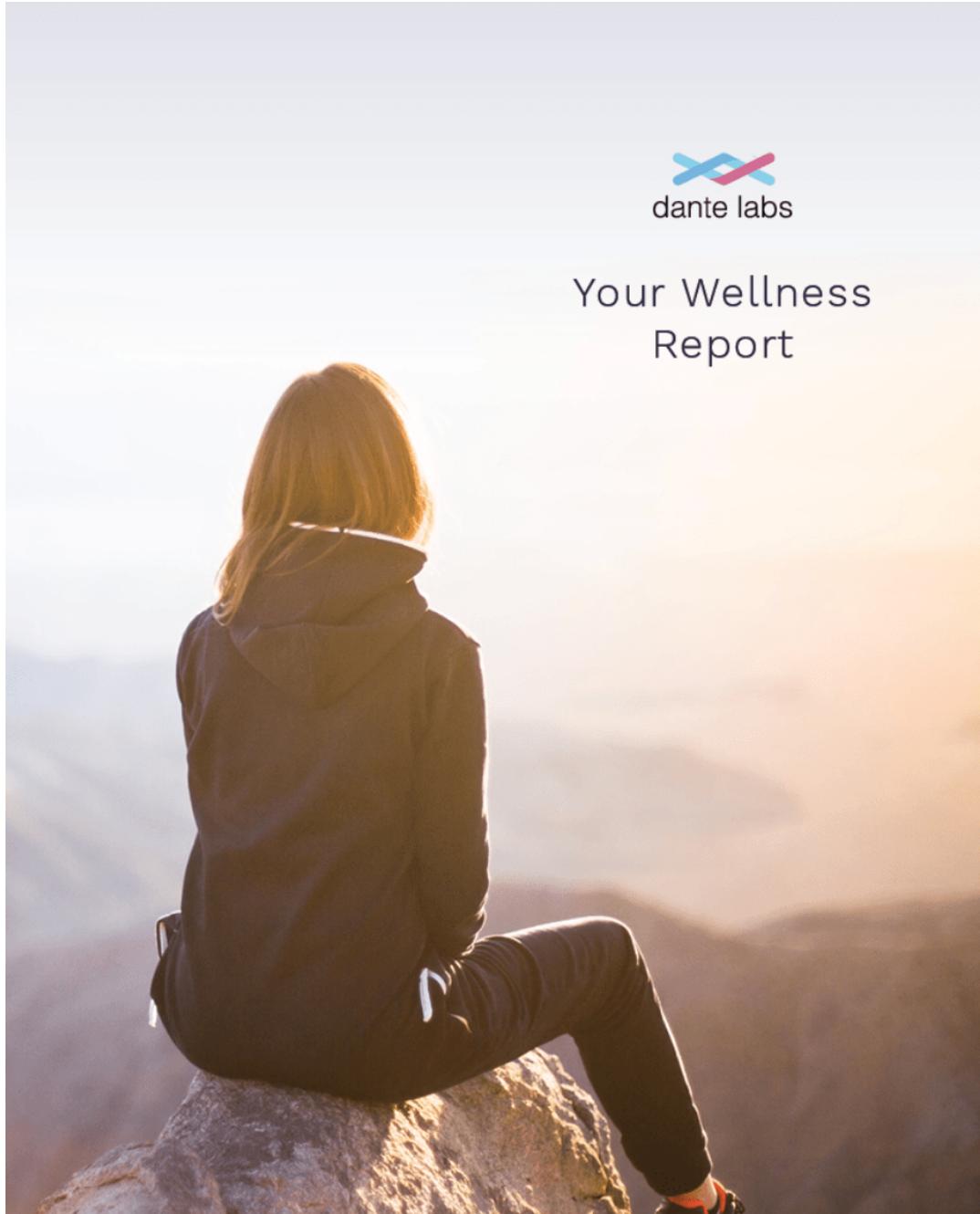




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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528





INTRODUCTION

This document is your genetic report, which is a straightforward and non-technical presentation of the results from your Dante Labs Genetic Wellness Test. It provides clear solutions to optimize your health and longevity. The insights obtained from learning about your genes may enable you, in partnership with your healthcare provider, formulate a plan to live a longer, more vibrant life. Our report tells you how your DNA can affect your chances of developing certain traits and health conditions.

Genetic variants are differences in DNA between people. Some variants may increase the risk of developing certain health conditions. However, not everyone with a risk variant will develop these health conditions. For many of these conditions, people without a risk variant can also develop them. Some variants are more common in certain ethnicities. The effect a variant has on risk for a health condition is often best studied in those ethnicities. Since families share DNA, having a family history of a condition can increase risk. If you have a variant, your family members may also have that variant. For certain conditions, genetics is just one part of a person's total risk. You may be able to manage your risk for some conditions by managing other risk factors. Our tests do not diagnose any health conditions.

Dante Labs recommends that you discuss your report with a healthcare provider/geneticist or Genetic Counselor in order to correctly interpret the results. As Science progresses, variants may be subject to score changes or reclassification.

QUICK SUMMARY

HEALTH	
CONDITION NAME	MAIN MESSAGE
Susceptibility to calcium deficiency	People with your genetic profile are likely to have a predisposition for a calcium deficiency.
Predisposition to iron deficiency	People with your genetic profile are likely to have regular iron levels.
Predisposition to Biotin deficiency	People with your genetic profile are likely to have regular biotin levels.
Predisposition to folic acid deficiency	People with your genetic profile are likely to have folic acid deficiency.
Predisposition to ascorbic acid deficiency	People with your genetic profile are likely to have regular ascorbic acid levels.
Predisposition to folate deficiency (homocysteine levels)	People with your genetic profile are likely to have a predisposition to develop folate deficiency.
Predisposition to a mineral salts deficiency (generic)	People with your genetic profile are likely to have regular levels of mineral salts.
Sodium balance (Na)	People with your genetic profile are likely to have a predisposition to have an enhanced sodium level.
Potassium balance (K)	People with your genetic profile are likely to have a regular potassium balance.
Balance of sodium / potassium ratio (Na / K)	People with your genetic profile are likely to have a regular balance ratio of sodium/potassium.
Morning / night chronotype	People with your genetic profile are likely to have a propensity to wake up early in the morning.
Predisposition to Coenzyme Q deficiency	People with your genetic profile are likely to have a predisposition to develop a Coenzyme -Q deficiency.
Predisposition to Choline deficiency	People with your genetic profile are not likely to have a predisposition to develop a choline deficiency.
Predisposition to copper deficiency	People with your genetic profile are not likely to have a predisposition to develop a copper deficiency.



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CONDITION NAME	MAIN MESSAGE
Predisposition to iron overload	People with your genetic profile are not likely to have a predisposition to experience an iron overload.
Predisposition to Lutein and Zeaxanthin deficiency	People with your genetic profile are likely to have a predisposition for a lutein and zeaxanthin deficiency.
Predisposition to Magnesium deficiency	People with your genetic profile are likely to have a predisposition for magnesium deficiency.
Predisposition to phosphorus deficiency	People with your genetic profile are likely to have regular phosphorus levels.
Predisposition to Selenium deficiency	People with your genetic profile are likely to have a predisposition for selenium deficiency.
Predisposition to Carotene deficiency	People with your genetic profile are likely to have regular carotene levels.
Predisposition to Retinol deficiency	People with your genetic profile are likely to have regular retinol levels.
Predisposition to Thiamine deficiency	People with your genetic profile are likely to have regular thiamine levels.
Predisposition to Riboflavin deficiency	People with your genetic profile are likely to have a predisposition for riboflavin deficiency.
Predisposition to Niacin or nicotinamide deficiency	People with your genetic profile are likely to have a predisposition for niacin deficiency.
Predisposition to the deficiency of pantothenic acid	People with your genetic profile are likely to have a predisposition for pantothenic acid deficiency.
Predisposition to Pyridoxine, Pyridoxal, Pyridoxamine deficiency	People with your genetic profile are likely to have regular pyridoxine levels.
Predisposition to Cobalamin deficiency	People with your genetic profile are likely to have regular cobalamin levels.
Predisposition to Myo-Inositol deficiency	People with your genetic profile are likely to have a regular Myo-Inositol level.
Predisposition to the deficiency of Paraminobenzoic Acid or PABA	People with your genetic profile are likely to have regular Para-aminobenzoic Acid (PABA) levels.
Predisposition to Pteroyl-L glutamic acid deficiency	People with your genetic profile are likely to have a predisposition for Pteryl-hepta glutamic acid deficiency.
Predisposition to Ergocalciferol and Lumisterol deficiency	People with your genetic profile are likely to have regular ergocalciferol and lumisterol levels.
Predisposition to Ergocalciferol deficiency	People with your genetic profile are likely to have regular ergocalciferol levels.
Predisposition to Cholecalciferol deficiency	People with your genetic profile are likely to have a predisposition for cholecalciferol deficiency.
Predisposition to Dihydroxy Ergocalciferol deficiency	People with your genetic profile are likely to have a regular dihydroxy ergocalciferol levels.
Predisposition to Tocopherol deficiency	People with your genetic profile are likely to have a predisposition for tocopherol deficiency.
Predisposition to Naftochinone deficiency	People with your genetic profile are likely to have regular naftochinone levels.
Predisposition to zinc deficiency	People with your genetic profile are likely to have a predisposition for zinc deficiency.
Predisposition to excessive alcohol consumption	People with your genetic profile are likely to have a predisposition to excessive alcohol consumption.
Itch intensity from mosquito bite	People with your genetic profile are likely to have a higher frequency of mosquito bites with severe itching.
Skin pigmentation	People with your genetic profile are likely to have a predisposition to develop hyperpigmentation.
Fear of minor pain	People with your genetic profile are not likely to have a fear of minor pain.
Skin aging	People with your genetic profile are likely to have regular aging of skin.



HEALTH

CONDITION NAME	MAIN MESSAGE
Gait speed in old age	People with your genetic profile are not likely to have a predisposition to walk slower with age.
Nicotine dependence	People with your genetic profile are not likely to have a predisposition to develop nicotine dependence.
Menarche (age at onset)	People with your genetic profile are likely to have an early onset of menarche.
Photic sneeze reflex	People with your genetic profile are not likely to have a predisposition to experience a photic sneeze reflex.

NUTRIGENOMICS

CONDITION NAME	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 respond positively to the Mediterranean diet	People with your genetic profile are likely to have a regular healthy response to the Mediterranean diet.
Predisposition to an altered metabolism of starch	People with your genetic profile are likely to have a regular starch metabolism.
Caffeine metabolism: plasma levels of paraxanthine	People with your genetic profile are likely to have a slower paraxanthine metabolic rate.
Caffeine metabolism: plasma levels of caffeine	People with your genetic profile are likely to have a regular caffeine metabolic rate.
Caffeine metabolism: plasma levels of theobromine	People with your genetic profile are likely to have an enhanced caffeine theobromine metabolic rate.
Caffeine metabolism: slow metabolizer	People with your genetic profile are likely to have a regular caffeine metabolic rate.
Perception of salty taste	People with your genetic profile are likely to have an enhanced taste for salty foods.
Propensity to choose sweet foods	People with your genetic profile are likely to have an enhanced propensity to prefer sweet foods.
Salty taste preference	People with your genetic profile are likely to have an enhanced propensity to prefer salty foods.

METABOLIC

CONDITION NAME	MAIN MESSAGE
Predisposition to excessive consumption of Carbohydrates	People with your genetic profile are likely to have a predisposition for excessive consumption of carbohydrates.
Predisposition to excessive consumption of fats	People with your genetic profile are likely to have a regular consumption of fat.
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 likely to have the predisposition to eat between meals.
High vs low carbohydrate diets	People with your genetic profile are likely to have a predisposition for an enhanced carbohydrate intake.
Predisposition to the accumulation of fat	People with your genetic profile are likely to have a predisposition for the accumulation of fat.
Predisposition to fatty food addiction	People with your genetic profile are likely to have a predisposition to develop addictions for fatty food.
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 (generic)	People with your genetic profile are likely to not develop an addiction to food.



METABOLIC

CONDITION NAME	MAIN MESSAGE
Predisposition to develop fat tissue over lean tissue	People with your genetic profile are not likely to have a predisposition to 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.
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.
Low resting metabolic rate	People with your genetic profile are likely to have a slow resting metabolic rate.
Predisposition to respond positively to a low-fat diet	People with your genetic profile are likely to have a predisposition to respond positively 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 predisposition to respond positively to a diet enriched in monounsaturated fats.
Positive response to a diet high in polyunsaturated fats	People with your genetic profile are likely to have a regular response to diet enriched in polyunsaturated fats.
Negative response to a diet high in unsaturated fats	People with your genetic profile are likely to have a predisposition to respond negatively to a diet enriched in unsaturated fats.
Negative response to a diet high in trans-fats	People with your genetic profile are likely to have a predisposition to develop adverse effects on the intake of trans fatty acids.
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.
Ratio of visceral vs. subcutaneous adipose tissue	People with your genetic profile are likely to have an unbalance between visceral adipose tissue/subcutaneous adipose tissue.
Prudent dietary pattern	People with your genetic profile are likely to have a healthy dietary pattern.

FOOD ALLERGY

CONDITION NAME	MAIN MESSAGE
Genetic sensitivity to gluten	People with your genetic profile are not likely to have gluten sensitivity.
Predisposition to lactose intolerance	People with your genetic profile are likely to have predisposition for lactose intolerance .
Predisposition to develop an egg allergy	People with your genetic profile are not 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 not likely to have a predisposition to develop a fish allergy.
Predisposition to develop a molluscs/crustaceans allergy	People with your genetic profile are not likely to have a predisposition to develop a shellfish allergy.
Predisposition to develop a soy allergy	People with your genetic profile are not likely to have a predisposition to develop a soy allergy.
Predisposition to develop fruit and vegetable allergies	People with your genetic profile are not 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 not likely to have a predisposition to develop a seed allergy.
Predisposition to develop a salicylate allergy (salicylic acid)	People with your genetic profile are not likely to have a predisposition to develop a salicylates allergy.



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FOOD ALLERGY

CONDITION NAME	MAIN MESSAGE
Predisposition to develop a Tartrazine allergy	People with your genetic profile are not likely to have a predisposition to develop a tartrazine allergy.
Predisposition to develop sensitivity to sulphites	People with your genetic profile are not likely to have a predisposition to develop a sulfites sensitivity.
Predisposition to develop sensitivity to metabisulphites	People with your genetic profile are not likely to have a predisposition to develop a metabisulphites sensitivity.
Predisposition to develop an allergy to bisulfites	People with your genetic profile are not 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 not 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.

SLEEP

CONDITION NAME	MAIN MESSAGE
Ease of getting up in the morning	People with your genetic profile are likely to have an easy time waking up in the morning.
Daytime nap	People with your genetic profile are likely to not take naps during the day.
Sleep duration	People with your genetic profile may have problems with sleep duration.
Snoring	People with your genetic profile are not likely to have a predisposition to snore.
Daytime sleepiness	People with your genetic profile are not likely to have a predisposition to experience excessive sleepiness.
Insomnia complaints	People with your genetic profile are not likely to have a predisposition to suffer from insomnia.
Obstructive sleep apnea	People with your genetic profile are likely to suffer from obstructive sleep apnea.

PERSONALITY

CONDITION NAME	MAIN MESSAGE
Conscientiousness	People with your genetic profile are likely to have a conscientious personality.
Extraversion	People with your genetic profile are likely to have regular levels of extraversion.

DETAILED INFORMATION



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SUSCEPTIBILITY TO CALCIUM DEFICIENCY

RESULTS



Calcium is an essential nutrient that is necessary for many functions in human health. Adequate calcium intake can reduce the risk of fractures, osteoporosis and others pathologic conditions. More than 99% (1.2-1.4 kg) is stored in the bones and teeth. A daily dietary intake of 1,000 mg of calcium would potentially result in 800 mg available for tissue nutrient requirements and 200 mg to maintain serum calcium levels. There are different population groups, such as the female athlete triad or postmenopausal, that are at highest risk for dietary calcium deficiency, also individuals with milk allergy or lactose intolerance are at risk groups for dietary deficiency intake. Calcium is classically associated with dairy products; milk, yogurt, and cheese are rich sources of calcium, providing the major share of calcium from foods in the general diet. The most common forms of supplemental calcium are calcium carbonate and calcium citrate. It is important to keep Calcium levels average because inadequate intake may change bone mineral density, particularly in the elderly.

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

Disorders of calcium metabolism are encountered pretty frequently in routine clinical practice. Hypocalcemia is not as frequently encountered as hypercalcemia, but it can be potentially life threatening if not appropriately recognized and treated promptly. The causes of hypocalcemia can be divided into three broad categories: Miscellaneous (Pseudohypocalcemia, Acidosis/Akcalosis, Acute pancreatitis, Severe sepsis, Hypomagnesemia/hypermagnesemia, Acute hyperphosphatemia, Drugs, Massive Blood transfusion), PTH deficiency (Post thyroidectomy, Autoimmune), High PTH (Chronic Kidney Disease (CKD), Absolute or relative Vitamin D deficiency, Pseudohypoparathyroidism). Work up of hypocalcemia can be thought of in following parts: Confirming the hypocalcemia: First part of evaluation should focus on confirming the hypocalcemia and requires checking a serum albumin level to correct the total calcium, or measuring directly the ionized calcium level (where available). An EKG should also be obtained for all suspected cases of hypocalcemia to look for QTc prolongation which if present is a risk factor for Torsades de pointes. Etiology of hypocalcemia: this part can be driven by the clinical picture obtained during previous steps. Usually entails checking electrolytes such as serum magnesium and phosphorus levels and at least a serum PTH level. If suspicion for vitamin D deficiency is high based on history then Vitamin D2 level should be measured as vitamin D3 can be affected by PTH levels. Other biomarkers may be obtained as indicated by history and physical eg. serum lipase in suspected pancreatitis. Management of Hypocalcemia can be divided into two broad categories: Symptomatic hypocalcemia: intravenous calcium is recommended for rapid repletion if there is any evidence of neuromuscular excitability. If the symptoms are mild such as paresthesias or psychiatric oral calcium can be attempted. Calcium gluconate is the preferred solution and can be given over 10-30 minutes depending on the severity of symptoms. Calcium chloride can be used if central venous access is available. Alkaline solution like bicarbonate and phosphorus containing solution need to be avoided through the same iv to avoid precipitation of calcium salts. Asymptomatic hypocalcemia: if corrected total serum calcium is below 7.5mg/dL, iv calcium should still be the preferred method. However, if corrected serum calcium is >7.5 mg/dL and patient is asymptomatic oral calcium can be used. Vitamin D supplementation is often recommended with calcium to promote absorption and because vitamin D deficiency is commonly encountered in most clinical scenarios leading to hypocalcemia. It is also important to address disease-specific problems and correct co-existing electrolyte disturbances eg. hypomagnesemia. Check a magnesium level when faced with hypocalcemia since its an important and easily correctable cause of hypocalcemia.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CARS	rs7481584	GA



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SCIENTIFIC DETAILS		
VWA8-AS1 - RPS28P8	rs7336933	GG
ARID1B	rs11967485	GG



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PREDISPOSITION TO IRON DEFICIENCY

RESULTS



Anemia is defined as a hemoglobin below two standard deviations of the mean for the age and gender of the patient. Iron is an essential component of the hemoglobin molecule. The most common cause of anemia worldwide is an iron deficiency, which results in microcytic and hypochromic red cells on the peripheral smear. Several causes of iron deficiency vary based on age, gender, and socioeconomic status. The patient often will have nonspecific complaints such as fatigue and dyspnea on exertion. Treatment is a reversal of the underlying condition as well as iron supplementation. Iron supplementation is most often oral, but certain cases may require intravenous iron. The cause of iron-deficiency anemia varies based on age, gender, and socioeconomic status. Iron deficiency may result from insufficient iron intake, decreased absorption, or blood loss. Iron-deficient anemia is most often from blood loss, especially in older patients. It may also be seen with low dietary intake, increased systemic requirements for iron such as in pregnancy, and decreased iron absorption such as in celiac disease. In newborns, breastfeeding is protective against iron deficiency due to the higher bioavailability of iron in breast milk compared to cow's milk; iron deficiency anemia is the most common form of anemia in young children on cow's milk. In developing countries, a parasitic infestation is also a significant cause of iron-deficiency anemia. Dietary sources of iron are green vegetables, red meat, and iron-fortified milk formulas.

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

Dietary iron requirements are estimated using multifactorial modeling. Factors that affect iron needs include the basal physiologic iron loss, periodic loss of iron in females with menstruation, fetal requirements in pregnancy, elevated requirements during growth stages of life, iron storage, etc. A normal individual loses about 1 mg of iron in feces daily. This loss increases in menstruating women by an additional 0.5 mg/day or approximately 14 mg of iron loss in 28 days. So women of childbearing age require higher iron intake than men. Bioavailability of iron differs in various food sources depending on the types of dietary iron as well as the presence or absence of iron absorption enhancers or inhibitors.



SCIENTIFIC DETAILS

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



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PREDISPOSITION TO BIOTIN DEFICIENCY

RESULTS



Biotin, a B vitamin, is an essential nutrient that is naturally present in some foods. It is highly involved in the metabolism of fatty acids, glucose, and amino acids. Chronic exposure to alcohol inhibits the absorption of biotin. At least a third of pregnant women develop marginal biotin deficiency in spite of normal biotin intakes. Signs of biotin deficiency include skin rashes, hair loss, and brittle nails. Therefore, biotin supplements are often promoted for hair, skin, and nail health.

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

Many foods contain some biotin. Foods that contain the most biotin include organ meats, eggs, fish, meat, seeds, nuts, and certain vegetables (such as sweet potatoes). The biotin content of food can vary; for example, plant variety and season can affect the biotin content of cereal grains, and certain processing techniques (e.g., canning) can reduce the biotin content of foods. Dietary avidin, a glycoprotein in raw egg whites, binds tightly to dietary biotin and prevents biotin absorption in the gastrointestinal tract. Cooking denatures avidin, making it unable to interfere with biotin absorption.



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



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PREDISPOSITION TO FOLIC ACID DEFICIENCY

RESULTS



Folic acid is a water-soluble type of vitamin B. Because folate is not stored in the body in large amounts, your blood levels will get low after only a few weeks of eating a diet low in folate. Folate is found in green leafy vegetables and liver. All women of reproductive age should get 400 mcg of folic acid each day to get enough folic acid to help prevent some birth defects. During early development, folic acid helps form the neural tube. Folic acid is very important because it can help prevent some major birth defects of the baby's brain (anencephaly) and spine (spina bifida). Folic acid is water-soluble. Leftover amounts of the vitamin leave the body through the urine. That means your body does not store folic acid. You need to get a regular supply of the vitamin through the foods you eat or through supplements. Folate has many functions in the body: Helps tissues grow and cells work. Works with vitamin B12 and vitamin C to help the body break down, use, and create new proteins, helps form red blood cells (helps prevent anemia), helps produce DNA, the building block of the human body, which carries genetic information. While, Folate deficiency may cause: Diarrhea; Gray hair; Mouth ulcers; Peptic ulcer; Poor growth; Swollen tongue (glossitis). The best way to get the daily requirement of essential vitamins is to eat a wide variety of foods. The Food and Nutrition Board of the Institute of Medicine Recommended that the Daily Reference Intakes (DRIs) for folate should be 65- 80 mcg/day* for infants and 400- 600 mcg/day for adults.

People with your genetic profile are likely to have folic acid deficiency.

All patients with folate deficiency should be offered supplemental folic acid for the correction of the deficiency. Typically, oral folic acid (1 to 5mg daily) suffices to treat folate deficiency. Intravenous, subcutaneous, or intramuscular formulations of folic acid can be used for patients unable to tolerate oral medications. Folinic acid (also called leucovorin), a reduced form of folate, is primarily used to prevent toxicities of methotrexate. The duration of therapy depends on whether the cause of initial deficiency persists. Patients with malabsorption or short gut syndromes may typically require long-term treatment. In patients who have a concomitant vitamin B12 deficiency, it is imperative to replete vitamin B12 as well. Folate treatment alone does not improve neurological symptoms and signs due to B12 deficiency, which, if untreated, may likely progress and cause permanent neurological damage. All patients should be encouraged to a diet rich in fruits and vegetables.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GA



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PREDISPOSITION TO ASCORBIC ACID DEFICIENCY

RESULTS



Vitamin C, also known as L-ascorbic acid, is a water-soluble vitamin that is naturally present in some foods. Vitamin C is required for the biosynthesis of several molecules and certain neurotransmitters, it is also involved in protein metabolism. Vitamin C is an important physiological antioxidant, indeed by limiting the damaging effects of free radicals might help prevent or delay the development of certain cancers, cardiovascular disease, and other diseases in which oxidative stress plays a causal role. Insufficient vitamin C intake causes scurvy, which is characterized by fatigue or lassitude, widespread connective tissue weakness, and capillary fragility. Fruits and vegetables are the best sources of vitamin C. Supplements typically contain vitamin C in the form of ascorbic acid, which has equivalent bioavailability to that of naturally occurring ascorbic acid in foods, such as orange juice and broccoli. Mean intakes of vitamin C are 105.2- 83.6 mg/day for adult males and females respectively, while mean intakes for children and adolescents aged 1-18 years range from 75.6 mg/day to 100 mg/day. Vitamin C inadequacy can occur with intakes that fall below the RDA (Recommended Dietary Allowances). The following groups are more likely than others to be at risk of obtaining insufficient amounts of vitamin C: smokers and passive "smokers", infants fed evaporated or boiled milk, Individuals with limited food variety, people with malabsorption and certain chronic diseases. Evidence suggests that higher consumption of fruits and vegetables is associated with lower risk of most types of cancer, perhaps, in part, due to their high vitamin C content . Vitamin C can limit the formation of carcinogens, modulate immune response and, through its antioxidant function, possibly attenuate oxidative damage that can lead to cancer.

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

Humans are unable to synthesize vitamin C, so it is strictly obtained through dietary intake of fruits and vegetables. Citrus fruits, berries, tomatoes, potatoes, and green leafy vegetables are excellent sources of vitamin C.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SLC23A1	rs33972313	CC



PREDISPOSITION TO FOLATE DEFICIENCY (HOMOCYSTEINE LEVELS)

RESULTS



Homocysteine (Hcy) is a sulfur-containing amino acid produced by the demethylation of the essential amino acid methionine. An elevated plasma Hcy level has been identified as a risk factor for ischemic strokes. The homocysteine (Hcy) level in blood is a sensitive indicator of vitamin B12 and folate deficiencies. Folate is a generic term for this water-soluble B-complex vitamin, which functions in single-carbon transfer reactions and exists in many chemical forms. In fact, the primary indicator used to estimate the Recommended Dietary Allowance (RDA) for folate is erythrocyte folate in conjunction with plasma homocysteine and folate concentrations. The folate coenzymes are involved in numerous reactions, such as, deoxyribonucleic acid (DNA) synthesis or the conversion of homocysteine to methionine serves as a major source of methionine for the synthesis of S-adenosyl-methionine, an important in vivo methylating agent. Inadequate folate intake first leads to a decrease in serum folate concentration, then to a decrease in erythrocyte folate concentration, a rise in homocysteine concentration, and megaloblastic changes in the bone marrow. The evidence is strong that the risk of having a fetus with an NTD decreases with increasing intake of folate during the periconceptional period. So, it was demonstrated that homocysteine is related to pregnancy complications, neural tube defects, cognitive impairment, and mental disorders in the elderly. Different studies report that Hcy is directly associated with cardiovascular risk. Plasma Hcy can be lowered with B vitamin supplementation. Persons with high plasma levels or dietary intake of folate and vitamin B6 have a decreased risk of CHD. However, no significant benefit or harm of B vitamins supplementation were found on the risk of CVD (cardiovascular disease) or MI (myocardial infarction). Dietary fatty acids have been demonstrated recently to be associated with plasma Hcy concentration, even so B vitamins appear to be the more effective Hcy-lowering nutrients. Determination of the mechanisms and magnitude of relationships of folate intake with risk reduction for the occurrence of neural tube defects (NTDs) and vascular disease and the influence of related factors, including genetic polymorphism, could be useful.

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

The folate deficiency may occur from malabsorption problems. Hereditary folate malabsorption (HFM) is characterized by folate deficiency with impaired intestinal folate absorption and impaired folate transport into the central nervous system. The diagnosis of Folate Malabsorption is established in a proband: with anemia, impaired absorption of an oral folate load, and low cerebrospinal fluid (CSF) folate concentration (even after correction of the serum folate concentration); and/or by the identification of biallelic pathogenic variants in SLC46A1 on molecular genetic testing. Parenteral (intramuscular) or high-dose oral 5-formyltetrahydrofolate (5-formylTHF, folic acid, Leucovorin®) or the active isomer of 5-formylTHF (Isovernin® or Fusilev®) can obviate the signs and symptoms of this condition. Dosing is aimed at achieving CSF folate trough concentrations as close as possible to the normal range for the age of the affected individual (infants and children have higher CSF folate levels than adults).



SCIENTIFIC DETAILS

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



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PREDISPOSITION TO A MINERAL SALTS DEFICIENCY (GENERIC)

RESULTS



Mineral salts are responsible for structural functions involving the skeleton and soft tissues and for regulatory functions including neuromuscular transmission, blood clotting, oxygen transport, and enzymatic activity. Calcium, phosphorus, and magnesium are required in relatively large amounts and are designated as macrominerals. Calcium is the most abundant mineral in the human body, making up 1.5 to 2% of the total body weight. Phosphorus, along with calcium, is essential for calcification of bones (85% of body phosphorus is located in the skeleton). The remainder of body phosphorus is needed in soft tissues as a cofactor essential in the metabolism of carbohydrates, lipids, and proteins. Of total body magnesium, 60 to 65% is found in bone and 27% is located in muscles. Magnesium is second only to potassium as the most predominant cation within cells and is essential both for the functions of many enzyme systems and for neuromuscular transmission. In contrast to cellular processes in bone, calcium, phosphorus, and magnesium play a more passive role in any mass changes that occur in bone. They must be present at physiological concentrations in extracellular fluids for bone mineralization (formation) to occur normally. Dietary minerals contribute to this physiological state by helping to replace minerals that have been lost by obligatory processes (in urine, feces, and sweat). Contraction of smooth muscle depends on the interaction among the contractile proteins—actin and myosin—and is the end result of a cascade of reactions initiated by a rise in cytosolic free calcium concentrations. This observation led to the hypothesis that dietary calcium influences blood pressure and possibly risk for hypertension. Low intakes of calcium, which occur commonly, have been associated with age-related osteoporosis. Maximum bone mass is achieved by approximately 25 to 30 years of age. It is maintained until 35 to 45 years of age and then declines. Once maximum bone mass is achieved, it is maintained without much change for 10 to 20 years. Calcium intake need not be greater than 800 mg/day during this period, because bone building has been completed and intestinal absorption of calcium is normal. However, men and women lose bone at a constant rate of 0.2 to 0.5% per year, starting at ages 40 to 45. For approximately 10 years immediately before, during, and after menopause, women lose bone more rapidly than men (2 to 5% per year). This rapid rate of bone loss in menopausal women returns to the slower rate shared by the sexes after this 10-year period. The association between decreased calcium intake and hypertension is suggestive but inconclusive. The epidemiologic and animal evidence relating calcium to colorectal cancer risk is also inconclusive. High-phosphorus diets may decrease calcium bioavailability, but they also reduce urinary calcium excretion and their influence on bone mass and the risk of osteoporosis is unknown.

People with your genetic profile are likely to have regular levels of mineral salts.

