
dante labs
Your Wellness Report

YOUR NUTRIGENOMIC REPORT

INTRODUCTION

This is your personal Nutrigenomic report, providing you with invaluable genetic insights into your unique nutritional needs. Based upon your own DNA, this report lists the recommended nutrients/nutritional supplements that you need due to your own specific genetic variations. Your report highlights any genetic predispositions that you might have when it comes to nutritional/vitamin needs, can help to remove the guesswork when selecting nutritional supplements, and it contributes to an empowering, holistic approach when it comes to your personal diet/food-consumption.

Do not modify diets/meal-plans or supplements recommended or prescribed by your doctor/qualified healthcare professional without first consulting them.

If you have any nutrition, diet, or healthcare related questions, please make sure that you promptly consult your doctor/qualified healthcare professional.

Genetic testing identifies and analyses your DNA and can help to rule out suspected genetic disorders and/or help determine your chances of developing or passing on a specific genetic disorder. Huge benefits from genetic testing also include:

1. Useful results for family planning

2. Guidance for your doctor/qualified healthcare professional in diagnosis and suitable treatments/interventions

3. Early interventions when it comes to medical conditions - which can even save a life.

When a family member has a trait for a disease it's wise to have genetic testing and screening to find out if you are a carrier or have the disease yourself.

Some genetic variations can increase our risk of developing certain health conditions/diseases, other genetic variations can reduce our risk, and some people will develop a health condition/disease when they haven't been at high genetic risk. Whichever way, it's wise to find out your own personal genetic composition and where possible, reduce your risks through lifestyle changes.

As we all differ so much in our characteristics and traits, so do we in our genetic makeup. Our genetic differences are shown through variations in our DNA: the building blocks of life.

As a species, we humans are incredibly genetically diverse and complex. We find that there are some genetic variations more pronounced in one ethnicity over another, for example. It's in us identifying our own personal genetic complexities and variations that we can work towards making constructive changes in our lives to better our health and wellbeing. Put simply: fully understanding your own genetic variations can help to provide a pathway to optimum health and a longer, more fruitful life.

Personal, appropriate genetic testing provides valuable insights but also has its limitations (see below).

LIMITATIONS AND OTHER IMPORTANT INFORMATION

Genetic testing provides you with personal information on your own genetic risk(s), based upon the assessment of specific genetic variants. However, it doesn't report on your entire genetic profile. It doesn't report on all of the genetic variants that relate to a specific disease or condition, and if a variant that is tested doesn't show up, that doesn't mean that there aren't any other genetic variants present that could be related. Indeed, other genetic risk tests could identify separate genetic variants that are related to the same disease/condition. In addition, your environment and lifestyle can be contributory risk factors affecting your risk of developing a disease or health condition.

Future versions of this report may include additional polymorphisms to provide a higher degree of accuracy than this SNP alone does.

Remember that testing is not a substitute for visits to your doctor/qualified healthcare professional. Any questions or concerns that you might have about your genetic test results, or your current health status must be brought up in an appointment with them.

Lots of people find speaking with a genetic counselor or board-certified clinical geneticist very helpful in their genetic journey. Genetic counselling can be valuable in helping you with worries and concerns that you might have about your genetic testing (both prior and post-results), and clinical geneticists can prove essential after you've received your results; to analyse them and potentially diagnose you with a condition/disease. You can access genetic counselors via the Genetic Counselor United Arab Emirates: http://www.geneticcounselor.ae

The results of a genetic test do not provide you with a diagnosis or provide you with information about your current state of health/wellbeing and they mustn't be used by you to make any medical decisions, such as altering any medications that you are taking.

Our genetic testing reports haven't been evaluated by the FDA and by the UAE Ministry of Health & Prevention.Our product isn't intended to diagnose, treat, cure or prevent any medical condition/disease.

INFORMATION FOR HEALTH CARE PROFESSIONALS

Our genetic testing is not intended to diagnose a patient with a disease, to determine suitable medical treatments, or to inform the patient of anything regarding their current or future state of health. Instead, the test is intended to provide patients with personal genetic information to help influence their conversations with clinicians and to help them make constructive lifestyle decisions.

Any decisions regarding patient diagnosis or treatment should be based upon clinical diagnostic testing and/or other information that you consider to be appropriate.

This report is NOT intended for US persons and was not submitted for approval to the US Food and Drug Administration (FDA).

The above information provides an overview of predicted genetic risks to the patient. All information is based solely on genotype data and does not replace a consultation with a doctor/qualified healthcare professional, or, indeed, a complete patient profile.

Doctors/qualified healthcare professionals should also consider family history, symptoms presented, current medical prescriptions, and other factors prior to making any clinical or therapeutic decisions.

KEY SUMMARY

This colour means that you could have an enhanced beneficial reaction linked to the associated trait

This colour means that you could have an average reaction linked to the associated trait

This colour means that you could have a reduced beneficial reaction linked to the associated trait

QUICK SUMMARY

VITAMIN A AND CAROTENOIDS

CONDITION NAME	RESULTS	MAIN MESSAGE
Retinol (A1) - deficiency		People with your genetic profile are likely to have regular retinol levels
Lutein + Zaexanthin – deficiency		People with your genetic profile are likely to have a predisposition for a lutein and zeaxanthin deficiency
Carotene - deficiency		People with your genetic profile are likely to have a predisposition for carotene deficiency

VITAMIN B COMPLEX

CONDITION NAME	RESULTS	MAIN MESSAGE
Thiamine (B1) - deficiency		People with your genetic profile are likely to have regular thiamine levels
Riboflavin (B2) – deficiency		People with your genetic profile are likely to have regular riboflavin levels
Niacin/Nicotinamide (B3) - deficiency		People with your genetic profile are likely to have regular niacin levels
Choline (B4) – deficiency		People with your genetic profile are likely to have a predisposition to develop a choline deficiency
Panthothenic Acid (B5) - deficiency		People with your genetic profile are likely to have regular pantothenic acid levels
Pyridoxine (B6) - deficiency		People with your genetic profile are likely to have a predisposition for pyridoxine deficiency
Biotin (B7) - deficiency		People with your genetic profile are likely to have regular biotin levels
Folic Acid (B9) – deficiency		People with your genetic profile are likely to have regular folic acid levels
Homocysteine levels - deficiency		People with your genetic profile are likely to have a predisposition to develop folate deficiency
Cobalamin (B12) – deficiency		People with your genetic profile are likely to have regular cobalamin levels

VITAMIN C

CONDITION NAME	RESULTS	MAIN MESSAGE
Ascorbic acid - deficiency		People with your genetic profile are likely to have regular ascorbic acid levels

VITAMIN D COMPLEX

CONDITION NAME	RESULTS	MAIN MESSAGE
Ergocalciferol (Vit D2) and Lumisterol (D2 precursor) - deficiency		People with your genetic profile are likely to have regular ergocalciferol and lumisterol levels
Ergocalciferol (Vit D2) - deficiency		People with your genetic profile are likely to have regular ergocalciferol levels
Cholecalciferol (D3) – deficiency		People with your genetic profile are likely to have regular cholecalciferol levels

VITAMIN E

CONDITION NAME	RESULTS	MAIN MESSAGE
Tocopherol - deficiency		People with your genetic profile are likely to have a predisposition for tocopherol deficiency

VITAMIN K COMPLEX

CONDITION NAME	RESULTS	MAIN MESSAGE
Naftochinone/Naphthoquinone (Vit K Precursor) - deficiency		People with your genetic profile are likely to have a predisposition for naftochinone deficiency

MACRONUTRIENTS

CONDITION NAME	RESULTS	MAIN MESSAGE
Calcium - deficiency		People with your genetic profile are not likely to have a predisposition for a calcium deficiency
Magnesium - deficiency		People with your genetic profile are likely to have a predisposition for magnesium deficiency
Phosphorus - deficiency		People with your genetic profile are likely to have regular phosphorus levels
Sodium - balance		People with your genetic profile are not likely to have a regular sodium balance
Potassium - balance		People with your genetic profile are likely to have a regular potassium balance
Na/K ratio		People with your genetic profile are likely to have a regular balance ratio of sodium/potassium

MICRONUTRIENTS

CONDITION NAME	RESULTS	MAIN MESSAGE
Selenium - deficiency		People with your genetic profile are likely to have a predisposition for selenium deficiency
Iron – deficiency		People with your genetic profile are likely to have regular iron levels
Zinc - deficiency		People with your genetic profile are likely to have regular zinc levels
Copper - deficiency		People with your genetic profile are not likely to have a predisposition to develop a copper deficiency

ANTI-OXIDANTS

CONDITION NAME	RESULTS	MAIN MESSAGE
CoE-Q - deficiency		People with your genetic profile are likely to have a predisposition to develop a coenzyme-Q deficiency

CARBOHYDRATES

CONDITION NAME	RESULTS	MAIN MESSAGE
Predisposition to respond positively to a low carbohydrate diet		People with your genetic profile are likely to have an enhanced response to a low carbohydrate diet
Predisposition to excessive consumption of Carbohydrates		People with your genetic profile are likely to have a regular consumption of carbohydrates

CARBOHYDRATES

CONDITION NAME	RESULTS	MAIN MESSAGE
High vs low carbohydrate diets	🟢	People with your genetic profile are likely to have a predisposition to a regular carbohydrate intake
Predisposition to an altered metabolism of starch	🟡	People with your genetic profile are not likely to have a regular starch metabolism

FATS

CONDITION NAME	RESULTS	MAIN MESSAGE
Predisposition to excessive consumption of fats	🔵	People with your genetic profile are likely to have a regular consumption of fat
Predisposition to the accumulation of fat	🟡	People with your genetic profile are likely to have a predisposition to the accumulation of fat
Predisposition to respond positively to a low fat diet	🟢	People with your genetic profile are likely to have a regular response to a low-fat diet
Predisposition to lipoprotein deficiency	🟡	People with your genetic profile are likely to have a predisposition for lipoprotein deficiency
Positive response to a diet high in monounsaturated fats	🟢	People with your genetic profile are likely to have a regular response to diet enriched in monounsaturated fats
Positive response to a diet high in polyunsaturated fats	🟢	People with your genetic profile are likely to have a predisposition to respond positively to a diet enriched in polyunsaturated fats
Negative response to a diet high in unsaturated fats	🟢	People with your genetic profile are likely to have a regular response to a diet enriched in unsaturated fats
Negative response to a diet high in trans-fat	🟢	People with your genetic profile are likely to have a regular response to the intake of trans fatty acids
Postprandial response of triglycerides to high-fat diet meals	🟡	People with your genetic profile tend to have a higher plasma fat concentration.

PROTEIN

CONDITION NAME	RESULTS	MAIN MESSAGE
Risk of gaining fat through the intake of high protein foods	🟢	People with your genetic profile are not likely to have a predisposition to accumulate fat mass due to the intake of high protein foods
Predisposition to feel full with protein intake	🟢	People with your genetic profile are likely to have a predisposition to develop enhanced satiety with daily protein intake

CAFFEINE METABOLISM

CONDITION NAME	RESULTS	MAIN MESSAGE
Plasma levels of paraxanthine	🔵	People with your genetic profile are likely to have a slower paraxanthine metabolic rate
Plasma levels of caffeine	🔵	People with your genetic profile are likely to have a regular caffeine metabolic rate
Plasma levels of theobromine	🟢	People with your genetic profile are likely to have a regular theobromine metabolic rate
Slow metabolizer	🟢	People with your genetic profile are likely to have a regular caffeine metabolic rate

TASTE

CONDITION NAME	RESULTS	MAIN MESSAGE
Perception of salty taste	🔵	People with your genetic profile are likely to have an enhanced taste for salty foods
Salty taste preference	🟢	People with your genetic profile are likely to have an enhanced propensity to prefer salty foods
Propensity to choose sweet foods	🔵	People with your genetic profile are likely to have a regular propensity to prefer sweet foods

ABNORMAL EATING PATTERNS

CONDITION NAME	RESULTS	MAIN MESSAGE
Predisposition to experience an altered sense of fullness	🟡	People with your genetic profile are likely to have an altered sense of satiety after a meal
Predisposition to eat between meals	🔵	People with your genetic profile are not likely to have a predisposition to eat between meals
Predisposition to eat when under stress	🟡	People with your genetic profile are not likely to have a predisposition to eat when under stress
Predisposition to develop addiction to food	🟡	People with your genetic profile are likely to have a predisposition to develop an addiction to food
Predisposition to fatty food addiction	🟡	People with your genetic profile are not likely to have a regular predisposition for fatty food intake
Prudent dietary pattern	🟢	People with your genetic profile are likely to have a healthy dietary pattern

METABOLISM

CONDITION NAME	RESULTS	MAIN MESSAGE
Predisposition to develop fat tissue over lean tissue	🟡	People with your genetic profile are likely to have a predisposition for the accumulation of fat mass vs lean mass
Genetic predisposition to high energy expenditure at rest	🔵	People with your genetic profile are likely to have a regular energy expenditure at rest
Low resting metabolic rate	🟢	People with your genetic profile are likely to have a slow resting metabolic rate
Leptin resistance	🔵	People with your genetic profile are likely to not have a genetic predisposition for leptin-resistance.
Predisposition to respond positively to the Mediterranean diet	🟢	People with your genetic profile are likely to have an enhanced healthy response to the Mediterranean diet

ALLERGY AND INTOLERANCES

CONDITION NAME	RESULTS	MAIN MESSAGE
Genetic sensitivity to gluten	🟡	People with your genetic profile are likely to have gluten sensitivity
Predisposition to lactose intolerance	🟢	People with your genetic profile are not likely to have predisposition for lactose intolerance
Predisposition to develop an egg allergy	🟡	People with your genetic profile are likely to have a predisposition to develop an egg allergy
Predisposition to develop a milk allergy	🟢	People with your genetic profile are not likely to have a predisposition to develop a milk allergy
Predisposition to develop a peanut allergy	🟢	People with your genetic profile are not likely to have a predisposition to develop a peanut allergy
Predisposition to develop a fish allergy	🟡	People with your genetic profile are likely to have a predisposition to develop a fish allergy
Predisposition to develop a molluscs/crustaceans allergy	🟡	People with your genetic profile are likely to have a predisposition to develop a shellfish allergy
Predisposition to develop a soy allergy	🟡	People with your genetic profile are likely to have a predisposition to develop a soy allergy
Predisposition to develop fruit and vegetable allergies	🟡	People with your genetic profile are likely to have a predisposition to develop an allergy to fruits and vegetables
Predisposition to develop seed allergy (generic)	🟡	People with your genetic profile are likely to have a predisposition to develop a seed allergy
Predisposition to develop a salicylate allergy	🟡	People with your genetic profile are not likely to have a predisposition to develop a salicylates allergy

ALLERGY AND INTOLERANCES

CONDITION NAME	RESULTS	MAIN MESSAGE
Tartrazine allergy	🟡	People with your genetic profile are likely to have a predisposition to develop a tartrazine allergy
Predisposition to develop sensitivity to sulphites	🟡	People with your genetic profile are likely to have a predisposition to develop a sulfites sensitivity
Predisposition to develop sensitivity to metabisulphites	🟡	People with your genetic profile are likely to have a predisposition to develop a metabisulphites sensitivity
Predisposition to develop an allergy to bisulfites	🟡	People with your genetic profile are likely to have a predisposition to develop a bisulfites sensitivity
Predisposition to develop a walnut allergy	🟢	People with your genetic profile are not likely to have a predisposition to develop a walnut allergy
Predisposition to develop a kiwi allergy	🟡	People with your genetic profile are likely to have a predisposition to develop a kiwi allergy
Predisposition to develop a pine nut allergy	🟢	People with your genetic profile are not likely to have a predisposition to develop a pine nuts allergy
Predisposition to develop a wheat allergy	🟢	People with your genetic profile are not likely to have a predisposition to develop a wheat allergy
Predisposition to develop a corn allergy	🔵	People with your genetic profile are not likely to have a predisposition to develop a corn allergy
Predisposition to develop an amaranth grain allergy	🔵	People with your genetic profile are not likely to have a predisposition to develop a amaranth allergy
Predisposition to develop a cassava allergy	🔵	People with your genetic profile are not likely to have a predisposition to develop a cassava allergy
Predisposition to develop a quinoa allergy	🔵	People with your genetic profile are not likely to have a predisposition to develop a quinoa allergy
Sensitivity to nickel	🔵	People with your genetic profile are likely to have regular nickel sensitivity

RETINOL (A1) - DEFICIENCY

RESULTS

Retinol (vitamin A1) is a fat-soluble vitamin. It is found in some foods and used as a dietary supplement to treat and prevent vitamin A deficiency. This deficiency can cause xerophthalmia in severe cases and is common in some geographic regions. A single dose by mouth or by intramuscolar injection is recomannded. Retinol is also used to stem complications in people with measles. At normal doses, retinol is well tolerated by the body, but at higher doses, it causes problems such as hepatomegaly, dry skin, and hypervitaminosis A. If high doses of vitamin A are taken during pregnancy, it can cause congenital disabilities. This vitamin is essential for visual acuity, the normal development of bones and teeth, reproductive function, and the health of skin and mucous membranes. It is also an important antioxidant [1]. Food-borne retinol is found in fish, dairy products and meat; it can be absorbed in an already biologically active form (normally present in animal derivatives) or in the form of provitamins that the body must convert before being usable. They are obtained from plants (especially red, yellow and dark green) and derivatives. However, vitamin A is mainly stored in the liver and released into the bloodstream to reach target cells or tissues when needed. Vitamin A deficiency is common in developing countries where it is responsible for thousands of night blindness causes. Night blindness is one of the earliest manifestations of vitamin A deficiency. Vitamin A deficiency contributes to blindness by making the cornea very dry, but it also damages the retina. Other deficiency symptoms are difficulty in conception/infertility, developmental delays, throat and chest infections, poor wound healing, acne and skin disorders [2].

