INTESTINOgenes

Genetic profile of intestinal diseases

Surname:	Ejemplo	**Reception date:**	DD/MM/YYYY
Name:	Paciente Modelo	**Validation date:**	DD/MM/YYYY
Birth Date:	DD/MM/YYYY	**Sample Type:**	-
ID nr:	-	**Physician:**	-
Gender:	Male	**Validation by:**	-
Order:	-		

INTESTINOgenes

The intestine is part of the **digestive system** and extends from the stomach to the anus. It allows **food** to be converted into **nutrients** and thus **absorbed**. At the same time, it also expels and **eliminates waste products** properly. Problems or alterations in these functions can compromise the health and well-being of the individual.

Intestinal disorders

Intestinal problems are of **multifactorial** etiology and are closely related to the **diet** and **lifestyle habits** of each person, although personal factors also play a role, some of them related to **genetics**. Intestinal disorders can manifest from **mild symptoms** such as diarrhea or constipation, to **more severe symptoms** such as microvilli alterations, ulcers or stenosis.

Lactose and celiac disease

In **lactose intolerance**, the **ability** to metabolize lactose into its two assimilable products, **glucose** and **galactose**, is **lost**. This impairs its absorption and intestinal pathology occurs. Although it usually manifests at the age of 4-6 years, there are many forms of late onset that are not related to it.
Celiac disease is a multifactorial disease of **immune** nature in which the etiological basis lies in an **immune hypersensitivity** to **gluten**. Both are **common genetic diseases** that cause gastrointestinal symptoms such as diarrhea, bloating and flatulence.

Inflammatory bowel diseases

They are a group of intestinal diseases characterized by a **chronic degree of inflammation**. Two types stand out: **ulcerative colitis** and **Crohn's disease**. The first one, is characterized by **inflammation** and **ulcers** in the **superficial** lining of the large intestine (colon) and rectum, while Crohn's disease is characterized by **inflammation** of the lining of the digestive tract, generally in the **deeper layers**.

Irritable bowel syndrome

It is a pathology for which the exact cause is not known, nor is there a specific test to diagnose it. It is a set of **functional disorders** characterized by the appearance of recurrent episodes of abdominal pain, bloating and alterations in the quantity and consistency of stool, **without altering the intestinal tissue**.

Selected genetic polymorphisms

The genetic variants studied for this report are shown below. They have been selected from the review of contrasted **scientific publications** and the implementation of **statistical models** so that their use in clinical care is considered useful.

Based on the results obtained, nutritional and lifestyle recommendations adapted to your profile are offered. It is essential that you follow your **prescriber's instructions** to avoid any **adverse reactions**, as **nutritional supplements may interact with medication**.

Gene	Rs identifier	Polymorphism	Results
6p21 (DQ2.5)	rs2187668	C>T	CC
6p21 (DQ8)	rs7454108	T>C	TT
AOC1	rs2052129	-691G>T	GT
AOC1	rs1049742	995C>T	CC
AOC1	rs10156191	47C>T	CT
CAT	rs1001179	-262C>T	CC
CYP2R1	rs10741657	-1127T>C	AG
FAAH	rs324420	385C>A	AA
FUT2	rs601338	Trp154Ter	GG
FUT2	rs1047781	418A>T	AA
GPX	rs1050450	599C>T	CC
IL10	rs1800896	-1082G>A	TT
IL10	rs1800872	-592T>G	TG
IL18	rs187238	-137C>G	CC
IL1A	rs1800587	-889G>A	AG
IL1B	rs1143634	315C>T	GA
IL23R	rs11465804	75358T>G	TT
IL6	rs1800795	-174C>G	GG
IL6	rs1800796	85+369G>C	GG
IRF1-AS1	rs2188962	131770805C>T	CT
LCT	rs4988235	-13910C>T	CC
NFE2L2	rs6721961	-617T>G	GG
NOD2	rs2066847	37732dup	DD
NOD2	rs2066845	30491G>C	GG
NQO1	rs1800566	C609T	GA

