MyNutriGenes®

Nutrigenetic Report

CASE INDEX	
Name:	**N.A.**
Gender:	**N.A.**
Date of birth:	**N.A.**
Age:	**N.A.**
Ethnicity:	**N.A.**
Referral number:	**N.A.**
Reason:	**Nutritional plan adequacy**
Purpose:	**Nutrigenetics**
Specimen type:	**N.A.**

CUSTOMER INSTITUTION	
Referring physician:	**N.A.**
Reference:	**N.A.**
Institution:	**N.A.**
Requisition date:	**N.A.**
Fulfillment date:	**N.A.**

1. WHAT IS ANALYZED IN THIS GENETIC TEST?

This genetic test analyses your DNA with the objective of evaluating 79 genetic variants in 53 genes which are decisively associated with nutrition and weight management.

The result achieved, called the genetic profile, is unique for each individual and could play a key role in delineating a personalised nutritional plan.

The associations identified between the genes studied and the body's response to food intake are corroborated by international standard scientific studies referred in this report.

2. IMPORTANT DISCLAIMER

Nutrigenetics is a science that studies the association between genes and each individual's response to nutrient intake. The use of information on genetic predisposition to establish a nutritional plan should be integrated with information on physical characteristics (e.g. age, gender, muscle mass index, etc.) and behavioural information (e.g. eating habits, physical activity, etc.).

The genetic test results cannot be used for clinical diagnostics, for disease prevention or for the identification of a clinical condition.

The genetic test result do not depend on the physical or clinical condition or on the therapeutic management of the individual tested.

3. WHAT YOUR GENES SAY ABOUT:

Your body

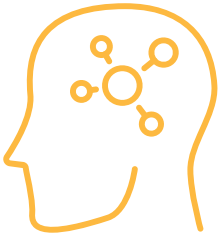
Discover your body structure genetic predisposition.

Body Mass Index (BMI), waist-hip ratio, abdominal fat, insulin resistance, LDL cholesterol, triglycerides, weight, appetite control

Your diet

Discover your actionable nutritional plan ideal for your body.

Carbohydrates, fiber, fats, protein, vitamins, salt, caffeine



Your behaviour

Discover your behavioural actionable plan ideal for healthy weight management.

Appetite control, sleep, physical exercise.

4. SUMMARY OF YOUR GENETIC PROFILE

Your body

Discover your body structure genetic predisposition.

Genetics influences the body structure of each individual, contributing to weight control and nutritional metabolism. The impact of your genetic predisposition on body composition can be modified by food and behaviour appropriate to your genetic profile.

Here you have your genetic predisposition for the following body characteristics. For more details, see the section 5.1.