People with your genetic profile are likely to have regular retinol levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
BCO1	rs119478057	CC

REFERENCES

[1] Dawson MI. The importance of vitamin A in nutrition. Curr Pharm Des. 2000 Feb;6(3):311-25.
[2] Wiseman EM, Bar-El Dadon S, Reifen R. The vicious cycle of vitamin a deficiency: A review. Crit Rev Food Sci Nutr. 2017 Nov 22;57(17):3703-3714.

LUTEIN + ZAEXANTHIN – DEFICIENCY

RESULTS

Lutein is a natural carotenoid in the family of xanthophyll. Lutein is quite essential for human dietary intake. It is synthesized only by plants and is easily found in green leafy vegetables such as spinach, kale but also in yellow carrots, but is also found in egg yolks and animal fat. Humans, and animals in general, obtain lutein from the diet and, although the mechanism is not yet fully understood, from the blood, lutein is carried into the retina where it is absorbed by the macula lutea [1]. Its vitality in animal fats is due to its fat solubility. A good supply of lutein can reduce the risk of developing eye diseases such as cataracts. According to some studies, consumption of more than 2.4 mg of lutein per day from food and supplements was significantly correlated with a reduced incidence of lens opacity, according to some studies [2,3].

People with your genetic profile are likely to have a predisposition for a lutein and zeaxanthin deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PDK1L2	rs9708919	CT
BCMO1	rs6564851	TG

REFERENCES

[1] Buscemi S, Corleo D, Di Pace F, Petroni ML, Satriano A, Marchesini G. The Effect of Lutein on Eye and Extra-Eye Health. Nutrients. 2018 Sep 18;10(9):1321.
[2] Manayi A, Abdollahi M, Raman T, Nabavi SF, Habtemariam S, Daglia M, Nabavi SM. Lutein and cataract: from bench to bedside. Crit Rev Biotechnol. 2016 Oct;36(5):829-39.
[3] Eisenhauer B, Natoli S, Liew G, Flood VM. Lutein and Zeaxanthin-Food Sources, Bioavailability and Dietary Variety in Age-Related Macular Degeneration Protection. Nutrients. 2017 Feb 9;9(2):120.

CAROTENE - DEFICIENCY

RESULTS

Carotene refers to a series of compounds having the brute formula C40Hx with no oxygen atom. These compounds usually cannot be synthesized by animals, with some exceptions in spiders and aphids. Carotenes are pigments with a crucial role in photosynthesis. Carotene is responsible for the typical orange pigmentation of some fruits and vegetables such as carrots, sweet potatoes, chanterelle, and orange cantaloupe melon. Pigmentation from carotenes is partially present in animals, as they are stored in fatty tissue, which takes a typical yellowish hue [1]. One of the most important carotenes is β-Carotene. β-Carotene is stored in the liver and converted when needed. [2]. β-carotene is mainly present in fruits (red,orange and yellow ones) and vegetables. Still, it is also possible to find it in other vegetables (green or dark green) with many other antioxidant compounds important for good health. Due to the lipophilic properties of Vitamin A, consuming these foods with other fatty foods can improve absorption. Food highest in beta carotene includes carrots, sweet potatoes, dark, leafy greens, kale and spinach, romaine lettuce, squash, cantaloupe, red and yellow, and peppers, apricots, peas, broccoli. Beta carotene is also found in herbs and spices such as paprika, cayenne, chili, parsley, cilantro, marjoram, sage, coriander. Pairing these foods, herbs, and spices with a healthy fat such as olive oil, avocado, or nuts and seeds can help carotene absorption [3].

People with your genetic profile are likely to have a predisposition for carotene deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PKD1L2 - BCO1	rs6564851	TG

REFERENCES

[1] Langi P, Kiokias S, Varzakas T, Proestos C. Carotenoids: From Plants to Food and Feed Industries. Methods Mol Biol. 2018;1852:57-71.
[2] Johnson EJ. The role of carotenoids in human health. Nutr Clin Care. 2002 Mar-Apr;5(2):56-65.
[3] Platel K, Srinivasan K. Bioavailability of Micronutrients from Plant Foods: An Update. Crit Rev Food Sci Nutr. 2016 Jul 26;56(10):1608-19.

THIAMINE (B1) - DEFICIENCY

RESULTS

Thiamine (Vitamin B1) is involved in a large number of physiological processes in the human body. It supports various reactions at the molecular level, plays a role in propagating nerve impulses, and participates in the maintenance of myelinated sheath. It is a water-soluble vitamin that can be easily found in meat, beef, pork, legumes, whole grains, and nuts. It is important to note that some food preparations or other foods or products can quickly destroy vitamin B1 due to the presence of thiaminase or the actual food preparation procedure. Thiamine deficiency can affect the cardiovascular, nervous and immune systems, commonly seen in wet beriberi, dry beriberi, or Wernicke-Korsakoff syndrome. This condition occurs mainly in populations that largely consume rice as their main food source and in patients with chronic alcohol abuse. Another reason for vitamin B1 deficiency is a poor dietary intake, malabsorption, increased vitamin B1 loss or use, and even some medications that can lead to thiamine deficiency, such as diuretics [1]. Generally, thiamine deficiency is linked to a limited food intake, especially in areas with a diet mainly focused on rice and controlled cereals. It is widely present in patients with chronic alcohol abuse or other chronic diseases. Other subjects at risk of thiamine deficiency are pregnant women, those in need of parental nutrition, people who have undergone bariatric surgery, those with poor general nutritional status and patients on chronic diuretic therapy. The human body can store an amount of vitamin B1, so the first symptoms begin to appear a month after the last intake of B1. If the central nervous system is involved, it can evolve into dry beriberi, with impaired reflexes and symmetrical motor and sensory deficits in the extremities; myelin loss is observed without any acute inflammation. In the worst case, it can evolve into Wernicke's encephalopathy [2].

People with your genetic profile are likely to have regular thiamine levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
NRG1	rs7817052	TT

REFERENCES

[1] Lonsdale D. Thiamin. Adv Food Nutr Res. 2018;83:1-56.
[2] Polegato BF, Pereira AG, Azevedo PS, Costa NA, Zornoff LAM, Paiva SAR, Minicucci MF. Role of Thiamin in Health and Disease. Nutr Clin Pract. 2019 Aug;34(4):558-564.

RIBOFLAVIN (B2) – DEFICIENCY

RESULTS

Riboflavin (Vitamin B2) is a vitamin characterized by its thermostability and water solubility. It is used to metabolize fats, proteins and carbohydrates into glucose. It is also involved in the proper functioning of the immune system, skin, and hair; It is also an excellent antioxidant factor. Riboflavin also helps convert tryptophan into niacin, which activates vitamin B6. The role of metabolism is possible because riboflavin is a crucial factor in the reactions of the FMN and FAD coenzymes. Without riboflavin, these two compounds cannot function, so there is a block in the metabolism of fats, proteins, and carbohydrates. Some preventable diseases manageable with adequate riboflavin are anemia, cataracts, migraines, and thyroid dysfunction. Usually, vitamin B2 is readily absorbed from the gastric tract, so it is important to make sure you have a good supply. Riboflavin deficiency can result from inadequate dietary intake or endocrine abnormalities and is usually linked to other vitamin B deficiencies. The main antioxidant riboflavin works as well as glutathione. Glutathione works to destroy free radicals and detoxify the liver, as free radicals can cause various diseases to develop. Riboflavin deficiency can also result from chronic diarrhea, liver disease, alcoholism, and hemodialysis [1].

People with your genetic profile are likely to have regular riboflavin levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GG

REFERENCES

[1] Thakur K, Tomar SK, Singh AK, Mandal S, Arora S. Riboflavin and health: A review of recent human research. Crit Rev Food Sci Nutr. 2017 Nov 22;57(17):3650-3660.

NIACIN/NICOTINAMIDE (B3) - DEFICIENCY

RESULTS

Vitamin B3, or niacin, is a water-soluble vitamin. It is found in bran, yeast, eggs, peanuts, poultry, red meat, fish, whole grains, legumes, and seeds. It is involved in lipid metabolism and can be linked to two conditions: vitamin B3 deficiency (also known as pellagra) and dyslipidemia. Pellagra indicates severe vitamin B3 deficiency, characterized by skin problems, vomiting, constipation or diarrhea, several neurological symptoms such as depression; apathy; headache; fatigue; memory loss that can progress to aggressive, paranoid and suicidal behaviors; and auditory and visual hallucinations [1]. The disease can progress to anorexia and eventually death. The leading cause of niacin deficiency is a low dietary intake [2].

People with your genetic profile are likely to have regular niacin levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
RBFOX3	rs12946859	CC

REFERENCES

[1] Meyer-Ficca M, Kirkland JB. Niacin. Adv Nutr. 2016 May 16;7(3):556-8.
[2] Redzic S, Gupta V. Niacin Deficiency. 2021 Feb 6. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.

CHOLINE (B4) – DEFICIENCY

RESULTS

Choline is a nutrient naturally found in many foods. It is widely needed by different organs such as the brain to regulate mood and memory, is involved in muscle control and many other functions. It is also common to find choline in the membrane surrounding cells. The human body can synthesize a small amount of choline in the liver daily, but the primary source is diet. The recommended daily intake for men and women is 550 mg/day and 425 mg/day, respectively [1]. Choline deficiency causes liver damage, and excess choline can increase cardiovascular disease risk [1]. Studies say that low plasma choline is associated with an increased risk of neural tube defects. Europeans have a higher probability of developing organ dysfunctions linked to a low choline intake [2].

People with your genetic profile are likely to have a predisposition to develop a choline deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PEMT	rs12325817	CC
PEMT	rs4646343	GT

REFERENCES

[1] Wortmann SB, Mayr JA. Choline-related-inherited metabolic diseases-A mini review. J Inherit Metab Dis. 2019 Mar;42(2):237-242. doi: 10.1002/jimd.12011. Epub 2019 Jan 25. Erratum in: J Inherit Metab Dis. 2020 Jan;43(1):156.
[2] Meyer KA, Shea JW. Dietary Choline and Betaine and Risk of CVD: A Systematic Review and Meta-Analysis of Prospective Studies. Nutrients. 2017 Jul 7;9(7):711.

PANTHOTHENIC ACID (B5) - DEFICIENCY

RESULTS

Pantothenic acid (Vitamin B5) is a water-soluble vitamin and an essential nutrient in animals. It is mainly involved in the synthesis of coenzyme-A but also in the biosynthesis and metabolism of proteins, carbohydrates, and fats [1]. Due to its many biological roles, dietary deficiency of pantothenic acid, although rare and usually associated with other nutrient deficiencies, has several adverse effects. Symptoms of vitamin B5 deficiency are generally similar to other deficiencies in the B vitamin groups. Reduced energy production, irritability, fatigue, and apathy are typical. Other symptoms may be linked to acetylcholine impairment, leading to a variety of neurological effects such as numbness, paraesthesia, and muscle cramps. This deficiency can cause hypoglycemia or increased insulin sensitivity. Additional symptoms could include restlessness, feeling unwell, sleep disturbances, nausea, vomiting, and abdominal cramps. More serious (but reversible) conditions, such as adrenal insufficiency and hepatic encephalopathy, have been observed in rare circumstances. A balanced diet can avoid the risk of deficiency. It is easy to find vitamin B5 in many foods such as highly fortified ready-to-eat cereals, formula milk, energy bars and dry foods, dried shiitake mushrooms, liver, kidneys, egg yolks, and sunflower seeds. For this reason, it is easy to have a sufficient daily food intake, and its deficiency is linked to severe malnutrition or genetic mutations. The average daily intake is 4.0 mg for women and 5.5 mg for men [2].

People with your genetic profile are likely to have regular pantothenic acid levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
IGFBP7	rs13141016	AA
RTEL1-TNFRSF6B, RTEL1	rs2738784	AA
PANK2	rs1131692166	GG

REFERENCES

[1] Sanvictores T, Chauhan S. Vitamin B5 (Pantothenic Acid). 2020 Sep 27. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.
[2] Kelly GS. Pantothenic acid. Monograph. Altern Med Rev. 2011 Sep;16(3):263-74.

PYRIDOXINE (B6) - DEFICIENCY

RESULTS

Vitamin B6 is a water-soluble widely found in many foods. Generally, pyridoxine is the most common form found in multivitamins. Vitamin B6 is involved as a cofactor in over 100 enzymatic reactions, including the metabolism of amino acids, in particular, that of homocysteine, in the metabolism of lipids and in that of carbohydrates. Vitamin B6 also plays a role in cognitive development through neurotransmitter synthesis, immune function with interleukin-2 production, hemoglobin formation, and fetal brain development. Vitamin B6 deficiency is rare with proper diet and is usually found in association with other B vitamin deficiencies such as folic acid and vitamin B12. Low plasma levels of active B6 are found in chronic alcohol dependence, obesity, pregnancy, preeclampsia and eclampsia, malabsorption states such as celiac disease, inflammatory bowel disease and bariatric surgery. It can also be found in patients with chronic renal failure (especially those undergoing hemodialysis or peritoneal dialysis) and autoimmune diseases such as rheumatoid arthritis, which causes increased B6 catabolism [1]. The human body cannot store vitamin B6, so a daily supply is required. Since animal sources have higher bioavailability, vegans and vegetarians may need supplementation. The main multivitamin supplement is pyridoxine hydrochloride. Amounts of vitamin B6 higher than 250 mg per day can cause toxicity with negative effects on the skin, intestines and brain system. There are many symptoms and clinical findings associated with vitamin B6 deficiency: some specific disease states with similar symptoms include porphyria, beriberi (thiamine deficiency), normocytic anemias, depression and various disorders of cognitive function, folic acid deficiency, isoniazid toxicity and neonatal convulsions. Vitamin B6 deficiency is effectively treated with adequate oral or parenteral supplementation [2].

People with your genetic profile are likely to have a predisposition for pyridoxine deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
NBPF3	rs4654748	CC

REFERENCES

[1] Abosamak NER, Gupta V. Vitamin B6 (Pyridoxine). 2021 Feb 6. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.
[2] Brown MJ, Ameer MA, Beier K. Vitamin B6 Deficiency. 2021 Feb 11. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.

BIOTIN (B7) - DEFICIENCY

RESULTS

Biotin, which is part of the Vitamin B group, is an essential nutrient. Its biological role is linked to the metabolism of lipids and fatty acids, glucose and amino acids [1]. A prevalent cause of biotin deficiency is alcohol abuse, as alcohol inhibits biotin absorption. It is common to find a slight deficiency in pregnant women even with normal biotin intake because pregnancy requires more biotin. Usually, biotin deficiency is shown by skin rashes, hair loss and brittle nails. Consequently, biotin supplements are often used to improve hair, nails and skin health. Normally biotin need varies depending on age, sex, habits and therapies. The primary way to treat biotin deficiency is to treat the cause, but it is common to use oral biotin supplements. Biotin supplements have a high bioavailability, so it is common to use a dose of 5mg/day regardless of the etiology of the deficiency [2].

People with your genetic profile are likely to have regular biotin levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
BTD	rs397514436	GG
BTD	rs146015592	GG
BTD	rs397514367	GG
BTD	rs28934601	AA
BTD	rs397514402	GG
BTD	rs80338685	AA
BTD	rs138818907	CC
BTD	rs104893688	CC
BTD	rs80338686	CC
BTD	rs146136265	CC

REFERENCES

[1] Mock DM. Biotin: From Nutrition to Therapeutics. J Nutr. 2017 Aug;147(8):1487-1492. doi: 10.3945/jn.116.238956. Epub 2017 Jul 12
[2] Saleem F, Soos MP. Biotin Deficiency. 2020 Apr 20. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.

FOLIC ACID (B9) – DEFICIENCY

RESULTS

Folic acid is part of the vitamin B family and is characterized by its water solubility. Due to this solubility, folic acid is easily excreted by urine and the body can not store the vitamin for a long time. Daily folic acid intake through food or supplements is needed to maintain adequate levels of folic acid in serum. Leafy vegetables and livers are rich sources of folic acid [1]. Women of reproductive age have a special need for this vitamin because it is primarily involved in the fetus'brain development. Optimal folic acid intake can prevent significant congenital disabilities such as spina bifida and anencephaly. A deficiency in folic acid can lead to diarrhea, greying of hair, ulcers (both mouth and peptic), growth disorders and glossitis [2].

People with your genetic profile are likely to have regular folic acid levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GG

REFERENCES

[1] Sijilmassi O. Folic acid deficiency and vision: a review. Graefes Arch Clin Exp Ophthalmol. 2019 Aug;257(8):1573-1580.
[2] Viswanathan M, Treiman KA, Kish-Doto J, Middleton JC, Coker-Schwimmer EJ, Nicholson WK. Folic Acid Supplementation for the Prevention of Neural Tube Defects: An Updated Evidence Report and Systematic Review for the US Preventive Services Task Force. JAMA. 2017 Jan 10;317(2):190-203.