Gene	Rs identifier	Polymorphism	Results
OPRM1	rs1799971	118A>G	AG
PRDM1	rs7746082	106435269G>C	GG
PTPN2	rs2542151	12779947G>T	TG
SLC22A23	rs17309827	655-17229T>G	TG
SOD2	rs4880	Ala16Val	AG
TAB2/MAP3K7IP2	rs7758080	43020A>G	AA
TNFa	rs1800629	-308G>A	AG
TNFSF15	rs4263839	6969T>C	GA
TNFSF15	rs11554257	117605070T>C	TC
TNXB	rs17207986	2585A>G	TT
VDBP	rs7041	1296 T>G	AA
VDR	rs1544410	BsmI	CT

High risk	Normal risk	N/A

Lactose intolerance

Lactose is the sugar in milk. It is a **disaccharide** composed of **glucose** and **galactose**. The **enzyme lactase** cleaves lactose into the two monosaccharides which are absorbed in the intestine. The lactase activity depends on the LCT gene. In mammals, as they do not drink milk in adulthood, the gene reduces its expression and eventually stops synthesizing lactose.

When man started to take milk regularly in the adulthood, there has been an **adaptive mutation** of the LCT gene, which causes it not to be deprogrammed. In Spain, 20% of the population continues having two ancestor alleles, therefore becoming lactose intolerant. Intolerance usually manifests itself in childhood, but can also appear in adults who can spend years with digestive disorders without being related to a genetic intolerance lactose. It is therefore of interest to perform the genetic test.

Gene	Rs identifier	Polymorphism	Results
LCT	rs4988235	-13910C>T	CC

Consequences

The detected genotype for **LCT** gene is associated with the presence of lactose intolerance. This genotype leads to a decrease or cessation of lactase expression in adulthood.

This test only refers to primary lactose intolerance. It does not exclude other pathologies such as secondary intolerance, intestinal dysbiosis or of an immunological nature.

Recommendations

The lactose intolerance can be of a **very variable magnitude**. The degree of intolerance can be **mild** and **not lead to discomfort** with a normal consumption of lactose, however it must be borne in mind that the deficiency can be made patent with a higher consumption of lactose, and this can **increase with age**, eventually manifesting a **full intolerance**.

Therefore, in spite of not presenting symptomatology, it is recommended to:

- **Avoid dairy products** (especially milk and cheese) in the diet.
- If in doubt whether there is lactose in the food, **supplement** with **lactase enzyme**.

Celiac disease

Celiac disease is an autoimmune inflammatory disease that affects between 1 and 2% of the population. It is caused by an abnormal activation of the lymphocytes in the membranes of the small intestine, which is triggered by the gluten protein (gliadin) and occurs in people who are positive for the genetic markers **HLA-DQ2 or HLA-DQ8**.

Having a negative result for both markers excludes, with a 98% probability, a coeliac condition; 70% of non-celiacs can be excluded in this way. 30% of the population has one or two positive markers, in homozygosis or heterozygosis and **such positivity is necessary, but not sufficient**, for a diagnosis of coeliac disease. The definitive diagnosis must be made by a specialist, taking into account, in addition to genetic positivity, the patient's clinic, other laboratory tests and in some cases an intestinal biopsy.

There is also the so-called "intolerance or sensitivity to gluten", which requires other tests and is more heterogeneous and difficult to diagnose.

Gene	Rs identifier	Polymorphism	Results
6p21 (DQ2.5)	rs2187668	C>T	CC
6p21 (DQ8)	rs7454108	T>C	TT

Consequences

The results obtained **exclude** the **possibility** of current or future **gluten intolerance** with high, although not absolute, reliability. Approximately 95% of gluten-intolerant individuals carry one of the DQ2.5 or DQ8 risk alleles, but in a small proportion of cases this is due to other factors not analyzed in this study.

Recommendations

• In **asymptomatic people** this results excludes a risk of developing celiac disease with high reliability, and therefore there is **no medical reason** to **remove gluten** from the diet.

• In case of celiac-like **symptoms**, a **differential diagnosis** with other compatible disease, such as dyspepsia, inflammatory bowel syndrome or inflammatory bowel disease should be considered.

Diamine oxidase enzyme (DAO)

The **AOC1** gene (also represented as ABP1 or DAO1) encodes the diamine oxidase enzyme. This enzyme catalyzes deamination reactions of different nitrogenous substrates, such as putrescine or histamine. The protein is present in the membrane of most cell types, and is responsible for the **metabolism and degradation of histamine** in almost all tissues. Its expression is particularly high in the intestinal mucosa, and therefore plays an important role in the control of exogenous histamine from food.