OVERWEIGHT

High BMI

High waist-hip ratio

Accumulation of abdominal fat

Low risk — High risk

METABOLISM

Insulin resistance

High LDL cholesterol

High triglycerides

Low risk — High risk

WEIGHT MANAGEMENT

Weight loss difficulty

Lost weight regain

Difficulty in controlling appetite

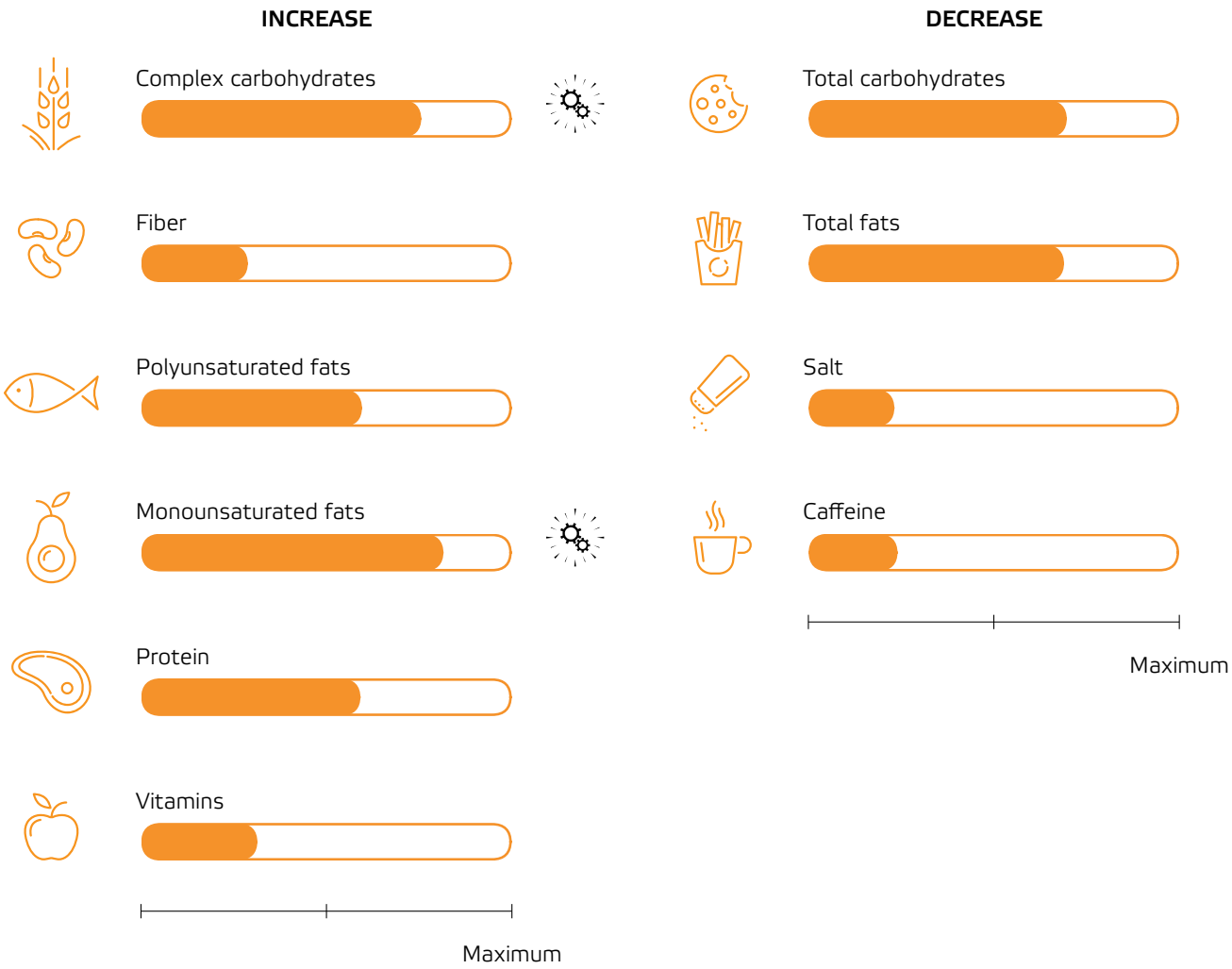
Low risk — High risk

Your diet

Discover your actionable nutritional plan ideal for your body.

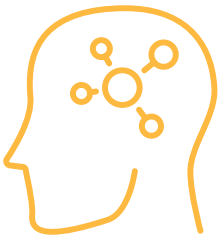
By adopting the most appropriate nutritional plan for your genetic predisposition you will be improving your body's performance. Find out the foods that are most suited to you, and which actions you should prioritise.

Your genes suggest the following nutritional plan for maintaining a healthy weight. For more details, see the section 5.2.



If you find this symbol next to one or more bars, it is because you have a favourable genetic predisposition in that area.

If you find this symbol next to one or more bars, consider the recommended actions to obtain more health benefits.