HOMOCYSTEINE LEVELS - DEFICIENCY

RESULTS

Homocysteine (Hcy) is an amino acid produced by the demethylation of the essential amino acid methionine, characterized by the presence of sulfur. Hcy level in blood is considered a good indicator for B12 and folate deficiency and a high concentration has been identified as a risk marker and factor for ischemic stroke events [1,2,3].

People with your genetic profile are likely to have a predisposition to develop folate deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GG
DPEP1 - CHMP1A	rs154657	GA
CPS1	rs1047891	CA

REFERENCES

[1] Hannibal L, Lysne V, Bjørke-Monsen AL, Behringer S, Grünert SC, Spiekerkoetter U, Jacobsen DW, Blom HJ. Biomarkers and Algorithms for the Diagnosis of Vitamin B12 Deficiency. Front Mol Biosci. 2016 Jun 27;3:27.
[2] Ebara S. Nutritional role of folate. Congenit Anom (Kyoto). 2017 Sep;57(5):138-141.
[3] Kronn D, Goldman ID. Hereditary Folate Malabsorption. 2008 Jun 17 [updated 2017 Apr 27]. In: Adam MP, Ardinger HH, Pagon RA, Wallace SE, Bean LJH, Stephens K, Amemiya A, editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2020.

COBALAMIN (B12) – DEFICIENCY

RESULTS

Vitamin B12 (cobalamin) is a water-soluble vitamin derived from animal products. Intrinsic factor is a glycoprotein produced by parietal cells in the stomach and required to absorb B12 in the terminal ileum. Once absorbed, B12 is used as a cofactor for enzymes involved in the synthesis of DNA, fatty acids and myelin [1]. Vitamin B12 deficiency can lead to hematological and neurological symptoms. Excess vitamin B12 is stored in the liver. However, when B12 cannot be absorbed for an extended period (eg, dietary deficit, malabsorption, lack of intrinsic factor), liver reserves are depleted and a deficiency occurs. Patients with normal intrinsic factor production, any damage to the terminal ileum, such as surgical resection due to Crohn's disease, will impair the absorption of B12 and lead to a deficiency. Other damage to the small intestine, such as celiac inflammation or tapeworm infection, can also result in a vitamin B12 deficiency. Patients who have followed a strict vegan diet for up to three years may develop a vitamin B12 deficiency due to a lack of dietary intake. Vitamin B12 occurs naturally in animal products and is generally not found in plant foods, but fortified foods can be a readily available source of the vitamin for vegetarians [2].

People with your genetic profile are likely to have regular cobalamin levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CLYBL, AL137139.2	rs41281112	CC
TCN1	rs34324219	CC

REFERENCES

[1] Drugs and Lactation Database (LactMed) [Internet]. Bethesda (MD): National Library of Medicine (US); 2006–. Vitamin B12. 2020 Apr 20.
[2] Ankar A, Kumar A. Vitamin B12 Deficiency. 2020 Jun 7. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.

ASCORBIC ACID - DEFICIENCY

RESULTS

L-ascorbic acid (Vitamin C) is a hydrophilic vitamin naturally found in various foods. It is an essential micronutrient required for the biosynthesis of several molecules, such as some neurotransmitters and proteins. Furthermore, vitamin C is an important antioxidant. It acts as a protective factor against free radicals helping to prevent certain types of cancer, cardiovascular disease, and other diseases linked to oxidative stress. Typical sources of vitamin C are fruits and vegetables such as grapefruits, oranges, lemons, limes, potatoes, spinach, broccoli, red peppers, and tomatoes. Up to 90% of vitamin C is consumed in the form of fruits and vegetables.On the other hand, supplements typically contain ascorbic acid, characterized by having the same bioavailability naturally present in foods such as oranges and broccoli. Average vitamin C intake depends on gender and age. For example, in adults, the recommended intake is 105.2- 83.6 mg/day for males and females, respectively, while the average intakes for children and adolescents aged 1 to 18 years range from 75.6 mg/day to 100 mg/day [1]. Vitamin C deficiency can occur in various situations, for example, in smokers (active and passive), in infants fed boiled or powdered milk, in people with poor dietary variety, in malabsorption or chronic diseases. In humans, vitamin C deficiency can lead to scurvy, characterized by fatigue, exhaustion, weakness of the connective tissues and capillary fragility. Due to this, vitamin C an essential micronutrient [2]. Humans cannot synthesize vitamin C because they lack the enzyme L-gluconolactone oxidase, so it is mandatory to obtain vitamin C from the diet. It is common to have deficiencies linked to an inadequate dietary intake. Vitamin C is sensitive to heat, so some preparations (such as boiling or cooking) can lead to degradation. Historically this led to epidemic events of scurvy in some populations, such as sailors. Due to its water solubility, it isn't easy to store vitamin C in the human body. Small quantities are stored in the leukocytes, in the adrenal glands, or the pituitary, but most of the vitamin in plasma is due to recent food intake [2].

People with your genetic profile are likely to have regular ascorbic acid levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SLC23A1	rs33972313	CC

REFERENCES

[1] Travica N, Ried K, Sali A, Scholey A, Hudson I, Pipingas A. Vitamin C Status and Cognitive Function: A Systematic Review. Nutrients. 2017 Aug 30;9(9):960.
[2] Deirawan H, Fakhoury JW, Zarka M, Bluth MH, Moossavi M. Revisiting the pathobiology of scurvy: a review of the literature in the context of a challenging case. Int J Dermatol. 2020 Dec;59(12):1450-1457. "

ERGOCALCIFEROL (VIT D2) AND LUMISTEROL (D2 PRECURSOR) - DEFICIENCY

RESULTS

Lumisterol is an inactive compound that is part of the vitamin D family of steroid compounds. Toxic levels of vitamin D do not occur from prolonged sun exposure. Thermal activation of previtamin D3 in the skin gives rise to multiple non-vitamin D forms, such as lumisterol, tachysterol and others; this limits the formation of vitamin D3 itself from vitamin D2 [1].

People with your genetic profile are likely to have regular ergocalciferol and lumisterol levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2R1	rs10832313	AA

REFERENCES

[1] Borel P, Caillaud D, Cano NJ. Vitamin D bioavailability: state of the art. Crit Rev Food Sci Nutr. 2015;55(9):1193-205.

ERGOCALCIFEROL (VIT D2) - DEFICIENCY

RESULTS

Ergocalciferol, also known as vitamin D2, is a type of vitamin D commonly found in plant sources, for example mushrooms. Mushrooms and yeast that have exposure to sunlight or UV radiation are some of the few foods that contain naturally high levels of vitamin D2 [1].

People with your genetic profile are likely to have regular ergocalciferol levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2R1	rs10832313	AA

REFERENCES

[1] Borel P, Caillaud D, Cano NJ. Vitamin D bioavailability: state of the art. Crit Rev Food Sci Nutr. 2015;55(9):1193-205.

CHOLECALCIFEROL (D3) – DEFICIENCY

RESULTS

Vitamin D3 (cholecalciferol) is synthesized in the skin of humans from 7-dehydrocholesterol and is also consumed in the diet via the intake of animal-based foods. Sources of Vitamin D3, include oily fish and fish oil, liver and egg yolk. A diet with not enough vitamin D in addition to inadequate sun exposure causes vitamin D deficiency. Severe vitamin D deficiency in children causes rickets, a softening and weakening of bones [1].

People with your genetic profile are likely to have regular cholecalciferol levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
VDBP	rs7041	AA

REFERENCES

[1] Borel P, Caillaud D, Cano NJ. Vitamin D bioavailability: state of the art. Crit Rev Food Sci Nutr. 2015;55(9):1193-205.

TOCOPHEROL - DEFICIENCY

RESULTS

Vitamin E is a family of 8 compounds. Of the eight, only alpha-tocotrienol satisfies the human dietary requirement. The vitamin E isoforms are absorbed in the small intestine and metabolized in the liver, but the alpha is retained, while the other seven are excreted. Vitamin E is fat-soluble and plays a vital role as an antioxidant factor in reducing atherosclerosis and lowering the risk of ischemic heart disease. In general, vitamin E deficiency is rare but can occur in the case of low dietary intake, at birth (as it is unable to cross the placenta), in an irregular dietary intake of lipids, genetic mutations in some transporters and chronic diseases (such as cystic fibrosis, Crohn's disease, short bowel syndrome and so on). It is a common deficiency in developing countries. Recommended daily vitamin E intake for adults is 15 mg/day. For vitamin E deficiency treatment, a diet rich in vegetables is recommended. Supplementation with 15 to 25 mg/kg of vitamin E once daily or 200 IU mixed tocopherols is also possible. You can also opt for intramuscular injections in case of problems affecting the small intestine [1].

People with your genetic profile are likely to have a predisposition for tocopherol deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP4F2	rs2108622	TT
CYP4F2	rs3093105	CC

REFERENCES

[1] Kemnic TR, Coleman M. Vitamin E Deficiency. 2020 Jul 10. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.

NAFTOCHINONE/NAPHTHOQUINONE (VIT K PRECURSOR) - DEFICIENCY

RESULTS

Vitamin K is a fat-soluble vitamin. Due to its lipophilic properties, dietary intake of vitamin K depends on bile, pancreatic enzymes and fat intake and absorption in the fasting and ileum, as well as on their transport in the form of chylomicrons. These vitamins play a vital role in blood clotting as they are cofactors of clotting factors II, VII, IX and X. Normal dietary intake levels are 90 ug/day for women and 120 ug/day for men. Vitamin K deficiency can lead to various clinical effects such as significant bleeding, impaired bone development, osteoporosis and increased cardiovascular disease [1]. With significant bleeding, prothrombin time lengthening occurs: therefore, prothrombin time is a good indicator of vitamin K status. However, it is non-specific and requires massive changes to be truly indicative [2].

People with your genetic profile are likely to have a predisposition for naftochinone deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP4F2	rs2108622	TT

REFERENCES

[1] Shearer MJ, Fu X, Booth SL. Vitamin K nutrition, metabolism, and requirements: current concepts and future research. Adv Nutr. 2012 Mar 1;3(2):182-95.
[2] Dong R, Wang N, Yang Y, Ma L, Du Q, Zhang W, Tran AH, Jung H, Soh A, Zheng Y, Zheng S. Review on Vitamin K Deficiency and its Biomarkers: Focus on the Novel Application of PIVKA-II in Clinical Practice. Clin Lab. 2018 Apr 1;64(4):413-424.

CALCIUM - DEFICIENCY

RESULTS

Calcium is an essential nutrient required for many functions in human health. Adequate calcium intake can reduce the risk of fractures, osteoporosis and other pathological conditions [1]. More than 99% of calcium is stored in the bones and teeth. Groups with the highest risk for dietary calcium deficiency include postmenopausal women, individuals with milk allergy or lactose intolerance, adolescents and the elderly [2]. Dairy products such as milk, yogurt and cheese are rich sources of calcium, providing the largest share of calcium from foods in the overall diet. Foods of non-animal origin containing calcium include: legumes (soy, chickpeas, beans), vegetables (broccoli, asparagus), fruit fresh such as oranges and raspberries and dehydrated fruit. Alternative foods that are good non-dairy calcium are: canned sardines, fortified soy, almond and rice milk, fortified orange juice, tofu made with calcium sulfate, canned pink salmon with bones, fortified cereals, greens and beans. Garbanzo, kidney, navy and even canned baked beans provide calcium; boiled green soybeans are another good option; canned shrimp, veggies like cooked broccoli, edamame, acorn squash, papaya, dried figs and oranges. The most common forms of calcium-based supplements are calcium carbonate and calcium citrate. It is important to keep calcium levels optimal because inadequate intake can change bone mineral density, particularly in the elderly.

People with your genetic profile are not likely to have a predisposition for a calcium deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CARS	rs7481584	GG
VWA8-AS1 - RPS28P8	rs7336933	GG
ARID1B	rs11967485	GG

REFERENCES

[1] Goyal A, Singh S. Hypocalcemia. 2020 Jun 22. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.
[2] Pepe J, Colangelo L, Biamonte F, Sonato C, Danese VC, Cecchetti V, Occhiuto M, Piazzolla V, De Martino V, Ferrone F, Minisola S, Cipriani C. Diagnosis and management of hypocalcemia. Endocrine. 2020 Sep;69(3):485-495.

MAGNESIUM - DEFICIENCY

RESULTS

Magnesium is an important micronutrient. It is involved in many physiological functions such as cellular function and signal conduction. Normal serum magnesium levels are between 1.46 and 2.68 mg/dL [1]. Hypomagnesemia is often a consequence of other conditions and chronic diseases, such as alcohol use disorders, starvation, aggressive use of some medication, parenteral nutrition, renal and/or gastrointestinal losses, Crohn's disease, and so on. Symptoms of hypomagnesemia include mild tremors, generalized weakness, cardiac arrest and even death. The therapeutic framework is not constant but must be planned based on patient's kidney function, symptoms and hemodynamic condition. Low levels of magnesium can also cause low levels of calcium and/or potassium as well. Furthermore, many other hormonal and electrolyte abnormalities can present with similar symptoms [2].

People with your genetic profile are likely to have a predisposition for magnesium deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC089987.1 - OR5BS1P	rs193153567	CC
MPPED2-AS1 - AL122014.1	rs3925584	TC
AC009988.1 - RPS15AP5	rs1219515	GG

REFERENCES

[1] Flink EB. Magnesium deficiency. W V Med J. 1990 Oct;86(10):459-63.
[2] Van Laecke S. Hypomagnesemia and hypermagnesemia. Acta Clin Belg. 2019 Feb;74(1):41-47.

PHOSPHORUS - DEFICIENCY

RESULTS

Phosphorus is one of the most common minerals in the human body, second only to calcium. Most of the phosphate compound is stored in the bones as hydroxyapatite (85%). A small amount is stored in the muscles (10%), and the remainder (5%) is found in body fluids. It is common within cells due to its role as an intracellular anion. Phosphate plays an essential role in many biological functions such as the formation of ATP, cyclic AMP and protein phosphorylation [1]. The kidneys handle most of the phosphorus excretion, but the gastrointestinal tract excretes a small percentage. Hypophosphatemia occurs when the level of phosphorus in the serum drops below 2.5 mg/dl [2]. Hypophosphatemia is most commonly induced by one of three causes: (1) inadequate phosphate intake, (2) increased phosphate excretion and (3) passage of phosphate from the extracellular to the intracellular compartment. Hypophosphatemia is typically asymptomatic and is present in up to 5% of humans. It is common in alcoholism and diabetic ketoacidosis [3,4].

People with your genetic profile are likely to have regular phosphorus levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
IP6K3	rs9469578	CC
CSTA	rs17265703	AG
IP6K3	rs73743323	CC

REFERENCES

[1] Calvo MS, Lamberg-Allardt CJ. Phosphorus. Adv Nutr. 2015 Nov 13;6(6):860-2.
[2] Chang AR, Anderson C. Dietary Phosphorus Intake and the Kidney. Annu Rev Nutr. 2017 Aug 21;37:321-346.
[3] Linglart A, Biosse-Duplan M. Hypophosphatasia. Curr Osteoporos Rep. 2016 Jun;14(3):95-105.
[4] Mornet E. Hypophosphatasia. Metabolism. 2018 May;82:142-155.

SODIUM - BALANCE

RESULTS

Na
Sodium

Sodium is an essential nutrient for humans. It is primarily used for maintaining the gradient between the two sides of the cell membrane. This equilibrium is controlled by the sodium-potassium (NA+ K+) pump situated in the cell membrane. This ATPase pump is vital for many physiological processes such as cell volume and signal transduction regulation. Sodium and potassium concentration play a vital role in the physiology of all organs and cells. For example, the kidneys use Na, K-ATPase for their role in filtering and reabsorbing nutrients and waste from the blood. Sperm cells use Na, K-ATPase to enhance their movement. The brain also needs Na, K-ATPase for neurons to reverse the postsynaptic ionic flux to re-establish the membrane potential. Na+ K+-ATPase and its endogenous regulators, the endogenous cardiac steroids (ECS), play a role in the etiology of bipolar disorder and are a potential target for drug development for the treatment. There is evidence of a Na/K-ATPase oxidant amplification loop in the process of aging, obesity, and cardiovascular disease. High levels of sodium in the blood can lead to hypertension (also called high blood pressure). Hypertension is characterized by a constant elevation of arterial pressure and is one of the most common chronic disease conditions. The linkage between salt and hypertension has been studied. It has been discovered that a genetic sensitivity to salt exists in 50 to 60% of patients with a higher risk of developing hypertension. Management of hypertension should address the pharmacological and nonpharmacological aspects of the condition. Patients need to know about non pharmacological elements of the disease, such as weight management, smoking restriction, stress reduction, exercise, salt restriction and a healthy diet. Lifestyle changes alone can account for a 15% reduction in all cardiovascular-related events. Smoking may not directly affect blood pressure, but its link with heart diseases has been established, so quitting can improve hypertension. Pharmacological therapy consists of angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARBs), diuretics, calcium channel blockers, and beta-blockers. Dosages and routines are usually adapted to the patient situation [1].