Several **polymorphisms** in the AOC1 gene have been linked to a **lower expression of the gene**, and may favour an **intolerance to histamine**.

Gene	Rs identifier	Polymorphism	Results
AOC1	rs2052129	-691G>T	GT
AOC1	rs1049742	995C>T	CC
AOC1	rs10156191	47C>T	CT

Consequences

The genotypes detected correspond to **reduced DAO enzyme activity**, which is associated with a **higher risk** of primary histamine intolerance compared to the general population.

Recommendations

Since your risk assessment falls into the category of increased risk for primary histamine intolerance, it would be especially beneficial for you to adopt the following recommendations:

• Follow a **low histamine diet** reducing those foods with concentrations higher than **20 mg/kg**. The right combination of foods, keeping histamine intake as low as possible, allows to cover all the nutritional needs of the patient.

• **Avoid**:
- **Dairy products**: fermented, cured and dry cheeses.
- **Bread** made with artificial colors, preservatives and yeast.
- **Legumes**: soybeans.
- **Vegetables**: eggplant, fermented cabbage (xukrut), spinach, fermented vegetable products soy derivatives.

- **Animal protein**: frozen fish, canned blue fish and seafood.
- **Sweets**: made with colorants, artificial yeasts, preservatives, margarine, cakes, industrial pastries, fermented syrups.
- **Beverages**: red wine, cider, alcoholic beverages.
- **Foods with flavorings or dressings**, soy sauce, vinegar and balsamic vinegar, prepared sauces (ketchup), processed fats or fats with preservatives.

• Avoid **high alcohol intake** as it competes for the same metabolic pathways as histamine, decreasing the amount of DAO enzyme in the intestinal mucosa even in healthy individuals without genetic deficiency of the enzyme.

• Increase the consumption of foods and/or supplements with antihistamine activity such as **beta-glucans** (present in mushrooms, cereals and algae), **bromelain** (present in pineapple), **quercetin** (onion, green tea, apples, grapes, berries and Brassica vegetables (cabbage, broccoli, cauliflower, turnips).

• Avoid deficiency of **vit. C** and **B6** and the minerals **zinc** and **magnesium**, which are cofactors of DAO.

• In case of ingesting food rich in histamine, it is recommended to take a **DAO supplement**.

• It is very important to **treat digestive problems** (**inflammatory** bowel disease, **dysbiosis**, **Crohn's disease**, ulcerative **colitis** or **celiac** disease) and any process involving **alterations** in **intestinal and mucosal permeability**. These alterations cause, in itself, a deficit of the enzymes in charge of metabolizing histamine.

• If necessary, your prescriber will assess modifications in treatments with certain drugs that could interact with DAO:

- **DAO activity inhibitors** such as: analgesics (metamizole, acetylsalicylic acid), antihistamines (diphenhydramine), antiasthmatics (theophylline), antidepressants (amitriptyline), diuretics (furosemide), expectorants and mucolytics (acetylcysteine), antimalarials (chloroquine), antibiotics (clauvanic acid), antiemetics and prokinetics (metoclopramide), tranquilizers (diazepam), ...

- **Drugs with endogenous histamine-releasing activity** such as: analgesics (diclofenac, naproxen), anesthetics (prolocaine), antitussives (codeine), cytostatics (cyclophosphamide), etc.

Irritable bowel syndrome

Irritable bowel syndrome (IBS) is a **chronic** and **benign functional disorder** of the gastrointestinal tract. Although the mechanisms involved in its development are not known, the main symptoms are **abdominal pain** or **discomfort**, **bloating** and **altered bowel habit**, with alternating episodes of diarrhea and constipation. Although the symptoms of IBS can cause a lot of discomfort, there is no damage to the **intestinal tissue**.

It is known that the **central nervous system** (CNS) is in **permanent relationship** with the **gastrointestinal tract** and that, this connection is **bidirectional**. This explains why it has been found that the presence of **psychological factors** can **aggravate** or **modulate** the symptomatology of IBS.