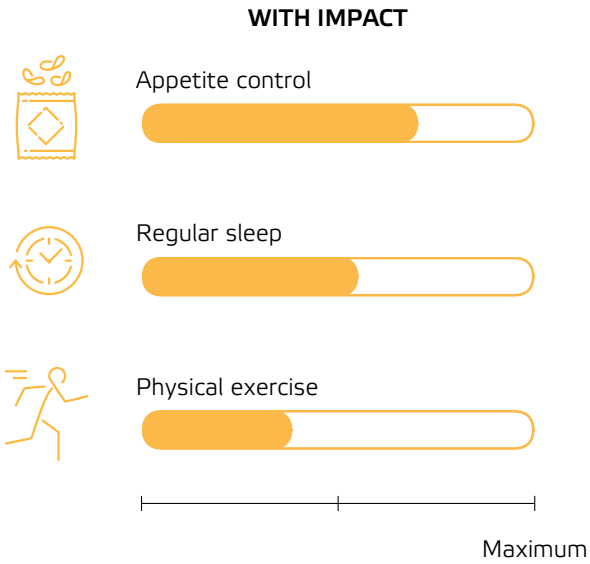


Your behaviour

Discover your behavioural actionable plan ideal for healthy weight management.

Controlling appetite, adopting a regular sleep pattern and practicing physical exercise are behaviours impacting weight management. Take your genetic predisposition into account for these traits and follow recommendations for the best results.

Your genes indicate how certain behaviours can have a significant impact on managing a healthy weight. For more details, see the section 5.3.



If you find this symbol next to one or more bars, it is because you have a favourable genetic predisposition in that area.

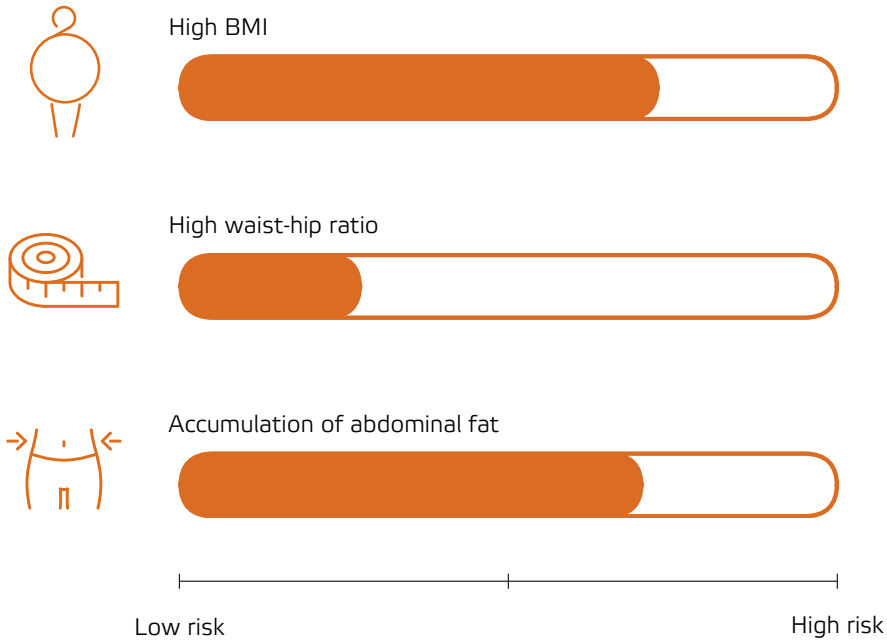
If you find this symbol next to one or more bars, consider the recommended actions to obtain more health benefits.

5. YOUR GENETIC PROFILE DETAILS

5.1. YOUR BODY

5.1.1. Overweight

Excess weight and an imbalance of body fat distribution result from a caloric intake that exceeds the body's needs. In assessing excess weight, the following parameters were considered: Body Mass Index (BMI), waist-hip ratio, and accumulation of abdominal fat. In order to maintain a healthy weight, it is essential to maintain the balance between calorie intake and energy expenditure.