People with your genetic profile are not likely to have a regular sodium balance

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AT1R	rs5186	CC
CYP11B2	rs3097	CT
BTBD7, BTBD7	rs2273640	GA
CUX2	rs79105258	CC
ARSG, SLC16A6	rs35397826	AG

REFERENCES

[1] Pirahanchi Y, Jessu R, Aeddula NR. Physiology, Sodium Potassium Pump. [Updated 2020 Aug 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

POTASSIUM - BALANCE

RESULTS

Potassium

The body maintains potassium concentration primarily through the action of the kidneys. Total-body potassium is determined by the balance between potassium intake and excretion. Potassium is one of the most important minerals in the body and it helps regulate fluid balance, muscle contractions and nerve signals. A high-potassium diet may help reduce blood pressure and water retention, protect against stroke and prevent osteoporosis and kidney stones. A low potassium level can make muscles feel weak, cramp, twitch, or even become paralyzed [1,2].

People with your genetic profile are likely to have a regular potassium balance

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PRDM8 - FGF5	rs12509595	TT
CLASP1	rs12465752	CC
NUP93	rs118070237	TT
AC104781.2, EML6	rs17046344	GG
WNT2B	rs12037987	TT

REFERENCES

[1] Epstein M, Lifschitz MD. Potassium homeostasis and dyskalemias: the respective roles of renal, extrarenal, and gut sensors in potassium handling. Kidney Int Suppl (2011). 2016;6(1):7-15.
[2] Kardalas E, Paschou SA, Anagnostis P, Muscogiuri G, Siasos G, Vryonidou A. Hypokalemia: a clinical update. Endocr Connect. 2018;7(4):R135-R146.

NA/K RATIO

RESULTS

Na/K
Sodium / Potassium

The Na+ K+ pump is an electrogenic transmembrane ATPase situated in the plasma membrane of the cells. Maintaining the gradient between those two ions is vital for lots of physiological processes such as regulation of membrane potentials, cell volume, and signal transduction. Kidneys are characterized by a high expression of NA-K ATPase pump, which is used to filter blood from waste and reabsorption of valuable nutrients, and they are involved in the pH regulation. With a different isoform, NA-K ATPase is used by sperm cells for their movement, so this pump is fundamental for male fertility. Even in the brain, it is widely present as it is involved in reversing the ionic flux to re-establish the action potentials. Na+ K+-ATPase and its endogenous regulators, the endogenous cardiac steroids (ECS), play a role in the etiology of bipolar disorder and are a potential target for drug development for the treatment. Lastly, there is evidence of a link between NA-K ATPase and other chronic diseases as cardiovascular disease, obesity and aging. Potassium and sodium are strictly related, as they are both essential nutrients that play a fundamental role in the homeostatic processes. Both nutrients have been linked to the risk of chronic disease, particularly cardiovascular disease, but unbalanced sodium is already linked to other chronic diseases [1].

People with your genetic profile are likely to have a regular balance ratio of sodium/potassium

SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADD1	rs4961	GG

REFERENCES

[1] Pirahanchi Y, Jessu R, Aeddula NR. Physiology, Sodium Potassium Pump. [Updated 2020 Aug 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

SELENIUM - DEFICIENCY

RESULTS

Selenium is a trace element and a micronutrient. This element is essential for the formation of selenoproteins, which perform many physiological functions. Studies say that selenium also plays a vital role in the immune system and inflammatory response and modulates HIV-AIDS infections [1]. It is also linked to cardiovascular disease, infertility, myodegenerative disease and cognitive decline [2]. Since selenium is present in the soil, it affects the selenium concentration in plant foods. Some excellent sources of selenium are seeds, Brazil nuts, green vegetables, shiitake mushrooms, and button mushrooms. Selenium yeast is also an excellent source and is used to make bread. Individuals who consume plant-based foods containing selenium, especially fish, seafood, beef, and poultry, are good sources of selenium from areas with adequate supply. Selenium deficiency can occur mainly due to poor availability in the area of interest, and this condition is typically associated with a vitamin E deficiency. Generally, the body's minimum amount of selenium demand is set at 70 and 55 micrograms (mcg) per day for men and women, respectively, but levels above 5.1mmol / day are considered toxic [3]. Selenium deficiency is more of a population problem than an individual problem because it affects entire populations due to the bioavailability of selenium in a particular region. Bio-fortification is a standard solution for large populations, and enriched foods have been introduced in some countries with good results. This approach is safer than oral supplementation because organic selenium, generally produced by the bacterium in the fortified food supply chain, is less likely to cause toxic reactions [4].

People with your genetic profile are likely to have a predisposition for selenium deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SLC39A11	rs891684	GG
DMGDH	rs248381	AA
AC076968.2	rs1596370	GG

REFERENCES

[1] Huang Z, Rose AH, Hoffmann PR. The role of selenium in inflammation and immunity: from molecular mechanisms to therapeutic opportunities. Antioxid Redox Signal. 2012 Apr 1;16(7):705-43.
[2] Roman M, Jitaru P, Barbante C. Selenium biochemistry and its role for human health. Metallomics. 2014 Jan;6(1):25-54.
[3] Zwolak I. The Role of Selenium in Arsenic and Cadmium Toxicity: an Updated Review of Scientific Literature. Biol Trace Elem Res. 2020 Jan;193(1):44-63.
[4] Nève J, Vertongen F, Molle L. Selenium deficiency. Clin Endocrinol Metab. 1985 Aug;14(3):629-56.

IRON – DEFICIENCY

RESULTS

Iron is an essential micronutrient. It is vital for the production of hemoglobin, and iron deficiency is one of the causes of anemia. Iron deficiency is linked to a variety of factors such as age, gender and socioeconomic status. Symptoms of iron deficiency anemia include fatigue and dyspnoea [1]. Iron supplementation is the treatment of choice for iron deficiency anemia; supplementation can be oral or intravenous. Iron deficiency may result from insufficient iron intake by diet, decreased iron absorption, or blood loss. Other predisposing conditions for iron deficiency are old age, pregnancy and celiac disease. In children, iron deficiency can be linked to the absence of breastfeeding as human milk is far richer in iron than cow's milk. In developing countries, parasitic infestations are a common cause of iron deficiency [2]. Dietary sources of iron are green vegetables, red meat and iron-fortified milk. Typically, humans lose about 1 mg of iron daily by expelling by digestion. The daily iron loss increases in menstruating women, so women of childbearing age require higher iron intake than men [3].

People with your genetic profile are likely to have regular iron levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
TMPRSS6	rs855791	GG
TF	rs8177240	TG
HFE	rs1799945	CC
ZDHHC14	rs181143083	TT
SCGN	rs115809796	AA

REFERENCES

[1] DeLoughery TG. Iron Deficiency Anemia. Med Clin North Am. 2017 Mar;101(2):319-332.
[2] Mantadakis E, Chatzimichael E, Zikidou P. Iron Deficiency Anemia in Children Residing in High and Low-Income Countries: Risk Factors, Prevention, Diagnosis and Therapy. Mediterr J Hematol Infect Dis. 2020 Jul 1;12(1):e2020041.
[3] Fernández-Gaxiola AC, De-Regil LM. Intermittent iron supplementation for reducing anaemia and its associated impairments in adolescent and adult menstruating women. Cochrane Database Syst Rev. 2019 Jan 31;1(1):CD009218.

ZINC - DEFICIENCY

RESULTS

Zinc, a divalent cation, is a micronutrient involved in many physiological processes. Zinc is involved in metabolism, gene transcription, reproductive function, immune functions and wound repair. Zinc is readily found in many foods, including meat, fish and legumes, although its absorption and bioavailability depend on the type of food source. Despite this, zinc deficiency is common worldwide and can be caused by a reduced intake or by problems with hereditary malabsorption, increased demand, or excessive loss. Zinc deficiency from an inadequate diet is more common in developing countries. Symptoms commonly involve the gastrointestinal system, immune response or skin. Usually, treatment is based on oral supplements, giving quick and effective results [1]. Serum alkaline phosphatase (a zinc-dependent metalloenzyme) and plasma zinc concentrations must be monitored to determine deficiency. Normal zinc levels are between 70 and 250 ug/dl in adults. A mild deficiency occurs clinically when the values drop to a range between 40 and 60 ug/dl. Supplementation therapy includes oral administration with different dosages, but generally with two doses of 3 mg/kg for up to two weeks. This therapy is sufficient to resolve all clinical manifestations. In people with genetic conditions such as enteropathic acrodermatitis, lifelong treatment with 1 to 2 mg/kg of zinc per day is enough to prevent symptoms [2].

People with your genetic profile are likely to have regular zinc levels

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PPCDC	rs2120019	TC

REFERENCES

[1] Maret W, Sandstead HH. Zinc requirements and the risks and benefits of zinc supplementation. J Trace Elem Med Biol. 2006;20(1):3-18.
[2] Muhamed PK, Vadstrup S. [Zinc is the most important trace element]. Ugeskr Laeger. 2014 Mar 3;176(5):V11120654. Danish.

COPPER - DEFICIENCY

RESULTS

Copper is a trace element that acts as an enzymatic cofactor in the human body. Its daily loss amounts to 1.3 mg so, to replenish it, it is necessary to take 2 mg / day. Copper deficiency occurs mainly in premature infants and adults on long-term parenteral feeding without adequate copper supplements [1].

People with your genetic profile are not likely to have a predisposition to develop a copper deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SMIM1	rs1175550	AA

REFERENCES

[1] Williams DM. Copper deficiency in humans. Semin Hematol. 1983 Apr;20(2):118-28.

COE-Q - DEFICIENCY

RESULTS

Coenzyme Q10 is a compound widely present in the human body but concentrated mainly in the heart, liver, kidneys and pancreas. This coenzyme is widely used by cells in various biological processes such as aerobic respiration, metabolism and oxidative metabolism. Q10 is also used as an antioxidant [1,2,3].

People with your genetic profile are likely to have a predisposition to develop a coenzyme-Q deficiency

SCIENTIFIC DETAILS

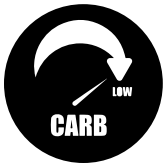
Gene	rsID	Genotype
NEGR1	rs55927656	TT
	rs11591201	GA
	rs146799867	CC
OLAH	rs12573070	GG
PRMT8	rs17769758	GG
	rs7141874	GG
TOM1L1	rs184812087	GG
DCC	rs74681568	CC

REFERENCES

[1] Sangsefidi ZS, Yaghoubi F, Hajiahmadi S, Hosseinzadeh M. The effect of coenzyme Q10 supplementation on oxidative stress: A systematic review and meta-analysis of randomized controlled clinical trials. Food Sci Nutr. 2020 Mar 19;8(4):1766-1776.
[2] Hargreaves I, Heaton RA, Mantle D. Disorders of Human Coenzyme Q10 Metabolism: An Overview. Int J Mol Sci. 2020 Sep 13;21(18):6695.
[3] Salviati L, Trevisson E, Doimo M, Navas P. Primary Coenzyme Q10 Deficiency. 2017 Jan 26. In: Adam MP, Ardinger HH, Pagon RA, Wallace SE, Bean LJH, Stephens K, Amemiya A, editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2020.

PREDISPOSITION TO RESPOND POSITIVELY TO A LOW CARBOHYDRATE DIET

RESULTS



Scientific studies have shown that a low carbohydrate diet produces more significant weight loss (within the first 6-12 months) than other types of diet. Reducing the amount of carbohydrates in meals inevitably produces an increase in fat and protein macronutrients in the diet itself. Fats and proteins increase satiety and have less concomitant hypoglycemia. This increase in satiety then reduces hunger and food intake in general, leading to a calorie deficit. The low-carb diet, and especially the ketogenic approaches, quickly lead to weight loss. This type of diet is recommended for those who want to keep glycemic control under control, for those who want to lose weight, take care of their health and those who want to improve physical performance [1].

People with your genetic profile are likely to have an enhanced response to a low carbohydrate diet

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AMY1-AMY2	rs11185098	GA

REFERENCES

[1] Oh R, Gilani B, Uppaluri KR. Low Carbohydrate Diet. [Updated 2020 Jul 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

PREDISPOSITION TO EXCESSIVE CONSUMPTION OF CARBOHYDRATES

RESULTS

Carbohydrates account for over 50% of the energy found in most human diets. They include monosaccharides, disaccharides and polysaccharides. The increase in the consumption of free monosaccharides is one of the factors most linked to the prevalence of obesity. Although carbohydrates are the only food constituents that directly raise blood sugar, population studies suggest that the total amount of carbohydrate (as a percentage of food energy) is less significant than the type of carbohydrate due to the risk of chronic disease [1]. Clinical studies have shown that short-term low-carb diets produce greater weight loss than low-fat diets, but this difference diminishes over time due to poor long-term compliance. Therefore, evidence suggests that the type of carbohydrate may have a greater effect on health than the total amount. However, specific groups may respond differently to the quantity and quality of carbohydrates. It can be argued that the consumption of high glycemic cereals and products based on potatoes and added sugars is causally related to obesity, diabetes, cardiovascular disease and some types of cancer. However, non-starchy vegetables, fruit whole, legumes and whole grains seem protective. However, the metabolic effects of total and high glycemic index carbohydrates can vary from individual to individual, depending on the degree of insulin resistance, glucose intolerance, or other inherited or acquired biological predispositions [1].

People with your genetic profile are likely to have a regular consumption of carbohydrates

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC092422.1	rs7619139	AA
RN7SL423P - AC009313.2	rs197273	AA

REFERENCES

[1] National Research Council (US) Committee on Diet and Health. Diet and Health: Implications for Reducing Chronic Disease Risk. Washington (DC): National Academies Press (US); 1989. 9, Carbohydrates.

HIGH VS LOW CARBOHYDRATE DIETS

RESULTS

Low-carb diets have gained international attention as an addition to weight loss management. With a low-carb diet, the body undergoes a metabolic process, known as ketosis. When the availability of carbohydrates is reduced, the body will oxidize fats to support energy needs. Low-carbohydrate diets have several beneficial effects compared to a high-carbohydrate diet. With a low-carb diet, improvement in blood pressure, rapid weight loss, reduced circulating triglyceride levels, decreased fasting glucose and insulin levels occur. However, some less desirable effects have been highlighted, such as an increase in plasma homocysteine levels, an increased loss of lean body mass, an increase in low-density lipoprotein cholesterol and a greater loss of urinary calcium. Fatty acid synthesis is stimulated by low-fat, high-carbohydrate diets in animals that have a positive calorie balance [1,2,3].

People with your genetic profile are likely to have a predisposition to a regular carbohydrate intake

SCIENTIFIC DETAILS

Gene	rsID	Genotype
ISCA1P1 - AC113420.1	rs16891077	GA
RPA2P2 - AL096706.1	rs9787485	CC
GCK	rs4607517	GG

REFERENCES

[1] Jung CH, Choi KM. Impact of High-Carbohydrate Diet on Metabolic Parameters in Patients with Type 2 Diabetes. Nutrients. 2017;9(4):322.
[2] Oh R, Gilani B, Uppaluri KR. Low Carbohydrate Diet. [Updated 2020 Jul 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls.
[3] Adam-Perrot A, Clifton P, Brouns F. Low-carbohydrate diets: nutritional and physiological aspects. Obes Rev. 2006 Feb;7(1):49-58.

PREDISPOSITION TO AN ALTERED METABOLISM OF STARCH

RESULTS

Individuals with certain genetic abnormalities might not be able to breakdown starch, a type of carbohydrate, which is a form of sugar. Individuals with Congenital sucrase-isomaltase deficiency (CSID), a genetic disorder that affects a person's ability to digest certain sugars are prone to this altered metabolism of starch. Individuals with such conditions are recommended to eat plain sources of protein, including beef, pork, lamb, fish, turkey, chicken, and eggs [1,2].

People with your genetic profile are not likely to have a regular starch metabolism

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AMY1-AMY2	rs11185098	GA

REFERENCES

[1] Heianza Y, Sun D, Wang T, et al. Starch Digestion-Related Amylase Genetic Variant Affects 2-Year Changes in Adiposity in Response to Weight-Loss Diets: The POUNDS Lost Trial. Diabetes. 2017;66(9):2416-2
[2] Aller EE, Abete I, Astrup A, Martinez JA, van Baak MA. Starches, sugars and obesity. Nutrients. 2011;3(3):341-369.

PREDISPOSITION TO EXCESSIVE CONSUMPTION OF FATS

RESULTS

Reduction of dietary saturated fat has been recommended for reducing cardiovascular disease (CVD) risk for decades. This recommendation was based on the classic "diet-heart" association that regards saturated fat and cholesterol as the main risk factors for atherosclerosis and coronary heart disease (CHD). The pathogenetic mechanism underlying this consists of the increase in LDL lipoproteins induced by a diet with an excess of fat. The recommended total fat intake is between 20 and 35% of total calories. A minimum of 20% is used to ensure adequate total energy consumption, essential fatty acids and fat-soluble vitamins. The 35% maximum is based on limiting saturated fat and observing that individuals on high-fat diets consume more calories, resulting in weight gain. Saturated and monounsaturated fatty acids are synthesized in the body for energy, physiological and structural functions and are present in many foods. For example, palmitic acid, the primary saturated fatty acid in the diet, is synthesized in the liver from starch and sugar via de novo lipogenesis and is the predominant fatty acid in dairy products and meats. Due to the positive linear relationship between total saturated fat intake and LDL concentrations, the recommendation is to limit saturated fat to <10% of calories [1].