Foods rich in minerals include nuts, fish, seeds, beans, mushrooms, whole grains, dark leafy greens and dried fruits as well as avocados, tofu, shellfish, cheese, lamb, low-fat dairy and even beef.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TRPV4	rs3742030	GG



SODIUM BALANCE (NA)

RESULTS



Electrolytes are essential for basic life functioning such as maintaining electrical neutrality in the cells, generation, and conduction of action potentials in the nerves and muscles. Sodium, potassium, and chloride are the significant electrolytes along with magnesium, calcium, phosphate, and bicarbonates. Electrolytes come from our food and fluids. These electrolytes can have an imbalance, leading to either high or low levels. A high or a low level of electrolytes disrupts the normal bodily functions and can lead to even life-threatening complications. Sodium, which is an osmotically active anion, is one of the most important electrolytes in the extracellular fluid. It is responsible for maintaining the extracellular fluid volume, and also for regulation of the membrane potential of cells. Sodium is exchanged along with potassium across cell membranes as part of active transport. Sodium regulation occurs in the kidneys. Sodium transport takes place via sodium-chloride symporters which is by the action of the hormone aldosterone. Among the electrolyte disorders, hyponatremia is the most frequent. It is diagnosed when the serum sodium level less than 135 mmol/L. Hyponatremia has neurological manifestations. Patients may present with headache, confusion, nausea, deliriums. Hypernatremia presents when the serum sodium levels greater than 145 mmol/L. Symptoms of hypernatremia include tachypnea, sleeping difficulty, and feeling restless. Rapid sodium corrections can have serious consequences like cerebral edema and osmotic demyelination syndrome.

People with your genetic profile are likely to have a predisposition to have an enhanced sodium level.

An enhanced blood sodium level leads to Hypertension. The current definition of hypertension (HTN) is systolic blood pressure (SBP) values of 130mmHg or more and/or diastolic blood pressure (DBP) more than 80 mmHg. Hypertension ranks among the most common chronic medical conditions characterized by a persistent elevation in the arterial pressure. It has long been suggested that an increase in salt intake increases the risk of developing hypertension. One of the described factors for the development of essential hypertension is the patient genetic ability to salt response. About 50 to 60% of the patients are salt sensitive and therefore tend to develop hypertension. There are various mechanisms described for the development of hypertension which includes increased salt absorption resulting in volume expansion, an impaired response of the renin-angiotensin-aldosterone system (RAAS), increased activation of the sympathetic nervous system. These changes lead to the development of increased total peripheral resistance and increased afterload which in turn leads to the development of hypertension. The management of hypertension subdivides into pharmacological and nonpharmacological management. Non-pharmacological and lifestyle management are recommended for all individuals with raised BPs regardless of age, gender, comorbidities or cardiovascular risk status. Patient education is paramount to effective management and should always include detailed instructions regarding weight management, salt restriction, smoking management, adequate management of obstructive sleep apnea and exercise. Patients need to be informed and revised at every encounter that these changes are to be continued lifelong for effective disease treatment. Weight reduction is advisable if obesity is present although optimum BMI and optimal weight range is still unknown. Weight reduction alone can result in decreases of up to 5 to 20mmHg in systolic blood pressure. Smoking may not have a direct effect on blood pressure but will help in reducing long term sequelae if the patient quits smoking. Lifestyle changes alone can account for up to 15% reduction in all cardiovascular-related events. Pharmacological therapy consists of angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARBs), diuretics (usually thiazides), calcium channel blockers (CCBs) and beta-blockers (BBs), which are instituted taking into account age, race and comorbidities such as presence of renal dysfunction, LV dysfunction, heart failure and cerebrovascular disease.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
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KIT ID: TPD14630189007528

SCIENTIFIC DETAILS		
AT1R	rs5186	AA
CYP11B2	rs3097	CC
BTBD7, BTBD7	rs2273640	GG
CUX2	rs79105258	CC
ARSG, SLC16A6	rs35397826	AG



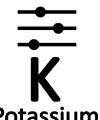
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KIT ID: TPD14630189007528

POTASSIUM BALANCE (K)

RESULTS



Potassium

Potassium is mainly an intracellular ion. The sodium-potassium adenosine triphosphatase pump is primarily responsible for regulating the homeostasis between sodium and potassium which pumps out sodium in exchange for potassium which moves into the cells. In the kidneys, filtration of potassium takes place at the glomerulus. Potassium secretion takes place at the distal convoluted tubule. Aldosterone increases potassium secretion. Potassium channels and potassium-chloride cotransporters at the apical membrane also secrete potassium. Potassium disorders are related to cardiac arrhythmias. Hypokalemia occurs when serum potassium levels under 3.6 mmol/L. Weakness, fatigue and muscle twitching present in hypokalemia. Hyperkalemia occurs when the serum potassium levels above 5.5 mmol/L which can result in arrhythmias. Muscle cramps, muscle weakness, rhabdomyolysis, myoglobinuria are presenting signs and symptoms in hyperkalemia.

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

Food Sources of Potassium are: bananas, oranges, cantaloupe, honeydew, apricots, grapefruit (some dried fruits, such as prunes, raisins, and dates, are also high in potassium); cooked spinach; cooked broccoli; potatoes; sweet potatoes, mushrooms, peas, cucumbers.



SCIENTIFIC DETAILS

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



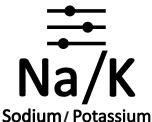
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KIT ID: TPD14630189007528

BALANCE OF SODIUM / POTASSIUM RATIO (NA / K)

RESULTS



The Na⁺ K⁺ pump is an electrogenic transmembrane ATPase situated in the plasma membrane of the cells. The sodium and potassium move against the concentration gradients. The sustained concentration gradient is crucial for physiological processes in many organs and has an ongoing role in stabilizing the resting membrane potential of the cell, regulation of the cell volume and in the cell signal transduction. It plays a crucial role on other physiological processes: sodium and potassium gradients function in various organ systems' physiologic processes. The kidneys have a high level of expression of the Na, K-ATPase. This sodium gradient is necessary for the kidney to filter waste products in the blood, reabsorb amino acids, reabsorb glucose, regulate electrolyte levels in the blood, and to maintain pH. Sperm cells also use the Na, K-ATPase, but they use a different isoform necessary for preserving fertility in males. The brain also requires NA, K ATPase activity. Neurons need the Na, K ATPase pump to reverse postsynaptic sodium flux to re-establish the potassium and sodium gradients which are necessary to fire action potentials. Na⁺ K⁺-ATPase and its endogenous regulators, the endogenous cardiac steroids (ECS), play a role in the etiology of bipolar disorder and is 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.

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

Sodium and potassium imbalances can cause cardiac arrhythmias and shock (a reduced flow of blood and oxygen to tissues throughout the body). Although diarrheal fluids deplete a number of electrolytes (sodium, potassium, chloride, calcium, phosphorus, and magnesium), the main concern in avoiding shock is replacing sodium and water, and all the other minerals when necessary.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADD1	rs4961	GG



MORNING / NIGHT CHRONOTYPE

RESULTS



The term "chronotype" refers to the variation of individual patterns of early or late beginning of daily activity. Variations in chronotype are associated with variations in the timing of numerous physiological and behavioral variables that have consequences for overall health. For example, the rhythms of body temperature and melatonin secretion peak. In terms of behavioral performance, early types are better at sentence recognition early in the day than late types and are more alert than late types early in the day. Because the environmental light-dark cycle is a major synchronizer of circadian rhythms, variations in exposure to bright light might be associated with variations in chronotype. Human sleep occurs with circadian periodicity. To synchronize physiological processes with the day-night cycle (called photoentrainment), the biological clock must detect decreases in light levels as night approaches. The receptors that sense these light changes are in the outer nuclear layer of the retina and project to the suprachiasmatic nucleus (SCN) of the hypothalamus, the site of the circadian control. The SCN's role as a sort of master biological clock and also governs other functions that are synchronized with the sleep-wake cycle, including body temperature, hormone secretion, urine production, and changes in blood pressure.

People with your genetic profile are likely to have a propensity to wake up early in the morning.

The prevalence of the various circadian rhythm sleep disorders is unknown. This disorder is characterized by excessive evening sleepiness and early morning awakening. Patients may get adequate quality and quantity of sleep if no external pressures dictate that patients stay awake in the evening, but often patients are distressed and sleep deprived because societal obligations require patients to stay awake longer than desired in the evening. Patients with advanced sleep-wake phase disorder will wake at the same early time whether they have forced themselves to stay up later, leading to sleep deprivation and daytime sleepiness. It is hypothesized that advanced sleep-wake phase disorder results from an intrinsic circadian cycle that is less than 24 hours. Advanced sleep-wake phase disorder is more prevalent in older adults and males. Diagnosis is made with history and sleep logs. Treatment is primarily achieved with evening bright light therapy. Pharmacotherapy is not indicated for this condition.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TRAF3IP1 - RNU6-234P	rs80271258	CC
ALG10B - AC087897.1	rs12427164	AA
CPNE8	rs7312879	GG
NF1P12 - Y_RNA	rs11182930	AA
AC087897.2	rs35817541	TT



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KIT ID: TPD14630189007528

PREDISPOSITION TO COENZYME Q DEFICIENCY

RESULTS



Coenzyme Q10 (CoQ10) is a substance that is naturally present in the human body, with the highest levels in the heart, liver, kidneys, and pancreas. Coenzyme Q10 is used by cells of the body in a process known variously as aerobic respiration, aerobic metabolism, oxidative metabolism, or cell respiration. Through this process, mitochondria produce energy for cell growth and maintenance. Coenzyme Q10 is also used by the body as an endogenous antioxidant. The term "primary coenzyme Q10 deficiency" refers to the group of conditions characterized by a reduction of coenzyme Q10 (CoQ10) levels in tissues or cultured cells associated with mutation of the nine genes involved in the biosynthesis of coenzyme Q10. As primary deficiency of coenzyme Q10 is a lipid component of the mitochondrial respiratory chain, it is classified as a mitochondrial respiratory chain disorder. This disease is inherited in an autosomal recessive manner and it is usually associated with multisystem involvement, including neurologic manifestations such as fatal neonatal encephalopathy with hypotonia; a late-onset slowly progressive multiple-system atrophy-like phenotype and dystonia, spasticity, seizures, and intellectual disability. Hypertrophic cardiomyopathy (HCM), retinopathy or optic atrophy, and sensorineural hearing loss can also be seen. Supplementation with high-dose oral CoQ10 can prevent progression of the renal disease and onset of neurologic manifestations.

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

Primary coenzyme Q10 (CoQ10) deficiency is usually associated with multisystem involvement, including neurologic manifestations such as fatal neonatal encephalopathy with hypotonia; a late-onset slowly progressive multiple-system atrophy-like phenotype (neurodegeneration with autonomic failure and various combinations of parkinsonism and cerebellar ataxia, and pyramidal dysfunction); and dystonia, spasticity, seizures, and intellectual disability. Steroid-resistant nephrotic syndrome (SRNS), the hallmark renal manifestation, is often the initial manifestation either as isolated renal involvement that progresses to end-stage renal disease (ESRD), or associated with encephalopathy (seizures, stroke-like episodes, severe neurologic impairment) resulting in early death. Hypertrophic cardiomyopathy (HCM), retinopathy or optic atrophy, and sensorineural hearing loss can also be seen. In individuals with primary CoQ10 deficiency early treatment with high-dose oral CoQ10 supplementation (ranging from 5 to 50 mg/kg/day) can limit disease progression and reverse some manifestations; however, established severe neurologic and/or renal damage cannot be reversed. ACE inhibitors may be used in combination with CoQ10 supplementation in persons with proteinuria; renal transplantation is an option for those with ESRD. Treatment of hypertrophic cardiomyopathy, retinopathy, and sensorineural hearing loss is per usual practice. Supplementation with high-dose oral CoQ10 can prevent progression of the renal disease and onset of neurologic manifestations. Periodic neurologic evaluation, urine analysis (for proteinuria) and renal function tests, ophthalmologic evaluation, and audiology. Presymptomatic diagnosis for the purpose of early treatment with CoQ10 supplementation is warranted for relatives at risk. Primary coenzyme Q10 deficiency is inherited in an autosomal recessive manner. At conception, each sib of an affected individual has a 25% chance of being affected, a 50% chance of being an asymptomatic carrier, and a 25% chance of being unaffected and not a carrier. Carrier testing for at-risk relatives, prenatal testing for pregnancies at increased risk, and preimplantation genetic diagnosis are possible if the pathogenic variants in a family are known.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
NEGR1	rs55927656	TT
	rs11591201	GG
	rs146799867	CC



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KIT ID: TPD14630189007528

SCIENTIFIC DETAILS		
OLAH	rs12573070	AA
PRMT8	rs17769758	GG
	rs7141874	GG
TOMM10L	rs184812087	GA
DCC	rs74681568	CC



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KIT ID: TPD14630189007528

PREDISPOSITION TO CHOLINE DEFICIENCY

RESULTS



Choline is a nutrient that is found in many foods. Your brain and nervous system need it to regulate memory, mood, muscle control, and other functions. You also need choline to form the membranes that surround your body's cells. You can make a small amount of choline in your liver, but most of the choline in your body comes from the food you eat. The AI (adequate intake) for adults is 550 mg/day of choline for men and 425 mg/day for women. Choline accelerates the synthesis and release of acetylcholine, an important neurotransmitter involved in memory storage, muscle control, and many other functions. It is also a precursor for the synthesis of phospholipids, a membrane constituent important for the structure and function of membranes and for intracellular signaling. Choline is a precursor for the formation of the methyl donor betaine. Betaine is also required by renal glomerular cells, which use betaine and glycerophosphocholine as organic osmolytes to adapt to osmotic stress. The liver is damaged when humans consume an otherwise adequate diet that is deficient in choline, resulting in elevated alanine aminotransferase levels in blood. Fatty infiltration of liver also occurs in choline deficiency. Some research shows that getting enough choline might help keep the heart and blood vessels healthy, partly by reducing blood pressure. Other research suggests that higher amounts of choline might increase cardiovascular disease risk.

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

Choline is a dietary component that is important for the structural integrity of cell membranes, methyl metabolism, cholinergic neurotransmission, transmembrane signaling, and lipid and cholesterol transport and metabolism. Human cells grown in culture have an absolute requirement for choline. The Dietary Guidelines for Americans describes a healthy eating pattern as one that: Includes a variety of vegetables, fruits, whole grains, fat-free or low-fat milk and milk products, and oils. Many vegetables, fruits, whole grains, and dairy products contain choline. Includes a variety of protein foods, including seafood, lean meats and poultry, eggs, legumes (beans and peas), nuts, seeds, and soy products. Fish, beef, poultry, eggs, and some beans and nuts are rich sources of choline. Limits saturated and trans fats, added sugars, and sodium. Stays within your daily calorie needs.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
PEMT	rs12325817	CC
PEMT	rs4646343	GG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO COPPER DEFICIENCY

RESULTS



Copper functions as a component of a number of metalloenzymes acting as oxidases to achieve the reduction of molecular oxygen. Nearly two-thirds of the body copper content is located in skeleton and muscle, but studies with stable isotopes have shown that the liver is a key site in maintaining plasma copper concentrations. Frank copper deficiency in humans is rare, but has been found in a number of special conditions. It has been observed in premature infants fed milk formulas, in infants recovering from malnutrition associated with chronic diarrhea and fed cow's milk, and in patients with prolonged total parenteral nutrition. Supplementation with copper resulted in rapid increases in serum copper and ceruloplasmin concentrations. Symptoms accompanying the copper deficiency included normocytic, hypochromic anemia, leukopenia, and neutropenia. Osteoporosis was observed in copper-deficient infants and growing children. Copper balance, which can be achieved over a broad range of dietary copper intakes, reflects prior dietary intake; thus long adaptation is required for results to be meaningful. Copper is widely distributed in foods. Organ meats, seafood, nuts, and seeds are major contributors of dietary copper. Wheat bran cereals and whole grain products are also sources of copper. The median intake of copper for women is approximately 1.0 to 1.1 mg/day, whereas the median intake for men ranges from 1.2 to 1.6 mg/day.

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

Copper is a mineral that your body requires in small quantities to maintain good health. It uses copper to form red blood cells, bone, connective tissue and some important enzymes. Copper is also involved in the processing of cholesterol, the proper functioning of your immune system and the growth and development of babies in the womb. Though it's only needed in tiny amounts, it's an essential mineral — meaning that you must obtain it from your diet because your body cannot produce it on its own. It's recommended that adults get 900 mcg of copper per day. Foods high in copper are: liver, oysters, Spirulina, Shiitake Mushrooms, Nuts and Seeds, Lobster, Leafy Greens, Dark Chocolate. To avoid a deficiency, be sure to include a variety of these sources in your diet.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SMIM1	rs1175550	AA



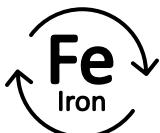
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO IRON OVERLOAD

RESULTS



Iron overload is defined as excess stores of iron in the body. The most notable organs with iron deposition are the liver, heart, and endocrine glands. Primary iron overload is often inherited. Hereditary hemochromatosis is the leading case of iron overload disease. Resulting symptoms and disease are related to specific organ damage. Damage to the liver can result in chronic liver disease, cirrhosis and lead to hepatocellular carcinoma. Damage to the heart muscle can lead to heart failure and irregular heart rhythms. Damage to the pancreas can lead to elevated blood glucose levels and "bronze" diabetes. Hypothyroidism and hypogonadism can result in fatigue, hair loss, infertility, and decreased libido. Joint involvement leads to arthritis. Neurological involvement can accelerate neurodegenerative diseases such as Alzheimer disease. The treatment for iron overload is reduction therapy. This is most commonly achieved through therapeutic phlebotomy. Complications of iron overload include liver damage, liver cirrhosis, pancreatic islet cell damage, diabetes, hypothyroidism, and hypogonadism. Consulting a gastroenterologist or hepatologist may be necessary if the patient develops cirrhosis and its sequelae.

People with your genetic profile are not likely to have a predisposition to experience an iron overload.

When you eat food with iron, iron is absorbed into your body mainly through the upper part of your small intestine. Very good sources of heme iron, with 3.5 milligrams or more per serving, include: Lean beef, Oysters, Chicken, Turkey, Beans and lentils, Tofu, Baked potatoes, Cashews, Dark green leafy vegetables such as spinach, Fortified breakfast cereals, Whole-grain and enriched breads.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
HFE	rs1800562	GG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO LUTEIN AND ZEAXANTHIN DEFICIENCY

RESULTS



Lutein is a xanthophyll and one of 600 known naturally occurring carotenoids. Lutein is synthesized only by plants, and like other xanthophylls is found in high quantities in green leafy vegetables such as spinach, kale and yellow carrots. In green plants, xanthophylls act to modulate light energy and serve as non-photochemical quenching agents to deal with triplet chlorophyll (an excited form of chlorophyll), which is overproduced at very high light levels, during photosynthesis. See xanthophyll cycle for this topic. Animals obtain lutein by ingesting plants. In the human retina, lutein is absorbed from blood specifically into the macula lutea, although its precise role in the body is unknown. Lutein is also found in egg yolks and animal fats. Lutein is isomeric with zeaxanthin, differing only in the placement of one double bond. Lutein and zeaxanthin can be interconverted in the body through an intermediate called meso-zeaxanthin. The principal natural stereoisomer of lutein is (3R,3'R,6'R)-beta,epsilon-carotene-3,3'-diol. Lutein is a lipophilic molecule and is generally insoluble in water. The presence of the long chromophore of conjugated double bonds (polyene chain) provides the distinctive light-absorbing properties. The polyene chain is susceptible to oxidative degradation by light or heat and is chemically unstable in acids. This xanthophyll, like its sister compound zeaxanthin, has primarily been used in food and supplement manufacturing as a colorant due to its yellow-red color. Lutein absorbs blue light and therefore appears yellow at low concentrations and orange-red at high concentrations. There is preliminary epidemiological evidence that increasing lutein and zeaxanthin intake lowers the risk of cataract development. Consumption of more than 2.4 mg of lutein/zeaxanthin daily from foods and supplements was significantly correlated with reduced incidence of nuclear lens opacities, as revealed from data collected during a 13- to 15-year period in one study. Lutein is a natural part of a human diet found in orange-yellow fruits and flowers, and in leafy vegetables. According to the NHANES 2013-2014 survey, adults in the United States consume on average 1.7 mg/day of lutein and zeaxanthin combined.

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

A deficiency in Lutein and Zeaxanthin can cause symptoms. These can include night blindness, fatigue, skin issues, and a weakened immune system. Severe problems can lead to blindness. This is a leading cause of blindness in some parts of the world. Clinical trials have repeatedly shown that supplementation with the macular carotenoids lutein, zeaxanthin, and meso-zeaxanthin results in augmentation of macular pigment carotenoids, and consequential benefits in visual performance such as improved contrast sensitivity and reduced glare disability. The importance of these findings extends to those involved in vision-dependent-specialized activities, such as pilots, vehicle drivers, military personnel, and athletes. Lutein, zeaxanthin, and meso-zeaxanthin are widely recommended as dietary supplements for the prevention of visual loss from age-related macular degeneration (AMD) and other ocular diseases, but the basic and clinical science supporting such recommendations is underappreciated by clinicians and vision scientists. Much has changed in ophthalmologists' management and treatment of AMD in the past few decades. A once largely ignored and poorly understood disease of aging now consumes billions of healthcare dollars in the United States and other developed countries, and with longer lifespans, its prevalence is rising in the developing world as well. Diet has been of particular interest to AMD epidemiologists because multiple laboratory studies have implicated oxidative stress as a major potential mechanism underlying damage generated in cell culture and animal models of AMD, and diet is the usual source of antioxidants and other protective nutrients for most individuals. The association of carotenoids and eye health extends back for centuries based largely on the recognition that consumption of certain foods such as carrots can help to treat and prevent symptoms of night blindness.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

SCIENTIFIC DETAILS		
PDK1L2	rs9708919	CC
BCM01	rs6564851	TG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO MAGNESIUM DEFICIENCY

RESULTS



Magnesium is an important electrolyte. It is a key part of many reactions that occur in the human body, affecting cellular function, nerve conduction, and other needs. Normal serum magnesium levels are between 1.46 and 2.68 mg/dL. Hypomagnesemia is an electrolyte disturbance caused when there is a low level of serum magnesium (less than 1.46 mg/dL) in the blood. Hypomagnesemia can be attributed to chronic disease, alcohol use disorder, gastrointestinal losses, renal losses, and other conditions. Signs and symptoms of hypomagnesemia include anything from mild tremors and generalized weakness to cardiac ischemia and death. Hypomagnesemia can be secondary to decreased intake, as seen in: Starvation, Alcohol use disorder, Critically ill patients who are receiving total parenteral nutrition. It also can be secondary to the following medications: Loop and thiazide diuretics, Proton pump inhibitors, Aminoglycoside antibiotics, Amphotericin B, Digitalis Chemotherapeutic drugs, such as cisplatin, cyclosporine. Lastly, hypomagnesemia can be induced by gastrointestinal and/or renal losses, including but not limited to the following conditions: Acute diarrhea, Chronic diarrhea (Crohn disease, ulcerative colitis), Hungry bone syndrome (an increased, magnesium uptake by renewing bone following parathyroidectomy or thyroidectomy, causing a decrease in serum magnesium), Acute pancreatitis, Gastric bypass surgery, Inherited tubular disorders (Gitelman syndrome, Bartter syndrome), Familial hypomagnesemia with hypercalciuria and nephrocalcinosis, Other rare genetic renal diseases.

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

The treatment of patients with hypomagnesemia is based on a patient's kidney function, the severity of their symptoms, and their hemodynamic stability. If a patient is hemodynamically unstable in an acute hospital setting, 1 to 2 grams of magnesium sulfate can be given in about 15 minutes. For symptomatic, severe hypomagnesemia in a stable patient, 1 to 2 grams of magnesium sulfate can be given over one hour. Non-emergent repletion of the adult patient is generally 4 to 8 grams of magnesium sulfate given slowly over 12 to 24 hours. In pediatric patients, the dose is 25 to 50 mg/kg (with a maximum of 2 grams). For an asymptomatic patient who is not hospitalized and can tolerate medications by mouth, sustained-release oral replacement should be tried first. After repletion, serum electrolyte levels must be rechecked (whether in an inpatient or outpatient setting) to ensure that the treatment was effective. Although serum magnesium levels rise quickly with treatment, intracellular magnesium takes longer to replete. Thus, patients with normal renal function should try to continue magnesium repletion for two days after the level normalizes. Use caution in repleting magnesium in patients with abnormal kidney function (defined as creatinine clearance less than 30 mL/min/1.73 m²). These patients are at risk of hypermagnesemia. Studies recommend reducing the magnesium dose by 50% and closely monitoring magnesium levels in these patients. The underlying cause of persistent hypomagnesemia should be addressed and treated. For example, if a patient is consistently having low levels of the electrolyte due to renal losses, they may benefit from amiloride, a potassium- and magnesium-sparing diuretic. Always check for other electrolyte abnormalities when suspecting or treating hypomagnesemia. Low levels of magnesium can, in turn, cause low levels of potassium and/or calcium as well. Furthermore, many other electrolyte and hormonal abnormalities can present with similar symptoms.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC089987.1 - OR5BS1P	rs193153567	CC
AC009522.1	rs7965584	AG
MPPED2-AS1 - AL122014.1	rs3925584	TC
AC009988.1 - RPS15AP5	rs1219515	GG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO PHOSPHORUS DEFICIENCY

RESULTS



Phosphate is an abundant mineral found in the body. The body store of phosphate is 500 to 800 g, with 85% of the total body phosphate present in crystals of hydroxyapatite in the bone — about 10% found in muscles and bones in association with proteins, carbohydrates, and lipids. The rest gets distributed in various compounds in the extracellular fluid (ECF) and intracellular fluid (ICF). Phosphate is predominantly an intracellular anion. The normal plasma inorganic phosphate (Pi) concentration in an adult is 2.5 to 4.5 mg/dL, and men have a slightly higher concentration than women. In children, the normal range is 4 to 7 mg/dL. A plasma phosphate level higher than 4.5 mg/dL is hyperphosphatemia. Phosphate plays an essential role in many biological functions such as the formation of ATP, cyclic AMP, phosphorylation of proteins, etc. Phosphate is also present in nucleic acids and acts as an important intracellular buffer. Normal adult dietary phosphate intake is around 1000 mg/day. 90% of this is absorbed primarily in the jejunum. In the small intestine, phosphate is absorbed both actively and by passive paracellular diffusion. Kidneys excrete ninety percent of the daily phosphate load while the gastrointestinal tract excretes the remainder. As phosphorus is not significantly bound to albumin, most of it gets filtered at the glomerulus. Phosphate homeostasis is under direct hormonal influence of calcitriol, PTH, and phosphatonins, including fibroblast growth factor 23 (FGF-23). Receptors for Vitamin D, FGF-23, PTH, and calcium-sensing receptor (CaSR) also play an important role in phosphate homeostasis. Serum phosphate level is maintained through a complex interaction between intestinal phosphate absorption, renal phosphate handling, and the transcellular movement of phosphate that occurs between intracellular fluid and bone storage pool. A transient shift of phosphate into the cells is also stimulated by insulin and respiratory alkalosis. Hypophosphatemia is defined as an adult serum phosphate level of less than 2.5 mg/dL. The normal level of serum phosphate in children is considerably higher and 7 mg/dL for infants. Hypophosphatemia is a relatively common laboratory abnormality and is often an incidental finding. Hypophosphatemia is most commonly induced by one of three causes: (1) Inadequate phosphate intake, (2) increased phosphate excretion, and (3) shift from extracellular phosphate into the intracellular space. Hypophosphatemia is typically asymptomatic and is present in up to 5% of patients. It is much more prevalent in alcoholism, diabetic ketoacidosis, or sepsis, with a frequency of up to 80%. The morbidity of hypophosphatemia is highly dependent on its etiology and severity.

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

Many different types of foods contain phosphorus, including dairy products, meats and poultry, fish, eggs, nuts, legumes, vegetables, and grains. In the United States, dairy products contribute about 20% of total phosphorus intakes, and bakery products (e.g., breads, tortillas, and sweet bakery products) contribute 10%. Vegetables and chicken contribute 5% each. The absorption rate for the phosphorus naturally contained in food is 40%-70%; phosphorus from animal sources has a higher absorption rate than that from plants. Calcium from foods and supplements can bind to some of the phosphorus in foods and prevent its absorption. According to one analysis, a very high calcium intake of 2,500 mg/day binds 0.61-1.05 g phosphorus.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO SELENIUM DEFICIENCY

RESULTS



Selenium (Se) is a trace mineral that is essential to human health. Selenium, in the form of selenoproteins, carries out various functions in normal health and metabolism. In particular, glutathione peroxidase (G-Px), a selenoprotein, performs antioxidant activities that protect against reactive oxygen and nitrogen species. Iodothyronine deiodinases that convert inactive thyroxine (T4) to active thyroid hormone, triiodothyronine (T3), are selenium-dependent. Selenium plays a role in the immune system functioning and the progression of HIV to AIDS. Selenium deficiency has been implicated in cardiovascular disease, infertility, myodegenerative diseases, and cognitive decline. The role of selenium in cancer treatment is currently being studied. In the 200 years since its discovery in 1817, the role of selenium in human health has evolved. Previously maligned as a carcinogen, it is now being understood as a vital nutrient, albeit one with a low window from being therapeutic to toxic. Selenium is present in the soil and dictates the concentrations in plant foods. Brazil nuts, seeds, especially young barley seedlings, green vegetables, shiitake mushrooms, and button mushrooms are excellent organic sources of selenium in regions with adequate selenium content in the soil. Selenium yeast is an excellent source as well and is used to make bread. Individuals that consume selenium containing plant-based foods, especially fish, seafood, beef, and poultry are good sources of selenium from areas with adequate supply. Selenium is present in plant food in organic form as selenomethionine, which has 90% bioavailability. Inorganic forms such as selenite and selenite are used in supplemental forms and are also highly bioavailable. Selenium deficiency occurs when there is inadequate dietary intake of selenium, typically due to a scarcity of selenium sources in a given region. Interestingly, many selenium-deficiency diseases are linked with concurrent vitamin E deficiency. The American Recommended Dietary Allowance (RDA) daily minimum requirement of selenium for optimal biological functioning is 70 and 55 micrograms (mcg) per day for men and women, respectively, per April 2000 recommendations. However, this level is considered low based on other studies, and some literature sets the minimum requirement at 90 mcg daily per adult. Per the World Health Organization, the tolerable upper intake level for selenium in adults 19 years or older is 400 micrograms or 5.1 micromoles per day. Levels above this are considered toxic. Selenium deficiency affects anywhere from 500 million to 1 billion people worldwide, due to inadequate intake. In the United States, selenium content in the soil and consequently plant sources is lowest in the Northwest, Northeast, Southeast, and areas of the Midwest abutting the Great Lakes. The Great Plains and the Southwest have adequate selenium content typically.