This disorder is characterized by **periods of exacerbation** alternating with **periods of remission** of symptoms. Its prevalence is 10-15% and it is more common in adults and young people, as it begins to decrease after the age of 50.

Gene	Rs identifier	Polymorphism	Results
FAAH	rs324420	385C>A	AA
IL10	rs1800896	-1082G>A	TT
OPRM1	rs1799971	118A>G	AG
TNFSF15	rs4263839	6969T>C	GA

Consequences

Genetic variants associated with **irritable bowel syndrome** have been detected in a higher than normal. This does not imply the diagnosis of any pathology at present, nor does it predict that you will have it in the future. However, if at any time you experience gastrointestinal problems without a clear cause, your doctor should consider irritable bowel syndrome as part of your diagnosis.

Inflammatory bowel diseases

These are disorders that have **chronic inflammation** of the **digestive tract** as a common symptom. They are characterized by being **immune-mediated**, **inflammatory** and **chronic diseases** that evolve in **outbreaks**, that is. with **active phases** and periods of **remission**. They have the capacity to alter food digestion and nutrient absorption and, in addition, they share clinical and pathological characteristics. Two of them stand out, **ulcerative colitis** and **Crohn's disease**:

• **Ulcerative colitis** causes **inflammation** and **ulcers** in the digestive tract, mostly affecting the more **superficial areas** of the colon and rectum.

• **Crohn's disease** can affect any part of the digestive tract and the **inflammation** usually reaches **deeper layers**.

Gene	Rs identifier	Polymorphism	Results
IL23R	rs11465804	75358T>G	TT
IRF1-AS1	rs2188962	131770805C>T	CT
NOD2	rs2066847	37732dup	DD
NOD2	rs2066845	30491G>C	GG
PRDM1	rs7746082	106435269G>C	GG
PTPN2	rs2542151	12779947G>T	TG
SLC22A23	rs17309827	655-17229T>G	TG
TAB2/MAP3K7IP2	rs7758080	43020A>G	AA
TNFSF15	rs11554257	117605070T>C	TC
TNXB	rs17207986	2585A>G	TT

Consequences

The overall **genetic variants** detected is within the **normal** range. The results do not suggest an increased risk of developing inflammatory bowel disease.

Inflammatory mediators

Interleukins (**IL**) and **tumor necrosis factor** (**TNF**) are a set of molecules called **cytokines** that have the function of establishing communication and coordinating the response of the immune system. They are involved in the activation and deactivation of cells of the immune system and in the inflammatory response.

Polymorphisms of some IL are known to affect their level of expression, so that, given the **same level of stimulus**, people can synthesize **more or less quantity of a certain IL**, depending on their genotype. Knowledge about the genotype allows to minimize the risk of suffering from certain inflammatory processes.

Gene	Rs identifier	Polymorphism	Results
IL10	rs1800872	-592T>G	TG
IL10	rs1800896	-1082G>A	TT
IL18	rs187238	-137C>G	CC
IL1A	rs1800587	-889G>A	AG
IL1B	rs1143634	315C>T	GA
IL6	rs1800795	-174C>G	GG
IL6	rs1800796	85+369G>C	GG
TNFa	rs1800629	-308G>A	AG

Consequences

The detected genotypes for the studied genes are associated with a **particularly higher** global **risk of inflammation** compared to the general population.

Recommendations

Because your risk assessment falls into the higher genetic risk category, you would benefit the most from adopting the following recommendations:

• **Avoid** unhealthy **weight gain**, as they directly generate low-grade inflammation.

• **Include omega-3** fatty acids (fish oil, krill, and / or chia or flax seeds) in the diet and / or assess the possibility of supplementation.

• **Avoid foods** rich in **trans fatty** acids present in **ultra-processed foods**, foods rich in **saturated fatty acids and omega-6 with arachidonic acid**, especially mammalian meat, poultry and dairy products and, to a lesser extent, eggs.

• **Reduce oxidative stress** by increasing the intake of **fruits** (citrus, red fruits ...) and **vegetables** (cruciferous, garlic, onion) rich in antioxidants (vitamins A, C and E, zinc, quercetin ...).

• **Reduce simple sugars** (white, cane or brown sugar), as they are inflammatory and acidifying.