People with your genetic profile are likely to have a regular consumption of fat

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FGF21	rs838133	AG

REFERENCES

[1] Liu AG, Ford NA, Hu FB, Zelman KM, Mozaffarian D, Kris-Etherton PM. A healthy approach to dietary fats: understanding the science and taking action to reduce consumer confusion. Nutr J. 2017;16(1):53.

PREDISPOSITION TO THE ACCUMULATION OF FAT

RESULTS

It appears that over time high food intake and lack of regular exercise can change the processes that regulate appetite and body fat distribution, making the person physiologically more likely to gain weight. Hormones are a crucial factor in the onset of obesity: obese people have hormone levels that encourage the accumulation of body fat. Our appetite, body fat distribution and metabolism are affected by hormones such as leptin and insulin, growth hormone and sex hormones. The increase or decrease in the production and secretion of hormones can lead to obesity and vice versa. Body fat distribution plays an essential role in some obesity-related conditions such as stroke, some forms of arthritis, and heart disease. It appears that body fat distribution is also affected by estrogen and androgen. Age plays a vital role in the distribution of body fat. Evidence suggests that excessive weight gain may be due to a lack of estrogen. It appears that healthy eating and regular exercise can retrain the body to shed excess body fat and keep it away. Weight loss is also associated with a reduced risk of developing heart disease, stroke, type II diabetes, and some cancers [1].

People with your genetic profile are likely to have a predisposition to the accumulation of fat

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FTO	rs9972653	GT
FTO	rs1421085	TC
FGF21	rs838133	AA
FTO	rs1558902	TA
FTO	rs9939973	GA
RNU4-17P - AC090771.1	rs663129	GG
CDK6	rs42235	CC

REFERENCES

[1] Bhandari P, Sapra A. Low Fat Diet. [Updated 2021 Feb 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

PREDISPOSITION TO RESPOND POSITIVELY TO A LOW FAT DIET

RESULTS

Most doctors agree that the average diet needs a reduction in fat to decrease the risk of cardiovascular problems. However, in humans, lipids are necessary for various roles, including energy storage and organs protection. Research has suggested that a decrease in serum cholesterol levels can prevent atherosclerosis. Therefore, a diet low in fat has been widely advocated by doctors for reducing cardiovascular-related problems [1].

People with your genetic profile are likely to have a regular response to a low-fat diet

SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADIPOQ	rs17300539	GG

REFERENCES

[1] Tobias DK, Chen M, Manson JE, Ludwig DS, Willett W, Hu FB. Effect of low-fat diet interventions versus other diet interventions on long-term weight change in adults: a systematic review and meta-analysis. Lancet Diabetes Endocrinol. 2015 Dec;3(12):968-79.

PREDISPOSITION TO LIPOPROTEIN DEFICIENCY

RESULTS

Lipoproteins are molecules that transport plasma lipids and are specific risk factors for cardiovascular and metabolic diseases. They consist of a central core composed of triglycerides and cholesterol esters, around which there is an outer layer of phospholipids, free cholesterol and apolipoproteins (apo). Apolipoproteins, i.e. the protein component of lipoproteins, allow their classification into one of the five main classes: chylomicrons, very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL), lipoproteins low density (LDL), and high density lipoprotein (HDL). HDL, known as "good cholesterol", participates in the reverse transport of cholesterol, while LDL, known as "bad cholesterol", promotes atherosclerosis. Alterations in lipoproteins have both genetic and environmental bases. Lipids, along with carbohydrates, proteins and nucleic acids, are one of the four main biological molecules in the human body. They are essential cellular components. They are involved in multiple processes such as the storage of energy, the transmission of chemical messages, the formation of cell membranes and the transport of fat-soluble vitamins such as vitamin E. For lipids to play these roles in the cell, they must travel to target cells after being absorbed in the gastrointestinal tract. Without lipoproteins, this transport would not be possible, as the hydrophilic environment of the blood is not compatible with the hydrophobic nature of the lipids. High levels of LDL cholesterol is a significant cause of atherosclerotic cardiovascular disease (ASCVD). The result is the oxidation of macrophages, which remain trapped in the vessel wall, thus promoting the onset of atherosclerotic lesions [1].

People with your genetic profile are likely to have a predisposition for lipoprotein deficiency

SCIENTIFIC DETAILS

Gene	rsID	Genotype
HERPUD1 - CETP	rs247616	CC
HERPUD1 - CETP	rs247617	CC
HERPUD1 - CETP	rs3764261	CC
HERPUD1 - CETP	rs821840	AA
HERPUD1 - CETP	rs56156922	TT
HERPUD1 - CETP	rs17231506	CC
CETP	rs7205804	GG

REFERENCES

[1] Schaefer EJ. Clinical, biochemical, and genetic features in familial disorders of high density lipoprotein deficiency. Arteriosclerosis. 1984 Jul-Aug;4(4):303-22.

POSITIVE RESPONSE TO A DIET HIGH IN MONOUNSATURATED FATS

RESULTS

Monounsaturated fat is a type of dietary fat. Monounsaturated fat is present in some plant foods, such as nuts, avocados and vegetable oils. Eating moderate amounts of monounsaturated (and polyunsaturated) fats have beneficial health effects. They can help lower your LDL cholesterol level. Keeping the LDL level low helps reduce the risk of heart disease and stroke. Dietary guidelines recommend consuming less than 10% of your daily calories as saturated fat. An easy way to do this is by replacing saturated fat with monounsaturated and polyunsaturated fats. Despite the beneficial effects on health, monounsaturated and polyunsaturated fats are still a concentrated source of calories. Therefore, they should be substituted for saturated fat while staying within the recommended limits for total calories and dietary fat [1].

People with your genetic profile are likely to have a regular response to diet enriched in monounsaturated fats

SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADIPOQ	rs17300539	GG

REFERENCES

[1] National Research Council (US) Committee on Diet and Health. Diet and Health: Implications for Reducing Chronic Disease Risk. Washington (DC): National Academies Press (US); 1989. 7, Fats and Other Lipids.

POSITIVE RESPONSE TO A DIET HIGH IN POLYUNSATURATED FATS

RESULTS

Fats and oils are essential components of a healthy diet as energy sources, liposoluble vitamins, and sources of essential fatty acids. They also contribute satiety, flavour, and palatability to the diet. Polyunsaturated fatty acids (PUFAs), such as linoleum acid, are classified as an essential nutrient since the body requires it but can't synthesise it. Also needed by the body is arachidonic acid. However, arachidonic acid can be synthesised from linoleum acid, commonly found in soybeans, corn, and safflower seeds. These fatty acids play vital roles in the structure and function of biological membranes in the central nervous system and the retina [1].

People with your genetic profile are likely to have a predisposition to respond positively to a diet enriched in polyunsaturated fats

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FADS2, FADS1	rs174547	TT

REFERENCES

[1] National Research Council (US) Committee on Diet and Health. Diet and Health: Implications for Reducing Chronic Disease Risk. Washington (DC): National Academies Press (US); 1989. 7, Fats and Other Lipids.

NEGATIVE RESPONSE TO A DIET HIGH IN UNSATURATED FATS

RESULTS

Lipids are hydrophobic compounds, that is, insoluble in water. Those essential for our health include fats and oils (triglycerides or triacylglycerols), fatty acids, phospholipids and cholesterol. Fats and oils are glycerol esters, linked to fatty acid chains. They are important as sources of energy and essential fatty acids and contribute to the absorption of fat-soluble vitamins. The relationship between plasma triglyceride levels and cardiovascular disease is somewhat controversial and unclear. In most population-based studies, plasma triglyceride levels are associated with increased cardiovascular risk without being independently predictive of CHD after statistical adjustment for closely associated attributes such as HDL-C, hypertension (also called high blood pressure), cigarette smoking, and obesity. However, studies have found plasma triglyceride level to be an independent predictor of CHD in women. However, the plasma level of total triglycerides, rather than being a direct cause of atherosclerotic disease, probably reflects the presence of some atherogenic lipoproteins. Numerous genetic disorders of lipoprotein metabolism have been identified and characterized. The study of these disorders has provided much information on the structure, metabolism and regulation of plasma lipoproteins and apolipoproteins. Many of these disorders are characterized by severe hypercholesterolemia, early atherosclerosis, and coronary artery disease. These disorders include familial hypercholesterolaemia (FH), familial combined hyperlipidaemia, and familial dysbetalipoproteinemia (type 3 hyperlipoproteinemia) [1,2].

People with your genetic profile are likely to have a regular response to a diet enriched in unsaturated fats

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FADS1, FADS2	rs174566	AA
FADS2, FADS1	rs174547	TT

REFERENCES

[1] Burdge GC, Calder PC. Introduction to fatty acids and lipids. World Rev Nutr Diet. 2015;112:1-16.
[2] Bhupathi V, Mazariegos M, Cruz Rodriguez JB, Deoker A. Dairy Intake and Risk of Cardiovascular Disease. Curr Cardiol Rep. 2020 Jan 29;22(3):11.

NEGATIVE RESPONSE TO A DIET HIGH IN TRANS-FAT

RESULTS

Fats are essential macromolecules for humans, but they must be consumed in limited quantities to avoid health risks. The four main types of dietary fat include polyunsaturated, monounsaturated, trans and saturated fats, which differ in their physical and chemical structure. While saturated and trans fats are solid at room temperature, mono and polyunsaturated fats are liquid. Regardless of their physical and chemical properties, these different forms of fat provide 9 kCal / g, a much higher amount of calories than that provided by the same amount of carbohydrates and proteins. Saturated and trans fats increase the production of low density lipoproteins (LDL) and are therefore considered unhealthy, while monounsaturated (MUFA) and polyunsaturated (PUFA) fats, which lower LDL, are considered beneficial. There is a general scientific consensus that the fat content of the human diet should be reduced to lower the risk of cardiovascular morbidity and mortality. Low-fat diets are those in which 30% or less of the calories come from fat. Common examples of low-fat foods include vegetables, fruits, whole grains, egg whites, chicken breast and skinless turkey, beans, lentils, peas, seafood, etc. Dietary cholesterol has also received considerable attention due to its direct connection with the risk of coronary heart disease. The level of LDL lipoproteins is the best predictor of cardiovascular risk. Trans fatty acids are also similar to saturated fatty acids in raising cholesterol, so their dietary intake should be low; the levels of polyunsaturated fatty acids, on the other hand, should be high. There is epidemiological evidence showing the association between dietary fat intake and breast, prostate, colon and even lung cancer. Of these various forms of breast cancer, breast cancer has been the most widely linked to excessive lipid consumption. Several mechanisms have been suggested to explain this phenomenon, including the conversion of essential fatty acids into hormone-like lipids and the production of reactive oxygen species capable of inducing mutations in genomic DNA. Other potential mechanisms include changes in the hypothalamic-pituitary axis, leading to alterations in hormone levels, the effect on enzyme functions that affect estrogen, changes in cell structure and functioning, as well as changes in immune function. Studies have also suggested that polyunsaturated fatty acids (especially omega-3s) have a protective effect against tumour development. Obesity is a chronic multifactorial disease associated with many comorbidities such as diabetes mellitus, dyslipidemia, fatty liver, and obstructive sleep apnea, among others. Of the many external factors, dietary fat intake is believed to have the strongest association with this condition [1].

People with your genetic profile are likely to have a regular response to the intake of trans fatty acids

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PLA2G2A	rs4654990	CC

REFERENCES

[1] National Research Council (US) Committee on Diet and Health. Diet and Health: Implications for Reducing Chronic Disease Risk. Washington (DC): National Academies Press (US); 1989. 7, Fats and Other Lipids.

POSTPRANDIAL RESPONSE OF TRIGLYCERIDES TO HIGH-FAT DIET MEALS

RESULTS

Adiponectin is the most abundant adipokine secreted by the adipocytes. There is a lesser concentration of circulating adiponectin in people who are obese or have Type 2 diabetes. This hormone is believed to play a vital role in the etiology of metabolic syndrome (MetS) because it may be an important regulator of insulin sensitivity and inflammation [1,2]. A high protein diet (HPD) results in short-term loss of body weight and fat mass. In contrast, lean mass is preserved, and leptin and GLP-1 concentrations are significantly reduced increasing postprandial glucagon concentrations. Long-term use of HPD appears to be an effective strategy to reduce energy intake and expenditure and maintain lean body mass. The reduction of glucose intake in the diet leads to a decrease in lipogenesis, and the concomitant increase in proteins contributes to a large extent to energy expenditure. Lowering carbohydrates in favor of proteins in obese subjects could be beneficial as dietary CHOs (cholesterol) could compromise the oxidation of fats. Diets high in protein and low in CHO reduce the development of fatty tissue. Additionally, a higher daily protein intake at the expense of fat intake could result in a healthier weight state. The mechanism by which increased long-term dietary protein intake affects body weight is not well understood [3].

People with your genetic profile tend to have a higher plasma fat concentration.

SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADIPOQ	rs266729	CC

REFERENCES

[1] Esfahani M, Movahedian A, Baranchi M, Goodarzi MT. Adiponectin: an adipokine with protective features against metabolic syndrome. Iran J Basic Med Sci. 2015;18(5):430-442.
[2] Nigro E, Scudiero O, Monaco ML, et al. New insight into adiponectin role in obesity and obesity-related diseases. Biomed Res Int. 2014;2014:658913.
[3] Cohen SS, Gammon MD, North KE, Millikan RC, Lange EM, Williams SM, Zheng W, Cai Q, Long J, Smith JR, Signorello LB, Blot WJ, Matthews CE. ADIPOQ, ADIPOR1, and ADIPOR2 polymorphisms in relation to serum adiponectin levels and BMI in black and white women. Obesity (Silver Spring). 2011 Oct;19(10):2053-62.

RISK OF GAINING FAT THROUGH THE INTAKE OF HIGH PROTEIN FOODS

RESULTS

The high protein diet (HPD) results in short-term loss of body weight and fat mass, while lean mass is preserved. Long-term use of HPD appears to be an effective strategy to reduce energy intake and expenditure and maintain lean body mass. The reduction of glucose intake in the diet leads to a decrease in lipogenesis, and the concomitant increase in proteins contributes to a large extent to energy expenditure. Lowering carbohydrates in favor of proteins in obese subjects could be beneficial as dietary CHOs (cholesterol) could compromise the oxidation of fats. Diets high in protein and low in CHO reduce the development of fatty tissue. Additionally, a higher daily protein intake at the expense of fat intake could result in a healthier weight state. The mechanism by which increased long-term dietary protein intake affects body weight is not well understood [1].

People with your genetic profile are not likely to have a predisposition to accumulate fat mass due to the intake of high protein foods

SCIENTIFIC DETAILS

Gene	rsID	Genotype
RMI2, PRM1	rs737008	GG

REFERENCES

[1] Pesta DH, Samuel VT. A high-protein diet for reducing body fat: mechanisms and possible caveats. Nutr Metab (Lond). 2014 Nov 19;11(1):53.

PREDISPOSITION TO FEEL FULL WITH PROTEIN INTAKE

RESULTS

A protein is made up of a chain of amino acids (AA) linked by peptide bonds. In the the gastrointestinal tract, enzymes such as proteases and peptidases hydrolyse dietary proteins to create amino acids, dipeptides and tripeptides. These digestion products are absorbed in enterocytes or used by bacteria in the small intestine. AAs that are not degraded by the small intestine via the portal vein are destined for skeletal muscle and other tissues for protein synthesis. AAs are also used for the cell-specific production of low molecular weight metabolites of enormous physiological importance. Therefore, protein malnutrition causes stunting, anemia, physical weakness, edema, vascular dysfunction and reduced immunity. Guidelines require a daily protein intake of 0.8 g of protein per kg of body weight (BW) per day for a healthy adult with minimal physical activity. A dietary intake of 1.0, 1.3 and 1.6 g of protein per kg body weight are recommended per day for people with minimal, moderate and intense physical activity, respectively. Long-term consumption of protein at 2 g per kg of body weight per day is safe for healthy adults, and the tolerable upper limit is 3.5 g per kg of body weight per day for well-adapted individuals. Chronic high protein intake (> 2 g per kg body weight per day for adults) can cause digestive, renal and vascular abnormalities and should be avoided. The quantity and quality of proteins are the determinants of their nutritional values. Therefore, adequate consumption of high-quality protein from animal products (for example, lean meat and milk) is essential for the optimal growth, development and health of humans [1].

People with your genetic profile are likely to have a predisposition to develop enhanced satiety with daily protein intake

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FTO	rs1421085	TC

REFERENCES

[1] Wu G. Dietary protein intake and human health. Food Funct. 2016 Mar;7(3):1251-65.

PLASMA LEVELS OF PARAXANTHINE

RESULTS

PARAXANTHINE

The production of caffeine can take place by extraction or by synthetic procedures. The extraction procedures include three methods: extraction from kola nuts; extraction from tea dust, waste and fragments of tea leaves and; direct decaffeination of green coffee beans through solvents. The synthetic production of caffeine, on the other hand, involves the methylation of various xanthines. The gastrointestinal tract absorbs caffeine rapidly, entirely and directly depending on pH. After oral doses of 5-8 mg/kg of body weight, peak plasma concentrations of 8-10 µg / ml were observed. After ingestion, the time required to reach maximum plasma concentration shows considerable variations, ranging from 15 to 120 min. Once absorbed, caffeine is rapidly and evenly distributed in body fluids and is eliminated by apparent first-order kinetics, described by a one-compartment open model system. Caffeine is metabolised mainly in the liver. The body eliminates caffeine more quickly than paraxanthine. Therefore, 8-10 hours after ingestion of caffeine, higher levels of the latter will be present in the plasma. The toxicological potency of paraxanthine is low: the human body converts 70-80% of caffeine and paraxanthine, with no apparent toxic effects, after caffeine doses of 300-500 mg / day. The effects of caffeine vary according to the dose: in low doses (up to 2 µg / ml in the blood), it stimulates the central nervous system, which consumers perceive as a benefit; a high concentration in the blood (10–30 µg / ml) can induce mood and sleep disturbances, produce restlessness, excitement, tremor, tinnitus, headache and insomnia; increase urine production and gastric acid secretion, alter myocardial function, induce hypertension (also called high blood pressure) and arrhythmia, and increase plasma catecholamine levels and plasma renin activity, especially when administered to non-users or recent abstainers. Excessive caffeine consumption can lead to an anxiety neurosis known as "caffeinism" [1,2].