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

Selenium deficiency is often a population-level problem rather than an individual one. It affects a community as a whole. Biofortification has been the approach used to tackle this problem by fertilizing the soil at the agricultural end of things. Alternately enrichment of food sources through fodder with selenium compounds, for instance, eggs (or rather, egg yolks) with more selenium, is also another approach that has been used to raise nutritional selenium content. In many countries, eggs, meat, and milk fortified with selenium have been introduced successfully. Using microorganisms for the production of functional foods such as selenium yeast is yet another approach that is being used. Organic selenium is less likely to reach toxic levels as quickly as supplementing with selenite and selenate (the inorganic salt forms of selenium supplements) can. However, using inorganic salts is a quick way to supplement selenium in the situation of gross and immediate deficiency. The goal to target is supplementation, achieving about 90 mcg/day for adults. Per the World Health Organization, the tolerable upper intake level for selenium in adults 19 years or older is 400 micrograms or 5.1 micromoles per day. Levels above this are considered toxic. Ultimately, a balanced diet is the best way to stave off selenium deficiency. If identified, this is a condition that can be treated with supplementation.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

SCIENTIFIC DETAILS		
SLC39A11	rs891684	GG
DMGDH	rs248381	GA
AC076968.2	rs1596370	GG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO CAROTENE DEFICIENCY

RESULTS



The term carotene (also carotin, from the Latin *carota*, "carrot") is used for many related unsaturated hydrocarbon substances having the formula C₄₀H_x, which are synthesized by plants but in general cannot be made by animals (with the exception of some aphids and spider mites which acquired the synthesizing genes from fungi). Carotenes are photosynthetic pigments important for photosynthesis. Carotenes contain no oxygen atoms. They absorb ultraviolet, violet, and blue light and scatter orange or red light, and (in low concentrations) yellow light. Carotenes are responsible for the orange colour of the carrot, for which this class of chemicals is named, and for the colours of many other fruits, vegetables and fungi (for example, sweet potatoes, chard and orange cantaloupe melon). Carotenes are also responsible for the orange (but not all of the yellow) colours in dry foliage. They also (in lower concentrations) impart the yellow coloration to milk-fat and butter. Omnivorous animal species which are relatively poor converters of coloured dietary carotenoids to colourless retinoids have yellowed-coloured body fat, as a result of the carotenoid retention from the vegetable portion of their diet. The typical yellow-coloured fat of humans and chickens is a result of fat storage of carotenes from their diets. β -Carotene is composed of two retinyl groups, and is broken down in the mucosa of the human small intestine by β -carotene 15,15'-monooxygenase to retinal, a form of vitamin A. β -Carotene can be stored in the liver and body fat and converted to retinal as needed, thus making it a form of vitamin A for humans and some other mammals. The carotenes α -carotene and γ -carotene, due to their single retinyl group (β -ionone ring), also have some vitamin A activity (though less than β -carotene), as does the xanthophyll carotenoid β -cryptoxanthin. All other carotenoids, including lycopene, have no beta-ring and thus no vitamin A activity (although they may have antioxidant activity and thus biological activity in other ways).

People with your genetic profile are likely to have regular carotene levels.

Beta carotene is predominantly found in fruits and veggies with a red, orange, or yellow color. However, don't shy away from dark leafy greens or other green veggies, as they contain a good amount of this antioxidant as well. Some studies have shown that higher amounts of beta carotene are found in cooked forms of fruits and veggies compared to raw. Because beta carotene converts to the fat-soluble vitamin A, it's important to consume this nutrient with a fat for best absorption. Foods highest in beta carotene include: carrots, sweet potatoes, dark, leafy greens, such as kale and spinach, romaine lettuce, squash, cantaloupe, red and yellow, 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 their absorption.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
PKD1L2 - BC01	rs6564851	TG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO RETINOL DEFICIENCY

RESULTS



Retinol, also known as Vitamin A1, is a vitamin found in food and used as a dietary supplement. As a supplement it is used to treat and prevent vitamin A deficiency, especially that which results in xerophthalmia. In regions where deficiency is common, a single large dose is recommended to those at high risk a couple of times a year. It is also used to reduce the risk of complications in those who have measles. It is used by mouth or injection into a muscle. Retinol at normal doses is well tolerated. High doses may result in an enlarged liver, dry skin, or hypervitaminosis A. High doses during pregnancy may result in harm to the baby. Retinol is in the vitamin A family. It is converted in the body to retinal and retinoic acid through which it acts. Dietary sources include fish, dairy products, and meat. This vitamin plays an essential role in vision, particularly night vision, normal bone and tooth development, reproduction, and the health of skin and mucous membranes (the mucus-secreting layer that lines body regions such as the respiratory tract). Vitamin A also acts in the body as an antioxidant, a protective chemical that may reduce the risk of certain cancers. There are two sources of dietary vitamin A. Active forms, which are immediately available to the body are obtained from animal products. These are known as retinoids and include retinaldehyde and retinol. Precursors, also known as provitamins, which must be converted to active forms by the body, are obtained from fruits and vegetables containing yellow, orange and dark green pigments, known as carotenoids, the most well-known being β-carotene. For this reason, amounts of vitamin A are measured in Retinol Equivalents (RE). One RE is equivalent to 0.001 mg of retinol, or 0.006 mg of β-carotene, or 3.3 International Units of vitamin A. In the intestine, vitamin A is protected from being chemically changed by vitamin E. Vitamin A is fat-soluble and can be stored in the body. Most of the vitamin A consumed is stored in the liver. When required by a particular part of the body, the liver releases some vitamin A, which is carried by the blood and delivered to the target cells and tissues. Vitamin A deficiency is common in developing countries but rarely seen in developed countries. Approximately 250,000 to 500,000 malnourished children in the developing world go blind each year from a deficiency of vitamin A. Vitamin A deficiency in expecting mothers increases the mortality rate of children shortly after childbirth. Night blindness is one of the first signs of vitamin A deficiency. Vitamin A deficiency contributes to blindness by making the cornea very dry and damaging the retina and cornea.

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

Retinol is found in meat, dairy and eggs, as well as red, orange, yellow and green plant foods. To make sure you get enough vitamin A, eat a variety of these foods. Too little vitamin A can lead to inflamed skin, night blindness, infertility, delayed growth and respiratory infections.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
BCO1	rs119478057	CC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO THIAMINE DEFICIENCY

RESULTS



Thiamine was the first vitamin identified (vitamin B1) many years ago. It functions as a catalyst in the generation of energy through decarboxylation of branched-chain amino acids and alpha-ketoacids and acts as a coenzyme for transketolase reactions in the form of thiamine pyrophosphate. Thiamine also plays an unidentified role in the propagation of nerve impulses and takes part in myelin sheath maintenance. This water-soluble vitamin is present in meat, beef, pork, legumes, whole grains, and nuts; however, milled rice and grains have little amounts of thiamine as the processing involved in creating these food products removes thiamine. Additionally, certain food products such as tea, coffee, raw fish, and shellfish, contain thiaminases - enzymes that destroy thiamine. Deficiency of thiamine can affect the cardiovascular, nervous, and immune system, as is commonly seen in wet beriberi, dry beriberi, or as Wernicke-Korsakoff syndrome. Worldwide it is most widely reported in populations where polished rice and milled cereals are the primary food source, and also in patients with chronic alcohol abuse. Dry beriberi presents as symmetrical peripheral neuropathy while wet beriberi presents with high-output heart failure. Wernicke-Korsakoff syndrome (WKS) can manifest with CNS symptoms such as gait changes, altered mental status, and ocular abnormalities. Deficiency of thiamine can be related to: Poor intake, Poor absorption, Increased loss, Increased thiamine utilization (pregnancy). Drugs that can lead to thiamine deficiency are Diuretics.

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

There are many natural ways to add thiamine-rich foods to an everyday diet. Food sources of thiamine include beef, liver, dried milk, nuts, oats, oranges, pork, eggs, seeds, legumes, peas and yeast. Foods are also fortified with thiamine. Some foods that are often fortified with B1 are rice, pasta, breads, cereals and flour.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
NRG1	rs7817052	TT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO RIBOFLAVIN DEFICIENCY

RESULTS



Riboflavin, vitamin B2, is a water-soluble and heat-stable vitamin that the body uses to metabolize fats, protein, and carbohydrates into glucose for energy. In addition to boosting energy, riboflavin functions as an antioxidant for the proper function of the immune system, healthy skin, and hair. These effects occur with the help of two coenzymes, flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD). Without an adequate amount of riboflavin, macronutrients like carbohydrates, fats, and proteins cannot be digested and maintain the body. With a healthy digestive system, the body can absorb most of the nutrients from the diet, so it is important to get most of the riboflavin from dietary sources. Riboflavin has a yellow-green fluorescent pigment, which causes urine to turn yellow, indicating the body is absorbing riboflavin. Riboflavin also helps convert tryptophan to niacin, which activates vitamin B6. Some preventable diseases manageable with adequate riboflavin are anemia, cataracts, migraines, and thyroid dysfunction. Riboflavin is necessary for normal development, lactation, physical performance, and reproduction. Riboflavin deficiency can result from inadequate dietary intake or by endocrine abnormalities. Riboflavin deficiency also correlates with other vitamin B complexes. Riboflavin naturally occurs in some food such as eggs, dairy products, meats, green vegetables, and grains. The main antioxidant riboflavin works as is glutathione. Glutathione works to destroy free radicals and detox the liver, as free radicals can cause to develop several diseases. Riboflavin deficiency can also result from chronic diarrhea, liver disorder, alcoholism, and hemodialysis.

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

Riboflavin deficiency is extremely rare in the United States. Riboflavin deficiency is most common in developing countries in Asia and Africa. Older adults, alcoholics, and women who take birth control pills are most likely to suffer from riboflavin deficiency since the body cannot absorb much riboflavin when on birth control pills. Riboflavin deficiency can be related to many developmental abnormalities such as cleft lip and palate, growth retardation, and cardiac disease. Pregnant and lactating women, people with Brown-Vialetto-Van Laere syndrome (BVVL), and vegan people are also at risk of riboflavin deficiency. Riboflavin supplements come in 25 mg, 50 mg, and 100 mg tablets. According to the National Institutes of Health, the recommended daily nutrient intake of riboflavin is 1.3 mg for men, 1.1 mg for women, 1.3 mg for male adolescents (age 14 to 18), and 1.0 mg for female adolescents (age 14 to 18). Recommendations are that pregnant women take 1.4 mg, and breastfeeding women take 1.6 mg. For infants age of 0 to 6 months old is 0.3 mg, 7 to 12 months is 0.4 mg, 1 to 3 years old is 0.5 mg, 4 to 8 years old is 0.6 mg, and 9 to 13 years is 0.9 mg. It is important to take riboflavin supplements with meals because absorption levels increase with food. If oral supplementation is not possible, then injections are an option. Taking certain medications such as anticholinergic, anticonvulsants, phenothiazines, and phenytoin can reduce the level of riboflavin by not being able to be absorbed effectively into the body. Riboflavin can also interfere with some medications such as tetracycline, which is an antibiotic and doxorubicin, a chemotherapy drug.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GA



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KIT ID: TPD14630189007528

PREDISPOSITION TO NIACIN OR NICOTINAMIDE DEFICIENCY

RESULTS



Niacin, known as vitamin B3, is a water-soluble, vitamin of the B complex group of vitamins. Food such as bran, yeast, eggs, peanuts, poultry, red meat, fish, whole-grain cereals, legumes, and seeds are rich sources of the vitamin. As a drug, it has two main indications: 1.Deficiency of the vitamin, also known as pellagra, 2.Dyslipidemia. Pellagra is the deficiency of vitamin B3. Niacin is indicated in the treatment of pellagra until the symptoms resolve, most commonly for the relief of skin symptoms. Therapeutic doses of niacin can reduce the level of total cholesterol up to 25%, low-density lipoprotein (LDL) up to 10% to 15%, and triglycerides by up to 20% to 50%. Niacin has the highest efficacy to raise high-density cholesterol (HDL), with an increase of 15% to 35%. With more evaluation, niacin may be in a grouping with hypolipidemic drugs used for patients with: - Concomitant hypercholesterolemia and statin intolerance, - Metabolic syndrome, - Patients unresponsive to hypolipidemic therapy of targeted LDL cholesterol values.

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

Severe niacin deficiency leads to pellagra, a disease characterized by a pigmented rash or brown discoloration on skin exposed to sunlight; the skin also develops a roughened, sunburned-like appearance. In addition, pellagra can cause a bright red tongue and changes in the digestive tract that lead to vomiting, constipation, or diarrhea. The neurological symptoms of pellagra can include depression; apathy; headache; fatigue; loss of memory that can progress to aggressive, paranoid, and suicidal behaviors; and auditory and visual hallucinations. As pellagra progresses, anorexia develops, and the affected individual eventually dies. Niacin inadequacy usually arises from insufficient intakes of foods containing niacin and tryptophan. It can also be caused by factors that reduce the conversion of tryptophan to niacin, such as low intakes of other nutrients. Individuals at risk are: people who are undernourished because they live in poverty or have anorexia, alcohol use disorder, AIDS, inflammatory bowel disease, or liver cirrhosis often have inadequate intakes of niacin and other nutrients; people who do not consume enough riboflavin (vitamin B2), pyridoxine (vitamin B6), or iron convert less tryptophan to niacin because enzymes in the metabolic pathway for this conversion depend on these nutrients to function; people with Hartnup disease which is a rare genetic disorder involving the renal, intestinal, and cellular transport processes for several amino acids, including tryptophan. The disease interferes with the absorption of tryptophan in the small intestine and increases its loss in the urine via the kidneys. As a result, the body has less available tryptophan to convert to niacin. The adult dose is nicotinamide 100 mg orally, every 6 hours for several days until relief of acute symptoms, followed by 50 mg every 8 to 12 hours until all skin lesions heal. In severe cases (marked neurological or gastrointestinal tract symptoms), 1 g three to four times a day can be given, initially by the parenteral route. For children, one can use 10 to 50 mg orally every 6 hours until symptoms of pellagra resolve. For mild endemic pellagra, smaller doses such as 10 mg per day are acceptable. Therapy should include other B vitamins, magnesium, and zinc as well as a calorie-rich diet. Topical emollients may reduce discomfort due to skin lesions. Sustained-release (SR) formulations have been developed which are available over-the-counter. Sustained-release niacin can be administered once daily and is less likely to cause flushing. However, it does not have approval for use in hyperlipidemia, and some studies showed a high likelihood of hepatotoxicity.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
RBFOX3	rs12946859	TT

KIT ID: TPD14630189007528

PREDISPOSITION TO THE DEFICIENCY OF PANTOTHENIC ACID

RESULTS



RESULTS



Pantothenic acid, also called vitamin B5 (a B vitamin), is a water-soluble vitamin. Pantothenic acid is an essential nutrient. Animals require pantothenic acid in order to synthesize coenzyme-A (CoA), as well as to synthesize and metabolize proteins, carbohydrates, and fats. The anion is called pantothenate. Coenzyme A may act as an acyl group carrier to form acetyl-CoA and other related compounds; this is a way to transport carbon atoms within the cell. CoA is important in energy metabolism for pyruvate to enter the tricarboxylic acid cycle (TCA cycle) as acetyl-CoA, and for α -ketoglutarate to be transformed to succinyl-CoA in the cycle. CoA is also important in the biosynthesis of many important compounds such as fatty acids, cholesterol, and acetylcholine. CoA is incidentally also required in the formation of ACP, which is also required for fatty acid synthesis in addition to CoA. Pantothenic acid in the form of CoA is also required for acylation and acetylation, which, for example, are involved in signal transduction, and various enzyme functions. Due to its numerous biological roles, dietary deficiency of pantothenic acid, although rare and usually occurring with other nutrient deficiencies, has diverse negative effects. Content of pantothenic acid varies among manufactured and natural foods, especially fortified ready-to-eat cereals, infant formulas, energy bars and dried foods. Major food sources of pantothenic acid are dried shiitake mushrooms, liver, kidney, egg yolks and sunflower seeds. Whole grains are another source of the vitamin, but milling removes much of the pantothenic acid, as it is found in the outer layers of whole grains. In animal feeds, the most important sources are alfalfa, cereal, fish meal, peanut meal, molasses, mushrooms, rice, wheat bran, and yeasts. The derivative of pantothenic acid, pantothenol (panthenol), is a more stable form of the vitamin and is often used as a source of the vitamin in multivitamin supplements. Another common supplemental form of the vitamin is calcium pantothenate. Calcium pantothenate is often used in dietary supplements because, as a salt, it is more stable than pantothenic acid. Symptoms of deficiency are similar to other vitamin B deficiencies. There is impaired energy production, due to low CoA levels, which could cause symptoms of irritability, fatigue, and apathy. Acetylcholine synthesis is also impaired; therefore, neurological symptoms can also appear in deficiency; they include numbness, paresthesia, and muscle cramps. Deficiency in pantothenic acid can also cause hypoglycemia, or an increased sensitivity to insulin. Insulin receptors are acylated with palmitic acid when they do not want to bind with insulin. Therefore, more insulin will bind to receptors when acylation decreases, causing hypoglycemia. Additional symptoms could include restlessness, malaise, sleep disturbances, nausea, vomiting, and abdominal cramps. In a few rare circumstances, more serious (but reversible) conditions have been seen, such as adrenal insufficiency and hepatic encephalopathy. Deficiency symptoms in other nonruminant animals include disorders of the nervous, gastrointestinal, and immune systems, reduced growth rate, decreased food intake, skin lesions and changes in hair coat, and alterations in lipid and carbohydrate metabolism.

People with your genetic profile are likely to have a predisposition for pantothenic acid deficiency.

Because some pantothenic acid is present in almost all foods, deficiency is rare except in people with severe malnutrition. When someone has a pantothenic acid deficiency, it is usually accompanied by deficiencies in other nutrients, making it difficult to identify the effects that are specific to pantothenic acid deficiency. The only individuals known to have developed pantothenic acid deficiency were fed diets containing virtually no pantothenic acid or were taking a pantothenic acid metabolic antagonist. On the basis of the experiences of prisoners of war in World War II and studies of diets lacking pantothenic acid in conjunction with administration of an antagonist of pantothenic acid metabolism, a deficiency is associated with numbness and burning of the hands and feet, headache, fatigue, irritability, restlessness, disturbed sleep, and gastrointestinal disturbances with anorexia. People with a pantothenate kinase-associated neurodegeneration 2 mutation are most likely to have inadequate pantothenic acid status. Pantothenic acid kinase is an enzyme that is essential for CoA and phosphopantetheine production. It is the principal enzyme associated with the metabolic pathway that is responsible for CoA synthesis. Mutations in the pantothenate kinase 2 (PANK2) gene cause a rare, inherited disorder, pantothenate kinase-associated neurodegeneration (PKAN). PKAN is a type of neurodegeneration associated with brain iron accumulation. A large number of PANK2 mutations reduce the activity of pantothenate kinase 2, potentially decreasing the conversion of pantothenic acid to CoA and thus reducing CoA levels. The manifestations of PKAN can include dystonia (contractions of opposing groups of muscles), spasticity, and pigmentary retinopathy. Its progression is rapid and leads to significant disability and loss of function. Treatment focuses primarily on reducing symptoms. Whether pantothenate supplementation is beneficial in PKAN is not known, but some anecdotal reports indicate that supplements can reduce symptoms in some patients with atypical PKAN. Few data on pantothenic acid intake in the United States are available. However, a typical mixed diet in the United States provides an estimated daily intake of about 6 mg, suggesting that most people in the United States consume adequate amounts. Some intake information is available from other Western populations. For example, a 1996–1997 study in New Brunswick, Canada, found average daily pantothenic acid intake of 4.0 mg in women and 5.5 mg in men.



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KIT ID: TPD14630189007528



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO PYRIDOXINE, PYRIDOXAL, PYRIDOXAMINE DEFICIENCY

RESULTS



Water-soluble vitamin B6 is widely present in many foods, including meat, fish, nuts, beans, grains, fruits and vegetables. Additionally, B6 is present in many multivitamin preparations for adults and children and added to foods as a supplement to breakfast foods, power bars, and powders. There are several active compounds or vitamers which fall under the generic B6. These include (1) pyridoxine an alcohol, (2) pyridoxal an aldehyde, (3) pyridoxamine which differs from the first two with an amine group, and (4) a 2,5' phosphate esters. The major esters are the active coenzyme form and are pyridoxal 5'phosphate(PLP) and pyridoxamine 5'phosphate(PMP). The major form of B6 in meats are the esters, and the major plant source is pyridoxine, which is less bioavailable. Pyridoxine is the most common form found in multivitamins. As a coenzyme, B6 is involved as a cofactor in over 100 enzyme reactions including amino acid metabolism, particularly homocysteine; carbohydrate metabolism, including gluconeogenesis and glycogenolysis; and lipid metabolism. B6 has a role in cognitive development thru neurotransmitter synthesis, immune function with interleukin-2 production, and hemoglobin formation. Fetal brain development requires adequate B6, and this continues throughout infancy. Vitamin B6 recommendations are made in accordance with age and life stage with pregnancy and breastfeeding involving the highest recommended daily allowance. In the United States and other western cultures, deficiency is rare with adequate diets, including B6 sources from poultry, fish, organ meats, potatoes, grains, legumes and non citrus fruits. Vitamin B6 deficiency is rare in isolation and usually found in association with other B vitamin deficiencies such as folic acid and B12. Low plasma levels of active B6 are found in chronic alcohol dependence, with obese states, pregnancy, preeclampsia and eclampsia, and malabsorptive states such as celiac, inflammatory bowel disease, and bariatric surgery. Additional at-risk groups with inadequate intake or increased metabolic requirements may become functionally deficient in B6. Included in this group are those with renal impairment, autoimmune disorders, and chronic alcohol use. Patients with chronic renal failure, especially those receiving hemodialysis or peritoneal dialysis, have low plasma levels of B6. Autoimmune disorders, such as rheumatoid arthritis, have increased catabolism of B6, resulting in higher demand for dietary supplementation of B6. Of great clinical importance in toxicology is that drug antagonists to vitamin B6 occurs with the tuberculosis medicine isoniazid. Also, penicillamine and levodopa, as well as some anticonvulsant medications, may interfere with B6 metabolism.

People with your genetic profile are likely to have regular pyridoxine levels.

Vitamin B6 is found in a wide variety of foods. The richest sources of vitamin B6 include fish, beef liver and other organ meats, potatoes and other starchy vegetables, and fruit (other than citrus). In the United States, adults obtain most of their dietary vitamin B6 from fortified cereals, beef, poultry, starchy vegetables, and some non-citrus fruits. About 75% of vitamin B6 from a mixed diet is bioavailable.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
NBPF3	rs4654748	CT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO COBALAMIN DEFICIENCY

RESULTS



Vitamin B12 (Cobalamin) is a water-soluble vitamin that is derived from animal products such as red meat, dairy, and eggs. Intrinsic factor is a glycoprotein that is produced by parietal cells in the stomach and necessary for the absorption of B12 in the terminal ileum. Once absorbed, B12 is used as a cofactor for enzymes that are involved in the synthesis of DNA, fatty acids, and myelin. As a result, a B12 deficiency can lead to hematologic and neurologic symptoms. B12 is stored in excess in the liver; however, in cases in which B12 cannot be absorbed for a prolonged period (e.g., dietary insufficiency, malabsorption, lack of intrinsic factor), hepatic stores are depleted, and deficiency occurs. Vitamin B12 deficiency has 3 primary etiologies: - Autoimmune: Pernicious anemia is an autoimmune condition in which antibodies to intrinsic factor are produced. Anti-intrinsic factor antibodies bind to and inhibit the effects of intrinsic factor, resulting in an inability of B12 to be absorbed by the terminal ileum. - Malabsorption: Parietal cells in the stomach produce intrinsic factor; therefore, any patient with a history of gastric bypass surgery may be at risk for developing a B12 deficiency because their new alimentary pathway bypasses the site of intrinsic factor production. In patients with normal intrinsic factor production, any damage to the terminal ileum, such as surgical resection due to Crohn disease, will impair the absorption of B12 and lead to a deficiency. Other damage to the small intestine, such as inflammation from Celiac disease or infection with the tapeworm *Diphyllobothrium latum*, may also result in a B12 deficiency. - Dietary Insufficiency: Vitamin B12 is stored in excess in the liver; however, patients who have followed a strict vegan diet for approximately three years may develop a B12 deficiency from a lack of dietary intake.

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

Vitamin B12 is naturally found in animal products, including fish, meat, poultry, eggs, milk, and milk products. Vitamin B12 is generally not present in plant foods, but fortified breakfast cereals are a readily available source of vitamin B12 with high bioavailability for vegetarians. Some nutritional yeast products also contain vitamin B12. Fortified foods vary in formulation, so it is important to read the Nutrition Facts labels on food products to determine the types and amounts of added nutrients they contain.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO MYO-INOSITOL DEFICIENCY

RESULTS



Inositol, or more precisely myo-inositol, is a carbocyclic sugar that is abundant in brain and other mammalian tissues, mediates cell signal transduction in response to a variety of hormones, neurotransmitters and growth factors and participates in osmoregulation. It is a sugar alcohol with half the sweetness of sucrose (table sugar). It is made naturally in humans from glucose. A human kidney makes about two grams per day. Other tissues synthesize it too, and the highest concentration is in the brain where it plays an important role making other neurotransmitters and some steroid hormones bind to their receptors. Since about 2008 myo-inositol gained importance in the management of polycystic ovary syndrome (PCOS) due to its efficacy, safety profile and worldwide availability. myo-Inositol plays an important role as the structural basis for a number of secondary messengers in eukaryotic cells, the various inositol phosphates. In addition, inositol serves as an important component of the structural lipids phosphatidylinositol (PI) and its various phosphates, the phosphatidylinositol phosphate (PIP) lipids. Inositol or its phosphates and associated lipids are found in many foods, in particular fruit, especially cantaloupe and oranges. In plants, the hexaphosphate of inositol, phytic acid or its salts, the phytates, serve as phosphate stores in seed, for example in nuts and beans. Phytic acid also occurs in cereals with high bran content. Phytate is, however, not directly bioavailable to humans in the diet, since it is not digestible. Some food preparation techniques partly break down phytates to change this. However, inositol in the form of glycerophospholipids, as found in certain plant-derived substances such as lecithins is well-absorbed and relatively bioavailable. Myo-Inositol (free of phosphate) was once considered a member of the vitamin B complex, called Vitamin B8 in this context. However, because it is produced by the human body from glucose, it is not an essential nutrient. myo-Inositol is naturally present in a variety of foods, although tables of food composition do not always distinguish between lecithin, the bioavailable form, and the unavailable phytate form in grains. Foods containing the highest concentrations of myo-inositol (including its compounds) include fruits, beans, grains, and nuts. Beans, nuts and grains, however, contain large amounts of phytate.

People with your genetic profile are likely to have a regular Myo-Inositol level.

Of nine possible structural isomers, Myo-Inositol is the most widely distributed in nature, being present in fresh fruits, vegetables, cereals, legumes, and nuts. Myo-inositol is a fundamental component of structural lipids in cell membranes, such as phosphatidylinositol (PI) and the various phosphatidylinositol phosphates (PIPs). Myo-inositol is endogenously synthesized from glucose-6-phosphate and represents, in some tissues, about 99% of intracellular inositol.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SLC5A11	rs4788439	CC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO THE DEFICIENCY OF PARAMINOBENZOIC ACID OR PABA

RESULTS



4-Aminobenzoic acid (also known as para-aminobenzoic acid or PABA because the number 4 carbon in the benzene ring is also known as the para position) is an organic compound with the formula H₂NC₆H₄CO₂H. PABA is a white solid, although commercial samples can appear gray. It is slightly soluble in water. It consists of a benzene ring substituted with amino and carboxyl groups. The compound occurs extensively in the natural world. PABA is an intermediate in the synthesis of folate by bacteria, plants, and fungi. Many bacteria, including those found in the human intestinal tract such as *E. coli*, generate PABA from chorismate by the combined action of the enzymes 4-amino-4-deoxychorismate synthase and 4-amino-4-deoxychorismate lyase. Plants produce PABA in their chloroplasts, and store it as a glucose ester (pABA-Glc) in their tissues. Humans lack the enzymes to convert PABA to folate, so require folate from dietary sources such as green leafy vegetables. In humans, PABA is considered nonessential and, although it has been referred to historically as "vitamin B_x", is no longer recognized as a vitamin, because most people have a microbiome that will generate PABA. Sulfonamide drugs are structurally similar to PABA, and their antibacterial activity is due to their ability to interfere with the conversion of PABA to folate by the enzyme dihydropteroate synthetase. Thus, bacterial growth is limited through folate deficiency. The potassium salt is used as a drug against fibrotic skin disorders, such as Peyronie's disease, under the trade name Potaba. PABA is also occasionally used in pill form by sufferers of irritable bowel syndrome to treat its associated gastrointestinal symptoms, and in nutritional epidemiological studies to assess the completeness of 24-hour urine collection for the determination of urinary sodium, potassium, or nitrogen levels. Despite the lack of any recognized syndromes of PABA deficiency in humans, except for those who lack the colonic bacteria that generate PABA, many claims of benefit are made by commercial suppliers of PABA as a nutritional supplement. The benefit is claimed for fatigue, irritability, depression, weeping eczema (moist eczema), scleroderma (premature hardening of the skin), patchy pigment loss in the skin (vitiligo), and premature grey hair.

People with your genetic profile are likely to have regular Para-aminobenzoic Acid (PABA) levels.

It may naturally occur in these foods: Brewer's yeast, Liver, Molasses, Mushrooms, Spinach, Whole grains. Other products may also contain PABA.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
NAT1	rs1057126	AA



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO PTEROYL-L GLUTAMIC ACID DEFICIENCY

RESULTS



Pteroyl-heptaglutamic acid, also known as Vitamin B-11, is a member of the B complex family of vitamins. Sometimes referred to as vitamin S or S factor, B-11 is also considered a folate (like B-9) and is a derivative of B-9. Folate and folic acid derive their names from the Latin word folium (which means "leaf"). Green leafy vegetables are a primary source of this nutrient, however, in Western diets bread and cereals fortified with folate may be a larger dietary source. An argument can be (and has been) made that enriched sources (bread and cereal) may not provide the absorption levels from leaf vegetables and in fact, be of no help at all. This is in part because of the other chemicals (namely bromine) used in the process of making bread and cereal. Bromine competes with other vitamins and minerals in absorption. A lack of folic acid leads to folate deficiency, an uncommon condition in the normal American diet. A complete lack of dietary folate takes months before deficiency develops. Most individuals have about 500–20,000 µg of folate stored in the body. It is not that uncommon to have depleted or less than optimal levels within the body for a variety of reasons, eating breads and cereals and no green leafy vegetables is only one of many. Stress, (whether from work, home, air pollution, or illness) also depletes stores of this nutrient. Any type of gut or intestinal illness (IBS, IBD, GERD, diarrhea, constipation, yeast infections, and parasites) blocks the manufacturing and absorption of B-11.

People with your genetic profile are likely to have a predisposition for Pteroyl-heptaglutamic acid deficiency.

Pteroyl-heptaglutamic acid is essential for the proper functioning of the nervous system and for maintaining its good state of health, it also stimulates and regulates gastric and pancreatic enzymes. Its absence can then lead to neuropsychological and gastric problems and disorders. For those who follow a vegetarian diet it is recommended to take all those plant foods that are rich in it. Vitamin B11 deficiency can lead to certain muscle diseases such as dystrophy. In the case of overdose with Vitamin B11, even if toxicity has not been demonstrated, the literature reports rare cases of gastritis, cramps, diarrhea, nausea, vomiting and increased platelet aggregation.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
MTHFR	rs1801133	GA



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO ERGOCALCIFEROL AND LUMISTEROL DEFICIENCY

RESULTS



Vitamin D is a fat-soluble vitamin used by the body for normal bone development and maintenance by increasing the absorption of calcium, magnesium, and phosphate. Vitamin D means a group of fat-soluble prohormones consisting of 5 different vitamins: vitamin D1 is composed from 1: 1 of ergocalciferol and lumisterol. Lumisterol is a compound that is part of the vitamin D family of steroid compounds. It is the ($9\beta,10\alpha$) stereoisomer of ergosterol and was produced as a photochemical by-product in the preparation of vitamin D1, which was a mixture of vitamin D2 and lumisterol. Vitamin D2 can be formed from lumisterol by an electrocyclic ring opening and subsequent sigmatropic hydride shift. Lumisterol has an analog based on 7-dehydrocholesterol, known as Lumisterol 3. Ergocalciferol, is a type of vitamin D found in food and used as a dietary supplement. As a supplement it is used to prevent and treat vitamin D deficiency.