The following table illustrates the genes that contribute to your genetic profile in the overweight subarea:

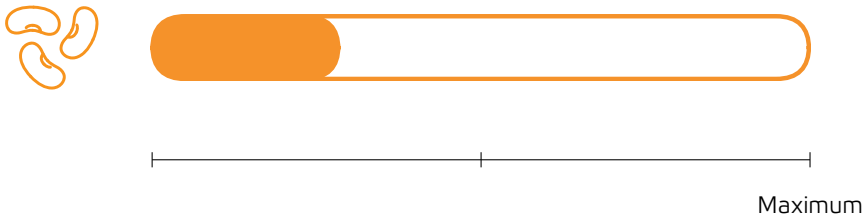
APOA1	*APOB*	*CLOCK*	*FTO*	*GIPR*	*GRB14*	*LYPLAL1*
MC4R	*MSRA*	*PCSK1*	*PER2*	*PPARG*	*PROX1*	*TCF7L2*
TFAP2B						

With impact Neutral

Your biggest risks associated with being overweight:

- Predisposition to a high body mass index (BMI).
- Predisposition to build up abdominal fat.

5.2.2. Increase fibers



Impact:

Your genetic results suggest that you benefit from a nutritional plan enriched in fiber.

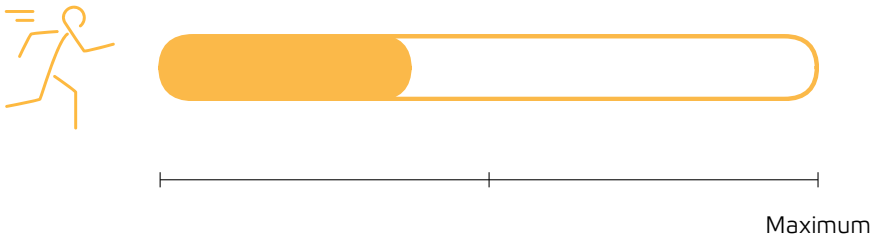
Your benefits associated with the increased consumption of fibers:

- Increased HDL cholesterol levels.
- Reduced levels of triglycerides, total cholesterol and LDL.
- Weight loss.

General information:

Dietary fibre is essential for the body's proper functioning, and is obtained from foods of plant origin. Insoluble fibre contributes intestinal transit regulation and provides beneficial effects for intestinal microflora. Soluble fibre fosters the feeling of satiety, since it absorbs large amounts of water in the intestinal tract and facilitates the formation of a type of gel that surrounds food, increasing its volume. This contributes to weight management, as it regulates the absorption of various nutrients, mainly sugars, fats and cholesterol.

5.3.3. Physical exercise impact



Impact:

Your genetic results suggest that you benefit from the practice of physical exercise.

Your benefits associated with the practice of physical exercise:

- Better weight loss results.
- Decreased BMI.
- Increased HDL cholesterol levels.

General information:

Physical exercise is important for weight management and the body's metabolic balance. Along with an appropriate diet, it enables the reduction of BMI, the loss of fat mass and excess weight and prevents the gain of lost weight. From a metabolic standpoint, it enables the reduction of LDL cholesterol and the reduction of insulin resistance. Various types of exercise can be recommended according to specific goals. For example, if the goal is to lose abdominal fat, resistance exercises will be the most suitable. On the other hand, more intense exercises contribute to the regulation of hormones associated with appetite. Genetics plays a very important role in the area of exercise associated with weight management. It is known that certain genes associated with body composition, lipid metabolism, insulin resistance and appetite control are conditioned by the practice of physical exercise.

of existing regulations applicable in its partners' home countries.

7. APPENDIX

7.1. GENETIC INFORMATION

The table below presents the genetic variants that have been identified as relevant for the design of a personalized nutritional plan. The results are described according to the HGVS nomenclature (`http:www.hgvs.org`), accessed on June 2018.

This genetic test identified 51 genetic variants (out of 79 analysed), with a significant impact for the design of a personalized nutritional plan.