People with your genetic profile are likely to have a slower paraxanthine metabolic rate

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2A6, AC008537.1	rs56113850	TT

REFERENCES

[1] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Coffee, Tea, Mate, Methylxanthines and Methylglyoxal. Lyon (FR): International Agency for Research on Cancer; 1991. (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 51.) Caffeine.
[2] Institute of Medicine (US) Committee on Military Nutrition Research. Caffeine for the Sustainment of Mental Task Performance: Formulations for Military Operations. Washington (DC): National Academies Press (US); 2001. 2, Pharmacology of Caffeine.

PLASMA LEVELS OF CAFFEINE

RESULTS

CAFFEINE

The production of caffeine occurs both by extraction and by synthetic procedures. The extraction procedures involve three methods: extraction from tea powders, waste and fragments of tea leaves and extraction from kola nuts and direct decaffeination of green coffee beans through solvents. In contrast, synthetic production involves the methylation of various xanthines. Caffeine (1,3,7-trimethylxanthine) is a plant alkaloid that structurally resembles purines. The mean plasma half-life of caffeine in healthy individuals is approximately 5 hours. The average half-life of caffeine is influenced both by inherent individual variation and by a variety of physiological and environmental factors that affect the metabolism of caffeine, such as obesity, use of oral contraceptives or altitude. Ingestion of caffeine doses up to 10 g resulted in convulsions and vomiting with complete recovery in 6 hours. The fatal acute oral dose of caffeine in humans is estimated at 10-14 g (150-200 mg/kg weight body). Maximum plasma concentrations occur between 15 and 120 minutes after oral ingestion. This large variation over time may be due to the different times required for gastric emptying and the presence of other dietary components, such as fiber [1,2].

People with your genetic profile are likely to have a regular caffeine metabolic rate

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP1A1 - CYP1A2	rs2472297	CC
CYP1A1 - CYP1A2	rs2470893	CC

REFERENCES

[1] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Coffee, Tea, Mate, Methylxanthines and Methylglyoxal. Lyon (FR): International Agency for Research on Cancer; 1991. (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 51.) Caffeine.
[2] Institute of Medicine (US) Committee on Military Nutrition Research. Caffeine for the Sustainment of Mental Task Performance: Formulations for Military Operations. Washington (DC): National Academies Press (US); 2001. 2, Pharmacology of Caffeine.

PLASMA LEVELS OF THEOBROMINE

RESULTS

THEOBROMINE

The production of caffeine takes place both by extraction, using three methods: direct decaffeination of green coffee beans with solvents, extraction from tea powders, waste and fragments of tea leaves and extraction from kola nuts; and by synthetic procedures, through the methylation of various xanthines. Theobromine is the principal alkaloid (1.5–3%) of the cocoa bean and is mainly used to make caffeine, is readily absorbed from food and evenly distributed in body fluids, or passes into the breastmilk of nursing mothers (half-life from 6.1 to 10 hours). As a metabolite of caffeine, theobromine was detected in varying amounts in plasma and urine. The apparent volumes of distribution were estimated at 0.761 / kg of body weight while those of clearance at 0.88 ml/min/kg of body weight. It has been stated that in excessive doses, theobromine can cause nausea and anorexia, and a daily intake of 50-100 g of cocoa (0.8-1.5 g of theobromine) has been associated with sweating, tremor and severe headache [1,2].

People with your genetic profile are likely to have a regular theobromine metabolic rate

SCIENTIFIC DETAILS

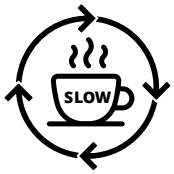
Gene	rsID	Genotype
CYP2A6, AC008537.1	rs56113850	TT

REFERENCES

[1] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Coffee, Tea, Mate, Methylxanthines and Methylglyoxal. Lyon (FR): International Agency for Research on Cancer; 1991. (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 51.) Caffeine.
[2] Institute of Medicine (US) Committee on Military Nutrition Research. Caffeine for the Sustainment of Mental Task Performance: Formulations for Military Operations. Washington (DC): National Academies Press (US); 2001. 2, Pharmacology of Caffeine.

SLOW METABOLIZER

RESULTS



The methods of caffeine production are extractive (direct decaffeination of green coffee beans using solvents, extraction from tea powders, waste and fragments of tea leaves and extraction from kola nuts) and synthetic through the methylation of various xanthines. Absorption of caffeine from the gastrointestinal tract is rapid and directly dependent on pH, in fact, after oral intake of doses of 5-8 mg per kg of body weight, peak plasma concentrations of 8-10 µg / ml were observed. with variations ranging from 15 to 120 minutes to reach the maximum peak. After absorption, caffeine is rapidly and evenly distributed in body fluids and then eliminated by apparent first-order kinetics, described by a one-compartment open model system. The metabolism of caffeine occurs mainly in the liver and is influenced by many factors (age, sex, hormones, liver disease, obesity, smoking and diet). The main element that acts on the metabolism of caffeine is the CYP1A2 isoform of cytochrome p450, the polymorphism of which explains the variability of pharmacokinetics between different subjects. Several loci have been identified and involved in caffeine consumption and have consequences on anxiety, sleep, and neurodegenerative or psychiatric disorders [1].

People with your genetic profile are likely to have a regular caffeine metabolic rate

SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP1A2	rs2069514	GG
CYP1A2	rs2069526	TT
CYP1A2	rs12720461	CC

REFERENCES

[1] Nehlig A. Interindividual Differences in Caffeine Metabolism and Factors Driving Caffeine Consumption. Pharmacol Rev. 2018 Apr;70(2):384-411.

PERCEPTION OF SALTY TASTE

RESULTS

Sodium is an essential element for the human body. It is used extensively as sodium chloride (table salt) in (processed) foods and consumed in excess by children and adults, exposing them to adverse health risks such as hypertension (also called high blood pressure) and cardiovascular diseases. Babies between the ages of three and four months can detect and prefer sodium chloride solutions over plain water, which is thought to be an unlearned biological response. The liking for water with sodium chloride decreases when infants enter early childhood, but the preference for foods (see soups or snacks) containing sodium chloride remains: children prefer higher salt concentrations than adults, so much so that adding salt to foods increases the consumption of these foods by children [1]. Although there is no correlation between the palatability of the taste of salt and the preference for salty foods by children, it is recommended to reduce exposure to salty foods during early childhood. Given that high salt intake is a significant risk factor for hypertension and is associated with cardiovascular events. Most countries have a high salt consumption; therefore, identifying an optimal strategy for reducing large-scale salt consumption can have a major impact on public health [2].

People with your genetic profile are likely to have an enhanced taste for salty foods

SCIENTIFIC DETAILS

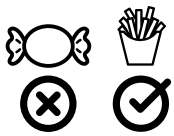
Gene	rsID	Genotype
TRPV1	rs8065080	CC

REFERENCES

[1] Liem DG. Infants' and Children's Salt Taste Perception and Liking: A Review. Nutrients. 2017 Sep 13;9(9):1011.
[2] Ha SK. Dietary salt intake and hypertension. Electrolyte Blood Press. 2014;12(1):7-18.

SALTY TASTE PREFERENCE

RESULTS



Saltiness is one of the basic tastes perceived by the taste buds. The ingredient that commonly stimulates the perception of saltiness on taste buds is table salt. However, the same sensation can be produced by any sodium salt, for example, sodium acetate, even if less intensely. Other non-sodium-containing salts, such as potassium or lithium salts, can also be perceived as salty; however, these salts can be toxic to human metabolism and induce a bitter taste [1].

People with your genetic profile are likely to have an enhanced propensity to prefer salty foods

SCIENTIFIC DETAILS

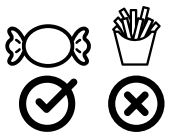
Gene	rsID	Genotype
TRPV1	rs150908	GA

REFERENCES

[1] Institute of Medicine (US) Committee on Strategies to Reduce Sodium Intake; Henney JE, Taylor CL, Boon CS, editors. Strategies to Reduce Sodium Intake in the United States. Washington (DC): National Academies Press (US); 2010. 3, Taste and Flavor Roles of Sodium in Foods: A Unique Challenge to Reducing Sodium Intake.

PROPENSITY TO CHOOSE SWEET FOODS

RESULTS



Sweetness is one of the five primary flavors, almost universally equated with a pleasant sensation [1]. Foods rich in simple carbohydrates, such as sugars, are generally associated with sweetness. Some molecules of natural origin or human invention are perceived as sweet but have much lower concentrations of sugars and can be used as low-calorie sweeteners. The chemical sensitivity of the perception of sweetness varies according to individuals and species and has been understood only in recent times. Some studies indicate that sensitivity to sugars and sweets has ancient evolutionary roots and is already present as chemotaxis in motile bacteria such as Escherichia coli. Babies prefer foods with a high concentration of sugars and, in particular, solutions with sugars sweeter than lactose, the sugar found in breast milk [2].

People with your genetic profile are likely to have a regular propensity to prefer sweet foods

SCIENTIFIC DETAILS

Gene	rsID	Genotype
PTGR1	rs117065036	TT
HMGN1P9 - HMGB3P13	rs151313984	CC
TAS1R2	rs35874116	TC
TAS1R3	rs307355	CC

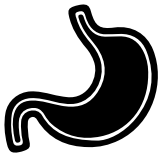
REFERENCES

[1] Eikemo M, Løseth GE, Johnstone T, Gjerstad J, Willoch F, Leknes S. Sweet taste pleasantness is modulated by morphine and naltrexone. Psychopharmacology (Berl). 2016 Oct;233(21-22):3711-3723.

[2] Forestell CA. Flavor Perception and Preference Development in Human Infants. Ann Nutr Metab. 2017;70 Suppl 3:17-25.

PREDISPOSITION TO EXPERIENCE AN ALTERED SENSE OF FULLNESS

RESULTS



Over the past few decades, the prevalence of overweight and obesity in societies worldwide has increased dramatically. Being overweight is a risk factor for many diseases such as type 2 diabetes, coronary heart disease, high blood pressure, and some forms of cancer. Increasing the filling capacity of foods can help people control their energy intake and weight. Total calorie intake can be reduced through meal replacement processes, whereby consumers learn to eat low-calorie products (such as vegetables) in place of foods with higher energy density. A promising path to increasing satiety is to make foods less caloric, for example, by replacing their fatty and / or carbohydrate components with water or air. You can also add extra fiber or change the structure of the food. It is now generally accepted among researchers that satiety is a complex interaction between physiological and non-physiological mechanisms. The actual choice of food is the result of a complex interaction between internal satiety cues and environmental cues such as health labels, portion sizes and perceived variety. Product characteristics that improve satiety need to be convincingly and responsibly communicated to consumers. This requires careful selection of the types of benefits to be communicated (e.g., prolonged fullness or delayed feelings of hunger). Significant changes in consumers' living environment are vital to end the overweight epidemic. Ultimately, the goal is to increase the satiety capacity of individual foods and make the environment less "toxic" by helping consumers control their energy intake in the short and long term [1].

People with your genetic profile are likely to have an altered sense of satiety after a meal

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FTO	rs9939609	TA
LEPR	rs1137101	AA
CCK	rs6809785	GG

REFERENCES

[1] Van Kleef E, Van Trijp JC, Van Den Borne JJ, Zondervan C. Successful development of satiety enhancing food products: towards a multidisciplinary agenda of research challenges. Crit Rev Food Sci Nutr. 2012;52(7):611-628.

PREDISPOSITION TO EAT BETWEEN MEALS

RESULTS

In achieving optimum health, it is important to limit the practice of eating between meals. Having such practice can lead to rapid weight gain in the consumption of excess calories. Experts recommend waiting three to five hours between meals [1].

People with your genetic profile are not likely to have a predisposition to eat between meals

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC116563.1 - AC131956.2	rs17049741	GA
FTO	rs9939609	TT

REFERENCES

[1] Njike VY, Smith TM, Shuval O, et al. Snack Food, Satiety, and Weight. Adv Nutr. 2016;7(5):866-878. Published 2016 Sep 15.

PREDISPOSITION TO EAT WHEN UNDER STRESS

RESULTS

Obesity is one of the most troubling diseases of our time. It is a multifactorial disease that is difficult to treat. Stress is one of the most critical factors in the development of food addiction disorder, and there are studies linking stress with the abuse of "comfort foods", which can help patients relieve stress. Stress refers to a challenge to natural homeostasis that the body naturally tends to resolve and return to its normal state. It is hypothesized that hyperpalatable foods can serve as "comfort food", which acts as a form of self-medication to dispel anxieties. In our current obesogenic environment where food is plentiful, palatable and easily accessible, the proliferation of stressors can drive non-homeostatic nutrition, in other words, eating without a real metabolic need. Repeated bouts of minor daily stressors that keep the stress system in a chronically activated state can alter the brain reward/motivation pathways involved in craving and seeking hyper-palatable foods and induce metabolic changes that promote weight and fat mass. The adaptations of the metabolic, neuroendocrine and neuronal pathways linked to weight can enhance food preference, desire and intake under stressful conditions. Individual differences in obesity susceptibility and types of stress can further moderate this process [1].

People with your genetic profile are not likely to have a predisposition to eat when under stress

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC096669.1	rs111940429	CC
AL359715.1	rs17810023	CC
AL136524.1 - RPL7P45	rs7337127	CC
SLC25A26, SLC25A26	rs145763646	GG
PRR5-ARHGAP8, ARHGAP8	rs726170	CC

REFERENCES

[1] Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. Nutrition. 2007 Nov-Dec;23(11-12):887-94.

PREDISPOSITION TO DEVELOP ADDICTION TO FOOD

RESULTS

Food addiction is a condition characterized by excessive consumption of comfort foods. It has a similar behavioral pattern to drug addiction. Psychological models play an important role in the development of food addiction. It has been shown that there is a two-way relationship between depression and obesity. Moreover, it has been observed that a depressed mood could change the food preferences of individuals and lead to an increase in the consumption of palatable foods to alleviate negative feelings. The effects of certain foods on the brain make it difficult for some people to avoid them. Food addiction works in a similar way to other addictions, which explains why some people fail to control themselves. These effects are mediated by neurotransmitters such as dopamine. The most problematic foods include candy and high-fat fried foods. No laboratory tests are available: the diagnosis is made only by studying the patient's behavioral patterns. To overcome food addiction, a person must be convinced that eliminating certain foods is the right thing. If you are unsure, writing down the pros and cons can help you make the decision [1].

People with your genetic profile are likely to have a predisposition to develop an addiction to food

SCIENTIFIC DETAILS

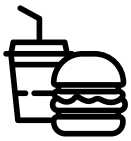
Gene	rsID	Genotype
AL356534.1	rs139878170	CA
PRKCA	rs74902201	GG
DRD2	rs1800497	GG

REFERENCES

[1] Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. Nutrition. 2007 Nov-Dec;23(11-12):887-94.

PREDISPOSITION TO FATTY FOOD ADDICTION

RESULTS



With food addiction, we refer to a situation where food intake is out of control and keeps increasing even if the negative consequences are visible, and it is impossible to reduce intake even if a reduction is desired. It has been observed that highly processed food was most likely to be involved in addiction [1].

People with your genetic profile are not likely to have a regular predisposition for fatty food intake

SCIENTIFIC DETAILS

Gene	rsID	Genotype
DRD2	rs1800497	GG
FTO	rs9939609	TA

REFERENCES

[1] Schulte EM, Avena NM, Gearhardt AN. Which foods may be addictive? The roles of processing, fat content, and glycemic load. PLoS One. 2015;10(2):e0117959. Published 2015 Feb 18.

PRUDENT DIETARY PATTERN

RESULTS

Two dietary models were identified: the Prudent model, characterized by a high intake of vegetables, fruit, whole grain products and low intakes of refined grain products, and the Western food model, based on a high intake of products such as refined cereals, desserts, sweets and processed meat. Findings from several studies support the role of a healthy eating pattern rather than the promotion of specific foods or nutrients. Furthermore, the gene expression profiles were different according to the dietary patterns. Understanding the motivational factors that guide food choices is important in addressing obesity, diabetes and cardiovascular disease. Eating behavior depends on physiological, psychological, social and genetic factors that influence meal times, the amount of food consumed, and food preferences [1].

People with your genetic profile are likely to have a healthy dietary pattern

SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC00466	rs76500500	TT
AL137026.3	rs76838052	CC

REFERENCES

[1] Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond). 2008 Aug;32 Suppl 3:S56-9.