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

Sources of Ergocalciferol and Lumisterol are: Fungus (Agaricus bisporus, Mushrooms, shiitake), Lichen, Plantae (Alfalfa).



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2R1	rs10832313	AA



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO ERGOCALCIFEROL DEFICIENCY

RESULTS



Vitamin D is a fat-soluble vitamin used by the body for normal bone development and maintenance by increasing the absorption of calcium, magnesium, and phosphate. Vitamin D means a group of fat-soluble prohormones consisting of 5 different vitamins: vitamin D2 is ergocalciferol. Ergocalciferol, also known as vitamin D2 and calciferol, is a type of vitamin D found in food and used as a dietary supplement. As a supplement it is used to prevent and treat vitamin D deficiency. This includes vitamin D deficiency due to poor absorption by the intestines or liver disease. It may also be used for low blood calcium due to hypoparathyroidism. It is used by mouth or injection into a muscle. Excessive doses can result in increased urine production, high blood pressure, kidney stones, kidney failure, weakness, and constipation. If high doses are taken for a long period of time, tissue calcification may occur. It is recommended that people on high doses have their blood calcium levels regularly checked. Normal doses are safe in pregnancy. It works by increasing the amount of calcium absorbed by the intestines and kidneys. Food in which it is found include some mushrooms.

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

Sources of Ergocalciferol are: Fungus (Agaricus bisporus, Mushrooms, shiitake), Lichen, Plantae (Alfalfa).



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2R1	rs10832313	AA



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO COLECALCIFEROL DEFICIENCY

RESULTS



Vitamin D is a fat-soluble vitamin used by the body for normal bone development and maintenance by increasing the absorption of calcium, magnesium, and phosphate. Vitamin D means a group of fat-soluble prohormones consisting of 5 different vitamins: vitamin D3 is cholecalciferol. Cholecalciferol, also known as vitamin D3 and colecalciferol, is a type of vitamin D which is made by the skin when exposed to sunlight; it is also found in some foods and can be taken as a dietary supplement. It is used to treat and prevent vitamin D deficiency and associated diseases, including rickets. It is also used for familial hypophosphatemia, hypoparathyroidism that is causing low blood calcium, and Fanconi syndrome. It is usually taken by mouth. Excessive doses in humans can result in vomiting, constipation, weakness, and confusion. Other risks include kidney stones. Normal doses are safe in pregnancy.] It may not be effective in people with severe kidney disease. Cholecalciferol is made in the skin following UVB light exposure. It is converted in the liver to calcifediol (25-hydroxyvitamin D) which is then converted in the kidney to calcitriol (1,25-dihydroxyvitamin D). One of its actions is to increase the uptake of calcium by the intestines. It is found in food such as some fish, cheese, and eggs. Certain foods such as milk have cholecalciferol added to them in some countries including the United States.

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

If vitamin D3 deficiency is found, we recommend that you investigate the other subcategories of vitamin D and consult your doctor for stable how and when to take food supplements.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
VDBP	rs7041	AC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DIHYDROXY ERGOCALCIFEROL DEFICIENCY

RESULTS



Vitamin D is a fat-soluble vitamin used by the body for normal bone development and maintenance by increasing the absorption of calcium, magnesium, and phosphate. Vitamin D means a group of fat-soluble prohormones consisting of 5 different vitamins: vitamin D4 is dihydroergocalciferol. 22-Dihydroxy Ergocalciferol is a form of vitamin D, also known as vitamin D4. It has the systematic name (5Z,7E)-(3S)-9,10-seco-5,7,10(19)-ergostatrien-3-ol. Vitamin D4 is found in certain mushrooms.

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

Cholecalciferol and ergocalciferol are the two forms of vitamin D. Vitamin D is not strictly a vitamin since it can be synthesized by the body. Therefore, vitamin D needs can be met by both dietary intake and endogenous synthesis by sun exposure. The main dietary sources of vitamin D are fatty fishes from cold sea: eel, salmon, mackerel and herring. Other dietary sources are mushrooms and dairy products. Fortified foods including milk/dairy products and cereals may also be important contributors to dietary vitamin D intake in many industrialized countries. For the supply of vitamin D lack of foods containing vitamin D can be compensated by endogenous synthesis from the action of sunlight on the skin.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP27B1	rs555068245	GG
CYP27B1	rs568165874	CC
CYP27B1	rs118204008	GG
CYP27B1	rs118204010	GG
CYP27B1	rs28934607	GG
CYP27B1	rs118204011	GG
CYP27B1	rs28934606	CC
CYP27B1	rs118204007	GG
CYP27B1	rs761780097	CC
CYP27B1	rs118204012	TT
CYP27B1	rs28934605	CC
CYP27B1	rs1057520815	CC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO TOCOPHEROL DEFICIENCY

RESULTS



Vitamin E is all the following eight compounds alpha, beta, gamma, and delta-tocopherol and alpha, beta, gamma, and delta-tocotrienol. Alpha-tocopherol is the only compound of the eight that are known to meet human dietary needs. All of the vitamin E forms are absorbed in the small intestine, and then the liver metabolizes only alpha-tocopherol. The liver then removes and excretes the remaining vitamin E forms. Vitamin E deficiency is extremely rare in humans as it is unlikely caused by a diet consisting of low vitamin E. Rather, it tends to be caused by irregularities in dietary fat absorption or metabolism. Vitamin E is a lipid-soluble nutrient. Vitamin E may have a role in reducing atherosclerosis and lowering rates of ischemic heart disease. Premature infants have low vitamin E reserves due to vitamin E only able to cross the placenta in small amounts. In developed countries, it is unlikely that vitamin E deficiency occurs due to diet intake insufficiency and the more common causes are below: - Premature low birth weight infants with a weight less than 1500 grams (3.3 pounds), - Mutations in the tocopherol transfer protein causing impaired fat metabolism, - Disrupted fat malabsorption as the small intestine requires fat to absorb vitamin E, Patients with cystic fibrosis patients fail to secrete pancreatic enzymes to absorb vitamins A, D, E, and K, - Short-bowel syndrome patients may take years to develop symptoms (surgical resection, mesenteric vascular thrombosis, and pseudo-obstruction are a few examples of this issue), - Chronic cholestatic hepatobiliary disease leads to a decrease in bile flow and micelle formation that is needed for vitamin E absorption, -Crohn's disease, exocrine pancreatic insufficiency, and liver disease may all not absorb fat, - Abetalipoproteinemia an autosomal-recessive disease causes an error in lipoprotein production and transportation, - Isolated vitamin E deficiency syndrome an autosomal recessive disorder of chromosome arm 8q. In developing countries, the most common cause is inadequate intake of vitamin E.

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

Serum levels of alpha-tocopherol in 0.1% of United States adults over the age of 20 have been found to be deficient. Surveys of the same data set have shown that 89.8% of men and 96.3% of women 19 years of age or older have insufficient intake of alpha-tocopherol. Some studies have shown alpha-tocopherol to be lower in pediatric populations and higher in pregnancy. A low alpha-tocopherol level or low ratio serum alpha-tocopherol to serum lipids measurement is the mainstay of diagnosis. In adults, alpha-tocopherol levels should be less than 5 mcg/mL. In an adult with hyperlipidemia, the abnormal lipids may affect the vitamin E levels and a serum alpha-tocopherol to lipids level, needing to be less than 0.8 mg/g) is more accurate. A pediatric patient with abetalipoproteinemia will have serum alpha-tocopherol levels that are not detectable. Treatment addresses the underlying cause of the deficiency (fat malabsorption, fat metabolism disorders, among others) and then provide oral vitamin E supplementation. Also, a modification in diet can assist in the supplementation, increase intake of leafy vegetables, whole grains, nuts, seeds, vegetable oils and fortified cereals is highly recommended. Though normally presented in our diets, adults need 15mg of vitamin E per day. A supplement of 15 to 25 mg/kg once per day or mixed tocopherols 200 IU can both be used. If a patient has issues with the small intestine and/or oral ingestion intramuscular injection is necessary. The recommended daily allowance of alpha-tocopherol is as follows: Age 0 to 6 months: 3 mg; Age 6 to 12 months: 4 mg; Age 1 to 3 years: 6 mg; Age 4 to 10 years: 7 mg; Adults and elderly patients: 10 mg.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP4F2	rs2108622	TT
CYP4F2	rs3093105	AA



KIT ID: TPD14630189007528

PREDISPOSITION TO NAFTOCHINONE DEFICIENCY

RESULTS



Vitamin K is a group of fat-soluble 2-methyl-1,4-naphthoquinone. There is a variable alkyl substituent at the third position and exists in two principal forms: K1 (phylloquinone) and K2 (menaquinone). There is a third, synthetic form K3 (menadione), the use of which has been replaced by a synthetic form of vitamin K1 due to the potential for toxicity in infants with glucose-6-phosphate dehydrogenase deficiency. The primary vitamin K1 is predominantly from leafy greens and vegetables, while the main source of Vitamin K2 is intestinal flora and fermented foods. Vitamins K1 and K2 have different bodily distributions and may have different impacts on enzyme activity. Vitamin K1 is the major source in the human diet and is absorbed in the jejunum and ileum, transported by chylomicrons in circulation, and is dependent on bile, pancreatic enzymes, and dietary fat content. These substances are necessary for adequate blood clotting because they are cofactors for gamma-glutamyl carboxylase and vitamin K2,3-epoxide reductase complex in modifying gamma-carboxyglutamic acid on clotting factors II, VII, IX, and X. This modification is required for cofactors to bind to phospholipids in the platelet membrane. Under-carboxylated clotting factors will lead to decreased protein activity and can lead to bleeding. Vitamin K is also a requirement for various other proteins including anti-coagulant proteins (C, S, and Z), osteocalcin, and matrix GLA protein. Under-carboxylated osteocalcin has shown to increase in individuals with decreased bone mineral density and with increased fracture rates in the elderly. Decreased levels of some vitamin K subtypes results in increased arterial calcification. Vitamin K deficiency can contribute to significant bleeding, poor bone development, osteoporosis, and increased cardiovascular disease. According to the National Academy of Science Food and Nutrition Board, the dietary requirements are based on the intake of healthy adults, and the adequate intake is 120 and 90 ug/day for men and women, respectively. Vitamin K Deficiency Bleeding (VKDB) in newborns can separate into three categories based on the timing of the presentation. Early VKDB presents within 24 hours after birth, classic VKDB presents within the first week, and late VKDB presents between one to twelve weeks of life.

People with your genetic profile are likely to have regular naftochinone levels.

Vitamin K is found in the following foods: Green leafy vegetables, such as kale, spinach, turnip greens, collards, Swiss chard, mustard greens, parsley, romaine, and green leaf lettuce. Vegetables such as Brussels sprouts, broccoli, cauliflower, and cabbage. Fish, liver, meat, eggs, and cereals (contain smaller amounts).



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP4F2	rs2108622	TT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO ZINC DEFICIENCY

RESULTS



Zinc is an essential nutrient for humans and is extensively involved in protein, lipid, nucleic acid metabolism, and gene transcription. Its role within the human body is extensive in reproduction, immune function, wound repair, and on the microcellular level, macrophage, neutrophil, natural killer cell, and complement activity. Zinc is found in multiple food groups including meat, fish, legumes, and other dietary sources although absorption varies by substrate. Zinc deficiency can be inherited as absorption difficulties or can manifest from a decreased intake. Zinc deficiency is common worldwide, but mostly in developing countries. It presents with infectious, inflammatory, gastrointestinal, or cutaneous involvement. Treatment is largely through oral replacement usually resulting in quick clinical improvement. Zinc is a divalent cation not synthesized within the human body and requires intake to maintain adequate levels. Deficiency can occur from decreased intake, inability to absorb the micronutrient, increased metabolic demand, or excessive loss.

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

Zinc deficiency is not common, but it occasionally occurs in people with restricted diets and malabsorption problems. Zinc deficiency can be prevented in the majority of cases by educating the public. Diagnosis can begin with establishing suspicion of inherited or acquired deficiency based on the above clinical features, and acquired disease can be suspected after evaluation of risk factors including geographical prevalence and age of presentation. Acrodermatitis enteropathica is suspected clinically and supported by laboratory findings and histopathology. Lab values will demonstrate low serum alkaline phosphatase (a zinc-dependent metalloenzyme) and low plasma zinc concentrations. Serum studies and the ideal collection includes using zinc-free vacuum tubes, stainless steel needles, avoiding contact with rubber stoppers, avoiding hemolysis, separating plasma or serum from cells within 45 minutes, use of anticoagulants low in zinc concentration, as well as morning fasting samples optimize accuracy. Normal zinc levels are between 70 to 250 ug/dl in adults, and mild deficiency can manifest clinically when values decrease to 40 to 60 ug/dl. Urine zinc levels vary widely and are not a reliable marker for the acute state. Hair zinc level is also an unreliable marker in acute changes. Punch biopsy and histopathology of affected tissue can support the diagnosis of necrolysis seen as cytoplasmic pallor, vacuolization, ballooning degeneration, and confluent necrosis of keratinocytes in the upper epidermis. Confluent parakeratosis is often present with loss of the granular layer and with dermal edema. An associated neutrophilic crust may be present. Individual keratinocytes often have pyknotic nuclei. These findings are non-specific and are often seen with pellagra and necrolytic migratory erythema. Late lesions of zinc deficiency may mimic psoriasis. Clinical improvement to zinc supplementation can also be confirmed. Treatment begins with oral replacement. Two to 3 mg/kg per day often cures all clinical manifestations within 1 to 2 weeks. Even in patients with acrodermatitis enteropathica, a disease of malabsorption, oral replacement with 1 to 2 mg/kg per day is still the standard of therapy with life-long supplementation. For preterm infants with zinc deficiency, normal breastfeeding is usually sufficient for correction, and the deficit usually resolves within weeks with no clinical symptoms. However, maternal breast milk can be zinc deficient if the mother's stores are depleted. Recommended daily dietary intake for lactating adult women increases from 11 mg per day to 12 mg per day. With treatment, there is often a rapid improvement of symptoms.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
PPCDC	rs2120019	TC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO EXCESSIVE ALCOHOL CONSUMPTION

RESULTS



Alcohol misuse has been linked to numerous social, economic, and health problems. Estimates vary but have suggested that up to 40% of patients have experienced complications of alcohol misuse. In the United States, 138.3 million people age 12 and older, surveyed, report that they actively use alcohol, according to the 2015 National Survey on Drug Use and Health. Of those, 48.2% report that they had binge drinking episode(s) within 30 days before taking the survey. Of those who reported binge drinking, 26% reported heavy alcohol use, defined as binge drinking five or more days in the previous 30 days, which accounts for 12.5% of total alcohol users. This means that 5.9%, or 15.7 million people in the United States aged 12 and older, meet criteria for an alcohol use disorder (see image for criteria). More than 85,000 deaths per year can be attributed to alcohol. In addition, motor vehicle accidents, dementia, depression, homicide and suicide have all been linked to alcoholism. Although the pathogenesis of alcohol use disorder is not strictly known, several factors are thought to contribute to its development. These include environmental influences, such as home environments, peer interactions, genetic factors, the level of cognitive functioning, and certain existing personality disorders. Some of the genes suspected include GABRG2 and GABRA2, COMT Val 158Met, DRD2 Taq1A, and KIAA0040. Personality disorders associated with the development of an alcohol use disorder include disinhibition and impulsivity-type disorders, as well as depressive and socialization-related disorders.

People with your genetic profile are likely to have a predisposition to excessive alcohol consumption.

Multiple theories have been suggested as to why some people develop alcohol use disorders. Some of the more evidence-supported theories include positive-effect regulation, negative-effect regulation, pharmacological vulnerability, and deviance proneness. Positive-effect regulation results in drinking for positive rewards (such as feelings of euphoria). Negative-effect regulation is seen when one drinks to cope with feelings of a negative nature, such as depression, anxiety, or feelings of worthlessness. Pharmacological vulnerability makes a note of an individual's varied response to both acute and chronic effects of alcohol intake and the individual differences in the body's ability to metabolize the alcohol. Deviance proneness speaks more to an individual's tendency towards deviant behavior established during their childhood, often due to a deficiency in socialization at an early age. Some treatment foci that have demonstrated promise include evidence-based motivational interviewing. This particular approach helps the patients explore the reasons behind their ambivalence with respect to changing their behavior or alcohol cessation to change their substance abuse-related behaviors with a personalized assessment of risks and needs. Other therapies include cognitive behavior therapy, 24-hour residential facilities that aim to treat medical as well as psychiatric complications or comorbidities associated with the alcohol use and process of cessation. There are also multiple programs, such as Alcoholics Anonymous or other 12-step programs that focus on group support/mentors that can provide a source of assistance with the maintenance of abstinence. Many patients have lapses during their lifetime and will require initiation of differing intensities of therapy throughout their lifetime.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
C4orf17	rs79139602	AA
ADH1B - ADH7	rs2165670	GG
FUT2 - MAMSTR	rs281379	GA
ADH5	rs1154414	TC



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

SCIENTIFIC DETAILS		
AP001961.1 - EMCN	rs4699791	GG
LINC01572	rs79616692	GG
ACTR1B	rs11692435	GG



ITCH INTENSITY FROM MOSQUITO BITE

RESULTS



Following a mosquito bite, mosquito salivary antigens elicit a cutaneous hypersensitivity reaction, predominated by mast cell degranulation of pruritic mediators, lymphocyte recruitment and localised inflammation. Longstanding evidence has suggested a natural history of sensitisation and subsequent desensitisation to mosquito saliva antigen in humans. As well as this within-individual variation, levels of mosquito antigen hypersensitivity are known to vary between individuals with bite reaction sizes ranging from mild wheals to large papules. Mosquito bites may cause a local cutaneous manifestation consisting of an immediate wheal and flare reaction that peaks after 20 minutes. Delayed pruritic indurated papules may arise within 24–36 hours and then diminish over several days or weeks. Larger local manifestations vary from pruritic, warm tumefaction to bullous reactions. The spectrum of manifestations may differ depending on subject susceptibility. Noteworthy there is no cross-reactivity between species of mosquitos. Therefore when a human is beaten by one species, there will be no sensitization to other species. Desensitization may occur during childhood or with continuous mosquito antigen exposure. Mosquitoes are a ubiquitous group of biting insects that commonly cause acute itch in humans. Pruritic manifestations can be debilitating, especially in individuals that are susceptible to an increased biting frequency. Despite the high frequency of the symptom, the pathogenesis of itch associated with mosquito bites is poorly understood. Although self-limited in the majority of cases, mosquito bites may impact the quality of life of certain populations that exhibit exaggerated cutaneous reactions. Moreover, the consequences of scratching can lead to superinfection, hyperpigmentation, and scarring.

People with your genetic profile are likely to have a higher frequency of mosquito bites with severe itching.

Factors that may predispose an individual to a higher frequency of biting include: lower microbial diversity on the skin, sweat, body odor, pregnancy, higher body temperature, blood O type, alcohol, applied scents, and dark clothing. Preventative measures can be taken in order to avoid the bite of a mosquito, which are summarized in. It is recommended to wear protective clothing and gear treated with permethrin, a pyrethroid agent. However some mosquitoes have been shown to develop pyrethroid resistance. Topical agents to help reduce inflammation and itching include calamine lotion and corticosteroid creams. In contrast, although topical antihistamines are widely available over-the-counter, they may put individuals at risk for allergic contact dermatitis, especially diphenhydramine, mepyramine, promethazine, and antazoline. Sensitization by topical diphenhydramine has resulted not only in cases of contact dermatitis, but also in photoallergic dermatitis and connubial dermatitis as well. Connubial dermatitis refers to dermatitis resulting from close contacts, in which the dermatitis-inducing agent has not been used by the patient but by his or her partner.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
P4HA2, SLC22A4	rs35260072	AA
IL3 - CSF2	rs11242103	CC
AC008695.1, FNIP1	rs35403253	CC
ARL2BPP4 - AC005741.1	rs113264677	AA
CHSY3 - RNU7-53P	rs971891	CT
LINC01862 - AC243312.1	rs2967678	CC



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SCIENTIFIC DETAILS

BBS12 - AC021205.2	rs79712192	GG
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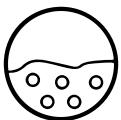
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KIT ID: TPD14630189007528

SKIN PIGMENTATION

RESULTS



Pigmentation means coloring. Skin pigmentation disorders affect the color of your skin. Your skin gets its color from a pigment called melanin. Special cells in the skin make melanin. When these cells become damaged or unhealthy, it affects melanin production. Some pigmentation disorders affect just patches of skin. Others affect your entire body. If your body makes too much melanin, your skin gets darker. Pregnancy, Addison's disease, and sun exposure all can make your skin darker. If your body makes too little melanin, your skin gets lighter. Vitiligo is a condition that causes patches of light skin. Albinism is a genetic condition affecting a person's skin. A person with albinism may have no color, lighter than normal skin color, or patchy missing skin color. Infections, blisters and burns can also cause lighter skin. Melasma is believed to be caused by hormonal changes and develops during pregnancy. Areas of hyperpigmentation can appear on any area of the body. They appear most commonly on the abdomen and face. Sunspots, also called liver spots or solar lentigines, are common. They're related to excess sun exposure over time. Generally, they appear as spots of hyperpigmentation on areas exposed to the sun, like the hands and face. Post-inflammatory hyperpigmentation is a result of an injury to the skin.

People with your genetic profile are likely to have a predisposition to develop hyperpigmentation.

Hyperpigmentation is caused by excess melanin production - the pigment that gives natural color to the skin, hair and eyes - in some areas of the skin. This excess production is stimulated by a number of factors, but the main ones can be attributed to sun exposure, genetic factors, age, hormonal influences, and skin wounds or inflammation. Sun exposure is the number one cause of hyperpigmentation, as it is primarily sunlight that stimulates melanin production. Melanin acts as a natural skin shield, protecting it from harmful UV rays, which is why people tan in the sun. But excessive sun exposure can ruin this process, leading to hyperpigmentation. Once dark spots have developed, sun exposure can also exacerbate them making freckles, age spots, melasma and post-inflammatory hyperpigmentation even darker. Limiting the time of sun exposure, wearing protective clothing, and using a broad spectrum sunscreen with a high FP can help reduce the risk of developing hyperpigmentation, and prevent dark spots from getting worse. Dermatological treatments - anti-pigmentation procedures are: Chemical peels involve applying an acid solution to the face, hands or feet to remove the surface layers of the skin. These chemicals cause blisters on the skin which can even be removed, revealing new and uniform complexion under the skin. Laser therapies have more or less the same effect, but tend to be more precise, as the dermatologist has greater control over the intensity of the treatment. They work by 'hitting' affected parts with high energy light. The most delicate treatments act only on the epidermis (superficial layer of the skin), while the more intense ones manage to penetrate into the deeper layers.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC092078.2 - SLC24A5	rs2675345	AA
AL590677.1 - PRPF18	rs6602666	AA
SLC24A5, MYEF2	rs2470102	AG
SST - RTP2	rs79592764	CC
PRTFDC1	rs151165649	GG
AC104574.2	rs532282237	TT



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SCIENTIFIC DETAILS		
AL590677.1 - PRPF18	rs6602665	TT
AC015911.11, SLFN12L	rs117307642	CC



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FEAR OF MINOR PAIN

RESULTS



Heightened fear and anxiety related to pain may result in emotional and behavioral avoidance responses causing disability, distress, and depression. Fear and anxiety associated with pain can potentially change the course of the pain experience. It is plausible that fear and anxiety related to pain affect the duration and frequency of pain experienced by the patient. Fear of pain (FOP) is associated with emotional reactions occurring in the anticipation of pain or during pain. FOP is related to increased pain perception and reduced capacity for pain inhibition, and is a central mechanism in several pain disorders. Fear of pain is experienced in acute and chronic pain populations, as well as in the general population, and it affects numerous aspects of the orofacial pain experience, including pain intensity, pain-related disability, and pain behavior (e.g., avoidance).

People with your genetic profile are not likely to have a fear of minor pain.

Fear is a primary defense emotion, caused by a dangerous situation that can be real, anticipated by prediction, evoked by memory or produced by fantasy. Fear is often accompanied by an organic reaction, for which the autonomic nervous system is responsible, which prepares the organism for the emergency situation, arranging it, even if in a non-specific way, for the preparation of the defenses that usually translate into attitudes fight and flight.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC02536 - THEMIS	rs72965720	AA
TNNI3K, FPGT-TNNI3K	rs114134414	AA
MYOCD, AC005358.1	rs56875752	AA
AC079313.2	rs61923480	AA



KIT ID: TPD14630189007528

SKIN AGING

RESULTS



Skin microtopography as a measure of photoaging is a non-invasive approach to measuring chronic ultraviolet radiation (UVR) exposure, and reflects the degree of dermal elastosis in populations of European descent in the subtropics. Less is known about the utility of this approach in populations at different latitudes, and whether it relates to skin cancer risk. The morphology of the skin surface has been studied extensively in the context of aging, disease, effect of sun exposure, and dermatological and cosmetic treatment. Skin micro-relief and wrinkles are manifestations of different etiology which in combination produce the overall skin roughness.

People with your genetic profile are likely to have regular aging of skin.

Apply moisturizer every day. As we age, skin becomes drier. Fine lines and wrinkles appear. Moisturizer traps water in our skin, giving it a more youthful appearance. For best results, use a facial moisturizer, body moisturizer, and lip balm.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SLC45A2	rs185146	CC
PEX3	rs3804540	AA
KIDINS220 - MBOAT2	rs191497052	CC
ARHGEF7	rs3825460	CC



KIT ID: TPD14630189007528

GAIT SPEED IN OLD AGE

RESULTS



Remaining years of life vary widely in older adults, and physicians should consider life expectancy when assessing goals of care and treatment plans. However, life expectancy based on age and sex alone provides limited information because survival is also influenced by health and functional abilities. There are currently no well-established approaches to predicting life expectancy that incorporate health and function, although several models have been developed from individual data sources. Gait speed, also often termed walking speed, has been shown to be associated with survival among older adults in individual epidemiological cohort studies and has been shown to reflect health and functional status.¹³Gait speed has been recommended as a potentially useful clinical indicator of well-being among the older adults. The purpose of this study is to evaluate the association of gait speed with survival in older adults and to determine the degree to which gait speed explains variability in survival after accounting for age and sex.

People with your genetic profile are not likely to have a predisposition to walk slower with age.

The skeletal system and the joints, with the progress of age, undergo various degenerative phenomena; among the various: bone demineralization, which leads to osteoporosis, fragility and susceptibility to fractures, rheumatic diseases, arthrosis etc. It is well known that, when the bones are stressed under load, they are able to have lower mineral losses and even a slight reconstruction (1% on a periodic basis), maintaining a good bone density. It has also been shown that, to keep your joints healthy, the best way is to keep them moving and avoid overloading them - to ward off negative effects on cartilage.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CDH13	rs11861812	CC
PTH2R	rs10210510	GG
GABRA1	rs6883877	CC
SIM1	rs17060534	GG



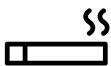
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NICOTINE DEPENDENCE

RESULTS



Nicotine dependence — also called tobacco dependence — is an addiction to tobacco products caused by the drug nicotine. Nicotine dependence means you can't stop using the substance, even though it's causing you harm. Nicotine produces physical and mood-altering effects in your brain that are temporarily pleasing. These effects make you want to use tobacco and lead to dependence. At the same time, stopping tobacco use causes withdrawal symptoms, including irritability and anxiety. While it's the nicotine in tobacco that causes nicotine dependence, the toxic effects of tobacco result from other substances in tobacco. Smokers have much higher rates of heart disease, stroke and cancer than nonsmokers do.

People with your genetic profile are not likely to have a predisposition to develop nicotine dependence.

It is possible that some individuals never feel the desire to try to smoke a cigarette in their life. Likewise, it is likely that while trying to smoke tobacco, they never develop addiction. In fact, it has been observed that the triggering of a mechanism of dependence, in addition to mental and environmental factors, is also strongly influenced by a genetic predisposition.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
FAM163B - AC002101.1	rs56116178	AA
HYKK	rs34684276	GG
CHRNA4	rs2273500	TT



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MENARCHE (AGE AT ONSET)

RESULTS



Menarche or first menstrual period is a landmark in reproductive life span and it is the most prominent change of puberty. The timing of menarche can be under the influence of genes as well as individual environmental factors interacting with genetic factors. Age at menarche, the time of first menstrual period, is an important biological and social event as well as a developmental milestone in females. Age at menarche has been identified as a risk factor for several traits including, depression, eating disorders, breast cancer, body image, osteoporotic fracture, risk of coronary heart disease, and conduct disorder. The latter is a common example of age of menarche being a complex trait, which is determined by an array of genetic and environmental factors. Early age of menarche has been associated with conduct disorders.

People with your genetic profile are likely to have an early onset of menarche.

Premature puberty is an event similar to physiological puberty but occurs earlier than the average of peers, i.e. before 8 years in the female and 9 years in the male. About 10% of girls who have premature thelarche go into early puberty. Early puberty has a low incidence (0.1-0.6% of the general population) for which it is classified in rare diseases. It is more frequent in the female in which the idiopathic form prevails (that is, not attributable to an identified cause), while in males the probability that early development is secondary to an organic pathology is greater. The changes that occur during puberty are related to the production of hormones produced by the pituitary, known as gonadotropins, which stimulate the function of the testicles and ovaries.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC019330.1	rs12617311	GG
LINC02073 - CA10	rs9635759	GA



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PHOTIC SNEEZE REFLEX

RESULTS



Photic sneeze reflex (PSR) is an interesting but yet mysterious phenotype featured by individuals' response of sneezing in exposure to bright light. Photic sneeze reflex (PSR), also known as autosomal dominant compulsive helio-ophthalmic outbursts (ACHOO) syndrome, refers to uncontrollable reflexive sneezing in response to sudden exposure to bright light. Sneezing is generally a protective reflex expelling particles and irritants from the nasal cavity, but it is a puzzle how bright light would stimulate the sneeze reflex, and evolutionarily speaking, whether it has any physiological relevance. Reflexive sneezing induced by light, and sunlight in particular, is estimated to occur in 18 to 35 percent of the population and is known as the photic sneeze reflex (PSR) or the ACHOO (autosomal dominant compulsive helio-ophthalmic outbursts of sneezing) syndrome. Its genetic nature has been known for at least the last 25 years; it is periodically discussed in the medical literature and lay press. Observations that emerge from dim light into sunlight or turning to face directly into the sun commonly triggers the reflex prompted early inquiries into the trait. The number of induced sneezes--which seems to be genetically mediated and can be predicted within a family--is constant from episode to episode and typically numbers two or three.

People with your genetic profile are not likely to have a predisposition to experience a photic sneeze reflex.