Gene	Genetic variant references		Nucleotidic change [1]	Aminoacidic change	Impact allele
	HGMD	Ensembl			
ADIPOQ	CR052432	rs17300539	c.-1138A>G	–	G
AHR	–	rs6968865	g.17287269A>T	–	TA
AHR	–	rs4410790	g.17284577T>C	–	CT
APOA1	CR900263	rs670	c.-113A>G	–	G
APOA5	CM023881	rs3135506	c.56G>C	p.Trp19Ser	G
APOA5	CR033141	rs662799	c.-620C>T	–	T
APOB	–	rs512535	c.-965A>G	–	G
APOE	CM860003	rs7412	c.526C>T	p.Arg176Cys	C
APOE	CM900020	rs429358	c.388T>C	p.Cys130Arg	T
CLOCK	CR984677	rs1801260	c.*213T>C	–	TC
CLOCK	CR121503	rs3749474	c.*897G>A	–	AG
CLOCK	–	rs1554483	c.982+247G>C	–	GC
CRY2	–	rs11605924	c.32+4259A>C	–	A
CYP1A1	–	rs2472297	g.74735539C>T	–	CT
CYP1A1	–	rs2470893	c.-1694G>A	–	GA
DHCR7	–	rs12785878	c.146+1233T>G	–	GT
FABP2	CM950433	rs1799883	c.163G>A	p.Ala55Thr	AG
FADS1	CR1510437	rs174546	c.*53A>G	–	GA
FTO	CS088104	rs8050136	c.46-27777C>A	–	CA
FTO	CR119358	rs1421085	c.46-43098T>C	–	CT
FTO	–	rs1121980	c.46-34805G>A	–	AG
FTO	CR119357	rs1558902	c.46-40478T>A	–	AT
FTO	CS076623	rs9939609	c.46-23525T>A	–	AT
FUT2	CM042988	rs602662	c.772A>G	p.Ser258Gly	GA
GC	–	rs2282679	c.*26-796A>C	–	CA
GIPR	–	rs2287019	c.886+14T>C	–	C
GRB14	–	rs10195252	g.165513091C>T	–	TC
GRK4	CM025430	rs1024323	c.425C>T	p.Ala142Val	TC
GRK4	CM025429	rs2960306	c.194G>T	p.Arg65Leu	GT
IL6	CR983402	rs1800795	c.-237G>C	–	CG
IRS1	CR096329	rs2943641	g.227093745TC>T	–	C
LIPC	CR971949	rs1800588	c.-557C>T	–	C
LPL	CS931395	rs320	c.1322+483G>T	–	T
LPL	CM900164	rs328	c.1421G>C	p.Term474Ser	C
LYPLAL1	–	rs2605100	g.219470882A>G	–	AG
MC4R	–	rs11152221	g.60350016C>T	–	TC
MC4R	–	rs17700633	g.60262199G>A	–	GA
MC4R	CM030481	rs2229616	c.307A>G	p.Ile103Val	G
MC4R	CM030483	rs52820871	c.751C>A	p.Leu251Ile	A
MMAB	–	rs2241201	c.*2701C>G	–	G
MTHFR	CM950819	rs1801133	c.665C>T	p.Ala222Val	TC
MTHFR	CM981315	rs1801131	c.1286A>C	p.Glu429Ala	CA
NR1D1	–	rs2314339	c.370+106A>G	–	AG

PCSK1	CM132638	rs6234	c.1993C>G	p.Gln665Glu	CG
PCSK1	CM1311914	rs6235	c.2069C>G	p.Thr690Ser	GC
PER2	–	rs2304672	c.-12C>G	–	CG
PPARD	CR035869	rs2016520	c.-87C>T	–	TC
PROX1	–	rs340874	c.-68+2590T>C	–	CT
SIRT1	–	rs1467568	c.1916-864A>G	–	AG
TCF7L2	CS065626	rs7903146	c.382-41435C>T	–	TC
TFAP2B	–	rs987237	c.602-724A>G	–	AG

[1] The numeric identification associated with each variant is indexed to a reference sequence obtained from Ensembl database (`http://www.ensembl.org/index.html`).

The table below presents the genetic variants that have no impact on the definition of your nutrition plan.