PREDISPOSITION TO DEVELOP FAT TISSUE OVER LEAN TISSUE

RESULTS

Body mass can be divided into two main components: body fat, with energy storage functions, and lean mass, including muscles, organs and bones. Generally, those two components keep a balance. In cases of obesity, there is an increase in both body fat and lean mass. Changes in body fat are followed by similar changes in the fat-free tissues of the body. This relationship can be predicted from the body composition of stable weight individuals with widely varying body fat content. A second influence is energy intake. The lower the energy intake is (and the greater the energy deficit) in individuals undergoing diet-induced weight reduction, the more significant the contribution of lean body mass to the total weight loss will be [1].

People with your genetic profile are likely to have a predisposition for the accumulation of fat mass vs lean mass

SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC01787	rs12409479	GG
RIN2	rs4813371	TA
ZBTB46	rs6011111	CC

REFERENCES

[1] Pomeroy E, Macintosh A, Wells JCK, Cole TJ, Stock JT. Relationship between body mass, lean mass, fat mass, and limb bone cross-sectional geometry: Implications for estimating body mass and physique from the skeleton. Am J Phys Anthropol. 2018;166(1):56-69.

GENETIC PREDISPOSITION TO HIGH ENERGY EXPENDITURE AT REST

RESULTS

Weight stability in adults is maintained by balancing energy intake and energy expenditure. When the weight is stable, the body's energy stores do not fluctuate much, as evidenced by consistency in body weight. Sedentary lifestyles, however, can change this balance. That leads to a larger amount of energy being stored than used. Energy intake occurs via macronutrients such as proteins, fats, carbohydrates, and alcohol. Daily energy expenditure consists of four elements: the thermic effect of food, sleeping metabolic rate, the energy cost of arousal, and the energy cost of physical activity. Energy expenditure varies throughout the day, depending on body composition and size. Resting energy expenditure is the metabolic rate required to maintain the body's vital physiological functions. When energy intake exceeds expenditure, the excess is usually stored as body fat [1].

People with your genetic profile are likely to have a regular energy expenditure at rest

SCIENTIFIC DETAILS

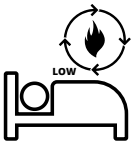
Gene	rsID	Genotype
KCNC1, SERGEF	rs142343672	GG
KLF12	rs61957289	TT
AC022167.3 - AC087190.2	rs146169233	CC
THSD7B	rs55691047	AA
VSNL1	rs62131523	AA

REFERENCES

[1] Galgani J, Ravussin E. Energy metabolism, fuel selection and body weight regulation. Int J Obes (Lond). 2008;32 Suppl 7(Suppl 7):S109-S119.

LOW RESTING METABOLIC RATE

RESULTS



To keep their temperature stable, humans use a lot of energy. This energy comes from the calories present in food. The resting metabolic rate is the amount of energy expended by the body at rest over a given period. The mean basal metabolic rate (BMR) for adult human males and females amounts to 1300-1500 kcal/day and 1600-1800 kcal/day, respectively. If the total loss of body heat overcomes the heat produced by the metabolic reactions, the body will be in a state of hypothermia. In such situations, the hypothalamus can increase the body's overall metabolic rate, generating more heat. Maintaining a stable body temperature requires a sufficient calorie intake and a correct metabolic response to external stimuli. If any of these requirements are not met, the body will not be able to maintain homeostasis. The purpose of metabolic reactions is to produce a usable and highly energetic final product, ATP. ATP feeds metabolic processes that maintain a stable temperature and is essential for the body to remain in homeostasis [1].

People with your genetic profile are likely to have a slow resting metabolic rate

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SULT2B1, AC008403.3	rs10401347	GA
AC092546.1 - AC006296.3	rs4698250	TC

REFERENCES

[1] Molé PA. Impact of energy intake and exercise on resting metabolic rate. Sports Med. 1990 Aug;10(2):72-87.

LEPTIN RESISTANCE

RESULTS

Leptin is a hormone that plays a vital role in the regulation of calorie intake and expenditure, including appetite and metabolism. Problems with the synthesis and secretion of leptin cause a lack of appetite control and can lead to obesity since the body appears to be in a permanent fasting state [1].

People with your genetic profile are likely to not have a genetic predisposition for leptin-resistance.

SCIENTIFIC DETAILS

Gene	rsID	Genotype
LEPR	rs1137101	AA

REFERENCES

[1] Ramos-Lobo AM, Donato J Jr. The role of leptin in health and disease. Temperature (Austin). 2017;4(3):258-291.

PREDISPOSITION TO RESPOND POSITIVELY TO THE MEDITERRANEAN DIET

RESULTS

The Mediterranean diet is a diet inspired by the foods common in the Mediterranean basin, which, according to doctors and nutritionists, represents a model diet for human health. The Mediterranean diet favors the consumption of healthy foods with low saturated fat content. The consumption of legumes, such as chickpeas, beans, lentils, peas, and whole grains, such as wheat, oats, amaranth, spelt, millet and barley, is encouraged. Fruits and vegetables play an important role in the Mediterranean diet, as long as they are in season. The use of olive oil is also a key feature of this diet. In 2010, the Mediterranean diet was recognized by UNESCO as a protected asset and included in the list of oral and intangible heritage of humanity. In addition to representing a healthy lifestyle and helping people keep fit, scientific research has highlighted how much this type of diet helps in the well-being of the body and the prevention of diseases. It is scientifically proven that a healthy and balanced diet such as the Mediterranean diet can decrease the risk of cancer, cardiovascular diseases or diabetes [1,2].

People with your genetic profile are likely to have an enhanced healthy response to the Mediterranean diet

SCIENTIFIC DETAILS

Gene	rsID	Genotype
TCF7L2	rs7903146	TT

REFERENCES

[1] Lăcătușu CM, Grigorescu ED, Floria M, Onofriescu A, Mihai BM. The Mediterranean Diet: From an Environment-Driven Food Culture to an Emerging Medical Prescription. Int J Environ Res Public Health. 2019;16(6):942. Published 2019 Mar 15.
[2] Tuttolomondo A, Simonetta I, Daidone M, Mogavero A, Ortello A, Pinto A. Metabolic and Vascular Effect of the Mediterranean Diet. Int J Mol Sci. 2019;20(19):4716. Published 2019 Sep 23.

GENETIC SENSITIVITY TO GLUTEN

RESULTS

Gluten is a protein that is found in wheat, barley, and rye grains; it is important to take an extremely close look at the food labels for individuals who have a genetic sensitivity to gluten [1]. Healthy and gluten-free options include brown rice, quinoa, oats, and millet.

People with your genetic profile are likely to have gluten sensitivity

SCIENTIFIC DETAILS

Gene	rsID	Genotype
RBFOX1	rs59325236	GA

REFERENCES

[1] Catassi C. Gluten Sensitivity. Ann Nutr Metab. 2015;67 Suppl 2:16-26. doi: 10.1159/000440990. Epub 2015 Nov 26. PMID: 26605537.

PREDISPOSITION TO LACTOSE INTOLERANCE

RESULTS

Lactose intolerance is a disorder characterized by the inability to digest lactose. Under normal conditions, lactose is broken down by the lactase enzyme into its two constituent sugars, glucose and galactose. People with lactose intolerance do not have enough quantities of the lactase enzyme. There are a number of lactose-free dairy products available to buy that are suitable for people with lactose intolerance. These contain the same vitamins and minerals as standard dairy products, but they also have an added enzyme called lactase, which helps digest any lactose so the products do not trigger any symptoms. Examples of lactose free products includes the following: soya yoghurts and cheeses, coconut-based yoghurts and cheeses, almond milk, yoghurts and cheeses, rice milk, oat milk and hazelnut milk [1,2,3].

People with your genetic profile are not likely to have predisposition for lactose intolerance

SCIENTIFIC DETAILS

Gene	rsID	Genotype
MCM6	rs4988235	GG
MCM6	rs776037433	TT
MCM6	rs138808270	GG

REFERENCES

[1] Di Costanzo M, Berni Canani R. Lactose Intolerance: Common Misunderstandings. Ann Nutr Metab. 2018;73 Suppl 4:30-37.
[2] Amiri M, Diekmann L, von Köckritz-Blickwede M, Naim HY. The Diverse Forms of Lactose Intolerance and the Putative Linkage to Several Cancers. Nutrients. 2015 Aug 28;7(9):7209-30.
[3] Swagerty DL Jr, Walling AD, Klein RM. Lactose intolerance. Am Fam Physician. 2002 May 1;65(9):1845-50. Erratum in: Am Fam Physician. 2003 Mar 15;67(6):1195.

PREDISPOSITION TO DEVELOP AN EGG ALLERGY

RESULTS

Egg allergy is an Ig-E mediated adverse immunological response triggered by exposure to egg white or egg yolk allergens. Mast cells and basophils are the main effectors of the immune response in this form of allergy [1]. Children with egg allergy typically present with urticaria or angioedema, which appear within minutes to hours after exposure. The severity of the immune reaction is variable, and although rare, fatal reactions have been reported. Egg allergy can also be associated with non-Ig-E mediated and mixed IgE- and non-IgE-mediated reactions, including atopic dermatitis and gastroenteropathy [2,3].

People with your genetic profile are likely to have a predisposition to develop an egg allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SERPINB7 - SERPINB2	rs1243064	TA
BMPR1B	rs17023017	TG

REFERENCES

[1] Mathew P, Pfleghaar JL. Egg Allergy. [Updated 2020 Jul 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

[2] Quirce S., Maranon F., Umpierrez A., de las Heras M., Fernandez-Caldas E., Sastre J. Chicken serum albumin (Gal d 5*) is a partially heat-labile-inhalant and food allergen implicated in the bird-egg syndrome. Allergy. 2001;56:754–762.

[3] Caubet JC, Wang J. Current understanding of egg allergy. Pediatr Clin North Am. 2011;58(2):427-xi.

PREDISPOSITION TO DEVELOP A MILK ALLERGY

RESULTS

Milk allergy is a common condition in infants and children. It occurs when there is an allergic reaction to proteins in cow milk. Milk allergy is characterized by a variety of symptoms. It can manifest within the first months of life and regress by the age of six years. Symptoms are divided into two categories, based on the time of onset: IgE mediated, rapid (which occur within an hour after ingestion) and non-IgE mediated, slow (which occur after hours or days). Rapid symptoms include urticaria, wheezing, itching, angioedema, coughing or shortness of breath, vomiting and anaphylaxis. Common slow symptoms are diarrhea, abdominal cramps and colic. Anaphylaxis is a critical condition that requires treatment with epinephrine to manage severe symptoms such as increased work of breathing, constriction of airways, swollen throat and facial flushing [1]. With the elimination of cow milk and its derivatives from the diet, the prognosis in infancy and young childhood is good. Milk allergy in children is typically associated with other forms of food allergy; associated adverse reactions can develop in up to 50% of them [2].

People with your genetic profile are not likely to have a predisposition to develop a milk allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC01909	rs17236768	TT

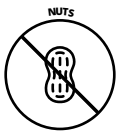
REFERENCES

[1] Edwards CW, Younus MA. Cow Milk Allergy. [Updated 2020 Nov 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

[2] Oranje AP, Wolkerstorfer A, de Waard-van der Spek FB. Natural course of cow's milk allergy in childhood atopic eczema/dermatitis syndrome. Ann Allergy Asthma Immunol. 2002 Dec;89(6 Suppl 1):52-5.

PREDISPOSITION TO DEVELOP A PEANUT ALLERGY

RESULTS



Peanut allergy is one of the most common forms of food allergy in infants and young children. Although the average age of onset is 18 months, it can manifest later in childhood or adulthood both as an allergy in itself and as part of the pollen-food allergy syndrome. Peanuts contain a high amount of protein, and their allergenicity can derive from the manufacturing processes. The immune reaction to peanut allergens is Ig-E mediated and generally occurs rapidly within two hours of exposure. Symptoms are due to the release of vasoactive cytokines from mast cells and basophils, ranging from moderate to severe. Cutaneous manifestations include urticaria and angioedema; gastrointestinal manifestations: abdominal pain and vomiting. The most severe symptoms are respiratory and cardiovascular ones and can manifest with chest tightness, difficulty breathing, difficulty swallowing, dizziness, collapse, pallor, floppiness/lethargy (young children) [1].

People with your genetic profile are not likely to have a predisposition to develop a peanut allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
MMP12 - BOLA3P1	rs144897250	CC
RNU6-92P - AC083873.1	rs78048444	AA

REFERENCES

[1] Frith, K. and Katelaris, C.H. (2019), Current perspectives on peanut allergy. Intern Med J, 49: 1480-1487.

PREDISPOSITION TO DEVELOP A FISH ALLERGY

RESULTS

Fish allergy is a condition that can lead to severe consequences due to adverse reactions to allergens contained in fish but also due to various toxins and parasites such as Anisakis [1]. Fish is an important food source of valuable substances such as omega-3 fatty acids and fat-soluble vitamins. Still, it also contains allergens that can lead to adverse reactions: the major allergen is parvalbumin, enolase, aldolase, vitellogenin and tropomyosin [2]. Predisposed subjects become sensitized when they contact allergens via the gastrointestinal tract (through ingestion), via the respiratory system, by inhaling aeroallergens or through skin contact [3]. The main clinical manifestations of fish allergy are urticaria, rhinitis, abdominal pain, diarrhea, angioedema, asthma and anaphylaxis. This form of food allergy is characterized by cross-reactivity among fish species, especially if they are very close [4].

People with your genetic profile are likely to have a predisposition to develop a fish allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Sharp MF, Lopata AL. Fish allergy: in review. Clin Rev Allergy Immunol. 2014 Jun;46(3):258-71.

[2] Dijkema, D., Emons, J.A.M., Van de Ven, A.A.J.M. et al. Fish Allergy: Fishing for Novel Diagnostic and Therapeutic Options. Clinic Rev Allerg Immunol (2020).

[3] Environmental exposure characterization of fish processing workers. Jeebhay MF, Robins TG, Seixas N, Baatjies R, George DA, Rusford E, Lehrer SB, Lopata AL Ann Occup Hyg. 2005 Jul; 49(5):423-37.

[4] Kuehn A, Swoboda I, Arumugam K, Hilger C, Hentges F. Fish allergens at a glance: variable allergenicity of parvalbumins, the major fish allergens. Front Immunol. 2014;5:179.

PREDISPOSITION TO DEVELOP A MOLLUSCS/CRUSTACEANS ALLERGY

RESULTS

Allergy to "shellfish," including molluscs and crustaceans, can give varying symptoms ranging from urticaria to anaphylactic reactions [1]. Shellfish poisoning is easily mistaken for an allergic reaction; five forms of this condition have been identified: 1) Paralytic 2) Neurotoxic 3) Diarrhetic 4) Amnesic 5) Azaspiracids shellfish poisoning [2]. Symptoms of shellfish allergy can appear a few minutes to an hour after ingestion. They may include atopic dermatitis, swelling of the lips, face, tongue and throat, nasal congestion or trouble breathing, abdominal pain, diarrhea, nausea or vomiting. The most serious reaction, anaphylaxis, is a medical emergency that requires immediate administration of epinephrine [3].

People with your genetic profile are likely to have a predisposition to develop a shellfish allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

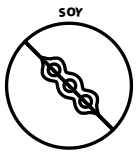
[1] Lopata, A.L., O'Hehir, R.E. and Lehrer, S.B. (2010), Shellfish allergy. Clinical & Experimental Allergy, 40: 850-858.

[2] Woo CK, Bahna SL. Not all shellfish "allergy" is allergy!. Clin Transl Allergy. 2011;1(1):3.

[3] Tintinalli JE, et al. Anaphylaxis, allergies, and angioedema. In: Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 8th ed. New York, N.Y.: The McGraw Hill Companies; 2016.

PREDISPOSITION TO DEVELOP A SOY ALLERGY

RESULTS



Soy, a product of soybeans, is a common allergen. For many people, the symptoms of soy allergy are bothersome but not severe. They include tingling in the mouth, itching, eczema, swelling of the lips, face, tongue and throat, breathing difficulty, abdominal pain, diarrhea, nausea or vomiting and skin redness (flushing). The worst reaction, anaphylaxis, is rare in this form of allergy [1]. Clinical manifestations of soy allergy are Ig-E mediated. Of the 28 soy proteins capable of binding Ig-E, only some of them have been identified as major antigens [2,3].

People with your genetic profile are likely to have a predisposition to develop a soy allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG

REFERENCES

[1] Burks W. Clinical manifestations of food allergy: An overview.

[2] Allergenicity of major component proteins of soybean. Shibasaki M, Suzuki S, Tajima S, Nemoto H, Kuroume T Int Arch Allergy Appl Immunol. 1980; 61(4):441-8.

[3] Purification and characterization of a soybean hull allergen responsible for the Barcelona asthma outbreaks. II. Purification and sequencing of the Gly m 2 allergen.
Codina R, Lockey RF, Fernández-Caldas E, Rama R. Clin Exp Allergy. 1997 Apr; 27(4):424-30.