Sneezing is an act induced by the activation of a trigeminal reflex. Sensitive fibers of the trigeminal nerve are in fact distributed also to the mucosa of the nasal cavities and to the mucosa of part of the upper respiratory tract; the stimulation (for example by small foreign bodies, such as dust or pollen) of these fibers induces their activation. They unload their impulses on the neurons of the main sensory nucleus of the trigeminal, which selectively recruits the bulbar respiratory center located in the parvocellular reticular area of the lateral parasagittal column of the reticular formation and motor neurons of the vagal ambiguous nucleus. In the first case, the projections are directed to medullary motor neurons delegated to the innervation of the diaphragm and intercostal muscles; in the second case, these are motor neurons responsible for innervating laryngeal and pharyngeal (striated) muscles. The result is therefore the violent expulsion of the air contained in the lungs and the coordinated contraction of the laryngeal and pharyngeal muscles.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
AC015574.1	rs1541995	CC
ESRRG	rs12755398	GG



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PREDISPOSITION TO RESPOND POSITIVELY TO A LOW CARBOHYDRATE DIET

RESULTS



Studies have shown low-carb approaches to be superior to other dietary approaches in producing rapid weight loss for the first 6-12 months. When lowering carbohydrates from the diet, the macronutrient intake of fat and protein generally increases to compensate for the reduction of carbohydrates. Fats and protein increase satiety and produce less concomitant hypoglycemia. This increase in satiety and less rebound hypoglycemia then reduces hunger and overall food intake and produces a caloric deficit. Recent reviews of low-carb diets on lipids demonstrate a neutral increase in LDL but a favorable triglycerides reduction and an increase in HDL cholesterol, particularly those assigned to the very low-carb intervention. Low-carb diet, and specifically ketogenic approaches, induces rapid induction of weight loss. Initial weight loss is due partly from water loss, but fat loss occurs with adherence to the low carb approach. Low-carb nutrition may be advisable for those who desire health or athletic performance, weight loss, improvement of glycemic control for type 1 or 2 diabetes, or for a seizure disorder.

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

According to your results, you are likely to have an enhanced response to a low carbohydrate diet in terms of weight loss. There are numerous ways to start a person on a low-carb diet. Low-carb nutrition may be advisable for those who desire health or athletic performance, weight loss, improvement of glycemic control for type 1 or 2 diabetes, or for a seizure disorder. First, an understanding of what macronutrients are and its relation to food is a critical part of the counseling. Secondly, determine the patient's desire for either small steps or a rapid induction phase through motivational interviewing and S.M.A.R.T goal setting. Limitation of added sugar (sucrose) and refined carbohydrates is critical in the overall improvement of food quality and will generally reach a moderate carbohydrate (< 45% carbohydrates) level. A way to initiate low-carb is through a rapid induction phase of 2 to 4 weeks, with 20 to 50 gms of carbohydrates to induce nutritional ketosis. Ad libitum vegetables that grow above the ground and are lower in carbohydrate content are encouraged. Additionally, carbs should be limited to those found in whole, unprocessed food. Finally, after the induction phase, depending on goals, people can remain in the keto phase or slowly add healthy carbohydrates from whole, unprocessed vegetables, and low-glycemic, high fiber fruit (i.e., berries).



SCIENTIFIC DETAILS

Gene	rsID	Genotype
ISCA1P1 - AC113420.1	rs16891077	GA
RPA2P2 - AL096706.1	rs9787485	CC
AMY1-AMY2	rs11185098	GG



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KIT ID: TPD14630189007528

PREDISPOSITION TO RESPOND POSITIVELY TO THE MEDITERRANEAN DIET

RESULTS



The term "Mediterranean diet" is used today to describe the traditional dietary habits of countries neighboring the Mediterranean Sea, mostly Greece and Southern Italy. The Mediterranean diet is not, in fact, a unique diet in today's meaning of the word "diet". The term "Mediterranean diet" could be best understood as a peculiar "dietary pattern" featuring an inter-related set of specific characteristics. Descriptions including only some foods present in the popular culture: first, olive oil plays a central role in the cooking process, and thus, represents the main source of dietary fat. Cheese is used in limited servings and usually within salads. Meat, milk, and eggs are consumed with a low frequency and in small amounts, and processed meat and sweets are practically non-existent. The Mediterranean diet hence represents, in fact, the only traditional dietary pattern where consumption of saturated and trans fats is inherently minimal. Second, olive oil consumption is associated with a higher vegetable intake, cooked as salads, and to an equally high legume intake in thermic-prepared foods. Other key components of the Mediterranean diet are the whole grains, nuts, fresh fruits, and a moderate fish intake. Grapes and their derivative products are also used, but one of the main features of the Mediterranean diet is the limited intake of alcohol, as red wine is consumed only with meals. However, some variations in food intake between various countries do exist. Besides cardiovascular, metabolic, cognitive, and possibly anti-neoplastic benefits, the Mediterranean diet seems to be associated with good adherence scores in some extra-Mediterranean populations and with an improved quality of life.

People with your genetic profile are likely to have a regular healthy response to the Mediterranean diet.

There are variations of the "Mediterranean diets" in different countries and among the individual populations of the Mediterranean basin, due to ethnic, cultural, economic and religious diversities. The "Mediterranean diet" as defined by dietitians generally includes the following components, which are not typical of diets in the Mediterranean basin: High intakes of olive oil (as the principal source of fat), vegetables (including leafy green vegetables, onions, garlic, tomatoes and peppers), fresh fruits (consumed as desserts or snacks), cereals (mostly whole grains), nuts and legumes. Moderate intakes of fish and other seafood, poultry, eggs, dairy products (principally cheese and yogurt) and red wine. Low intakes of red meat, processed meat, refined carbohydrates and sweets. In contrast to the dietary recommendation, olive oil is not the staple fat in much of the Mediterranean basin: in northern and central Italy, lard and butter are commonly used in cooking, and olive oil is reserved for dressing salads and cooked vegetables; in both North Africa and the Middle East, sheep's tail fat and rendered butter (samna) are traditional staple fats.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TCF7L2	rs7903146	CC



PREDISPOSITION TO AN ALTERED METABOLISM OF STARCH

RESULTS



The rising prevalence of obesity is one of the most important public health problems. Low-fat/high carbohydrate diets seem to be effective for body weight management. However the amount and type of carbohydrates included influence the metabolic responses. The dietary carbohydrates can be divided into three main groups, sugars, oligosaccharides and polysaccharides. Polysaccharides can be divided into starch and non-starch polysaccharides. Starch can be divided into rapidly digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS). Several studies have shown that higher intakes of SDS and RS are associated with increased satiety, reduced hunger and/or reduced body weight. The starch composition of food and its rate of digestion are determinants of blood glucose and insulin levels. Several studies have shown that higher intakes of slowly digested and resistant starches are associated with a reduced glycemic response and insulin resistance, while rapidly digestible starch may lead to hyperglycemic episodes, being associated with an increased risk of insulin resistance and type 2 diabetes. All these properties make RS and SDS attractive dietary targets for the development of weight maintenance diets and diets for the prevention and treatment of metabolic syndrome and cardiovascular risk factors. Data indicate that a high RS diet leads to a lower weight of fat depots and can reduce serum total cholesterol triacylglycerol concentrations. Thus, suggested that replacement of total dietary carbohydrate with RS increases postprandial lipid oxidation and may decrease fat accumulation in the long term. Thus, slowly absorbable and non-absorbable carbohydrates may all influence serum lipids and modify risk factors for cardiovascular disease. Resistant starch is associated with several changes in metabolism, which may confer some health benefits.

People with your genetic profile are likely to have a regular starch metabolism.

Starchy foods are a good source of energy and the main source of a range of nutrients in our diet. As well as starch, they contain fibre, calcium, iron and B vitamins. Some people think starchy foods are fattening, but gram for gram they contain fewer than half the calories of fat.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
AMY1-AMY2	rs11185098	GG



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KIT ID: TPD14630189007528

CAFFEINE METABOLISM: PLASMA LEVELS OF PARAXANTHINE

RESULTS



Caffeine is produced commercially by both extraction and synthetic procedures. Extraction procedures involve three methods: direct decaffeination of green coffee beans with solvents, extraction from tea dusts and wastes and fragments of tea leaves, and extraction from cola nuts. Synthetic production of caffeine involves the methylation of various xanthines. Caffeine absorption from the gastrointestinal tract is rapid, virtually complete and directly dependent on pH. After oral doses of 5–8 mg/kg bw, peak plasma concentrations of 8–10 µg/ml were observed. After oral ingestion, the time to reach peak plasma concentration exhibits wide variations, ranging from 15 to 120 min. After absorption, caffeine is rapidly and uniformly distributed into body fluids. Caffeine is eliminated by apparent first-order kinetics, described by a one-compartment open model system. Caffeine metabolism occurs primarily in the liver, catalyzed by hepatic microsomal enzyme systems. In healthy humans, repeated caffeine ingestion does not alter its absorption or metabolism. It is metabolized in the liver to dimethylxanthines, uric acids, di- and trimethylallantoin, and uracil derivatives. In humans 3-ethyl demethylation to paraxanthine is the primary route of metabolism. This first metabolic step accounts for approximately 75–80 percent of caffeine metabolism and involves cytochrome P4501A2. Paraxanthine is the dominant metabolite in humans, rising in plasma to concentrations 10 times those of theophylline or theobromine. Caffeine is cleared more quickly than paraxanthine, so 8 to 10 hours after caffeine ingestion, paraxanthine levels exceed caffeine levels in plasma. The human body converts 70–80 percent of caffeine into paraxanthine with no apparent toxic effects following caffeine doses of 300–500 mg/day suggests that paraxanthine's toxicological potency is low. Formation of paraxanthine and its excretion in the urine appears to be the major pathway for caffeine metabolism. The variability in the production and excretion rates of acetylated urinary metabolites was related to acetylation polymorphism. At low doses (up to 2 µg/ml in blood), caffeine stimulates the central nervous system, and this effect is perceived by many caffeine users as beneficial. High blood concentration (10–30 µg/ml) of caffeine may produce restlessness, excitement, tremor, tinnitus, headache and insomnia. Caffeine can induce alterations in mood and sleep patterns, increase urine production and gastric acid secretion, alter myocardial function, induce hypertension and arrhythmia, and increase plasma catecholamine levels and plasma renin activity, especially when administered to non-users or recent abstainers. Excessive consumption of caffeine may lead to an anxiety neurosis known as 'caffeinism'.

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

Paraxanthine is not known to be produced by plants and is only observed in nature as a metabolite of caffeine in animals and some species of bacteria. After intake, roughly 84% of caffeine is demethylated at the 3-position to yield paraxanthine, making it the primary metabolite of caffeine in humans. According to your genetic result you are predisposed to have a slower paraxanthine metabolism, which leads to higher plasma level for a longer time.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2A6, AC008537.1	rs56113850	TT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

CAFFEINE METABOLISM: PLASMA LEVELS OF CAFFEINE

RESULTS



Caffeine is produced commercially by both extraction and synthetic procedures. Extraction procedures involve three methods: direct decaffeination of green coffee beans with solvents, extraction from tea dusts and wastes and fragments of tea leaves, and extraction from cola nuts. Synthetic production of caffeine involves the methylation of various xanthines. Caffeine (1,3,7-trimethylxanthine) is a plant alkaloid. Structurally, caffeine resembles the purines. The mean half-life of caffeine in plasma of healthy individuals is about 5 hours. However, caffeine's elimination half-life may range between 1.5 and 9.5 hours, while the total plasma clearance rate for caffeine is estimated to be 0.078 L/h/kg. This wide range in the plasma mean half-life of caffeine is due to both innate individual variation, and a variety of physiological and environmental characteristics that influence caffeine metabolism (e.g., pregnancy, obesity, use of oral contraceptives, smoking, altitude). The fatal acute oral dose of caffeine in humans is estimated to be 10–14 g (150–200 mg/kg body weight [BW]). Ingestion of caffeine in doses up to 10 g has caused convulsions and vomiting with complete recovery in 6 hours. Peak plasma concentrations occur between 15 and 120 minutes after oral ingestion. This wide variation in time may be due to variation in gastric emptying time and the presence of other dietary constituents, such as fiber.

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

The caffeine molecule is structurally similar to adenine (the nitrogen base of adenosine) and binds to nucleoside receptors on cell membranes. There is therefore a competitive inhibition; that is, caffeine influences a process of regulation of the nerves by discharging the post-synaptic potential. An increase in adrenaline and norepinephrine levels results. Through these, caffeine therefore indirectly stimulates the sympathetic nervous system and leads to an increase in heart rate and blood flow to the muscles, a decrease in the flow of blood to the skin and internal organs and to the release of glucose from the liver.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

CAFFEINE METABOLISM: PLASMA LEVELS OF THEOBROMINE

RESULTS



Caffeine is produced commercially by both extraction and synthetic procedures. Extraction procedures involve three methods: direct decaffeination of green coffee beans with solvents, extraction from tea dusts and wastes and fragments of tea leaves, and extraction from cola nuts. Synthetic production of caffeine involves the methylation of various xanthines. Theobromine is the principal alkaloid (1.5-3%) of the cacao bean (*Theobroma cacao*); it is usually extracted from the husks of cacao beans. Theobromine is used principally to make caffeine. As a metabolite of caffeine, theobromine has been detected in variable amounts in plasma and urine. Theobromine is readily absorbed from food and evenly distributed in body fluids also has been reported that it is able to pass into the breastmilk of nursing mothers. The mean half-time of theobromine in human serum ranged from 6.1 to 10 h. The apparent volumes of distribution and clearance were estimated to be 0.761/kg bw and 0.88 ml/min/kg bw, respectively. It has been stated that 'in large doses' theobromine may cause nausea and anorexia and that daily intake of 50-100g cocoa (0.8-1.5 g theobromine) by humans has been associated with sweating, trembling and severe headache.

People with your genetic profile are likely to have an enhanced caffeine theobromine metabolic rate.

According to your genetic results you are predisposed to have an enhanced theobromine metabolic rate, which leads to lower plasma levels in a shorter time. High theobromine levels usually is not a problem because in men it has negligible toxicity, since it is metabolized very quickly.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
CYP2A6, AC008537.1	rs56113850	TT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

CAFFEINE METABOLISM: SLOW METABOLIZER

RESULTS



Caffeine is produced commercially by both extraction and synthetic procedures. Extraction procedures involve three methods: direct decaffeination of green coffee beans with solvents, extraction from tea dusts and wastes and fragments of tea leaves, and extraction from cola nuts. Synthetic production of caffeine involves the methylation of various xanthines. Caffeine absorption from the gastrointestinal tract is rapid, virtually complete and directly dependent on pH. After oral doses of 5–8 mg/kg bw, peak plasma concentrations of 8–10 µg/ml were observed. After oral ingestion, the time to reach peak plasma concentration exhibits wide variations, ranging from 15 to 120 min. After absorption, caffeine is rapidly and uniformly distributed into body fluids. Caffeine is eliminated by apparent first-order kinetics, described by a one-compartment open model system. Caffeine metabolism occurs primarily in the liver, catalyzed by hepatic microsomal enzyme systems. Metabolism of caffeine is affected by many factors (age, gender, hormones, liver disease, obesity, smoking, and diet). CYP1A2 isoform of cytochrome p450 mainly metabolizes it. Polymorphism at the level of this isoform explains the variability of pharmacokinetics among the individuals. Several loci have been identified and involved in caffeine consumption, and they have consequences for sleep, anxiety, and neurodegenerative/psychiatric disorders.

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

Caffeine influences a process of regulation of the nerves by discharging the post-synaptic potential. An increase in adrenaline and norepinephrine levels results. Through these, caffeine therefore indirectly stimulates the sympathetic nervous system and leads to an increase in heart rate and blood flow to the muscles, a decrease in the flow of blood to the skin and internal organs and to the release of glucose from the liver.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PERCEPTION OF SALTY TASTE

RESULTS



Sodium is an essential nutrient for the human body. It is widely used as sodium chloride (table salt) in (processed) foods and over consumed by both children and adults, placing them at risk for adverse health effects such as high blood pressure and cardiovascular diseases. Three -to- four month old infants are able to detect and prefer sodium chloride solutions over plain water, which is thought to be a biological unlearned response. Liking for water with sodium chloride mostly decreases when infants enter early childhood, but liking for sodium chloride in appropriate food contexts such as soup and snack foods remains high. The increased acceptance and preference of sodium chloride rich foods coincides with infants exposure to salty foods, and is therefore thought to be mostly a learned response. Children prefer higher salt concentrations than adults, but seem to be equally sensitive to salt taste. The addition of salt to foods increases children's consumption of those foods. However, children's liking for salt taste as such does not seem to correlate with children's consumption of salty foods. Decreasing the exposure to salty tasting foods during early infancy is recommended. Salt plays an important role in children's liking for a variety of foods. It is, however, questionable if children's liking for salt per se influences the intake of salty foods. High salt intake is a major risk factor for hypertension and is associated with cardiovascular events. Most countries exhibit a traditionally high salt intake; thus, identification of an optimal strategy for salt reduction at the population level may have a major impact on public health.

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

According to your genetic profile, your salt perception has improved. This means that you need less salt in food to have a good flavor of the dish. Reducing the amount of salt used for cooking has many health benefits. Salt reduction in foods is becoming an important challenge to protect population health from severe diseases as recommended by different health agencies worldwide. Infact, excess sodium in diet increases blood pressure because it holds excess fluid in the body, and that creates an added burden on the heart. Too much sodium will increase your risk of stroke, heart failure, osteoporosis, stomach cancer and kidney disease.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TRPV1	rs8065080	TT



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PROPENSITY TO CHOOSE SWEET FOODS

RESULTS



Sweet is one of the five basic flavors, almost universally considered a pleasant sensation. Foods rich in simple carbohydrates, such as sugars, are normally associated with sweetness. There are also some molecules of natural origin or of human invention that are perceived as sweets at much lower concentrations and can be used as low-calorie sweeteners. Finally, some substances, although not directly activating the sweet receptors, can modify their perception. The chemical sensitivity of the perception of the sweet, which varies for individuals and species, has only been understood in recent times. The currently accepted theoretical model involves multiple binding sites between the receptors and the sweet substance. Some studies indicate that sensitivity to sugars and sweets has very ancient evolutionary roots, already present as chemotaxis even in mobile bacteria such as *Escherichia coli*. Babies show preference for foods with a high concentration of sugars and, in particular, prefer sweeter solutions than lactose, the sugar found in breast milk.

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

The excess of added sugars can be very harmful to health. A diet of about 2.000kcal per day, containing 250ml of whole milk, 120g of skimmed yogurt, 400g of fresh fruit (apples and oranges), 400g of vegetables (lettuce and zucchini), free of any food with added sugars (such as jam, cakes, sweets, sweetened drinks etc.), contains about 77g of soluble sugars, equivalent to 14.4% of the total calories. The total soluble glycides are composed of: fructose, lactose and maltose (residue from cooking starch-containing foods). Adding sweetened foods at the end of meals, inserting some drinks instead of water and estimating 7g of sugar (1 sachet) for 2 coffees / teas per day, the increase is 100%, reaching more or less total 140g, i.e. 29% of the daily energy.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

SALTY TASTE PREFERENCE

RESULTS



Salty is one of the fundamental tastes perceived by the taste buds present in the human mouth. The most common ingredient to stimulate the perception of salty is the common table salt; however, the same sensation can be stimulated by any sodium salt, for example sodium acetate albeit less intensely. The salty is also stimulated by other salts not containing sodium, replaced by potassium or lithium, which however can be toxic to the human metabolism and also induce a perceptible bitter sensation.

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

On average, 10-15 grams per day of salt are consumed, or three times more than the amount recommended by the WHO (World Health Organization). This mineral is present in many foods, especially processed ones, therefore if you exceed it, we could risk suffering from various diseases in the future. The most common signs following a high percentage of sodium are the following: Sensation of thirst, Sensation of swelling, Retention of liquids, It hinders the function of the kidneys, High blood pressure. To reduce the consumption of salt, several precautions can be followed: We carefully read the nutritional label to choose, in each category, the products with lower salt content and look for products with low salt content, that is less than 0.3 grams per 100 g (corresponding to 0.12 g of sodium); We reduce the use of added salt in the kitchen, preferring, however, where necessary, minimum quantities of iodized salt; We limit the use of other condiments containing sodium (broth nuts, mayonnaise, sauces, etc.) and alternatively use spices, aromatic herbs, lemon juice or vinegar to flavor and enhance the flavor of the food; We do not bring salt or salty sauces to the table, so that we do not acquire the habit of adding salt to foods, especially among the youngest members of the family; We reduce the consumption of processed foods rich in salt (salty snacks, potato chips in the bag, some cold cuts and cheeses, canned foods); We drain and rinse canned vegetables and legumes before consuming them; We avoid adding salt to baby food, at least for the first year of life.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TRPV1	rs150908	GA



PREDISPOSITION TO EXCESSIVE CONSUMPTION OF CARBOHYDRATES

RESULTS



Carbohydrates represent more than 50% of the energy sources present in most human diets, include monosaccharides, common disaccharides, polysaccharides, and oligosaccharides. The increased consumption of free monosaccharides is one of the environmental factors that have been linked to the increased prevalence of obesity. Although carbohydrates are the only food constituents that directly increase blood glucose, population studies suggest that the total amount of carbohydrate as a percentage of dietary energy is less important than the carbohydrate type for risk of chronic disease. Clinical trials have shown that low carbohydrate diets produce greater weight loss than lower fat diets in the short term, but this difference diminishes with time because of poor long term compliance. Thus evidence suggests that the type of carbohydrates may have a greater effect on health outcomes than total amount for the general population. However, specific groups may respond differently to the carbohydrate quantity and quality. A strong case can be made for consumption of high GI grains, potato products, and added sugars (especially in drinks) being causally related to obesity, diabetes, cardiovascular disease, and some cancers; whereas non-starchy vegetables, whole fruits, legumes, and whole kernel grains appear protective. Nevertheless, the metabolic effects of total and high GI carbohydrate may vary among individuals, depending on the degree of insulin resistance, glucose intolerance, or other inherited or acquired biological predispositions.

People with your genetic profile are likely to have a predisposition for excessive consumption of carbohydrates.

Carbohydrates are usually the main source of dietary calories, yet body stores of glycogen are very limited: 500-1000 grams on average. Daily intake of carbohydrate corresponds to about 50-70% of the carbohydrate stores compared to about 1% for protein and fat, so that over a period of hours and days, the carbohydrate stores fluctuate markedly compared with those of protein and fat. However, as with protein stores, carbohydrate stores are tightly regulated. Even if all the details of this regulation remain to be established, the control is based on humoral and/or neural signals exchanged between the muscle, the liver and the brain. Dietary carbohydrate stimulates both glycogen storage and glucose oxidation and suppresses fat oxidation. Carbohydrates which are not stored as glycogen are oxidized (not converted to fat), and carbohydrate balance is achieved. Therefore, as with the other non-fat nutrients, a chronic imbalance between carbohydrate intake and oxidation cannot be the basis of weight gain because storage capacity is limited and controlled, conversion to fat is an option which only occurs under extreme conditions in humans, and oxidation is increased to match intake. However there is one exception to this rule. In situations of high acetyl-CoA (excess intake of carbohydrate in the face of excess energy intake overall), acetyl-CoA will be converted to citrate, and an accumulation of citrate will cause it to be transported out of the mitochondria into the cytosol where it is converted back to acetyl-CoA and acetylated to form malonyl-CoA – which is the first step in de novo lipogenesis. Therefore in situations of excess carbohydrate and energy intake, carbohydrate stores remain in balance and excess carbohydrates are converted to fat which can contribute to weight gain.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO EXCESSIVE CONSUMPTION OF FATS

RESULTS



Reducing dietary saturated fat has been a cornerstone of recommendations for reducing cardiovascular disease (CVD) risk for decades, largely based on the classic diet-heart hypothesis which proposes that dietary saturated fat and cholesterol play a primary role in the development of atherosclerosis and coronary heart disease (CHD). This hypothesis was informed by two key observations: 1) controlled feeding trials demonstrated that dietary saturated fatty acids and cholesterol raised serum total cholesterol and low density lipoprotein cholesterol (LDL-C) levels, and 2) increased serum total cholesterol and LDL-C predicted risk of CHD. The percentage of energy consumed as fat can vary widely, and the diet can still meet energy and nutrient needs. Intakes recommend a total fat intake between 20 and 35% of total calories. The minimum of 20% is to ensure adequate consumption of total energy, essential fatty acids, and fat-soluble vitamins. The maximum of 35% was based on limiting saturated fat and also the observation that individuals on higher fat diets consume more calories, resulting in weight gain. Saturated and monounsaturated fatty acids are synthesized in the body for energetic, physiological, and structural functions, and they are present in many foods. For example, palmitic acid, the major saturated fatty acid in the diet, is synthesized in the liver from starch and sugar via de novo lipogenesis, and it is the predominant fatty acid present in dairy and meats. Due to the positive linear relationship between total saturated fat intake and LDL-C concentrations, the recommendation is to limit saturated fat to <10% of calories.

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

There is a general consensus among all clinical specialties that the fat content of the average diet should be lowered to decrease the risk of cardiovascular morbidity and mortality. Low-fat diets are food where 30% or less of the calories come from fat. Multiple correlational studies have related a country's cardiovascular mortality to the food consumption of its population. A general rule is that if a provides 100 calories and it has 3 grams or less of fat, then it is a low-fat food. Common examples include vegetables, fruits, whole grain cereals, egg whites, chicken and turkey breast without skin, beans, lentils, peas, seafood, and low-fat dairy, among others.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
FGF21	rs838133	AG



PREDISPOSITION TO EXPERIENCE AN ALTERED SENSE OF FULLNESS

RESULTS



During the last decades, the prevalence of overweight and obesity (extreme overweight) in societies worldwide has increased dramatically. Being overweight is a serious risk factor for many diseases such as Type 2 diabetes, coronary heart diseases, hypertension, and certain forms of cancer. Enhancing the satiating capacity of foods may help people control their energy intake and weight. Reduction in food intake can be achieved through targeting two complementary processes: satiation and satiety. Satiation and satiety are components of the appetite control system and are involved in limiting energy intake. In addition, total intake may be reduced through substitution processes within the meal, whereby consumers learn to eat lower energy dense products (such as vegetables) as a substitute for foods with a higher energy density. Promising route to satiety enhancement is to make foods less energy dense. This is typically achieved by replacing fat and/or carbohydrate components by water or air. It can also be achieved by adding extra fibers or changing the structure of foods. It is now generally agreed among researchers that satiety is a complex interaction of physiological and non-physiological mechanisms. Actual food choice is the end result of a complex interaction between internal satiety signals, other food benefits, and environmental cues such as health labels, portion size, and perceived variety. Satiety enhancing product features need to be convincingly and responsibly communicated to consumers. This requires a careful selection of the types of benefits to communicate (e.g., prolonged fullness or delayed feelings of hunger), the level of detail provided to consumers. It is clear that fundamental changes in the environment of consumers are greatly needed to bring the overweight epidemic to an end. Ultimately, the goal is not only to enhance the satiety capacity of single foods, but to make the environment less "toxic" by helping consumers to control their energy intake at the shorter and longer term.

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

It has been demonstrated that children and adolescents with FTO rs9939609 obesity-risk alleles report more frequent loss of control (LOC) over eating episodes and select foods higher in fat at a buffet meal. Both LOC eating and more frequent selection of energy-dense, palatable foods may be mechanisms through which variant FTO alleles lead to excess body weight. Binge-eating episodes, defined as discrete time periods during which the consumption of a large amount of food is accompanied by a sense of loss of control (LOC) over eating, are common among overweight adults. LOC eating—defined as the experience of LOC while eating, regardless of whether the reported amount of food consumed is unambiguously large—is common in youth. We strongly recommend asking a doctor for advice.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO EAT BETWEEN MEALS

RESULTS



Different studies have shown that eating several smaller meals at frequent intervals ("grazing") is associated with better cognitive performance and mood than eating fewer and larger meals at longer intervals. Snacking is often defined as consuming a food or drink between regular meals. Satiety, the feeling of fullness that persists after eating, is an important factor in suppressing overconsumption. Identifying eating patterns and foods that promote satiety without considerably increasing overall energy intake is important for promoting more healthful eating behaviors. Eating snacks between meals can potentially promote satiety and suppress overconsumption at the next meal. Scientist suggests that the judicious selection of snack foods has the potential to contribute valuable nutrients to the daily diet. Furthermore, snack foods have the potential to contribute to satiety, with higher-protein snack foods having the strongest effect. For example, the consumption of high-protein, high-fiber snack foods can lead to reduced caloric intake at a subsequent meal when compared with high-fat, high-sugar snack foods. Consequently, thoughtful selection of snack foods may contribute to body-weight maintenance or reduction.

People with your genetic profile are likely to have the predisposition to eat between meals.

The imbalance between energy expenditure and energy intake that results in a positive energy balance is a contributing factor to the development of obesity. However, the impact of specific dietary factors has not been sufficiently examined, with the contribution of specific types of snacks subject to controversy. Some findings suggest that the relation of eating frequency with BMI z score differs from that of changes in BMI, obesity-related eating behaviors, such as the number of eating occasions, have been considered for their contribution to higher energy intake. Snack foods have the potential to contribute to satiety, with higher-protein snack foods having the strongest effect. For example, the consumption of high-protein, high-fiber snack foods can lead to reduced caloric intake at a subsequent meal when compared with high-fat, high-sugar snack foods. Consequently, thoughtful selection of snack foods may contribute to body-weight maintenance or reduction.



SCIENTIFIC DETAILS

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



HIGH VS LOW CARBOHYDRATE DIETS

RESULTS



Diets low in carbohydrate content have become a matter of international attention because of the WHO recommendations to reduce the overall consumption of sugars and rapidly digestible starches. One of the common metabolic changes assumed to take place when a person follows a low-carbohydrate diet is ketosis. It is assumed that when carbohydrate availability is reduced in short term to a significant amount, the body will be stimulated to maximize fat oxidation for energy needs. The scientific data shows that low-carbohydrate diets acutely induce a number of favourable effects, such as a rapid weight loss, decrease of fasting glucose and insulin levels, reduction of circulating triglyceride levels and improvement of blood pressure. On the other hand some less desirable immediate effects such as enhanced lean body mass loss, increased urinary calcium loss, increased plasma homocysteine levels, increased low-density lipoprotein-cholesterol have been reported. However, these undesirable effects may be counteracted with consumption of a low-carbohydrate, high-protein, low-fat diet. It is well known that fatty acid synthesis is stimulated by low fat, high carbohydrate diets in animals which are normally in positive caloric balance. A single day of high carbohydrate intake or a high carbohydrate breakfast increased the fraction of de novo VLDL palmitate, which have been associated with an increased risk of cardiovascular disease.

People with your genetic profile are likely to have a predisposition for an enhanced carbohydrate intake.

High-carbohydrate diets (> 60% of total dietary energy) that consist predominantly of high glycemic carbohydrates have detrimental metabolic effects. These diets increase serum triglyceride and insulin resistance, having the greatest adverse effect in insulin-resistant states, such as type 2 diabetes or pregnancy. As a consequence of this, the American Diabetic Association recommends limiting dietary carbohydrates to 45% in diabetic pregnancies and to 80% of total energy derived from carbohydrate and fat outside pregnancy. By contrast, the British Diabetic Association's guidelines advocate that 55% of total dietary energy should come from dietary carbohydrates.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO THE ACCUMULATION OF FAT

RESULTS



People who are obese have hormone levels that encourage the accumulation of body fat. It seems that behaviours such as overeating and lack of regular exercise, over time, 'reset' the processes that regulate appetite and body fat distribution to make the person physiologically more likely to gain weight. Hormones are one factor in causing obesity. The hormones leptin and insulin, sex hormones and growth hormone influence our appetite, metabolism (the rate at which our body burns kilojoules for energy), and body fat distribution. Excesses or deficits of hormones can lead to obesity and, on the other hand, obesity can lead to changes in hormones. Body fat distribution plays an important role in the development of obesity-related conditions such as heart disease, stroke and some forms of arthritis. It seems that oestrogens and androgens help to decide body fat distribution. The changes with age in the sex hormone levels are associated with changes in body fat distribution. While women of childbearing age tend to store fat in their lower body ('pear-shaped'), older men and postmenopausal women tend to increase storage of fat around their abdomen ('apple-shaped'). It was demonstrated that also a lack of oestrogen leads to excessive weight gain. There is evidence to suggest that long-term behaviour changes, such as healthy eating and regular exercise, can re-train the body to shed excess body fat and keep it off. Studies have also shown that weight loss as a result of healthy diet and exercise or bariatric surgery leads to improved insulin resistance, decreased inflammation and beneficial modulation of obesity hormones. Weight loss is also associated with a decreased risk of developing heart disease, stroke, type II diabetes and some cancers.