Gene	Genetic variant references		Nucleotidic change [1]	Aminoacidic change	Non-impact allele
	HGMD	Ensembl			
ADD1	CM021240	rs4961	c.1378G>T	p.Gly460Trp	G
ADRB2	CM950016	rs1042713	c.46A>G	p.Arg16Gly	A
ALPL	–	rs4654748	c.134-9113T>C	–	T
APOA2	CR024268	rs5082	c.-323T>C	–	TC
APOA5	CM032546	rs2075291	c.553G>T	p.Gly185Cys	G
BCMO1	CM091858	rs7501331	c.1136C>T	p.Ala379Val	TC
BCMO1	CM091857	rs12934922	c.801A>T	p.Arg267Ser	A
CLCNKA	–	rs848307	n.530+427C>T	–	C
CRY1	–	rs2287161	c.-562G>C	–	CG
CYP1A2	CR993820	rs762551	c.-9-154C>A	–	A
DRD2	CM041241	rs1800497	c.2137G>A	p.Glu713Lys	G
GHSR	CR084002	rs490683	g.172175074C>G	–	C
LIPC	CR002154	rs2070895	c.-293G>A	–	G
LPL	CS890131	rs285	c.1019-1582C>T	–	CT
MC4R	–	rs12970134	g.60217517G>A	–	G
MC4R	–	rs17782313	g.60183864T>C	–	T
MSRA	–	rs545854	g.9860080C>G	–	C
MTNR1B	CR110512	rs10830963	c.223+5596C>G	–	C
NR1D1	–	rs12941497	c.31+723C>T	–	C
OPRM1	CM003770	rs1799971	c.118A>G	p.Asn40Asp	AG
PCSK1	CM083013	rs6232	c.661A>G	p.Asn221Asp	A
PER2	–	rs4663302	g.238295120C>T	–	C
PPARG	CM981614	rs1801282	c.34C>G	p.Pro12Ala	C
PPM1K	–	rs1440581	n.133-6526T>C	–	TC
RS12272004	–	rs12272004	g.116733008C>A	–	C
SLC23A1	CM0911294	rs33972313	c.790G>A	p.Val264Met	G
SLC2A2	CM941277	rs5400	c.329C>T	p.Thr110Ile	C
SOD2	CM962694	rs1799725	c.47T>C	p.Val16Ala	TC

[1] The numeric identification associated with each variant is indexed to a reference sequence obtained from Ensembl database (`http://www.ensembl.org/index.html`).

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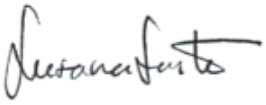

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(Validation responsibility)

The CLOCK protein is a transcriptional activator of several key genes that regulate the circadian rhythm. It thereby influences the balance between energy expenditure and fat, carbohydrate and protein metabolism, among other biological processes. Carriers of the G allele have a predisposition to higher Body Mass Index (BMI) [17].

CLOCK, CR121503 / rs3749474

The CLOCK protein is a transcriptional activator of several key genes that regulate the circadian rhythm. It thereby influences the balance between energy expenditure and fat, carbohydrate and protein metabolism, among other biological processes. Studies of genotype-phenotype association show that A allele carriers have a predisposition to an increased caloric intake and to a higher Body Mass Index (BMI) [18, 19, 20]. They benefit from a hypoenergetic diet with decreased fat intake, in order to achieve better weight loss results [21].

CLOCK, CR984677 / rs1801260

The CLOCK protein is a transcriptional activator of several key genes that regulate the circadian rhythm. It thereby influences the balance between energy expenditure and fat, carbohydrate and protein metabolism, among other biological processes. Studies of genotype-phenotype association show that C allele carriers have a predisposition to higher Body Mass Index (BMI) and greater difficulty in losing weight [18, 20, 22]. This allele is also associated with short-time sleepers with increased levels of the hunger-inducing hormone ghrelin, and with increased saturated fat intake [18, 20, 22]. Sleeping an appropriate number of hours and having a regular sleep pattern+D91 is beneficial to decrease ghrelin levels in those carrying this allele [23].