PREDISPOSITION TO DEVELOP FRUIT AND VEGETABLE ALLERGIES

RESULTS

Fruit and vegetable allergies are a common form of food allergy in young people and adults. They can result from gastrointestinal sensitization to food allergens or sensitization to inhalant allergens [1]. One of the main characteristics of this form of food allergy is its cross-reactivity with pollen due to IgE antibodies directed against "panallergens," which are proteins of the vegetable kingdom implicated in important biological functions. The syndrome resulting from this condition is called oral allergy syndrome, defined by localized symptoms in the oral mucosa. Another term used for this syndrome is food allergy to pollen. This syndrome consists of a subject, previously sensitized with pollen by inhalation, who then exhibits an allergic reaction following contact with antigens structurally similar to pollen in fruit and vegetables. Foods of plant origin most capable of triggering allergic reactions are apple, apricot, carrot, cherry, hazelnut, kiwi, banana, watermelon, zucchini, broccoli, onion, garlic and others. Symptoms include itching or tingling of the mouth, tongue or lips, swelling of the mouth, tongue, and/or throat (this is known as angioedema). It rarely causes vomiting, stomach cramps, or diarrhea and, very rarely, anaphylaxis [2].

People with your genetic profile are likely to have a predisposition to develop an allergy to fruits and vegetables

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Fernández-Rivas M, Benito C, González-Mancebo E, de Durana DA. Allergies to fruits and vegetables. Pediatr Allergy Immunol. 2008 Dec;19(8):675-81.

[2] Kondo Y, Urisu A. Oral allergy syndrome. Allergol Int. 2009 Dec;58(4):485-91.

PREDISPOSITION TO DEVELOP SEED ALLERGY (GENERIC)

RESULTS

Seed allergies are among the most common food allergies across all age groups (1.4%). Allergic reactions to seeds are the most common cause of fatal food allergy reactions. Allergy to different types of seeds often coexists and is estimated to be between 20% and 50% based on self-reported questionnaires, IgE test results (by specific IgE measurements or skin prick testing), or both. However, data from these tests could overestimate the actual prevalence of allergic reactions by overreporting allergic symptoms. Sesame seed allergy often coexists with peanut allergy. About 58% to 84% of children with sesame seed sensitization or reported sesame seed allergy were also sensitized or had reported allergic reactions to peanut and 25% of children with peanut allergy were reported to have sesame seed allergy [1].

People with your genetic profile are likely to have a predisposition to develop a seed allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Helen A. Brough, Jean-Christoph Caubet, Angel Mazon, Diab Haddad, Marcel M. Bergmann, Jacqueline Wassenberg, Valentina Panetta, Rosalynd Gourgey, Suzana Radulovic, Maria Nieto, Alexandra F. Santos, Antonio Nieto, Gideon Lack, Philippe A. Eigenmann, Defining challenge-proven coexistent nut and sesame seed allergy: A prospective multicenter European study, Journal of Allergy and Clinical Immunology, Volume 145, Issue 4, 2020, Pages 1231-1239.

PREDISPOSITION TO DEVELOP A SALICYLATE ALLERGY

RESULTS

Salicylates are a group of chemicals derived from salicylic acid. They are found naturally in some foods and are also synthetically produced for various products such as aspirin, toothpaste, and food preservatives. Adverse reactions can occur both after contact with natural and synthetic forms. Plants naturally produce salicylates as a form of defense against insects, fungi and diseases [1]. These molecules are found in a wide variety of foods, including fruits, vegetables, coffee, tea, nuts, spices and honey. Compared to foods, salicylate medications such as aspirin contain high amounts of it; therefore, salicylate intolerance is more commonly linked to medication. For example, the dietary intake of salicylates is on average 10-200 mg per day, compared to 325-650 mg contained in aspirin, depending on the type [2].

People with your genetic profile are not likely to have a predisposition to develop a salicylates allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
HLA-DPB1, HLA-DPB1, HLA-DPB1, HLA-DPB1, HLA-DPB1, HLA-DPB1, HLA-DPB1	rs1042151	AG

REFERENCES

[1] Duthie GG, Wood AD. Natural salicylates: foods, functions and disease prevention. Food Funct. 2011 Sep;2(9):515-20.
[2] Baenkler HW. Salicylate intolerance: pathophysiology, clinical spectrum, diagnosis and treatment. Dtsch Arztebl Int. 2008;105(8):137-142.

TARTRAZINE ALLERGY

RESULTS

Tartrazine is an azoic, orange-colored, water-soluble food dye. It is widely used in food, pharmaceuticals, cosmetics and textiles. Tartrazine can potentially cause adverse health effects in humans: common conditions are hyperactivity in children, allergy and asthma [1].

People with your genetic profile are likely to have a predisposition to develop a tartrazine allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Rovina K, Siddiquee S, Shaarani SM. A Review of Extraction and Analytical Methods for the Determination of Tartrazine (E 102) in Foodstuffs. Crit Rev Anal Chem. 2017 Jul 4;47(4):309-324.

PREDISPOSITION TO DEVELOP SENSITIVITY TO SULPHITES

RESULTS

The sulfite ion is an anion composed of sulfur and oxygen. Treatment of wine with sulphites (which exploits the biocidal and antioxidant properties of sulfur dioxide SO2) can involve repeated exposures of operators to significant quantities of SO2. Sulfites are chemicals found naturally in some foods or added as additives [1].

People with your genetic profile are likely to have a predisposition to develop a sulfites sensitivity

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Testud F, Matray D, Lambert R, Hillion B, Blanchet C, Teisseire C, Thibaudier JM, Raoux C, Pacheco Y. Manifestations respiratoires dues à l'anhydride sulfureux en cave de vinification: 6 observations [Respiratory manifestations after exposure to sulfurous anhydride in wine-cellar workers: 6 case reports]. Rev Mal Respir. 2000 Feb;17(1):103-8. French.

PREDISPOSITION TO DEVELOP SENSITIVITY TO METABISULPHITES

RESULTS

Sodium metabisulfite is a sodium salt of metabisulfite acid. It is a harmful and irritating compound used for the preservation of alcoholic beverages and as a preservative and antioxidant for foods. It can cause allergic reactions in subjects sensitive to sulfites, adverse respiratory reactions in asthmatics and anaphylaxis. It is used to clean equipment for the production of domestic beverages and the production of wine or beer and water desalination plants for the production of drinking water. In medicine, it is added in local anesthetic solutions to extend the shelf-life and used as an excipient in some tablets [1].

People with your genetic profile are likely to have a predisposition to develop a metabisulphites sensitivity

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] National Center for Biotechnology Information (2021). PubChem Compound Summary for CID 656671, Sodium metabisulfite.

PREDISPOSITION TO DEVELOP AN ALLERGY TO BISULFITES

RESULTS

Sodium bisulfite is a chemical compound with the formula NaHSO3. In the presence of acids, it releases sulfur dioxide, a toxic gas. It reacts rapidly with oxygen to form sodium bisulfate. Sodium bisulfite is used in wines as an alternative to sodium metabisulphite and by releasing sulfur dioxide it kills the microorganisms present in the must before fermentation, then it is added again as a preservative during the bottling of the wine. It is also used in fruit juices and preserves to prevent oxidation and microbial proliferation. Sodium bisulfite can cause an allergic reaction in people with a sulfite allergy [1].

People with your genetic profile are likely to have a predisposition to develop a bisulfites sensitivity

SCIENTIFIC DETAILS

Gene	rsID	Genotype
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] National Center for Biotechnology Information (2021). PubChem Compound Summary for CID 23665763, Sodium bisulfite.

PREDISPOSITION TO DEVELOP A WALNUT ALLERGY

RESULTS

Nut and seed allergies can also be severe, causing life-threatening reactions. There is also a milder form of nut allergy associated with birch pollen allergy, the symptoms of which are largely limited to the oral mucosa, causing a condition called "oral allergy syndrome" (OAS). This condition is linked to cross-reactivity phenomena between the antigens present in nuts and those present in pollen. Reactions to nuts and seeds can also occur due to hidden ingredients related to their handling [1].

People with your genetic profile are not likely to have a predisposition to develop a walnut allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
MMP12 - BOLA3P1	rs144897250	CC
RNU6-92P - AC083873.1	rs78048444	AA

REFERENCES

[1] Price A, Ramachandran S, Smith GP, Stevenson ML, Pomeranz MK, Cohen DE. Oral allergy syndrome (pollen-food allergy syndrome). Dermatitis. 2015 Mar-Apr;26(2):78-88.

PREDISPOSITION TO DEVELOP A KIWI ALLERGY

RESULTS

Kiwi is an edible berry, the two main varieties: green and yellow (or gold). Kiwi is a fruit rich in vitamin C (85 mg / 100 g), potassium, magnesium, vitamin E, copper, iron and fiber. The high potassium content and sodium poverty make it the ideal fruit for athletes, because it reduces the risk of muscle cramps. It is recommended for those with digestive problems and promotes intestinal transit. Ig-E mediates the clinical symptoms of kiwi allergy and, as happens in allergic forms to other fruits, falls within the Oral Allergic Syndrome (OAS). Skin manifestations include urticaria and atopic dermatitis, but asthma and gastrointestinal symptoms may also occur, as well as anaphylactic shock [1].

People with your genetic profile are likely to have a predisposition to develop a kiwi allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Gawrońska-Ukleja E, Różalska A, Ukleja-Sokołowska N, Zbikowska-Gotz M, Bartuzi Z. Anaphylaxis after accidental ingestion of kiwi fruit. Postepy Dermatol Alergol. 2013;30(3):192-194.

PREDISPOSITION TO DEVELOP A PINE NUT ALLERGY

RESULTS

Pine nuts are edible seeds deriving from some species of pine. They are rich in protein, dietary fiber, vitamins (especially E, B and K), calcium, magnesium and iron. Despite the numerous beneficial health effects of consuming pine nuts, they can be responsible for severe allergic reactions, which appear to be characterized by low Ige cross-reactivity with other similar foods such as walnuts [1].

People with your genetic profile are not likely to have a predisposition to develop a pine nuts allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
MMP12 - BOLA3P1	rs144897250	CC
RNU6-92P - AC083873.1	rs78048444	AA

REFERENCES

[1] Cabanillas B, Novak N. Reactions to Pine Nut: A Review. Department of Dermatology and Allergy, University of Bonn Medical Center, Bonn, Germany, J Investig Allergol Clin Immunol 2015; Vol. 25(5): 329-333 Allergic

PREDISPOSITION TO DEVELOP A WHEAT ALLERGY

RESULTS

Despite its many benefits, soft wheat is recognized as a food allergen capable of activating immune responses mediated by Ig-E and not Ig-E. Children experience wheat allergy more frequently than adults, especially if this food is introduced after six months of life. Wheat allergy is the consequence of releasing mediators such as histamine, platelet activating factor, leukotrienes, mast cells and basophils. People with wheat allergies can develop diarrhea, constipation, bloating, abdominal pain, anorexia, flatulence, weight loss, poor growth in childhood, anemia, dermatitis herpetiformis, fatigue, and even osteoporosis when exposed to wheat. The first-line treatment in managing wheat allergies is to avoid dietary intake and the inhalation of wheat allergens. In cases of anaphylaxis, the administration of adrenaline is a life-saving treatment. If you suspect you have a wheat allergy, you should see a doctor as soon as possible as this condition can have serious consequences [1].

People with your genetic profile are not likely to have a predisposition to develop a wheat allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
IL-18	rs181720	GG

REFERENCES

[1] Patel N, Samant H. Wheat Allergy. [Updated 2020 Oct 15]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-.

PREDISPOSITION TO DEVELOP A CORN ALLERGY

RESULTS

Corn is the traditional food of many people around the world. From Latin America to Europe to parts of North America and Africa, corn is a staple. Corn is used in the food industry to extract starch and oil or for fermentation to produce alcoholic beverages or bioethanol for energy purposes. The nutritional properties of corn intended for human consumption are modest. In addition to a good amount of carbohydrates, it contains some nutrients and B group vitamins. Furthermore, its protein component is low in lysine and tryptophan, two essential amino acids. Allergic reactions to corn can present with: hives, itching, particularly in or around the mouth (oral allergy syndrome), flushing of the skin, hay fever-like symptoms, wheezing, asthma, headaches, abdominal pain, nausea and/or vomiting and diarrhea [1].

People with your genetic profile are not likely to have a predisposition to develop a corn allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
SERPINB7	rs12964116	AG
AL035415.1 - AC099796.2	rs12121623	GG
AL445255.1	rs976078	AA

REFERENCES

[1] American College of Allergy, Asthma & Immunology. Corn Allergy. Updated March 8, 2019.

PREDISPOSITION TO DEVELOP AN AMARANTH GRAIN ALLERGY

RESULTS

Amaranth is a plant with about sixty different species, but only three are considered good seed producers: Caudatus, Cruentus and Hypochondriacus. It is rich in proteins (up to 16%), with a high biological value. Compared to cereals, it contains double the amount of lysine, an essential amino acid that almost all cereals lack. It has a high content of calcium, phosphorus, magnesium and iron. Thanks also to the high fiber content, it has a positive effect on digestion. Being gluten-free, it is suitable for the diet of those who have celiac disease or intestinal problems and children during the weaning period. Allergic reactions to amaranth are rare [1].

People with your genetic profile are not likely to have a predisposition to develop a amaranth allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG

REFERENCES

[1] R. Vaswani, V. Garg, B. Khim, Y. Huang, S. Vaswani. ANAPHYLACTIC REACTION TO AMARANTH (AMARANTHUS PANICULATUS). Annals of Allergy, Asthma & Immunology, Volume 121, Issue 5, Supplement, 2018, Pages S113-S114.

PREDISPOSITION TO DEVELOP A CASSAVA ALLERGY

RESULTS

Cassava is known also as manioc, mandioca, yucca, or tapioca, and it is the common name for the tuber Manihot esculenta Crantz. It belongs to the Euphorbiaceae family from the West Indies, Latin America, and Africa. A staple food in South America and Africa regions, cassava is eaten cooked, fried, or in the form of flour for bread, pastry, and cakes. It is necessary to remove the skin and then grind the flesh, soak it repeatedly, and cook it to avoid an excess of hydrocyanic acid. The ingestion of poorly processed cassava may cause tropical pancreatic diabetes, tropical ataxic neuropathy, and goiter. People who are allergic to latex (Hevea brasiliensis) may exhibit cross-hypersensitivity with foods containing cassava [1].

People with your genetic profile are not likely to have a predisposition to develop a cassava allergy

SCIENTIFIC DETAILS

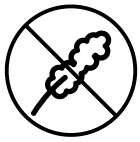
Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG
AL445255.1	rs976078	AA

REFERENCES

[1] Ibero M, Castillo MJ, Pineda F. Allergy to cassava: a new allergenic food with cross-reactivity to latex. J Investig Allergol Clin Immunol. 2007;17(6):409-12.

PREDISPOSITION TO DEVELOP A QUINOA ALLERGY

RESULTS



Quinoa (Chenopodium quinoa Willd) was known as the "golden grain" by the native Andean people in South America. Quinoa has been a source of valuable food for thousands of years, and it can produce a variety of secondary metabolites with broad spectra of bioactivities. In the past 40 years, at least 193 secondary metabolites have been identified from quinoa. They include flavonoids, terpenoids, steroids, phenolic acids, and nitrogen-containing compounds. These metabolites have many properties such as insecticidal, molluscicidal and antimicrobial activities. Quinoa also contains metabolites with antioxidant, cytotoxic, anti-diabetic and anti-inflammatory properties. There are many scientific reviews on quinoa. Most of them focused on the nutritional, functional and antinutritional aspects, abiotic stress responses, biodiversity and sustainability, or only a specific topic of quinoa secondary metabolites and their biological activities such as steroids and triterpenoid saponins. No review covers almost all secondary metabolites and their biological [1].

People with your genetic profile are not likely to have a predisposition to develop a quinoa allergy

SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC104088.1 - AC078883.3	rs115218289	CC
ANGPT4	rs523865	AA
SKAP1	rs200314279	TA
AL035415.1 - AC099796.2	rs12121623	GG
SERPINB7	rs12964116	AG

REFERENCES

[1] Lin M, Han P, Li Y, Wang W, Lai D, Zhou L. Quinoa Secondary Metabolites and Their Biological Activities or Functions. Molecules. 2019 Jul 9;24(13):2512.

SENSITIVITY TO NICKEL

RESULTS

Sensitivity to nickel is one of the most common metal sensitivities in humans. Nickel sensitivity tends to persist throughout life. Sensitivity to nickel is more common in females than males. Few patients develop vesicular-type hand eczema following dietary nickel ingestion. Such hand eczema also flares up when these patients are treated with oral nickel sulfate [1].

People with your genetic profile are likely to have regular nickel sensitivity

SCIENTIFIC DETAILS

Gene	rsID	Genotype
FLG	rs61816761	GG

REFERENCES

[1] Sharma AD. Low nickel diet in dermatology. Indian J Dermatol. 2013;58(3):240.

GLOSSARY

ALLELE	An allele is a variant form of a gene that is located at a specific position, or genetic locus, on a specific chromosome. Humans have two alleles at each genetic locus, with one allele inherited from each parent.
CHROMOSOME	A chromosome is a condensed thread-like structure of DNA that carries hereditary information, or genes. Human cells have 22 chromosome pairs plus two sex chromosomes, giving a total of 46 per cell.
GENOTYPE	The genetic makeup of an individual organism. It may also refer to just a particular gene or set of genes carried by an individual. The genotype determines the phenotype, or observable traits of the organism.
SNP	Single nucleotide polymorphisms, frequently called SNPs, are the most common type of genetic variation among people. A SNP is a variation in a single nucleotide that occurs at a specific position in the genome.
GENE	Genes are the basic units of inheritance. They contain DNA and are present in two forms called alleles.