium absorption. 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 tissues and organs in the body, including the skin. Indeed, vitamin D protects skin cells from UV-associated damage. While vitamin D is synthesized by the skin upon exposure to UV light, exercising caution around sun exposure is important to prevent accelerated aging and diminish the risk of skin cancer. Vitamin D can also be obtained from the diet. Vitamin D deficiency is diagnosed by measuring the most common form of vitamin D in the blood, which is 25-hydroxy vitamin 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)
Atlantic salmon (75g)	680
Vitamin D mushrooms, raw (1/2 cup)	532
Whitefish (75g)	448
Rainbow trout (75g)	192
Smoked salmon (40g)	168
Halibut (75g)	144
Fortified soy 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, Canadian Nutrient File and Food Standards Australia New Zealand (NUTTAB2010)

Your Results

Gene	Marker	Risk Variant	Your Variant
APOA5	rs12272004	CC	CA

Your Risk: **Typical**

Recommendation: Since you possess the typical intergenic risk variant near APOA5, you are likely to have adequate alpha-tocopherol levels in your blood to support skin health, as long as you meet the RDA for vitamin E of 15 mg/day (21 IU/day). Good sources of vitamin E include almonds, sunflower seeds, sunflower oil, hazelnuts, and grapeseed oil.

7 in 10 Africans with Risk Variant

1 in 4 Hispanics with Risk Variant

9 in 10 Europeans and East Asians with Risk Variant

Vitamin E

Vitamin E is a potent fat-soluble antioxidant essential to protect skin against the aging-related damage induced by lipid peroxidation and collagen cross-linking. Most vegetable oils, nuts and seeds are excellent sources of vitamin E. Grapeseed oil, sunflower oil, canola oil, and flaxseed oil are very high in vitamin E. Research has shown that variation in an intergenic region near the apolipoprotein A5 (APOA5) gene is associated with blood concentrations of alpha-tocopherol, the most abundant form of vitamin E in the blood*. Individuals who carry two copies of the C variant in this region have lower alpha-tocopherol concentrations in their blood than those with only one or zero copies of the C variant**.

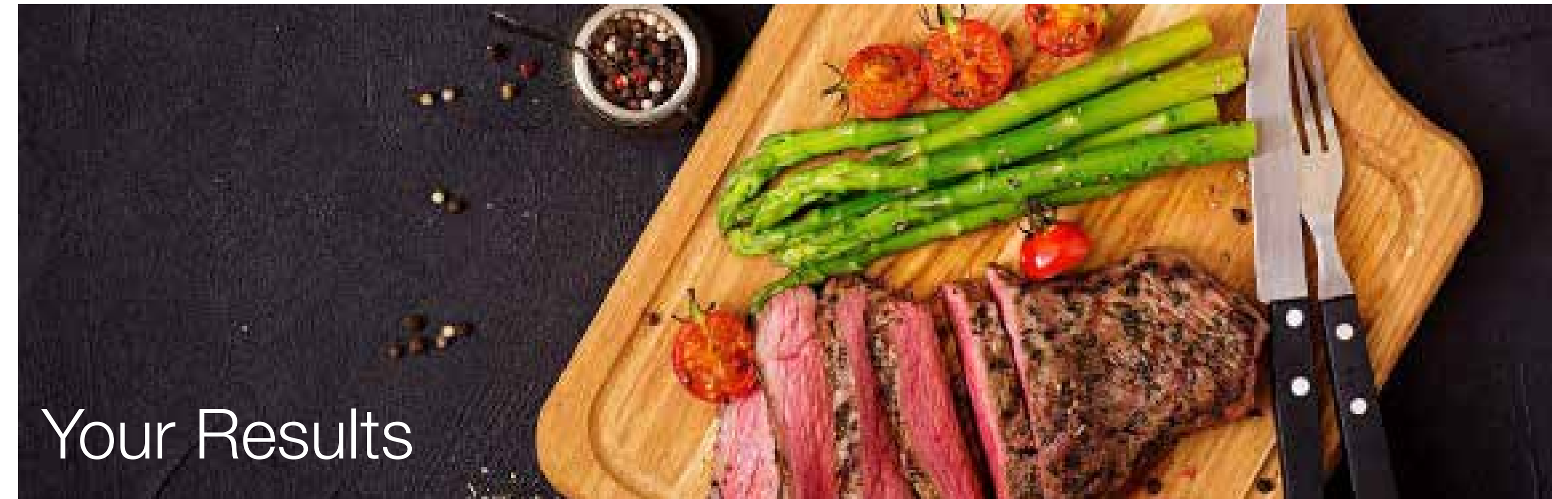
*Ferrucci L et al. Common Variation in the - Carotene 15,15 -Monooxygenase 1 Gene Affects Circulating Levels of Carotenoids: A Genome-wide Association Study. American Journal of Human Genetics. 2009;84(2):123-33.
**Major JE et al. Genome-wide association study identifies common variants associated with circulating vitamin E levels. Human Molecular Genetics. 2011;20(19):3876–83.