CRY2, – / rs11605924

The CRY2 protein participates in the regulation of the circadian rhythm and influences the balance between energy expenditure and food intake. Genotype-phenotype association studies show that A allele carriers have a predisposition to higher energy expenditure. They benefit from a regular sleep pattern of more than 6-7 hours to increase HDL cholesterol levels [24, 25].

CYP1A1, – / rs2470893

The SNP rs2470893 is localized in the bidirectional promoter of the *CYP1A1* and *CYP1A2* genes. Variations in this region may alter CYP1A2 expression, the main enzyme of the caffeine metabolism. Meta-analysis studies indicate that individuals carrying the C allele tend to ingest less caffeine [2, 3, 5, 26]. This allele is associated with decreased caffeine metabolism, as assessed by metabolite analysis [4].

CYP1A1, – / rs2472297

The SNP rs2470893 is localized in the bidirectional promoter of the *CYP1A1* and *CYP1A2* genes. Variations in this region may alter CYP1A2 expression, the main enzyme of caffeine metabolism. Meta-analysis studies indicate that individuals carrying the C allele tend to ingest less caffeine [2, 5, 27]. This allele is associated with decreased caffeine metabolism, as assessed by metabolite analysis [4].

CYP1A2, CR993820 / rs762551

The CYP1A2 enzyme is associated with caffeine metabolism. Studies of genotype-phenotype association show that CYP1A2 mutations result in different levels of enzymatic activity and therefore in different levels of caffeine metabolism, namely fast and slow metabolism. Carriers of the AA genotype have increased caffeine metabolism, i.e. they are fast metabolizers [28, 29, 30, 31].

DHCR7, – / rs12785878

The DHCR7 enzyme is involved in the production of cholesterol from 7-dehydrocholesterol, a precursor of vitamin D. Increased DHCR7 activity reduces the 7-dehydrocholesterol that is available for the production of vitamin D through sunlight exposure. The G allele is associated with increased activity of the DHCR7 enzyme, and therefore carriers of this allele present lower plasma levels of vitamin D. For those, this vitamin should be obtained from external sources [32]. A hypoenergetic protein-rich nutrition plan has a positive influence on vitamin D levels and promotes a decrease in insulin resistance [33].

FABP2, CM950433 / rs1799883

The FABP2 protein is involved in the regulation of the intestine's fat absorption, also influencing insulin sensitivity. Studies of genotype-phenotype association show that A allele carriers have a predisposition to a higher absorption of fats by the intestine [34]. Carriers of this allele have a predisposition to higher LDL-cholesterol levels and greater difficulty in losing weight [35, 36, 37]. They benefit from a nutritional plan with low saturated fat and enriched in complex carbohydrates and monounsaturated fat as it reduces insulin resistance [38, 39].

FADS1, CR1510437 / rs174546

Long-chain polyunsaturated fatty acids (LC-PUFAs) play a role in several biological processes, as components of cell membranes and acting as signalling molecules, regulating gene expression and body's inflammatory response [40]. LC-PUFAs can either be acquired through dietary intake or synthesised, by desaturases and elongases, from precursor essential fatty acids (linoleic and α-linoleic acids) [41]. Fatty acid desaturase 1 (or Delta-5 desaturase; D5D), encoded by the *FADS1* gene, is a key enzyme in the biosynthesis of LC-PUFAs. The rs174546 polymorphism, located in the 3'UTR of the *FADS1* gene, has been associated with D5D activity, which decreases with the number of copies of the A allele, with carriers of the AA genotype thus presenting the lowest activity [40, 42, 43]. Consequently, those harbouring an A allele have increased levels of desaturase substrates, i.e. precursor essential fatty acids, and decreased levels of products (LC-PUFAs) [42, 43]. These carriers are therefore likely to benefit from an increased intake of foods rich in LC-PUFAs. Western diets tend to be deficient in fatty acids from the omega-3 family and enriched in those