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

The management of this condition should include dietary modification, behavior interventions, medications, and surgical intervention if needed. The dietary modification should be individualized with close monitoring of regular weight loss. Low-calorie diets are recommended. Low calorie could be carbohydrate or fat restricted. A low-carbohydrate diet can produce greater weight loss in the first months compared to a low-fat diet. The patient's adherence to their diet should frequently be emphasized. Management of fat accumulation should also include prevention strategies with physical activity, exercise, nutrition, and weight maintenance.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
FTO	rs9972653	GG
FTO	rs1421085	CC
FTO	rs1558902	AA
FTO	rs9939973	AA
RNU4-17P - AC090771.1	rs663129	GG
CDK6	rs42235	CT



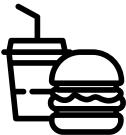
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KIT ID: TPD14630189007528

PREDISPOSITION TO FATTY FOOD ADDICTION

RESULTS



"Food addiction" is characterized by symptoms such as loss of control over consumption, continued use despite negative consequences, and an inability to cut down despite the desire to do so. Addictive-like eating has been associated with increased impulsivity and emotional reactivity. Highly processed foods, with added amounts of fat and/or refined carbohydrates (e.g., sugar, white flour), were most likely to be associated with behavioral indicators of addictive-like eating. Additionally, foods with high GL were especially related to addictive-like eating problems for individuals endorsing elevated symptoms of "food addiction." Individuals endorsing symptoms of addictive-like eating behavior may be more susceptible to the large blood sugar spike of high GL foods, which is consistent with the importance of dose and rate of absorption in the addictive potential of drugs of abuse. The majority of studies reported evidence for symptoms related to neurological changes and impaired control, with fewer studies evaluating preoccupation, chronicity, relapse, social impairment, and risky use. Behavioral and substance-related aspects of food addiction appear to be intertwined, but we suggest that the substance (highly-palatable food) component may be more salient to the diagnostic classification of this phenomenon than the behavior (eating).

People with your genetic profile are likely to have a predisposition to develop addictions for fatty food.

Heart disease and cancer, this nation's two leading killers, are linked to diets high in fat, and other chronic health problems may be exacerbated by high-fat diets. And yet our national diet contains as much as one-third more fat than it should. Eating a diet high in fat can increase the risk of developing cancer, particularly cancers of the colon and breast. Studies of cancer rates and eating habits among the different people of the world show a consistent relationship between high-fat diets and high overall cancer rates. None of these studies, though, are as conclusive as those linking high-fat diets to heart disease. Studies of the relationship of dietary fat to the development of cancer are confounded by several factors. High-fat diets tend to be low in complex carbohydrates, fiber, and fruits and vegetables—all thought to help prevent cancer. High-fat diets are also associated with higher caloric intake and obesity, factors that are suspected to encourage the development of some cancers. It has been difficult, too, to pinpoint any connections between dietary fat and specific cancers, or between specific types of fat and cancer. Studies of breast cancer, for example, tend to support a weak link between dietary fat and the risk of developing breast cancer. Some of these studies also single out saturated fatty acids, while others do not. For all these reasons we advise you to fight your desire for fatty foods and adopt a healthy lifestyle.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
DRD2	rs1800497	GG
FTO	rs9939609	AA



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KIT ID: TPD14630189007528

PREDISPOSITION TO EAT WHEN UNDER STRESS

RESULTS



Obesity is a heterogeneous construct that, despite multiple and diverse attempts, has been difficult to treat. Stress has long been considered a critical risk factor in the development of addictive disorders and relapse to addictive behaviors. However, studies have reviewed links between stress and food intake, particularly of hyperpalatable or "comfort" foods that may be consumed to reduce stress. The term "stress" refers to processes involving perception, appraisal, and response to noxious events or stimuli. Stress experiences can be emotionally or physiologically (e.g., food deprivation, illness, drug withdrawal states) challenging. Stress is a challenge to the natural homeostasis of an organism; in turn, the organism may react to stress by producing a physiological response to regain equilibrium lost by the impact of the stressor. One such homeostasis that is disrupted is that of feeding behavior. The foods eaten during times of stress typically favor those of high fat and/or sugar content. These findings suggest that stress may promote irregular eating patterns and strengthen networks towards hedonic overeating; these effects may be exacerbated in overweight and obese individuals. Chronic stress is often accompanied by anxiety, depression, anger, apathy, and alienation. Threatening and cognitively meaningful stimuli activate the emotional nervous system which, in part, determines behavioral output. Stress-induced elevations of GC secretion can intensify emotions and motivation. It is hypothesized that hyperpalatable foods may serve as "comfort food" that acts as a form of self-medication to dispel unwanted distress. In our current obesogenic environment where food is plentiful, palatable and easy accessible, the proliferation of stressors may drive non-homeostatic feeding – in other words, eating without metabolic need. Repeated bouts of minor daily stressors that keep the stress system in a chronically activated state may alter brain reward/motivation pathways involved in wanting and seeking hyperpalatable foods and induce metabolic changes that promote weight and body fat mass. Weight-related adaptations of the metabolic, neuroendocrine, and neuronal pathways can together potentiate food preference, craving and intake under conditions of stress. Individual differences in susceptibility to obesity and types of stress may further moderate this process.

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

Your results suggest that you are an emotionally balanced person. The key to a healthy diet life style is to eat the right amount of calories for how active you are so you balance the energy you consume with the energy you use. If you eat or drink more than your body needs, you'll put on weight because the energy you do not use is stored as fat. If you eat and drink too little, you'll lose weight. You should also eat a wide range of foods to make sure you're getting a balanced diet and your body is receiving all the nutrients it needs. Choose higher fibre or wholegrain varieties, such as whole wheat pasta, brown rice or potatoes with their skins on. Try to include at least 1 starchy food with each main meal. Some people think starchy foods are fattening, but gram for gram the carbohydrate they contain provides fewer than half the calories of fat. Keep an eye on the fats you add when you're cooking or serving these types of foods because that's what increases the calorie content – for example, oil on chips, butter on bread and creamy sauces on pasta.



SCIENTIFIC DETAILS

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



KIT ID: TPD14630189007528

SCIENTIFIC DETAILS

PRR5-ARHGAP8, ARHGAP8	rs726170	CC
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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP ADDICTION TO FOOD (GENERIC)

RESULTS



Food addiction (FA) term has been used as an abnormal pattern of excessive consumption of palatable and highly processed foods similar to addiction triggered by traditional addictive substances. Although it is a controversial issue, it is recommended by some authors that FA is considered as a separate phenotype of obesity. Preliminary evidence suggests that dopaminergic brain circuits commonly associated with substance dependence are also implicated in abnormal eating behaviors. There is evidence indicating the association between insulin signaling, dopamine-mediated reward circuits, and FA. It has been shown that there was a bidirectional relationship between depression and obesity also it was reported that depressive mood could change individuals' food preferences and increased consumption of palatable foods to alleviate negative feelings. Both rewarding and hedonic effects of foods may cause positive emotional reactions that play a major role in overeating. It was found that the frequency of FA was very high in obese patients who were referred to a specialized tertiary obesity center. The results of this study also indicate that FA was associated with high depression rates and lower quality of life scores, but not with the metabolic parameters except FPG (fasting plasma glucose). FA in the development of obesity, characteristics of patients with FA, and their responses to weight-loss treatments need to be investigated in large prospective studies to yield better prevention and treatment strategies.

People with your genetic profile are likely to not develop an addiction to food.

We suggest staying balanced in food intake in order to keep a good body weight and healthy lifestyle.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP FAT TISSUE OVER LEAN TISSUE

RESULTS



Body mass can be divided into two major components: body fat (energy stores) and lean mass (including muscle, organs, and bone), each of which has distinct biological significance and was likely subject to different selective pressures during human evolution. Obese individuals generally exhibit a modest increase in lean body mass (LBM) as well as excess body fat in comparison to their normal-weight peers, and among the obese, LBM tends to rise with increasing degrees of obesity. An induced change in fat content would, generally speaking, be accompanied by a change in LBM, the magnitude of the change in LBM being inversely related to the initial body fat content. Studies of subjects who have experienced dietary-induced changes in weight show that such changes do involve a large proportion of LBM in thin individuals, and a large proportion of fat in those who have larger amounts of body fat. Changes in fatness are followed by changes in the same direction of the fat-free adipose tissue and the adipose-free body. This relationship could have been predicted from the body composition of stableweight individuals with widely varying body fat content. A second influence is energy intake, for the lower the energy intake is (and hence the greater the energy deficit) in individuals undergoing diet-induced weight reduction, the greater will be the contribution of LBM to the total weight loss.

People with your genetic profile are not likely to have a predisposition to the accumulation of fat mass vs lean mass.

A key component of fitness is increasing lean body mass. Increased lean body mass offers aesthetic, metabolic and fitness benefits. Although exercise is the cornerstone for increasing lean body mass, there are five simple steps one can take to increase lean body mass faster: 1.Protein consumption helps build lean body mass. 2.Recovery is the most important part of your workout. 3.Drink a protein shake before bed. 4.Consume protein during your workout. 5.Lose fat not muscle when you diet.



SCIENTIFIC DETAILS

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



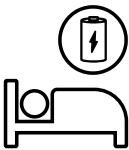
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

GENETIC PREDISPOSITION TO HIGH ENERGY EXPENDITURE AT REST

RESULTS



Adult humans maintain weight stability through a balance between energy intake and energy expenditure. When weight is stable, the energy store of the body does not fluctuate much, as evident by constancy in body weight. This weight constancy can be achieved through the balanced control of energy intake and expenditure. Under sedentary living conditions the energy balance is maintained at about 1.5 times basal metabolic rate (BMR), while during sustained exercise levels of 4.5 times BMR are reached. Disturbances of energy balance result in energy mobilization from, or energy storage in, body reserves. Energy intake occurs via macronutrients consumed in meals in the form of carbohydrate, protein, fat and alcohol. During positive energy balance, excess energy is stored as carbohydrate in glycogen, primarily in the liver, and as fat in adipose depots. Daily energy expenditure consists of four components: sleeping metabolic rate, the energy cost of arousal, the thermic effect of food (or diet induced energy expenditure (DEE)), and the energy cost of physical activity or activity-induced energy expenditure (AEE). Overnight when one sleeps quietly, food intake and physical activity are generally low or absent and energy expenditure gradually decreases to a daily minimum before increasing upon awakening. Energy expenditure varies throughout a day as a function of body size and body composition. Resting energy expenditure is defined as the metabolic rate required to maintain vital physiological functions of an individual that is in rest, awake, in a fasted state, and in a thermoneutral environment. Additionally, underfeeding causes a metabolic adaptation as reflected in a reduction of maintenance expenditure below predicted values and defined as adaptive thermogenesis. When intake exceeds energy requirements, the excess is primarily stored as body fat. As a substrate for energy metabolism, fat is less likely than carbohydrate or protein to be oxidized for fuel. Consumed fat is mostly stored before oxidation, especially in heavier people, increasing the likelihood of creating a positive energy balance. An activity-induced increase in energy requirement is typically followed by an increase in energy intake, whereas a reduction in physical activity does not result in an equivalent reduction of energy intake. Thus, preventing weight gain is more effectively reached by eating less than by moving more.

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

The average energy expenditure on various physical activities is different. Walking, watching TV and going downstairs are low-intensity physical activities; brisk walking, cycling and climbing stairs are medium-intensity physical activities. These sports activities are useful to increase your daily energy expenditure, in order to help your body lose weight.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

RISK OF GAINING FAT THROUGH THE INTAKE OF HIGH PROTEIN FOODS

RESULTS



High protein diet (HPD) resulted in short-term body weight and fat mass loss, but throughout the study preserved body lean mass and significantly reduced energy intake and expenditure as well as leptin and GLP-1 concentrations while elevating postprandial glucagon concentrations. This study suggests that long-term use of HPD may be an effective strategy to decrease energy intake and expenditure and to maintain body lean mass. Increasing protein leads to increased contribution of amino acids to energy expenditure with a concomitant decrease in lipogenesis due to decreased supply of dietary glucose and likely has a negative impact on exercise performance and training intensity. For obese subjects, lowering carbohydrate in favor of protein might be advantageous as dietary CHOs (cholesterols) might impair fat oxidation whereas low-CHO, high-protein diets reduce adipose tissue development. Higher daily protein intake at the expense of fat intake could substantially reduce total energy intake, which could possibly translate to a healthier weight status. The mechanisms by which increased long-term dietary protein intake regulate body weight are not well understood but are most likely multifactorial.

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

Protein intake is usually about 15% of calories and the protein stores in the body represent about one-third of the total stored calories in a 70 kg man. The daily protein intake amounts to a little over 1% of the total protein stores. Protein consumption must be balanced in the diet. Considering protein as the only food source thinking of losing weight is a mistake often made in DIY diets.



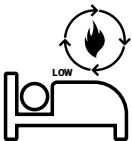
SCIENTIFIC DETAILS

Gene	rsID	Genotype
RMI2, PRM1	rs737008	GG



LOW RESTING METABOLIC RATE

RESULTS



Humans are endotherms, animals that keep their body temperature within a stable range using heat production and heat dissipation. The ability to produce heat from calories is an essential mechanism all endotherms need to survive. The molecules in food contain energy, or calories, and metabolic reactions which occur in human body can extract energy from these chemical bonds and use it to power other metabolic reactions that maintain the body's homeostasis. Metabolic rate is the amount of energy expended over a period. The rate is measured in joules, cal, or Kcal per unit time. Adult human females and males have an average BMR of 1300-1500 kcal/day and 1600-1800 kcal/day, respectively. If the heat produced by these reactions is exceeded by total body heat loss, the body is in a state of hypothermia. To counteract hypothermia, the hypothalamus can increase the body's overall metabolic rate generating more heat. A stable body temperature requires sufficient intake of calories and proper metabolic response to outside stimuli. If either of these requirements is not met, the body will be unable to maintain homeostasis. Sufficient intake of calories involves the body absorbing nutrients, such as glucose, and transforming them into useable end products, ATP. This energy fuels the metabolic processes that maintain a stable temperature. A stable balance is required for the body to remain in homeostasis.

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

Metabolism is partly genetic and largely outside of one's control. Changing it is a matter of considerable debate. Some people are just lucky. They inherited genes that promote a faster metabolism and can eat more than others without gaining weight. Others are not so lucky and end up with a slow metabolism. One way to think about metabolism is to view your body as a car engine that is always running. When you're sitting still or sleeping, your engine is idling like a car at a stop light. A certain amount of energy is being burned just to keep the engine running. Of course, for humans, the fuel source is not gasoline. It's the calories found in foods we eat and beverages we drink — energy that may be used right away or stored (especially in the form of fat) for use later. How fast your body's "engine" runs on average, over time, determines how many calories you burn. If your metabolism is "high" (or fast), you will burn more calories at rest and during activity. A high metabolism means you'll need to take in more calories to maintain your weight. That's one reason why some people can eat more than others without gaining weight. A person with a "low" (or slow) metabolism will burn fewer calories at rest and during activity and therefore has to eat less to avoid becoming overweight.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SULT2B1, AC008403.3	rs10401347	GG
AC092546.1 - AC006296.3	rs4698250	TT



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KIT ID: TPD14630189007528

PREDISPOSITION TO RESPOND POSITIVELY TO A LOW-FAT DIET

RESULTS



There is a general consensus among all clinical specialties that the fat content of the average diet should be lowered to decrease the risk of cardiovascular morbidity and mortality. Fats are essential to us, but we need to consume them in a limited amount. There is abundant literature to suggest that a decrease or modification of serum cholesterol is a possible way to prevent atherosclerosis. Decreasing the amount of fat intake is an effective means of lowering the serum cholesterol concentration. Hence, a low-fat diet has been widely advocated by clinicians for reducing the cardiovascular-related morbidity and mortality of their patients. Current National Cholesterol Education Program (NCEP) guidelines for adults based on ATP III (Adult Treatment Panel III) recommends reducing intake of saturated fats to less than 7 % of the total calories and cholesterol to less than 200 mg/day. Guidelines also recommend that polyunsaturated fat constitute up to 10% of total calories, and monounsaturated fats constitute up to 20% of total calories. People should receive advice to follow a dietary pattern with emphasizes increased intake of vegetables, fruits, whole grains, low-fat dairy, poultry, fish, legumes, nontropical vegetables and oils, and limits consumption of sweets, sugar-sweetened beverages, and red meats. Adults should also engage in aerobic physical activity to reduce LDL cholesterol and non-HDL cholesterol to counter the obesity epidemic and its various co-morbidities. The team of primary care providers, nurse practitioners, dietitians, and internists must be aware of the perils of high-fat content in the diet and form a multidisciplinary approach to manage the patient.

People with your genetic profile are likely to have a predisposition to respond positively to a low-fat diet.

There is abundant literature to suggest that a decrease or modification of serum cholesterol is a possible way to prevent atherosclerosis. Decreasing the amount of fat intake is an effective means of lowering the serum cholesterol concentration. Hence, a low-fat diet has been widely advocated by clinicians for reducing the cardiovascular-related morbidity and mortality of their patients. Anyway, there have also been multiple issues of concern and controversies around the concept of a low-fat diet. The biggest concern with the promotion of the low-fat diet has been that manufacturing companies are touting products labeled as low-fat products, where they are replacing the fat with large amounts of refined carbohydrates, which increase the risk of metabolic disorders and hypertriglyceridemia. Studies are also reporting that diets rich in carbohydrates, and low in unsaturated fat, can also negatively impact lipoprotein risk factors, and increase the cardiovascular risks. There is also a proposed theory that the refined carbohydrates decrease the cardioprotective action of HDL by altering its metabolic functions. There has undoubtedly been a focus on replacing the carbohydrates for fats, but the specificities of the replaced carbohydrates remain poorly defined. These concerns have led to the development of alternative dietary approaches.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADIPOQ	rs17300539	GA



PREDISPOSITION TO LIPOPROTEIN DEFICIENCY

RESULTS



Lipoproteins are lipid transport molecules that transport plasma lipids. Specific lipoproteins are risk factors for cardiovascular disease and other metabolic diseases. Understanding lipoproteins and the different ways in which to manipulate their metabolism is an essential step towards preventing disease and morbidity in the general population. In this review, we will highlight the cellular and molecular function of lipoprotein metabolism, how it is useful in diagnostic testing, its role in disease pathology, and its clinical significance. Lipoproteins are complex molecules that involve several different components. They contain a central core made of triglycerides and cholesterol esters. Fatty acids that are cleaved from triglycerides can be used for energy storage or production, and cholesterol is critical for steroid synthesis, cellular membrane formation, and bile acids. Surrounding this core is a mix of phospholipids, free cholesterol, and apolipoproteins (apo). The apolipoproteins are particularly important, as they play a role in classifying the lipoprotein into one of five main classes: chylomicrons, very low-density lipoprotein, (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) provide structure and function in lipid metabolism. The differentiation between these classes depends on the size of the molecule, its lipid content, and the type of apolipoprotein it features. HDL, colloquially known as "good cholesterol," participates in reverse cholesterol transport, while LDL, colloquially known as "bad cholesterol," promotes atherosclerosis. An impairment in lipoprotein metabolism could lead to catastrophic implications in an affected individual. A pathologic increase in LDL, for example, is a known risk factor in cardiovascular disease as it leads to premature atherosclerotic changes of vessels. Disorders of lipoproteins have both genetic and environmental underpinnings. In Western countries specifically, lifestyle has been identified as an insidious precipitant of these disorders. Studies, campaigns, and public health interventions are underway to encourage positive lifestyle changes in an effort to prevent lipoprotein disorders from becoming prevalent. Lipids are one of the four main biological molecules of the human body, along with carbohydrates, proteins, and nucleic acids. Lipids are essential components of life on a cellular level, as they are involved in multiple processes such as storing energy, serving as chemical messengers, and forming cell membranes and transporting fat-soluble vitamins such as Vitamin E. For lipids to carry out these roles in the cell, however, they must travel to their destination 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 lipids like cholesterol. Therefore, lipoproteins play an integral role in the ability of the human body to utilize lipids, and the metabolism of these lipoproteins has a direct effect on the level of lipids in the serum and on the subsequent processes that involve lipids within the cell. LDL-cholesterol is a leading cause of atherosclerotic cardiovascular disease (ASCVD); this involves many processes that ultimately result in ASCVD. LDL accumulates in circulating macrophages, following modifications such as oxidation of LDL. The oxidized LDL acts as a chemoattractant for monocytes, which become macrophages. These macrophages become trapped in the vessel wall, likely due to abnormal endothelial leukocyte adhesion molecule-1, vascular cell adhesion molecule-1, and intercellular adhesion molecule-1. The LDL particle promotes an inflammatory response, leading to increased cytokines and antibodies. The modified LDL in macrophages, now called foam cells, have the potential to rupture, which can release oxidized LDL, enzymes, and free radicals, which can all further destroy the vessel wall. Additionally, the modified LDL prevents the release of nitric oxide through dysfunctional endothelial function. This process reduces the vessels ability to vasodilate appropriately. Lastly, oxidized LDL causes increased platelet aggregation and thromboxane release. The net result is that platelet activity and aggregation becomes enhanced.

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

An impairment in lipoprotein metabolism could lead to catastrophic implications in an affected individual. Disorders of lipoproteins have both genetic and environmental underpinnings. Screening for dyslipidemia is through a routine test performed during health maintenance visits, most often by primary care physicians. Routine testing generally includes total cholesterol, triglycerides, LDL-C, HDL-C, with desirable values for adults being below 200 mg/dL, under 150 mg/dL, less than 100 mg/dL, greater than 60 mg/dL, respectively. Plasma or serum measurements of lipoprotein levels are useful. Plasma is typically collected in ethylene diamine-tetra acetic acid (EDTA), so it can be rapidly cooled and prevent lipid peroxidation and inhibit bacterial enzymes. Values from plasma for cholesterol and triglycerides are about 3% lower than in the serum. At least two lipid assessments, ideally two weeks apart, are performed before a diagnosis of dyslipidemia is made.



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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



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POSITIVE RESPONSE TO A DIET HIGH IN MONOUNSATURATED FATS

RESULTS



Monounsaturated fat is a type of dietary fat. Monounsaturated fats are found in plant foods, such as nuts, avocados, and vegetable oils. Eating moderate amounts of monounsaturated (and polyunsaturated) fats in place of saturated and trans fats can benefit your health. They can help lower your LDL (bad) cholesterol level. Cholesterol is a soft, waxy substance that can cause clogged, or blocked, arteries (blood vessels). Keeping your LDL level low reduces your risk for heart disease and stroke. The Dietary Guidelines for Americans recommends consuming less than 10% of your calories per day from saturated fat by replacing saturated fat with monounsaturated and polyunsaturated fats. Although monounsaturated and polyunsaturated fats can have a beneficial effect on your health, they are still a concentrated source of calories. Therefore, they should be eaten in place of saturated fat (rather than added to the diet) while staying within recommended limits for calories and total dietary fat. Use the Nutrition Facts Label as your tool for replacing saturated fat with monounsaturated and polyunsaturated fats. The Nutrition Facts Label on food and beverage packages shows the amount in grams (g) and the Percent Daily Value (%DV) of total fat and saturated fat in one serving of the food.

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

Like all dietary fats, monounsaturated fats provide calories and help the body absorb certain vitamins, cushion and insulate the body, and support many body processes. They contribute vitamin E to the diet. These fats are considered essential because they are required for normal body functioning, but they cannot be made by the body and must be obtained from food. Essential fats play a role in many body processes, including immune and nervous system function, blood clotting, and blood pressure regulation. Monounsaturated fats can lower the levels of total cholesterol and low-density lipoprotein (LDL or "bad") cholesterol in the blood — which, in turn, can reduce the risk of developing cardiovascular disease. The Dietary Guidelines for Americans recommends consuming less than 10% of your calories per day from saturated fat by replacing saturated fat with monounsaturated fats. Although monounsaturated fats can have a beneficial effect on your health, they are still a concentrated source of calories. Therefore, they should be eaten in place of saturated fat (rather than added to the diet) while staying within recommended limits for calories and total dietary fat.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
ADIPOQ	rs17300539	GA



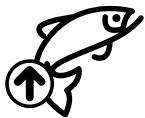
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

POSITIVE RESPONSE TO A DIET HIGH IN POLYUNSATURATED FATS

RESULTS



Fats and oils are esters of glycerol and three fatty acids. They are important in the diet as energy sources and as sources of essential fatty acids and fat-soluble vitamins, which tend to associate with fats. They also contribute satiety, flavor, and palatability to the diet. Polyunsaturated fatty acids (PUFAs), such as linoleic acid is classified as an essential nutrient, since the body requires it but cannot synthesize it. Arachidonic acid is also required by the body but can be synthesized from linoleic acid, which is abundant in oils from corn, soybeans, and safflower seeds. These fatty acids appear to play distinctive roles in the structure and function of biologic membranes in the retina and central nervous system. Whereas most epidemiological and clinical trial data support the replacement of SFAs with polyunsaturated fatty acids (PUFAs), particularly omega-3 (ω -3) fatty acids, for cardiovascular benefit, replacement of SFAs with dietary carbohydrates (CHOs) has been associated with no improvement or even a worsening of Cardiovascular Disease risk. This may be attributable at least in part to adverse effects on atherogenic dyslipidemia, a common trait characterized by elevated triglyceride (TG), reduced HDL cholesterol (HDL-C), and increased concentrations of small, dense LDL (sdLDL) particles.

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

Polyunsaturated fats are found in a variety of foods, including: Fish (such as herring, mackerel, salmon, trout, and tuna), Mayonnaise and oil-based salad dressings, Nuts (such as pine nuts and walnuts), Seeds (such as flax, pumpkin, sesame, and sunflower seeds), Soft margarine (liquid, spray, and tub), Vegetable oils (such as corn, cottonseed, soybean, and sunflower oils).



SCIENTIFIC DETAILS

Gene	rsID	Genotype
FADS2, FADS1	rs174547	TC



NEGATIVE RESPONSE TO A DIET HIGH IN UNSATURATED FATS

RESULTS



Lipids are compounds that are insoluble in water but are soluble in organic solvents such as ether and chloroform. Lipids that are important to our discussion include fats and oils (triglycerides or triacylglycerols), fatty acids, phospholipids, and cholesterol. Fats and oils are esters of glycerol and three fatty acids. They are important in the diet as energy sources and as sources of essential fatty acids and fat-soluble vitamins, which tend to associate with fats. They also contribute satiety, flavor, and palatability to the diet. Fatty acids generally consist of a straight alkyl chain, terminating with a carboxyl group. The number of carbons in the chain varies, and the compound may be saturated (containing no double bonds) or unsaturated (containing one or more double bonds). Unsaturated fatty acids form geometric isomers, i.e., the carbon chains are on the same side of the double bond in a cis isomer and on opposite sides of the bond in a trans isomer. Naturally occurring geometric isomers in food are mainly cis isomers, but hydrogenation of oils in the manufacture of margarine and shortening results in formation of some trans isomers. This latter process occurs naturally in the rumen of ruminants. The relationship between plasma triglyceride levels and cardiovascular diseases is somewhat controversial and unclear. In most within-population studies, plasma triglyceride levels were positively associated with increased risk for cardiovascular diseases but they were not independently predictive for CHD after statistical adjustment for closely associated -attributes such as HDL-C, hypertension, cigarette smoking, and obesity. However, the plasma triglyceride level was found to be an independent predictor of CHD in women. Even so, the plasma total triglyceride level, rather than being a direct cause of atherosclerotic disease, probably reflects the presence of certain atherogenic lipoproteins. A number of genetic disorders of lipoprotein metabolism have been identified and characterized. The study of these disorders has provided many insights into the structure, metabolism, and regulation of the plasma lipoproteins and apolipoproteins. Several of these disorders are characterized by severe hypercholesterolemia and premature atherosclerosis and CHD. Such disorders include familial hypercholesterolemia (FH), familial combined hyperlipidemia, and familial disbeta lipoproteinemia (type 3 hyperlipoproteinemia).

People with your genetic profile are likely to have a predisposition to respond negatively to a diet enriched in unsaturated fats.

In studies, it has been shown that consuming unsaturated fats instead of trans fats and saturated fats lowers the risk of vascular disease, heart disease, and stroke. The mechanisms by which unsaturated fats affect lipids is not fully known, but studies have shown that they can modestly lower your LDL cholesterol and increase your HDL cholesterol levels. Some polyunsaturated fats (PUFAs), such as omega-3 fatty acids, can also help lower your triglyceride levels. Negative effects may occur when there is a strong adverse metabolic effect to fats in general. In this case it is recommended to ask a nutritionist to follow your case.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

NEGATIVE RESPONSE TO A DIET HIGH IN TRANS-FATS

RESULTS



RESULTS



Fats are essential to us, but we need to consume them in a limited amount. The main four types of dietary fats include polyunsaturated, monounsaturated, trans, and saturated fats. These four varieties of fats differ in their physical and chemical structures. The saturated and the trans fats are considered solid at room temperature, whereas the mono and polyunsaturated fats are liquid at room temperature. Regardless of their physical and chemical properties, these all different forms of fat provide nine calories for every gram consumed, which is much higher than the amount of energy supplied per gram of carbohydrates or proteins. The saturated and the trans fats raise the low-density lipoproteins (LDL) and are considered unhealthy, whereas the monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) which lower LDL are considered beneficial. There is a general consensus among all clinical specialties that the fat content of the average diet should be lowered to decrease the risk of cardiovascular morbidity and mortality. Low-fat diets are food where 30% or less of the calories come from fat. Multiple correlational studies have related a country's cardiovascular mortality to the food consumption of its population. A general rule is that if a provides 100 calories and it has 3 grams or less of fat, then it is a low-fat food. Common examples include vegetables, fruits, whole grain cereals, egg whites, chicken and turkey breast without skin, beans, lentils, peas, seafood, and low-fat dairy, among others. There has been a direct relationship between dietary fat intake and cardiovascular disease (CVD). Besides, dietary cholesterol has been a focus of considerable attention due to a direct connection between diet and blood cholesterol levels and the subsequent risk for coronary artery disease. The level of LDL particles is the best predictor of cardiovascular risk. Studies have concluded that saturated fatty acids raised blood cholesterol levels, whereas PUFA's reduced serum cholesterol levels and MUFA's were neutral. Studies have also found myristic and palmitic acid to have cholesterol elevating effects, whereas stearic acid did not affect the levels. Trans fatty acids are similar to saturated fatty acids in raising cholesterol, as well. The level of saturated fats, trans-fatty acids should be low, and the levels of polyunsaturated fatty acids should be high. The results from the Nurses' Health Study, in which the women who consumed diets low in saturated and trans fatty acids and relatively high in unhydrogenated mono- and polyunsaturated fatty acids had the least risk for cardiovascular outcomes. The association between dietary fat and the risk of cancer development has had consistent support through multiple studies. There is epidemiologic evidence demonstrating associations between dietary fat intake and breast, prostate, colon, and even lung cancers in humans. Of those cancers, dietary fat intake has been the most extensively linked with breast cancer. Various mechanisms have been suggested, including conversion of essential fatty acids to short-lived hormone-like lipids, the production of reactive oxygen species which carry the potential to induce changes in the genomic DNA changes, leading to alterations in gene expression. Other potential mechanisms include modifications in the hypothalamus-pituitary axis leading to alterations in hormone levels, the effect on enzyme functions affecting the estrogen, changes in the structure and functioning of the cells as well as changes in immune function. Studies have also suggested a positive effect of polyunsaturated fatty acid, especially the omega -3 fatty acids to have a protective effect against the development of the cancers and high animal fat to have the strongest positive correlation for the development of these cancers. Obesity is a chronic disease that is associated with a plethora of comorbidities like diabetes mellitus, dyslipidemia, hypertension, fatty liver, obstructive sleep apnea, to name a few. It has multiple external and internal influences. Among the many environmental impacts, dietary fat intake is thought to have the strongest association. Energy imbalances result from excessive nutritional intakes along with low levels of physical activity. If we use BMI as the criterion to define obesity, more than one-third of adults in the United States gets categorized as overweight or obese.