Intergenic variant near APOA5

APOA5 is involved in lipid metabolism, particularly affecting triglyceride levels. As a lipid-soluble micronutrient, vitamin E is transported together with triglycerides in the bloodstream. The intergenic variant examined in this report is strongly associated with APOA5 variation that influences triglyceride concentrations, in turn affecting the amount of alpha-tocopherol present in the circulation.

Sources of Vitamin E	Amount (mg)
Almonds (1/4 cup)	9.3
Sunflower seeds, roasted (1/4 cup)	8.5
Sunflower oil (1 Tbsp)	7.7
Hazelnuts, dry roasted (1/4 cup)	5.2
Grapeseed oil (1 Tbsp)	4.0
Peanut butter (2 Tbsp)	2.9
Peanuts, dry roasted (1/4 cup)	2.6
Flaxseed oil (1Tbsp)	2.4
Canola oil (1 Tbsp)	2.4
Halibut (75g)	2.2
Eggs, hard boiled (2 large)	1.0

Source: Health Canada's Nutrient Value of Some Common Foods and Nutrient Tables for Use in Australia (NUTTAB2010)



Your Results

Gene	Markers	Risk Variant	Your Variants
SLC30A3	rs11126936	CC	CC

Your Risk: **Elevated**

Recommendation: Since you possess the elevated risk variant of SLC30A3, you are at a greater risk of zinc deficiency if you do not meet the RDA. This may make it more difficult for your skin to heal from UV ray damage and wounds, and it may also impact your general health. Ensure that you meet the RDA for zinc (8mg/day for women and 11 mg/day for men) and focus on eating foods with a high zinc bioavailability for effective absorption. Red meat and seafood, especially oysters, are great sources of bioavailable zinc. Most dairy products also provide zinc. Plant foods such as beans, nuts, whole grain bread, and fortified breakfast cereals also contain zinc. The body absorbs plant-sourced zinc less efficiently, so you may need to eat more servings of these foods to meet your needs.

1 in 10 Africans with Risk Variant

1 in 3 Hispanics with Risk Variant

6 in 10 Europeans with Risk Variant

3 in 4 East Asians with Risk Variant

Zinc

The mineral zinc plays a crucial role in numerous metabolic processes. It possesses antioxidant properties and is involved in DNA and protein synthesis and cell division, as well as immune function and wound healing. Zinc also helps protect the skin against UV ray damage*. Dietary sources of zinc include animal foods such as oysters, red meat, poultry and dairy, as well as beans, nuts, whole grains, and fortified products such as breakfast cereals. Importantly, zinc from plant sources is less bioavailable than from animal foods. Research shows that variation in a gene called solute carrier 30A3 (SLC30A3) is associated with zinc concentrations in the blood.

* Park K. Role of Micronutrients in Skin Health and Function. Biomolecules & Therapeutics. 2015;23(3):207-17.

SLC30A3

Given zinc's key role across physiological processes, several types of proteins are involved in its absorption and transport across the body. The SLC30A3 gene encodes one of several different zinc transporters, and it helps regulate zinc concentrations by controlling its release from cells. Different studies have shown that carrying two copies of the C variant of SLC30A3 is linked to lower zinc concentrations in the blood*.

Sources of Zinc	Amount (mg)
Oysters, cooked, breaded and fried, 3 ounces	74.0
Beef chuck roast, braised, 3 ounces	7.0
Lobster, cooked, 3 ounces	3.4
Baked beans, ½ cup	2.9
Chicken, dark meat, cooked, 3 ounces	2.4
Pumpkin seeds, dried, 1 ounce	2.2
Yogurt, 8 ounces	1.7
Cashews, dry roasted, 1 ounce	1.6
Chickpeas, cooked, ½ cup	1.3
Oatmeal, instant, cooked with water, ½ cup	1.1
Milk, 1 cup	1.0

Source: USDA Nutrient Database

*Da Rocha TJ et al. SLC30A3 and SEP15 gene polymorphisms influence the serum concentrations of zinc and selenium in mature adults. Nutrition Research. 2014;34:742–8.
Fujihara J et al. Association of SNPs in genes encoding zinc transporters on blood zinc levels in humans. Legal Medicine. 2018.30:28-33.

International Science Advisory Board

Ahmed El-Sohemy, PhD

Dr. Ahmed El-Sohemy is the Founder of Nutrigenomix Inc. and serves as the President and Chief Scientific 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 is currently a Professor and holds a Canada Research Chair in Nutrigenomics at the University of Toronto. Dr. El-Sohemy has published in the top scientific and medical journals with more than 120 peer-reviewed publications and has given more than 200 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.

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 glycaemic index of foods and demonstrate the breadth of metabolic effects of viscous soluble fiber. 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.