• **Consume green tea** that is rich in flavonoids and anti-inflammatory catechins.

• **Incorporate spices** such as **turmeric**, rich in curcumin, **ginger**, **oregano**, **basil**, **thyme**, **peppers** or **stevia** in the diet.

• **Red grapes**and **red wine** are a source of **resveratrol**, which is a powerful antioxidant.

• **Consume** foods rich in **L-arginine** in oily fish (longfin tuna, mackerel or pompano), walnuts and grapes. In the case of a deficient diet, your physician may assess the use of supplements.

• **Avoid toxic habits** such as excessive consumption of **alcohol** and **tobacco** as well as **minimize** exposure to environmental **pollutants** and **toxins**.

• **Reduce stress** and get a minimum of 8 quality hours of sleep, as well as **regular moderate exercise**.

• **Treat digestive problems** and any process that causes alteration of intestinal permeability and mucous membranes (dysbiosis, irritable bowel, ulcerative colitis, celiac disease ...).

• In case of acute inflammation, your physician will assess prescribing **supplements** that reduce inflammation such as: **willow, devil's claw, boswellia, cat's claw, arnica** ... among others.

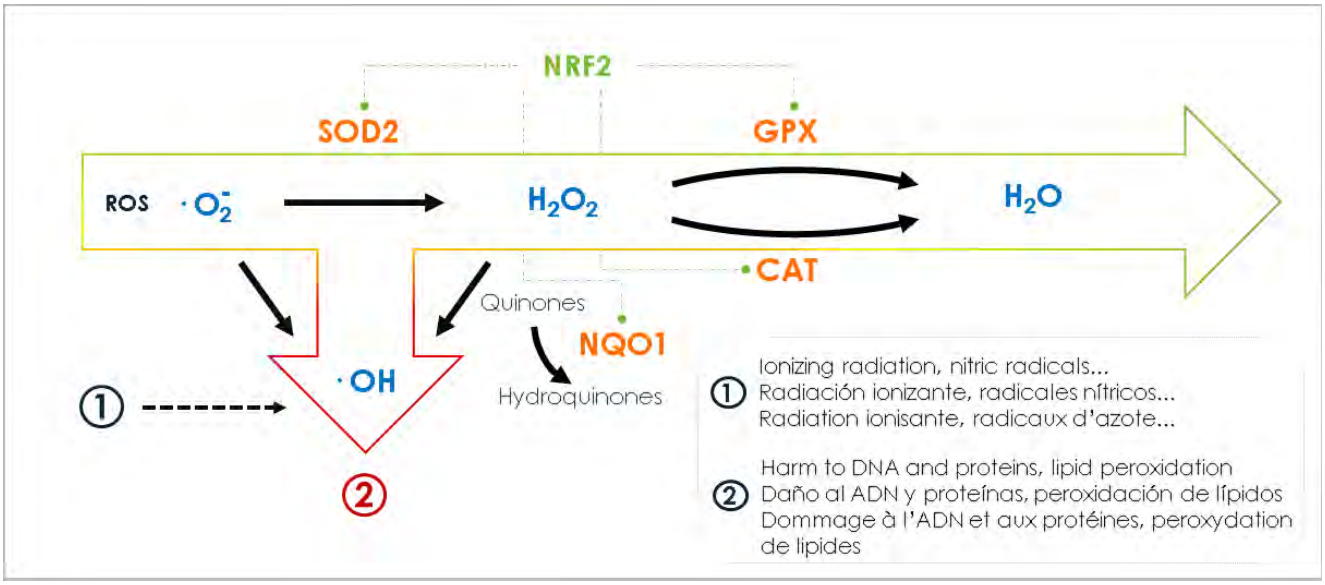
Antioxidant defense

Oxidative stress occurs through the generation of **reactive oxygen species** (ROS) that can damage cells, DNA and proteins, influencing the development of **degenerative, cardiovascular diseases** or various types of **cancer**. **ROS** are **generated** in particular metabolic processes, such as **inflammation**, but also from the simple act of **breathing oxygen**.