People with your genetic profile are likely to have a predisposition to develop adverse effects on the intake of trans fatty acids.

Trans fats raise your bad (LDL) cholesterol levels and lower your good (HDL) cholesterol levels. Eating trans fats increases your risk of developing heart disease and stroke. It's also associated with a higher risk of developing type 2 diabetes. Eat a dietary pattern that emphasizes fruits, vegetables, whole grains, low-fat dairy products, poultry, fish and nuts. Also limit red meat and sugary foods and beverages. Use naturally occurring, unhydrogenated vegetable oils such as canola, safflower, sunflower or olive oil most often. Look for processed foods made with unhydrogenated oil rather than partially hydrogenated or hydrogenated vegetable oils or saturated fat. Use soft margarine as a substitute for butter, and choose soft margarines (liquid or tub varieties) over harder stick forms. Look for "0 g trans fat" on the Nutrition Facts label and no hydrogenated oils in the ingredients list. Doughnuts, cookies, crackers, muffins, pies and cakes are examples of foods that may contain trans fat. Limit how frequently you eat them. Limit commercially fried foods and baked goods made with shortening or partially hydrogenated vegetable oils. Not only are these foods very high in fat, but that fat is also likely to be trans fat.



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KIT ID: TPD14630189007528



SCIENTIFIC DETAILS

Gene	rsID	Genotype
PLA2G2A	rs4654990	GG



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KIT ID: TPD14630189007528

PREDISPOSITION TO FEEL FULL WITH PROTEIN INTAKE

RESULTS



A protein consists of amino acids (AA) linked by peptide bonds. Dietary protein is hydrolyzed by proteases and peptidases to generate AA, dipeptides, and tripeptides in the lumen of the gastrointestinal tract. These digestion products are utilized by bacteria in the small intestine or absorbed into enterocytes. AA that are not degraded by the small intestine enter the portal vein for protein synthesis in skeletal muscle and other tissues. AA are also used for cell-specific production of low-molecular-weight metabolites with enormous physiological importance. Thus, protein undernutrition results in stunting, anemia, physical weakness, edema, vascular dysfunction, and impaired immunity. The Recommended Dietary Allowance of protein for a healthy adult with minimal physical activity is currently 0.8 g protein per kg body weight (BW) per day. To meet the functional needs such as promoting skeletal-muscle protein accretion and physical strength, dietary intake of 1.0, 1.3, and 1.6 g protein per kg BW per day is recommended for individuals with minimal, moderate, and intense physical activity, respectively. Long-term consumption of protein at 2 g per kg BW per day is safe for healthy adults, and the tolerable upper limit is 3.5 g per kg BW per day for well-adapted subjects. Chronic high protein intake (>2 g per kg BW per day for adults) may result in digestive, renal, and vascular abnormalities and should be avoided. The quantity and quality of protein are the determinants of its nutritional values. Therefore, adequate consumption of high-quality proteins from animal products (e.g., lean meat and milk) is essential for optimal growth, development, and health of humans.

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

Proteins are essential macronutrients that contribute to structural and mechanical function, regulate processes in the cells and body, and provide energy if necessary. Proteins are composed of amino acids and in food sources like meats, dairy foods, legumes, vegetables, and grains. 1 gram of protein contains 4 kcal of energy. The recommended protein intake is 0.8 to 1 gram per kilogram of body weight per day. For healthy children ages 1 to 3, ages 4 to 18, and adults, approximately 5 to 20%, 10 to 30%, and 10 to 35% of daily energy intake should come from protein, respectively, based on the adequate amount for nitrogen equilibrium. The protein stores increase in size in response to growth stimuli such as growth hormone, androgens, physical training, and weight gain, but do not simply increase from increased intake of dietary protein. Protein stores are, therefore, tightly controlled and on a day-to-day basis protein balance is achieved. Protein imbalance is therefore, not implicated as a direct cause of obesity although, as with the other non-fat nutrients, protein intake may affect fat balance.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
FTO	rs1421085	CC



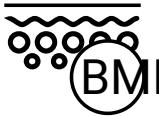
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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

RATIO OF VISCERAL VS. SUBCUTANEOUS ADIPOSE TISSUE

RESULTS



Body mass index (BMI) is a quotient that is used to identify adults and adolescents that have an abnormal weight in proportion to their height. It is the calculation of weight divided by height, and it is universally expressed in kg/m². It is important for a clinician to understand BMI due to the extensive research that is being done correlating BMI to various disease pathophysiology, and because of its use as stratification measure in many clinical treatment guidelines. Body mass index has been a useful tool due to its universal acceptance as a categorizing factor of body fatness. BMI is considered to be an indication of the relative amount of body fat on an individual's frame. Since it does not measure adipose tissue, it has the potential for inaccuracy. People with significant lean body mass, for example, could be classified as "overweight" while they would likely have a low body fat percentage. You will see this in bodybuilders and other athletes. In these cases, other anthropometric measurements may offer more clinical relevance. Another caveat is that the physical distribution of adipose tissue has been shown in many studies to affect morbidity and mortality. BMI has no way to account for this variable. In the calculation, height is squared to reduce the contribution of leg length in taller people. This was done because the majority of mass remains within the trunk. However, with this normalization, the equation distributes equal mass to each level of height. This does subtract from the utility of BMI in studies that differentiate body types. Even with these weaknesses, BMI is an excellent tool that is easy to work with, and useful in most patient populations.

People with your genetic profile are likely to have an unbalance between visceral adipose tissue/subcutaneous adipose tissue.

Body mass index is a calculation. The mechanism of this measurement is done by obtaining an individual's weight in kilograms and dividing that number by their height in meters squared. The resulting unit is kg/m². When individuals are identified as an abnormal weight, certain testing should be done. For people with a BMI greater than 30 kg/m², a lipid panel, thyroid level, and diabetes screening should always be done. These patients should be counseled about a healthy diet and exercise. For people with a BMI less than 18 kg/m², thyroid level, comprehensive metabolic panel, psychiatric screening for an eating disorder, and conditions of malabsorption should be assessed. These tests are considered standards of care in the American Diabetes Association Diabetes Care annual update. If their weight loss was rapid and unintentional, a cancer workup should also be done. Studies looking at the heredity of BMI showed a strong relationship. This led to further evaluation with large population genetic screening tools, to isolate genes that are associated with BMI and with obesity specifically. Abnormal BMI, as a majority of other chronic conditions, is the result of environmental and genetic factors working hand-in-hand. Further research is being done to identify more genes associated with abnormal BMI.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
ROBO1	rs17377726	GG
LGALS12	rs933186	TT
AL139390.1 - RREB1	rs2842895	GG
AC116362.1	rs10060123	CC



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KIT ID: TPD14630189007528

PRUDENT DIETARY PATTERN

RESULTS



Two dietary patterns were identified. The Prudent dietary pattern was characterised by high intakes of vegetables, fruits, whole grain products and low intakes of refined grain products and the Western dietary pattern, by high intakes of refined grain products, desserts, sweets and processed meats. The dietary patterns mainly composed of fruits and vegetables, natural juices, potato/cassava/cooked cornmeal, fish, and chicken, which was negatively associated with the Hcy level in this population. These findings support the role of a healthy dietary pattern in health outcomes, rather than promoting specific foods or nutrients, for policy-based health promotion strategies. Gene expression profiles were different according to dietary patterns, which probably modulate the risk of chronic diseases. Understanding why we eat and the motivational factors driving food choices is important to addressing the epidemics of obesity, diabetes and cardiovascular disease. Eating behavior is a complex interplay of physiologic, psychological, social, and genetic factors that influence meal timing, quantity of food intake, and food preference. Here we review the current and emerging knowledge of the genetic influences of eating behavior and how these relate to obesity with particular emphasis on the genetics of taste, meal size and selection, and the emerging use of functional magnetic resonance imaging to study neural reactions in response to food stimuli in normal, overweight and obese individuals.

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

Eating well is a lifestyle that pays off in terms of longevity and physical and mental well-being.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC00466	rs76500500	TT
AL137026.3	rs76838052	CC



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KIT ID: TPD14630189007528

GENETIC SENSITIVITY TO GLUTEN

RESULTS



Celiac disease (CD) is an immune-mediated disorder triggered in genetically susceptible individuals by ingestion of foods containing gluten, a family of proteins found in wheat, rye, barley, and related grains. Susceptibility to CD is linked to certain HLA class II alleles, especially in the HLA-DQ region. Approximately 95 percent of patients with CD have the HLA-DQ2 heterodimer, while the remaining 5 percent have the HLA-DQ8 heterodimer. Lack of these heterodimers all but rules out CD and genetic susceptibility for the disorder. These genetic tests are part of the diagnostic algorithms recommended by the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN).

People with your genetic profile are not likely to have gluten sensitivity.

Gluten (from Latin gluten, meaning glue) is a composite of storage proteins termed prolamins and glutelins that are stored together with starch in various cereal (grass) grains. It is found in wheat, barley, rye, oat, related species, and hybrids (such as spelled, Khorasan, emmer, among others) and the products of these such as malt. Gluten gives elasticity to dough, allowing for the puffy and chewy texture. About 80% of the protein in bread wheat is gluten. Pasta has a lower degree of gluten. Imitation meats, beer, soy sauce, and occasionally, ice cream and ketchup have gluten from the included stabilizing agents. Contamination of other food products with gluten is also a common problem. Hair products and cosmetics sometimes contain gluten, as well.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
RBFOX1	rs59325236	GG



PREDISPOSITION TO LACTOSE INTOLERANCE

RESULTS



Lactose intolerance is a clinical syndrome that manifests with characteristic signs and symptoms upon the consumption of food substances containing lactose, a disaccharide. Normally upon the consumption of lactose, it is hydrolyzed into glucose and galactose by lactase enzyme, which is found in the small intestinal brush border. Deficiency of lactase due to primary or secondary causes results in clinical symptoms. Disease severity varies among individuals. Lactose is present in dairy, milk products, and mammalian milk. It is also sometimes referred to as lactose malabsorption. Lactase enzyme deficiency can occur in individuals, and they have lower levels of this enzyme which results in failure to hydrolyze lactose into absorbable glucose and galactose components. There is a decrease or absence of lactase enzyme activity since birth due to autosomal recessive inheritance. It manifests in the newborn after ingestion of milk. It is a rare cause of the deficiency. The prevalence of lactose intolerance is variable among different ethnicities. It is most common in African Americans, Hispanics/Latinos, and Asians, while least prevalent in people of European descent. Ethnic groups with a higher prevalence of lactose intolerance also are more likely to have lactose non-persistence.

People with your genetic profile are likely to have predisposition for lactose intolerance .

Management of lactose intolerance consists of dietary modification, lactase supplementation and the treatment of an underlying condition in people with secondary lactase deficiency. Lactase containing milk products and calcium supplements are recommended. Limiting dietary intake of lactose by avoiding intake of lactose-containing products improves the symptoms of the disease. Following products contain lactose and therefore must be avoided: Soft and processed cheese, Buttermilk, Cream, Milk, Ice cream, Sour cream, Whey, Yogurt, Pancakes and waffles, Mashed potatoes, Butter, Margarine, Custard and pudding. It is also possible to introduce lactase enzyme supplements in your diet. Lactase enzyme supplements contain lactase which breaks down lactose in milk and milk containing products. There are available solutions such as lactase enzyme tablets or drops. Lactose intolerance has an excellent prognosis. Most patients have a considerable improvement in signs and symptoms with dietary modification alone.



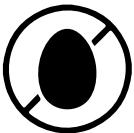
SCIENTIFIC DETAILS

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



PREDISPOSITION TO DEVELOP AN EGG ALLERGY

RESULTS



The term "egg allergy" refers to an adverse immunological response to exposure to allergens found in egg white or egg yolk. Egg allergy is IgE mediated, and so it is classified (like all food allergies) as a type one hypersensitivity reaction. The process of binding of IgE to human mast cells and basophils is termed sensitization. The process of sensitization prepares mast cells and basophils for antigen-specific activation. In the activation phase, reexposure to egg protein allergens initiates degranulation of mast cells and basophils with subsequent release of pharmacologically active mediators, such as heparin, histamine, leukocyte chemotactic factors, and leukotrienes; these mediators are responsible for the clinical manifestations of egg. It is believed that there may be a genetic component to the development of egg allergy as the progeny of atopic individuals are more likely to suffer from allergies themselves. The consensus is that IgE responses are genetically controlled by MHC-linked genes that are found on chromosome six. Other components that may be associated with atopy and allergy include the IgE Fc receptor located on chromosome eleven. Avoidance of egg exposure is the most effective form of egg allergy management but is not equivalent to cure and may not always be feasible. Unfortunately, efforts of avoiding exposure can pose a significant psychosocial stressor on both the child and the parents. Also, avoidance of eggs (or any food allergen) can place children at higher risk for nutritional deficiencies. Oral immunotherapy (OIT) involves oral administration of allergenic egg white with an edible vehicle in gradually increasing dosages. OIT has demonstrated the success of desensitization in patients with egg allergy. However, it remains relatively time-consuming and often requires long term maintenance therapy.

People with your genetic profile are not likely to have a predisposition to develop an egg allergy.

Eggs are among the most nutritious foods on the planet. In fact, a whole egg contains all the nutrients needed to turn a single cell into an entire chicken. However, eggs have gotten a bad reputation because the yolks are high in cholesterol. Your liver produces large amounts of cholesterol. When you eat cholesterol-rich foods such as eggs, your liver compensates by producing less. Nevertheless, you should still avoid eating excessive amounts of cholesterol if your blood levels are raised. A high intake may cause a moderate increase in blood cholesterol levels.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
SERPINB7 - SERPINB2	rs1243064	TT
BMPR1B	rs17023017	TT



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PREDISPOSITION TO DEVELOP A MILK ALLERGY

RESULTS



Milk allergy is a common diagnosis in infants and children. It presents as an allergic reaction to the protein found in milk. Cow's milk allergy commonly develop in infants and can regress by the age of 6. It can be a source of parental and family stress due to a milk-free diet and can lead to a subsequent nutritional deficiency if not treated appropriately. Cow's milk allergy reactions classify into rapid onset; usually, IgE mediated, where symptoms occur within an hour after ingestion, and slow onset, non-IgE mediated, where symptoms take hours or days to present. The clinician must recognize the difference between milk allergy and milk intolerance. The major difference is that intolerance does not involve the immune system. Common symptoms of milk intolerance include gas, bloating, or diarrhea after ingesting milk. The treatment of intolerances and allergies are different. The definitive treatment for all food allergies is the strict elimination of the food from the diet. If a child starts on a milk-free diet, the doctor or dietitian can help plan nutritionally balanced meals. The parent or child may need to take supplements to replace calcium and nutrients found in milk, such as vitamin D and riboflavin. The prognosis for cow's milk protein allergy in infancy and young childhood is good. Approximately 50% of affected children develop tolerance by the age of 1 year, more than 75% by the age of 3 years, and over 90% are tolerant at 6 years of age. Cow's milk allergy requires an interprofessional team approach, including physicians, specialists (most notably an allergist), specialty-trained nurses, and pharmacists, all collaborating across disciplines to achieve optimal patient results. The family may be educated by the pediatric nurse, who provides updates to the rest of the team. Pharmacists may be involved in formula selection and assist in medication review.

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

You may not need to avoid foods and beverages that contain lactose—such as milk or milk products. If you avoid all milk and milk products, you may get less calcium and vitamin D than you need.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC01909	rs17236768	TT



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A PEANUT ALLERGY

RESULTS



Peanut allergy is one of the most common allergies in children and although the allergy will improve with time for some, for others it will become worse. People who are allergic to peanuts will not necessarily be allergic to tree nuts or seeds. Symptoms of peanut, tree nut or seed allergies vary and range from milder reactions to a severe allergic reaction (anaphylaxis). The most common symptom of a nut allergy is raised red bumps of skin (hives) and other allergic symptoms such as runny nose, cramps, nausea or vomiting. The best way to manage peanut, tree nut and seed allergies is to avoid all products containing these foods. Food allergies can be life threatening and peanuts, tree nuts and seeds are some of the most common food triggers for life-threatening severe allergic reactions. Do not stand or walk . Administer adrenaline (epinephrine) via auto injector, if available.

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

Peanut seeds are usually eaten after toasting. The most frequent applications are the accompaniment of aperitifs, where they are consumed toasted and salted, in beer, toasted and sweetened or caramelized. They are also reduced to pasta to make peanut butter, sweet or savory spreads, ice cream, caramel crunchy and added, in pasta or flour, to various baked goods such as biscuits, cakes or snacks. Furthermore, peanut oil is obtained from peanut seeds, which is widely used in the kitchen thanks to a high smoke point. Peanut is also isolated from the seeds.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A FISH ALLERGY

RESULTS



The list of fish that can cause an allergic reaction is quite long and includes, but is not limited to: barramundi, cod, flake, salmon, snapper, trout, tuna, whiting. The specific molecule in fish that triggers your allergy may be present in a range of foods, and you may then have an allergic reaction to all foods containing that molecule. Some people who are allergic to one type of fish may be allergic to another type of fish or they may have allergies to several crustaceans, such as prawn, crab and lobster. This is known as cross-reactivity. Speak to your doctor about cross-reactivity because it is difficult to predict.

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

The nutritional properties of fish, it cannot be overlooked that it represents one of the most recommended foods in our diet. First of all, fish is rich in proteins, which have a high biological content and make up 15-20% of its composition. These are easily digestible substances. There are also many fats present, but they are not "bad" fats, which increase blood cholesterol. These are mostly unsaturated fats, which contain a high concentration of omega 3. The latter are important for the prevention of cardiovascular diseases and act as real antioxidants, which keep our body young. The fats in fish vary, in quantity, from 0.5% to 27%. Not to be underestimated are phospholipids, important fats for nervous function. Fish also contains mineral salts, such as calcium, phosphorus and iodine. Among the vitamins, A is more present, but the concentrations of vitamins B and D are also remarkable.



SCIENTIFIC DETAILS

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



PREDISPOSITION TO DEVELOP A MOLLUSCS/CRUSTACEANS ALLERGY

RESULTS



Crustaceans are a subcategory of shellfish. In recent years, there has been a steady growth in the production and consumption of seafood and specifically to shellfish. The high higher rate of consumption has led to an increase in adverse health problems among consumers, including allergic reactions. The pattern of allergic symptoms after ingestion of crustaceans appears similar to the symptoms experienced due to other foods. Reactions are immediate, and reported mostly within two hours; however, late phase reactions have been reported up to eight hours after ingestion, particularly to snow crab, cuttlefish, limpet, and abalone. Patients may have a single symptom, but often there is a multi-organ involvement. Importantly, respiratory reactions are often seen after ingestion of allergenic seafood and frequently anaphylactic reactions]. In particular, the oral allergy syndrome (OAS) seems to be very often experienced by crustacean allergic subjects. Shrimp has also been implicated in food-dependent exercise-induced anaphylaxis. Currently, 2 % of the general world population is affected by shellfish allergy, with much higher rates in countries with high seafood consumption. Unlike many other food allergies, most shellfish allergy persists for life in the affected individual.

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

Molluscs contain many proteins and few fats, and have many beneficial properties of which little is said. In fact, molluscs are rich in iodine, useful for activating the thyroid gland. It is essential to know, however, that molluscs contain omega3 and polyunsaturated fatty acids: essential for the prevention of cardiovascular diseases and diabetes. Crustaceans have positive nutritional characteristics: some of them are rich in iron, others of calcium, of thiamine (vit. B1) of riboflavin (vit. B2), of niacin (vit. PP) and (red ones) of astaxanthin, one provitamin A strongly antioxidant. Unlike most animal products (with few exceptions), crustaceans contain an energy portion of available carbohydrates (0.5% of the TOT kcal) and, exclusively, another of NON-available sugars: chitin; this is an OMO-polysaccharide consisting of long chains of acetyl-glucosamine (a derivative of glucose) also present in the carapace of insects. Unlike meat and fish, crustacean proteins contain more arginine than creatine.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A SOY ALLERGY

RESULTS



Soybeans are particularly problematic because of their widespread inclusion in processed foodstuffs which greatly limits consumer choices even if properly labeled. An additional primary use of soybeans is in infant formulas as a replacement for milk protein, and therefore, with the exception of milk, soybeans are the most likely FALCPA-listed allergen to be ingested by infants. With possible wide-spread exposure at an early age, it is surprising that soybean food allergenicity receives less attention than many of the others on the FALCPA list. This lack of emphasis likely results from the fact that initial allergic responses tend to be less severe and more antigen (soy protein) is required to elicit a response. Soybean exposure most often induces atopic skin reactions and gastrointestinal distress but rarely produces the fatal anaphylaxis associated with peanut and tree nut exposure. Sensitized individuals often become tolerant to soybeans over time, often as toddlers or young children while peanut allergy often persists throughout life. Although the soy allergic response can be mild and generally decreases with age, a growing body of evidence suggests alterations in gut-barrier function early in life predisposes an individual to a variety of diseases later in life.

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

Soybean is a legume. Like all legumes, soy is also rich in vegetable proteins, does not contain cholesterol, has several fibers and minerals (calcium, phosphorus, potassium) important for our health. Here is a short list of the main properties of soy, thanks to the molecules contained in it: regulates the intestine, blood sugar and cholesterol, thanks to the fiber and lecithins contained; helps fight menopause disorders, thanks to isoflavones, molecules similar to estrogen; protects bones: in soy we find another isoflavone called daidzein which helps prevent bone decalcification. In addition, 250 grams of soybeans provide about 50% of an adult's daily calcium needs; supports against premenstrual syndrome: thanks to the isoflavones genistein and daidzein, soybeans can positively influence the hormonal changes present before the menstrual cycle.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP FRUIT AND VEGETABLE ALLERGIES

RESULTS



Fruit and vegetable allergy is a reaction that occurs soon after contact to fruit and vegetables. These reactions usually occur within minutes after contact, but sometimes can take up to 1-2 hours. The reactions occur against proteins that are in a number of different fruits or vegetables. The Rosaceae (apple, pear, cherry, peach, and plum) and Cucurbitaceae (cucumber, melon, watermelon, zucchini, pumpkin) plant groups and kiwi fruit are particularly likely to cause allergies. In some cases, the proteins are also found in pollens of these plant groups leading to symptoms of pollen and food allergy (The Pollen-Food Allergy syndrome). Profilins are a type of plant protein that is known to cause allergy. They are proteins that affect cell shape and function and have been identified in trees, grass and weed pollens as well as many fruits and vegetables. Fruit and vegetable allergies are often due to an allergy to Profilins. Allergies to melon, watermelon, citrus fruits, tomato, and banana or a combination of these suggest an allergy to Profilins. Approximately 3% of teenagers have fruit or vegetable allergy. It is less common in young children. Sometimes symptoms occur only in the teenage years after developing hay fever. Some young children do however have allergy to banana, kiwi fruit and avocado, and more rarely to other fruits and vegetables.

People with your genetic profile are not likely to have a predisposition to develop an allergy to fruits and vegetables.

According to the World Health Organization, adequate consumption of fruit and vegetables would change the world map of cardiovascular diseases. The famous 5 portions a day reach an average of 400 grams, the minimum recommended quantity, therefore, for a healthy menu.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP SEED ALLERGY (GENERIC)

RESULTS



Seed allergies are among the most common food allergies across ages (1.4%) and have increased in children in the United States (2.1%) and United Kingdom (3%, including sesame) over the last 3 decades. Anaphylactic and fatal food allergy reactions are most commonly caused by seed. Coexistent seed allergy was reported 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, questionnaire-based data and IgE test results might overestimate the rate of clinical allergy by overreporting allergic symptoms. Sesame seed allergy often coexists with peanut allergy, 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.

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

Oil seeds, also known as dried fruit, are useful for cardiovascular diseases and diabetes thanks to their many properties. Let's find out better when and how to use them. Useful supplements in the nutrition of vegetarians and sportsmen, these fruits provide our body with many advantageous properties, thanks also to the presence of proteins, vitamins, minerals, essential fats, fibers and sugars. The fats they contain (unsaturated and polyunsaturated fats, in particular omega-3 and omega-6) are not only free of cholesterol but even capable of reducing the amount of the latter in the body, protecting the arteries from atherosclerosis thanks to their ability to lower the levels of "bad" cholesterol (LDL) and promote the increase in "good" cholesterol (HDL).



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A SALICYLATE ALLERGY (SALICYLIC ACID)

RESULTS



Salicylates are a group of chemicals derived from salicylic acid. They are found naturally in certain foods and also synthetically produced for use in products like aspirin, toothpaste and food preservatives. Both natural and synthetic forms can cause adverse reactions in some people. In their natural form, plants produce salicylates to defend against harmful elements like insects, fungus and disease. This form is found in a wide array of foods, including fruits, vegetables, coffee, teas, nuts, spices and honey. Meanwhile, the synthetic form is commonly used as a food preservative and found in medications like aspirin and Pepto-Bismol. Compared to foods, medications like aspirin contain high amounts of salicylates, which is why salicylate intolerance is most commonly linked to medications. For example, dietary intake of salicylates is usually 10–200 mg per day. Comparatively, a single dose of aspirin can contain 325–650 mg, depending on the type.

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

Foods that have salicylates include: Almonds, Apples, Apricots, Berries, Cherries, Coffee, Cucumbers and pickles, Grapes and raisins, Nectarines and oranges, Peaches, Peppers, Plums, Tea, Tomatoes.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A TARTRAZINE ALLERGY

RESULTS



Tartrazine is an azo food dye, which is orange-colored and water soluble. It is usually used in foods, pharmaceuticals, cosmetics, and textiles. Tartrazine has the potential to cause an adverse health effect on humans, such as hyperactivity in children, allergy, and asthma. Many researchers have detected the presence of tartrazine for monitoring the quality and safety of food products.

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

Tartrazine is a dye. It is found in confectionery, cotton candy, soft drinks, instant puddings, flavored chips (Doritos, Nachos), cereals (corn flakes, muesli), cake mixes, pastries, custard powder, soups (particularly instant or "cube" soups), sauces, some rices (paella, risotto, etc.), Kool-Aid, Mountain Dew, Gatorade, ice cream, ice pops, candy, chewing gum, marzipan, jam, jelly, gelatins, marmalade, mustard, horseradish, yogurt, noodles, pickles and other pickled products, certain brands of fruit squash, fruit cordial, chips, biscuits, and many convenience foods together with glycerin, lemon, and honey products. It is also found in soaps, cosmetics, shampoos and other hair products, moisturizers, crayons, and stamp dyes. The pharmaceutical industry uses this in vitamins, antacids, and prescription drugs.



SCIENTIFIC DETAILS

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



PREDISPOSITION TO DEVELOP SENSITIVITY TO SULPHITES

RESULTS



The sulfite ion is an anion composed of sulfur and oxygen, the first with oxidation state +4 and the second with oxidation state -2. Sulfite treatment of wine [a process exploiting the biocidal and anti-oxidant properties of sulfur dioxide (SO₂)] involves the use of liquified gas, aqueous solutions or bisulfites, i.e. the salts of sulfurous acid which slowly release SO₂. This procedure can result in repeated exposures of operators to significant amounts of SO₂. However, risks associated with the use of SO₂ are greatly underestimated by wine producers and wine-cellars workers. We report on 6 cases of respiratory symptoms attributable to SO₂ identified during a survey of wine-cellars in the French Beaujolais district. Their pathogenesis is discussed after an overview of the occupational toxicology of SO₂. Sulfites are chemicals that are in some foods, either naturally or as additives. It's rare, but some people (about 1 in 100, according to the FDA) are sensitive to these compounds. Their reaction can range from mild to life-threatening. Sulfites aren't used on most fresh foods, but they're still in some cooked and processed ones. And they can also happen naturally in the process of making wine and beer.

People with your genetic profile are not likely to have a predisposition to develop a sulfites sensitivity.

Foods containing sulphites include dried fruits, dried vegetables, pickled onions and bottled soft drinks and cordials. The addition of sulphite additives to beer and wine is permitted in most countries, and although the use of sulphites in fresh salads, fruit salads, mincemeat or sausage meat, is illegal in many countries, it may occur illegally. In addition to food, exposure to sulphites can occur through the use of cosmetics and medicines. Cosmetics containing sulphites include hair colours and bleaches, creams, and perfumes. Medicines containing sulphites include eye drops, topical medications, and parenteral medications such as adrenaline, phenylephrine, corticosteroids and local anaesthetics. The sulphites also have a number of industrial uses, including in the photographic and textile industries, and consequently, occupational exposures to these additives may also occur.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP SENSITIVITY TO METABISULPHITES

RESULTS



Sodium metabisulphite is a sodium salt of metabisulphurous acid H₂S₂O₅. At room temperature it appears as a colorless solid with a slightly pungent odor. It is a harmful, irritating compound. It is used for the storage of alcoholic beverages. It is easily digested by the human body. It is used as a preservative and antioxidant in food and is known by the abbreviation E223. It can cause allergic reactions in those sensitive to sulphites, it can cause respiratory reactions in asthmatics, anaphylaxis and other allergic reactions in sensitive individuals. It is commonly used for cleaning equipment for home beverage production and wine or beer production. It is used in water desalination systems in reverse osmosis membranes for the production of drinking water. It is also used to remove chloramine from water after treatment. It is added in local anesthesia solutions to prevent oxidation of vasoconstrictor adrenaline and lengthen the period of use of the solution. It is used as an excipient in some tablets such as those based on acetaminophen. It is used in waste treatment to chemically reduce hexavalent chromium to trivalent chromium which can be precipitated and removed from waste by decanting and filtering.

People with your genetic profile are not likely to have a predisposition to develop a metabisulphites sensitivity.

Foods containing metabisulphites include dried fruits, dried vegetables, pickled onions and bottled soft drinks and cordials. The addition of sulphite additives to beer and wine is permitted in most countries, and although the use of sulphites in fresh salads, fruit salads, mincemeat or sausage meat.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP AN ALLERGY TO BISULFITES

RESULTS



Sodium bisulfite, or sodium hydrogen sulfite, is a chemical compound with the formula NaHSO₃. In the presence of acids, it releases sulfur dioxide, a toxic gas. Reacts rapidly with oxygen to form sodium bisulfate. Sodium bisulfite is used in wines as an alternative to sodium metabisulfite, releasing sulfur dioxide which kills the microorganisms present in the must before fermentation and added again as a preservative during the bottling of the wine. It is also used in fruit juices and fruit preserves to prevent oxidation (browning) and the proliferation of microbes. It is also used in green leafy vegetables to preserve its apparent freshness, as well as in dehydrated potatoes. Sodium bisulfite can cause an allergic reaction in people with sulphite allergy, which can culminate in death. The substance is one of those compounds whose use in fresh vegetables and fresh fruit was banned in the United States by the FDA during the 1980s following the death of 13 people attributed to the presence of sulphites in these foods.