These harmful substances are neutralized thanks to the action of various enzymes. Among them, **SOD2** constitutes the **first step** in this **antioxidant pathway**. It transforms **superoxide radicals** (a type of ROS) that are produced by many redox reactions into **hydrogen peroxide** (H2O2), which is then converted to **water** (H2O) by the **CAT** and **GPX** enzymes. These three form the **first line** in the antioxidant defense.

Furthermore, the **NQO1** enzyme is involved in the **reduction of quinones** to hydroquinones. This prevents these quinones from producing more ROS from hydrogen peroxide and therefore results in a **protection of cells from oxidative stress** .

Finally, the activation of these and many other enzymes is **modulated** by the **NRF2 factor**, which is considered a **master regulator** of the antioxidant system. This allows increasing the production of enzymes in the face of **oxidative overload** by activating their genes. Polymorphisms in the NRF2 gene (NFE2L2) decrease its regulatory capacity and, thus, the response under **high oxidative stress.**



Gene	Rs identifier	Polymorphism	Results
CAT	rs1001179	-262C>T	CC
GPX	rs1050450	599C>T	CC
NFE2L2	rs6721961	-617T>G	GG
NQO1	rs1800566	C609T	GA
SOD2	rs4880	Ala16Val	AG

Consequences

The detected genotype for the enzyme genes is related to a slightly reduced antioxidant defense compared to non-variant homozygotes.

Recommendations

To increase the antioxidant activity:

• **Avoid** the **induction of Phase I detoxification** produced by **benzopyrenes**, **polycyclic aromatic hydrocarbons** (PAH), **heterocyclic aromatic amines** (AAH), **acrylamides**, **furans**, **nitrosamines**, **insecticides**, **herbicides**, **pesticides**, **heavy metals**, **PVC plastics**, **aflatoxins**... because they generate a lot of oxidative and nitrosative stress.

• **Follow** a diet rich in **micronutrients** and **antioxidants**, increasing the consumption of **group B vitamins:** B6, B9, B12, B2, B3, **vitamins C and E and resveratrol**.

• **Avoid foods rich in trans fats** present in industrially produced foods with vegetable oils such as chips, salted snacks, pastries, cookies...

• **Avoid excessive physical activity** in time and intensity (marathon, triathlon, etc.), as it increases oxidative stress. However, **moderate physical activity** (walking, swimming, jogging), is **beneficial** to health.

• **Avoid alcohol consumption** because it is one of the main factors affecting the redox mechanism (oxidation and reduction reactions) that generates high production of free radicals derived from oxygen and nitrogen and that affects the respiratory chain of the mitochondria.

Supplementation

Although the risk model with the results obtained does not indicate a need for the use of supplements, they may be useful in case of insufficient intake through the diet.

- Betacarotenes
- Lycopene
- Resveratrol
- Vitamin E
- Tumeric
- Fermented Papaya
- Vitamin C

Vitamin D

Vitamin D, also called calciferol, is an essential fat-soluble vitamin. It can be obtained in three ways:

- By **exposing** the skin cells to the sun's rays (UV). However, when wearing clothes and using sunscreens the synthesis by the action of the sun's rays is very low.
- By eating Vitamin D**rich food**, such as sardines, tuna, mackerel and salmon, as well as milk and eggs.
- Nutritional **supplements**, the most common way to maintain adequate levels.

Vitamin D deficiency is related to many pathologies among which **osteoporosis**, **hypocalcemia** and **osteomalacia** stand out.

The action of vitamin D may be influenced by polymorphisms in several genes: **CYP2R1** (Cytochrome P2R1) responsible for synthesizing active vitamin D, **VDBP** (Vitamin D Binding Protein) to which it is binded for transport, and **VDR** (Vitamin D Receptor) responsible to codfy its receptor in the tissues.

Gene	Rs identifier	Polymorphism	Results
CYP2R1	rs10741657	-1127T>C	AG
VDBP	rs7041	1296 T>G	AA
VDR	rs1544410	BsmI	CT

Consequences

The detected genotype for **CYP2R1** is associated with a slightly reduced levels of endogenous synthesis of active vitamin D, which may increase the risk of vitamin D deficiency.

The detected genotype for **VDBP** is related to higher affinity of the binding protein for vitamin D, which will result in a lower than expected levels of free vitamin D, which may increase the risk of a functional vitamin D deficit.

The detected genotype for **VDR** is related to a slightly reduce affinity of the receptor for vitamin D, which results in a decrease vitamin D efficiency.

Recommendations

Recommendations

• **Ensure** adequate **vitamin D** intake. If it is difficult to obtain it through the diet, an additional intake through nutritional supplements is suggested.

• **Consume** foods rich in **vitamin D3**.

• **Prefer calcium-rich products** to optimize bone mineralization.

• **Limit** consumption of products rich in **phosphorus** to a maximum of 700 mg per day. Also **avoid** foods bearing the code **E 338-452**, under which phosphates are hidden on food labels.

• **Consume** foods rich in **flavonoids** (present in tea, garlic, onions, blueberries, grapes...).

• **Practice** regular **physical activity**.

• Unless there are other risk factors that contraindicate it, it is good to **expose yourself to sunlight** without protection for about 10-15 minutes a day, on a regular basis and with the maximum number of cells exposed to the sun. Afterwards, apply sunscreen and avoid excessive sun exposure in the middle of the day.

FUT2 - Fucosyltransferase 2

FUT2 gene encodes the enzyme 2-galactoside alpha-L-fucosyltransferase 2. The enzyme catalyzes the transfer of L-fucose to glycoproteins on the cell surface. In the gastrointestinal system, these surface molecules condition the intestinal microbiota, while in blood cells it affects the synthesis of the H antigen, which is the final step in the secretion of soluble **ABO blood group** antigens into the body fluids. For this reason, the phenotype defined by this variant is called the **secretory state**.

The presence or not of these fucosylated structures in the intestine modifies the interaction between the tissue and the microorganisms and conditions the **composition of the intestinal biota**, as well as the **resistance to some pathogens**. In addition, the secretory phenotype is also strongly related to **circulating levels of vitamin B12**.

Gene	Rs identifier	Polymorphism	Results
FUT2	rs601338	Trp154Ter	GG
FUT2	rs1047781	418A>T	AA

Consequences

The genotype detected for **FUT2** predicts a **secretory phenotype**. This implies:

- **Lower natural resistance** to norovirus, rotavirus or *Helicobacter pylori* infections.

- Tendency to **lower vitamin B12 levels**, and **increased vulnerability** to **vitamin B12 deficiency**.

Bibliography

To create this report, we have been reviewed contrasted scientific publications , which are available upon request. Below there is a selection of the most relevant ones.

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Additional information

The genetic polymorphisms included in this report have been selected on the basis of scientific publications that endorse their interpretative value for predicting individual health risks.

There are changes in genes, which do not directly cause disease, but which alter the activity of an enzyme, a transport protein or a receptor, which may themselves condition metabolic dysfunction, or in association with other variants. Therefore, they can predispose to diseases or health alterations, if life habits are not implemented and if appropriate nutritional or pharmacological supplements are not used.

Its objective is to make a risk prediction, i.e. Predictive Medicine, in order to implement a Personalized Preventive Medicine.

The genetic polymorphisms that appear in this report are not directly a specific diagnosis, but a complementary help for the health professional who has requested them. Consequently, it is solely responsible for the conclusions and recommendations to the patient that it deems appropriate in each case, regardless of what can be stated in general terms in this report.

It is the responsibility of the health care professional to incorporate the data in this report and any recommendations that may arise from the interpretation of these polymorphisms into the patient's medical record, along with other results from conventional analyses or other complementary explorations.

This report may contain lists of suggested foods based on their nutrient content that may be beneficial to the patient. However, such foods may not be indicated for food intolerances, allergies, specific diets, or medications that the patient may be taking. Therefore, this report and its contents should be reviewed together with the prescribing physician and decide within the suggested foods which to take. If not, establish other food supplements.

The processing laboratory is responsible for the accuracy of the results obtained, but the interpretation of the results is the responsibility of the health professional who requested them.

The genetic results presented do not allow us to conclude with certainty about the development of a disease or its susceptibility, because the tests carried out do not allow us to consider all the factors that contribute to the relative risk of a given susceptibility or of the possible evolution of a disease. Complex variables such as the degree of risk to develop adverse effects to drugs, or to suffer from multifactorial diseases in which genetic factors are not totally determinant are also relevant.


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