People with your genetic profile are not likely to have a predisposition to develop a bisulfites sensitivity.

Foods containing bisulfites include dried fruits, dried vegetables, pickled onions and bottled soft drinks and cordials. The addition of sulphite additives to beer and wine is permitted in most countries, and although the use of sulphites in fresh salads, fruit salads, mincemeat or sausage meat.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A WALNUT ALLERGY

RESULTS



Allergies to tree nuts and seeds tend to be of a more severe nature, causing life-threatening and sometimes fatal reactions. People with tree nut allergies also often suffer from reactions triggered by a number of different types of nuts, even though they do not come from closely related plant species. In general these allergies are triggered by the major proteins found in nuts and seeds which are resistant to processes such as cooking. There is also a milder form of tree nut allergy which is associated with birch pollen allergy, where symptoms are confined largely to the mouth, causing a condition called "oral allergy syndrome" (OAS). This condition is triggered by molecules found in tree nuts which are very similar to pollen allergens like the major birch pollen allergen called Bet v 1. These molecules tend to be destroyed by cooking, which can reduce the allergenicity of nuts and seeds for these allergic consumers. Reactions to nuts and seeds can also occur as a consequence of hidden nut ingredients or traces of nuts and certain seeds introduced as a consequence of food handling or manufacturing. As a result tree nuts and seeds have been included in Annex IIIa of the EU food labelling directive. The following (including products thereof) must be declared on a label if they have been deliberately included in a food: Almond, hazelnut, Walnut, Cashew, Pecan nut, Brazil nut, Pistachio nut, Macadamia nut, Queensland nut, Mustard and Sesame seeds.

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

Fresh or dried walnuts can be consumed directly as dried fruit, breaking the shell. The kernels can be used in the kitchen to flavor salads, desserts but also to prepare jams. Walnuts also enter the composition of many processed foods: walnut bread, walnut cheese, walnut honey, jams, liqueurs, aperitifs, etc. Walnut oil is also extracted from the walnut by pressure and heating, with a yield of about 50% with the current pressing, that is, for every 40 kg of kernel it is possible to obtain up to twenty liters of oil. It is a good quality oil, cooked nutty flavor sometimes pronounced depending on the amount of heat applied and beneficial nutritional properties (many Omega-3 fatty acids for a little Omega-6 fatty acids). The "walnut wine" is also made, just as walnut husk enters the preparation of the numerous types of Nocino, generally drunk as digestives.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A KIWI ALLERGY

RESULTS



The kiwi or kivi is an edible berry, produced by numerous species of lianas of the genus Actinidia, family of the Actinidiaceae. The two main varieties of this berry are: green and yellow (or gold). The first, the most common, has dark brown skin with lint and bright green flesh, small and black seeds arranged in a radial pattern around the center of the berry, the shape is similar to an egg or a small potato. The gold variety has a more elongated shape, the pulp is yellow and has no lint on the skin. There are other varieties, but they are not very common, such as kiwi with red flesh and brick-colored skin. Kiwifruit is a fruit rich in vitamin C (85 mg / 100 g), potassium, magnesium, vitamin E, copper, iron and fiber. The high content of potassium and the poverty of sodium make it the ideal fruit for athletes, since it reduces the risk of cramps. It is recommended for those who have digestive problems and help the work of the intestine such as vegetables and plums. The calorie intake is very low: 44 kcal per 100 g approximately; this is because it is made up of about 84% water, 9% carbohydrates and traces of fats and proteins.

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

A portion of kiwifruit provides 44-132 kcal (on average 88 kcal) and is among the sweet foods, therefore with a prevalence of carbohydrates. The sugars it contains are simple and more precisely composed of fructose; those few fats that can be found are triglycerides made up of unsaturated chains, while proteins are NOT characterized by a good biological value and mainly contain amino acids: aspartic acid, glutamic acid and arginine. Kiwifruit is a good carrier of water, fiber (admirable the soluble content), potassium (K) and ascorbic acid (vit. C)



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A PINE NUT ALLERGY

RESULTS



Pine nuts (or pignoli) are the edible seeds of some pine species. Yellowish white in color and elongated. Pine nuts are rich in proteins and have been consumed in Europe since the Paleolithic period. They are also a source of dietary fiber. Pine nuts are essential for pesto and various other dishes including cakes, such as castagnaccio. The oldest recipes originate in the areas where the two European pines grow from which pine nuts are obtained: for example the strudel was formerly made with the seeds of the Swiss stone pine (a typical species of the high Trentino mountains), while the pesto from those of the pine domestic (species that grows in Mediterranean areas). Pine nuts are rich in vitamins (especially E, B and PP), calcium, magnesium and iron. The incidence of food-induced anaphylaxis (FIA) is increasing in young children. Although the commonest culprits are cow's milk and egg, FIA to tree nuts (TNs) have been increasing.

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

The pine nuts are rich in fatty acids, in particular monounsaturated which, according to numerous studies, help to lower cholesterol levels by decreasing the risk of heart attacks. They have a high content of proteins and phosphorus which play an important role in cell turnover and energy production mechanisms. For this reason pine nuts are particularly suitable for those who follow a vegetarian or vegan diet, but also for those who practice sports. Pine nuts are also enriched with antioxidant content able to slow down the aging process by destroying and attacking free radicals present in the body. Pine nuts do not contain gluten and are therefore indicated in the preparation of food for coeliacs and are a valid alternative in the diet of people with food allergies to wheat.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A WHEAT ALLERGY

RESULTS



Despite many benefits, common wheat is recognized as an immune-mediated food allergen because it activates immunoglobulin E (IgE) and non-IgE immune responses. Children have a higher prevalence of wheat allergies compared to adults and are more likely to develop an allergy if wheat is introduced after 6 months of life. Wheat allergy is the manifestation of mediator release, such as histamine, platelet activator factor, and leukotrienes, from mast cells and basophils. The IgE production is thought to be due to a breach of oral tolerance, and as a result, of Th2-biased immune dysregulation that causes sensitization and B-cell IgE production. Celiac disease is characterized by a specific autoantibody against tissue transglutaminase 2 (anti-tTG2), endomysium, and/or deamidated gliadin peptide. When gliadin peptides pass through the epithelial barrier, they activate CD4 T-lymphocytes which produce high levels of pro-inflammatory cytokines. Patients with wheat dependent exercise-induced anaphylaxis (WDEIA) usually have symptoms that include pruritus, urticaria, angioedema, flushing, shortness of breath, dysphagia, chest tightness, profuse sweating, syncope, headache, diarrhea, nausea, throat closing, abdominal pain, and hoarseness that occurs during intense physical exercise following wheat intake in the prior 4 hours before symptom onset. Those with celiac disease will present with diarrhea, constipation, bloating, abdominal pain, anorexia, flatulence, weight loss, poor growth in childhood, anemia, dermatitis herpetiformis, fatigue, and even osteoporosis. Primary treatment and management of wheat allergies is avoidance of both food and inhaled wheat allergens. In cases of exposure and an anaphylactic reaction, the administration of epinephrine is the lifesaving treatment. Patients who have signs or symptoms of a wheat allergy should see a doctor as soon as possible as it can be a life-threatening disease. Those that are already diagnosed should make sure they avoid wheat and to always carry epinephrine.

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

Wheat is an important source of carbohydrates. Globally, it is the leading source of vegetable protein in human food, having a protein content of about 13%, which is relatively high compared to other major cereals but relatively low in protein quality for supplying essential amino acids. When eaten as the whole grain, wheat is a source of multiple nutrients and dietary fiber.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
IL-18	rs181720	GG



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A CORN ALLERGY

RESULTS



Corn is the traditional food base of the populations of Latin America and some regions of Europe and North America. In temperate regions it is mainly intended for the feeding of domestic animals, in the form of grain, flour or other feed, or as silage, generally collected when waxy ripening. It is also intended for industrial transformations for the extraction of starch and oil or for fermentation, in order to produce alcoholic drinks or bioethanol for energy purposes by distillation. The nutritional properties of maize for human consumption are modest. Aside from a good amount of carbohydrates, it contains few nutrients and few vitamins of group B and group PP, which are present in a non-assimilable form. Furthermore, its protein component is low in lysine and tryptophan, two essential amino acids.

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

Corn is a gluten-free cereal, therefore it can enter the diet of those suffering from celiac disease. It is a source of folic acid and vitamin B1; it is therefore suitable for feeding during pregnancy and for children, starting from early childhood: corn cream is one of the first foods to be introduced into the diet during weaning. Furthermore, corn has a good share of iron and of other minerals; it is therefore useful in case of anemia. It is particularly digestible and is rich in dietary fiber, for this reason it is a valuable ally for the stomach and intestines. Finally, the fibers contained in corn slow down the absorption of sugars, thus helping to keep blood glucose levels low. Thanks to its properties, it also helps to keep LDL cholesterol values low; the so-called "bad". Corn is therefore an excellent food; however, it is also used in herbal medicine, to obtain products useful to fight diabetes, hypotension, hypokalaemia, overweight, water retention and spasms of the colon.



SCIENTIFIC DETAILS

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



PREDISPOSITION TO DEVELOP AN AMARANTH GRAIN ALLERGY

RESULTS



Amaranth is a plant that has several species, originally from Central America, where it was already cultivated by pre-Columbian civilizations. The genus Amaranthus includes about 60 species, but only 3 are considered to be good producers of seeds: Caudatus, Cruentus and Hypochondriacus. Not being part of the grasses, it is not a cereal, as buckwheat, quinoa, sago and cassava are not. Rich in proteins, up to 16%, with high biological value, amaranth, compared to cereals, contains twice as much 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 an explosive effect on digestion and replacement. Being gluten-free, it is indicated for feeding those who suffer from celiac disease, or have intestinal problems, but also for children during the weaning period. It is conveniently used often as a base for baby food or as a valuable ingredient in vegetable soups for convalescents and the elderly.

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

Not being part of the grasses, amaranth is not a cereal, as buckwheat, quinoa, sago and cassava are not. Rich in proteins, up to 16%, with high biological value, amaranth, compared to cereals, contains twice as much 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 very beneficial effect on digestion. Being gluten-free, it is indicated for feeding those who suffer from celiac disease, or have intestinal problems, but also for children during the weaning period. It is conveniently used often as a base for baby food or as a valuable ingredient in vegetable soups for convalescents and the elderly.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

PREDISPOSITION TO DEVELOP A CASSAVA ALLERGY

RESULTS



Cassava, also known as manioc, mandioca, yucca, or tapioca, is the common name for the tuber *Manihot esculenta* Crantz, which belongs to the Euphorbiaceae family from the West Indies, Latin America, and Africa. It is a staple food in regions of South America and Africa and 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 in order to avoid an excess of hydrocyanic acid. It has been reported that the ingestion of poorly processed cassava may cause tropical ataxic neuropathy, tropical pancreatic diabetes, and goitre. Patients who are allergic to latex (*Hevea brasiliensis*) may exhibit cross-hypersensitivity with foods.

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

Individuals with food allergies often benefit from using cassava root in cooking and baking because it is gluten-free, grain-free and nut-free. One important note is that cassava root must be cooked before it is eaten. Raw cassava can be poison. A 3.5-ounce (100-gram) serving of boiled cassava root contains 112 calories. 98% of these are from carbs and the rest are from a small amount of protein and fat. This serving also provides fiber, as well as a few vitamins and minerals. Boiled cassava root also contains small amounts of iron, vitamin C and niacin.



SCIENTIFIC DETAILS

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



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KIT ID: TPD14630189007528

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, and has been a source of valuable food over thousands of years. It can produce a variety of secondary metabolites with broad spectra of bioactivities. At least 193 secondary metabolites from quinoa have been identified in the past 40 years. They mainly include phenolic acids, flavonoids, terpenoids, steroids, and nitrogen-containing compounds. These metabolites exhibit many physiological functions, such as insecticidal, molluscicidal and antimicrobial activities, as well as various kinds of biological activities such as antioxidant, cytotoxic, anti-diabetic and anti-inflammatory properties. In science, there are many reviews on quinoa, most of them are 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, but no review covers almost all secondary metabolites and their biological activities.

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

It is very rich in betaine, a substance that is extracted from sugar beet. Quinoa, not containing gluten, can be eaten by celiacs. Quinoa can be consumed after decortication in soups or risotto. Quinoa flour is indicated alone or mixed with cereal flours, for all normal uses of flour, therefore: sweets, bread, pasta.



SCIENTIFIC DETAILS

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



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DANTE LABS™ TEST RESULTS

KIT ID: TPD14630189007528

EASE OF GETTING UP IN THE MORNING

RESULTS



Brightening an otherwise dark room, the numbers on the alarm clock glow with a steady message: Time to wake up. The bed is cozy, though, and the day promises to be a busy one. It's moments like this where getting up in the morning isn't so easy. It's not an uncommon experience, but for some people, waking up in the morning can be a unique challenge. There are many reasons for this, one of which may have to do with the HCRTR2 gene. We usually don't give it much thought, but being awake is not a simple thing—it's a very active process. In the awake state, our brain has to process incoming sensory information, coordinate body movement, recall memories, and formulate new thoughts. In contrast, while sleeping, our bodies become temporarily paralyzed, we become less aware of external stimuli, and our brains' focus turns inward. Both states of being—awake and asleep—are actively promoted by processes in our body, in the same way that we have to actively turn and hold the steering wheel of a car to make it move in one direction or another. In order to stay awake, our bodies rely heavily on the orexin signaling cascade. Daily, 5 am is best time to walk up.

People with your genetic profile are likely to have an easy time waking up in the morning.

The wake-up success correlation does not lie and leaves little to the imagination. Benefits-of-Waking Up-Presto tant so much that even science seems to show that the benefits of getting up early correspond to the truth. In fact, it seems that most of the most influential personalities of our day love to start their day well before the cock crow. Apple CEO Tim Cook, for example, sets the alarm at 3:45 am and Virgin founder Richard Branson and Vogue director Anna Wintour wake up at 5:45 am. Everyone uses the very first hours of the day to train, get informed, to carve out precious moments to devote to self-care before professional and social commitments demand their attention.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LRRTM4	rs4853283	AA
LINC01107	rs4483990	AA
EXD3	rs77641763	CC
PER2	rs116298301	CC
AC001226.2, FBXL3, CLN5	rs7332608	AA



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KIT ID: TPD14630189007528

DAYTIME NAP

RESULTS



If you're sleep deprived or just looking for a way to relax, you might be thinking about taking a nap. Napping at the wrong time of day or for too long can backfire, though. Understand how to get the most out of a nap. Napping offers various benefits for healthy adults, including: Relaxation, Reduced fatigue, Increased alertness, Improved mood, Improved performance, including quicker reaction time and better memory. Napping isn't for everyone. Some people simply can't sleep during the day or have trouble sleeping in places other than their own beds, which napping sometimes requires. Napping can also have negative effects, such as: Sleep inertia: you might feel groggy and disoriented after waking up from a nap. Night time sleep problems: short naps generally don't affect night time sleep quality for most people. But if you experience insomnia or poor sleep quality at night, napping might worsen these problems. Long or frequent naps might interfere with night time sleep. The optimum nap time is 3pm. As a nation, the United States appears to be becoming more and more sleep deprived. And it may be our busy lifestyle that keeps us from napping. While naps do not necessarily make up for inadequate or poor quality night time sleep, a short nap of 20-30 minutes can help to improve mood, alertness and performance. Nappers are in good company: Winston Churchill, John F. Kennedy, Ronald Reagan, Napoleon, Albert Einstein, Thomas Edison and George W. Bush are known to have valued an afternoon nap. A recent study in the research journal Sleep examined the benefits of naps of various lengths and no naps. The results showed that a 10-minute nap produced the most benefit in terms of reduced sleepiness and improved cognitive performance. A nap lasting 30 minutes or longer is more likely to be accompanied by sleep inertia, which is the period of grogginess that sometimes follows sleep.

People with your genetic profile are likely to not take naps during the day.

The nap is not for everyone. It should be considered that for many individuals it is impossible to sleep in the middle of the day. Others find it difficult to sleep in broad daylight if they don't have their own bed.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LMOD1	rs2820313	AA
KRT18P24 - AL512634.1	rs34799682	AA
FGF4	rs182197129	CC
NLN	rs755927998	CC
ZNF385D	rs541594711	CC
CCR3	rs114515123	GG



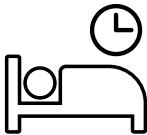
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KIT ID: TPD14630189007528

SLEEP DURATION

RESULTS



Sleep is a vital indicator of overall health and well-being. We spend up to one-third of our lives asleep, and the overall state of our "sleep health" remains an essential question throughout our lifespan. Most of us know that getting a good night's sleep is important, but too few of us actually make those eight or so hours between the sheets a priority. For many of us with sleep debt, we've forgotten what "being really, truly rested" feels like. To further complicate matters, stimulants like coffee and energy drinks, alarm clocks, and external lights—including those from electronic devices—interferes with our "circadian rhythm" or natural sleep/wake cycle. Sleeping less than seven hours a night is associated with an increased risk of developing conditions such as obesity, diabetes, high blood pressure, heart disease, stroke, and frequent mental distress, the CDC states. The panel revised the recommended sleep ranges for all six children and teen age groups. A summary of the new recommendations includes: Newborns (0-3 months): Sleep range narrowed to 14-17 hours each day (previously it was 12-18). Infants (4-11 months): Sleep range widened two hours to 12-15 hours (previously it was 14-15). Toddlers (1-2 years): Sleep range widened by one hour to 11-14 hours (previously it was 12-14). Preschoolers (3-5): Sleep range widened by one hour to 10-13 hours (previously it was 11-13). School age children (6-13): Sleep range widened by one hour to 9-11 hours (previously it was 10-11). Teenagers (14-17): Sleep range widened by one hour to 8-10 hours (previously it was 8.5-9.5). Younger adults (18-25): Sleep range is 7-9 hours (new age category). Adults (26-64): Sleep range did not change and remains 7-9 hours. Older adults (65+): Sleep range is 7-8 hours (new age category).

People with your genetic profile may have problems with sleep duration.

To pave the way for better sleep, follow these simple yet effective healthy sleep tips, including: stick to a sleep schedule, even on weekends; practice a relaxing bedtime ritual, exercise daily, evaluate your bedroom to ensure ideal temperature, sound and light; sleep on a comfortable mattress and pillows; beware of hidden sleep stealers, like alcohol and caffeine; turn off electronics before bed.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
BANK1	rs13109404	TT
SEMA6D	rs13329140	GG
TCF4	rs12607679	TT
ZBED9	rs34388845	AA
LINC00240	rs12215241	GG
MAPT, MAPT, MAPT	rs62061734	TT
AL645939.5	rs1633063	CT
ADCK1 - FXNP1	rs11621908	CT



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KIT ID: TPD14630189007528

SNORING

RESULTS



Snoring is a common condition that can affect anyone, although it occurs more frequently in men and people who are overweight. Snoring has a tendency to worsen with age. Occasional snoring is usually not very serious and is mostly a nuisance for your bed partner. However, if you are a habitual snorer, you not only disrupt the sleep patterns of those close to you, but you also impair your own sleep quality. Medical assistance is often needed for habitual snorers (and their loved ones) to get a good night's sleep. Snoring is the hoarse or harsh sound that occurs when air flows past relaxed tissues in your throat, causing the tissues to vibrate as you breathe. Nearly everyone snores now and then, but for some people it can be a chronic problem. Sometimes it may also indicate a serious health condition. In addition, snoring can be a nuisance to your partner. According to the latest data, roughly 40% of adult men and 24% of adult women experience chronic snoring. Half the U.S. population will snore at some point during their adult lives. Snoring is also somewhat linked to age. While most habitual snorers are adults, the majority experience less nightly snoring after the age of 70. Children with at least one parent who snored frequently were three times as likely to also be frequent snorers compared with children of silent sleepers. African-American children were three times more likely to snore frequently than other children. Children who tested positive for allergies were more than twice as likely to be frequent snorers.

People with your genetic profile are not likely to have a predisposition to snore.

Not snoring allows you to have a continuous and better rest. It can also sometimes happen to those who do not snore to take uncomfortable positions and have breathing difficulties that cause the characteristic noise of snoring. In these cases it is advisable to take a comfortable position to rest at best.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC01876	rs72906130	CC
LACTB2-AS1, LACTB2	rs7007887	CC
MSRB3	rs10878269	CC
LINC02210-CRHR1, LINC02210-CRHR1	rs57222984	AA
AC044784.1 - LINC00709	rs11256034	CC



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KIT ID: TPD14630189007528

DAYTIME SLEEPINESS

RESULTS



Excessive sleepiness often goes unrecognized in the primary care setting despite its high prevalence and deleterious effects on both individual and public safety. Patients with neurologic and psychiatric illnesses, as well as those with acute and chronic medical conditions, plus those with sleep disorders, often have symptoms of excessive sleepiness, tiredness, and fatigue. Recognition and prompt treatment of these symptoms are important, even though their etiology may not be immediately understood. This review focuses on the underlying causes, consequences, identification, and treatment of excessive sleepiness. Patients with excessive daytime sleepiness (EDS) have impaired function due to difficulty maintaining wakefulness or alertness at appropriate times during the day. Complaints of EDS, or related terms such as tiredness, fatigue, and lack of energy, constitute some of the most common issues presented to clinicians. EDS is important to recognize because it can signal an undiagnosed sleep disorder or other treatable conditions. In addition, EDS can have a negative impact on a broad range of activities and raise safety risks while driving or operating other machinery.

People with your genetic profile are not likely to have a predisposition to experience excessive sleepiness.

Not suffering from drowsiness is an advantage that allows you to have a very high energy peak during the day. Preventing sleepiness is possible by adopting a lifestyle that respects the correct circadian rhythms and therefore giving our body and brain the right recovery time during the night.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
TMEM132B	rs142261172	GG
AC108752.1 - MRPS17P3	rs182765975	GG
BMI1P1 - AL157700.1	rs73536079	GG
TENT5A	rs189689339	CC
CPEB1, CPEB1, AC245033.1, AC245033.2	rs17507216	GG
LINC01358, AL592431.1	rs192315283	TT



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INSOMNIA COMPLAINTS

RESULTS



Insomnia is the most common sleep disorder, yet little is known about the etiology, pathophysiology, and clinical course of this highly prevalent and chronic disorder. In the few published general population longitudinal studies, the estimated rate of persistent insomnia varies from 40% to 70%. Factors such as gender, age, body weight, physical disorders, depression, or alcohol consumption have been proposed to be associated with persistent insomnia, but the results are inconsistent. Furthermore, these studies have not included polysomnography (PSG), and, for example, the longitudinal association of sleep duration or sleep disordered breathing (SDB) with persistent insomnia has not yet been studied. Insomnia is frequently associated with physical and mental health disorders. Insomnia is a risk factor for the development of depression, and depression is a risk factor in the persistence of insomnia. As far as the association of chronic insomnia with the second most common sleep disorder (SDB), the findings have been mixed. Some but not all investigators have suggested that SDB is associated with chronic insomnia. However, no longitudinal study to date has examined whether SDB is a risk factor for the persistence of insomnia.

People with your genetic profile are not likely to have a predisposition to suffer from insomnia.

Good sleep habits, also called sleep hygiene, can help you get a good night's sleep and prevent insomnia.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
MEIS1	rs113851554	GG
MEIS1	rs11679120	GG
TRPC7	rs55972276	CC
AC099520.1	rs2431108	TT
PTPRD	rs118166957	CC
SLC39A8	rs13135092	AA
TMEM161B-AS1	rs16903122	CC



OBSTRUCTIVE SLEEP APNEA

RESULTS



Obstructive sleep apnea (OSA)—also referred to as obstructive sleep apnea-hypopnea—is a sleep disorder that involves cessation or significant decrease in airflow in the presence of breathing effort. It is the most common type of sleep-disordered breathing and is characterized by recurrent episodes of upper airway collapse during sleep. These episodes are associated with recurrent oxyhemoglobin desaturations and arousals from sleep. OSA that is associated with excessive daytime sleepiness is commonly called obstructive sleep apnea syndrome—also referred to as obstructive sleep apnea-hypopnea syndrome. These non-anatomical traits include: (1) poor pharyngeal muscle responsiveness during sleep, (2) an overly sensitive ventilatory control system (i.e., high loop gain [LG]), and (3) a low respiratory arousal threshold that leads to arousal rather than stable ventilation. Obstructive sleep apnea is a common disorder associated with increased risk for cardiovascular disease, diabetes, and premature mortality.

People with your genetic profile are likely to suffer from obstructive sleep apnea.

For adults, the use of continuous positive airway pressure (CPAP) is the most effective treatment, and diligent adherence to nightly CPAP use can result in near complete resolution of symptoms. For patients unable or unwilling to use CPAP or those who will be unable to access electricity reliably, custom fitted and titrated oral appliances can be used to bring the lower jaw forward and relieve airway obstruction. This typically works best for candidates deemed to have appropriate dentition and mild to moderate sleep apnea. Severe obstructive sleep apnea can be treated with BiPAP as well and is better tolerated by patients who require higher pressure settings (greater than 15 cm to 20 cm H₂O). For all patients, it is important to address any concomitant nasal obstruction with nasal steroids for allergic rhinitis or surgically for nasal valve collapse. For obstructive sleep apnea with a strong positional component, a positioning device to keep a patient on his or her side can be an option. Although weight loss is recommended and can often decrease the severity of obstructive sleep apnea, it is not usually curative by itself. The primary treatment for obstructive sleep apnea in a child is tonsillectomy and adenoidectomy. The consideration for surgery should be balanced with the severity of symptoms, physical exam, and age. In mild cases, a trial of montelukast and nasal steroids may be enough to reduce the apnea-hypopnea index to goal. There are surgical options for adults, but these are usually reserved for severe obstructive sleep apnea and patients unable to tolerate noninvasive treatment modalities due to surgical risks and varying efficacy. Uvulopalatopharyngoplasty (UPPP) is a term used to surgically remove the uvula and tissue from the soft palate to create more space in the oropharynx. This is sometimes done in conjunction with a tonsillectomy and adenoidectomy. More recently, drug-induced sleep endoscopy (DISE) has been used for preoperative planning to identify multiple levels of obstruction that are often present in these patients. This allows surgeons to address any nasal, soft palate and hypopharyngeal obstructions that may be present during a single surgery. Another surgical option is maxillomandibular advancement (MMA) in which both the upper and lower jaws are detached and surgically advanced anteriorly to increase space in the oropharynx. This is best for patients with retrognathia and is less successful in older patients or those with larger neck circumferences. A newer option is the implantable hypoglossal nerve stimulator. It works by stimulating the genioglossus (upper airway dilator muscle) during apneas resulting in protrusion of the tongue and relief of the obstruction. To be considered a candidate patient must meet the following criteria: BMI less than 32, more than 22 years of age, apnea-hypopnea index 2 to 65 with less than 25% central apneas, unable to tolerate CPAP and no complete concentric collapse at the palate on drug induced sleep endoscopy. The use of a permanent surgically-implanted device with limited time on the market and limited published long-term data on safety and efficacy should be pursued in specialty centers with experience in the surgical treatment of sleep-disordered breathing, in appropriately selected patients. In extreme cases, obstructive sleep apnea can also be treated with a tracheostomy to bypass the oropharyngeal obstruction. This management option is also best addressed at academic or specialty sleep centers that are experienced in the care of patients with tracheostomy. Such patients will encounter numerous challenges with home care and durable medical equipment and family/partner education on tracheostomy management. Many patients with severe sleep disordered breathing requiring tracheostomy have comorbidities and require long term home-based mechanical ventilation, the management of which will be outside the scope of most community sleep medicine practices. Patients should be counseled to avoid alcohol, benzodiazepines, opiates, and some antidepressants which may worsen their condition. Most importantly, patients should reflect on the impact of sleep duration and their health, and place a priority on getting at least 7 to 8 hours of sleep per night. No treatment for OSA per se will correct insufficient sleep.



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SCIENTIFIC DETAILS

Gene	rsID	Genotype
ST8SIA6-AS1, ST8SIA6	rs12415421	TT
LMOD2 - WASL	rs4731117	TT
HECW1	rs9648078	CC
ASB15	rs12669165	TT
LINC01428	rs6117669	GG
ATP2B4 - LAX1	rs116133558	CC



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CONSCIENTIOUSNESS

RESULTS



Conscientiousness is one of the five personality traits described in the Big Five model of personality psychology. It's used to describe a person's tendency to be organized and goal-oriented. Someone with a high degree of conscientiousness is self-disciplined, efficient, orderly and methodical. They place a lot of importance on getting stuff done - and getting it done properly. They turn up on time, meet deadlines and follow the rules. Yet research suggests that your conscientiousness score is the single biggest predictor of success. It influences everything from grades at school to your chances of becoming addicted to drugs. According to the Big Five theory, there are five basic traits or dimensions that serve as the building blocks of personality. The five traits are specified by the helpful anagram OCEAN: O - Openness to experience (your level of curiosity) C - Conscientiousness (your level of work ethic) E - Extraversion (your level of sociability) A - Agreeableness (your level of kindness) N - Neuroticism (your level of anxiety or shame). According to twin studies, around 40-60% of the variance in the Big Five is heritable, with some overlap in heritability between personality traits themselves.

People with your genetic profile are likely to have a conscientious personality.

A person's level of conscientiousness is generally assessed using self-report measures, although peer-reports and third-party observation can also be used. Self-report measures are either lexical or based on statements.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LINC00461	rs3814424	CC
KATNAL2	rs2576037	CC



EXTRAVERSION

RESULTS



Extraversion (versus introversion) reflects talkativeness, assertiveness and a high activity level. In the big 5 theory of personality, extroversion (often known as extraversion) is one of the five core traits believed to make up human personality. Extroversion is characterized by sociability, talkativeness, assertiveness, and excitability. People who are high in extroversion tend to seek out social stimulation and opportunities to engage with others. These individuals are often described as being full of life, energy, and positivity. In group situations, extroverts (extraverts) are likely to talk often and assert themselves. Introverts, on the other hand, are people who are low in extroversion. They tend to be quiet, reserved and less involved in social situations. It is important to note that introversion and shyness are not the same things.

People with your genetic profile are likely to have regular levels of extraversion.

The extent of extroversion and introversion is generally assessed by means of self-regulatory measures, although peer relations and third party observations can also be used. Self-reporting measures are lexical or statement-based. The type of measurement is determined by an evaluation of the psychometric properties of the method used.



SCIENTIFIC DETAILS

Gene	rsID	Genotype
LOC105377179	rs57590327	GG
MTMR9	rs2164273	AA

GLOSSARY

ALLEL	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.
GENOME	A genome is an organism's complete set of DNA, including all of its genes. Each genome contains all of the information needed to build and maintain that organism. In humans, a copy of the entire genome—more than 3 billion DNA base pairs—is contained in all cells that have a nucleus.
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.
ODDS RATIO	The odds ratio is a way of comparing whether the odds of a certain outcome is the same for two different groups. In this report, the odds ratio estimates the probability of a condition occurring in a group of people with a certain genetic variant compared to a group of people without that variant. An odds ratio of 1 means that the two groups are equally likely to develop the condition. An odds ratio higher than 1 means that the people with the



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	genetic variant are more likely to develop the condition, while an odds ratio of less than 1 means that the people with the variant are less likely to develop the condition.
PHENOTYPE	A description of an individual's physical characteristics, including appearance, development and behaviour. The phenotype is determined by the individual's genotype as well as environmental factors.
POPULATION ALLELE FREQUENCY	The allele frequency represents the incidence of a variant in a population. Alleles are variant forms of a gene that are located at the same position, or genetic locus, on a chromosome.
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.