NUTRITION Solutions

Smart DNA - Nutrition Package

Doctor report

Smart DNA - Nutrition Package

CONTENT

Smart DNA - Nutrition Package

CONTENT

1

Patient identification data

Patient identification data

1

Patient's name and last name	**SmartSalem Test Patient**
Date of Birth	**03-03-1973**
Gender	**Male**
Sample type	**Buccal mucosa**
Sample code	**NUT22926AA**
Sample date	**03-07-2024**
Report date	**07-08-2024**

Doctor's name and last name	**SmartSalem test doctor**
Email address	**testsmartsalemdoctor@mail.com**

DISCLAIMER

Fagron Genomics, S.L.U carries out genetic tests upon request by healthcare professionals, in relation to biological samples from patients obtained by the healthcare professional. Our tests do not replace a medical consultation, nor do they make up a diagnostic or treatment, nor should they be interpreted this way. Only healthcare professionals can interpret the results of said tests, based on their knowledge of the clinical records of the patients and other relevant factors and, under their responsibility, give a diagnostic or prescribe treatment to the patient. We decline all responsibility derived from the use and interpretation of the results of our tests by the solicitant healthcare professional. Fagron Genomics, S.L.U expressly reserves any legal actions in case of an inappropriate, negligent or incorrect use or interpretation of the results of our tests. It is the responsibility of the healthcare professional who requests a test to guarantee to the patient the appropriate genetic advice as foreseen by Law 14/2007, of 3rd July, of biomedical research. As Fagron Genomics, S.L.U does not have access to the personal identifiable information about the patient from whom the sample comes, it is the responsibility of the requesting healthcare professional to comply with the applicable data protection Laws and regulations.

Fagron Genomics,
SRN: ES-MF-000001092
C/ de les Cosidores, 150
08226 Terrassa, Barcelona (Spain)

CE IVDD
Self-Declared
98/79/EC

REF FGMS-Nutri

IVD In Vitro Diagnostic MedicalDevice

UDI 8437024682FGMS-NutriVC

2

Patient characteristics

Patient characteristics

2

Weight related variables

Age (years)	**51**
Height (cm)	**180**
Current weight (kg)	**90**
Goal weight (kg)	**75**
Current BMI	**27,77**
Goal BMI	**23,14**
Weight type	**Pre-obesity**

Physical exercise and metabolism related factors

Daily sport activity	**Without activity**

- Basal metabolism -

Current (cal)	**1.775**
Target (cal)	**1.625**

- Current DEE -

Current (Kcal)	**2.130**
Target (Kcal)	**1.950**
Variation (Kcal)	**-180**

IMPORTANT

In case of underweight, Obesity Type I, II, III, IV and/or existing pathologies, the results of this test must be evaluated and implemented ONLY by a doctor / endocrinologist / specialist / medical practitioner.

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3

Genetic results overview

Genetic results overview

3

3.1.

CATEGORY	DESCRIPTION	RESULTS
Morphological genetics for weight control	Medium-high genetic predisposition to being overweight. In case of overweight or obesity, it is caused mainly by inherited genetics. Following the recommendations of this DNA analysis will improve outcomes.	28.69%

• Genetic risk of overweight/obesity	MEDIUM-LOW RISK OF OVERWEIGHT/OBESITY	Pg. 29
• Risk of rebound weight gain	HIGH REBOUND EFFECT	Pg. 30
• Risk of increased BMI	MEDIUM-LOW RISK OF INCREASED BMI	Pg. 31
• Basal metabolic rate (burn calories at rest)	LOW BURNER AT REST	Pg. 32
• Weight loss capability during diet interventions	SLOW WEIGHT LOSS	Pg. 33

3.2.

CATEGORY	DESCRIPTION	RESULTS
Behavioural genetics in food intake	Medium-low dysregulation of food intake behaviour. Slight predisposition to being overweight. In case of excessive quantity or compulsive intake, strategies to reduce anxiety should be considered.	63.57%

• Appetite and anxiety risk	INCREASED APPETITE AND ANXIETY RISK	Pg. 34
• Satiety: Feeling Full	NORMAL SATIETY	Pg. 35

3.3.

CATEGORY	DESCRIPTION	RESULTS
Flavour sensitivities	Normal or average flavour sensitivity.	99.67%

• Bitter taste sensitivity	NORMAL BITTER TASTE SENSITIVITY	Pg. 36
• Salt sensitivity	LOW SALT SENSITIVITY	Pg. 37
• Sweet flavour preference	NORMAL SWEET PREFERENCE	Pg. 38

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

3

3.4.

CATEGORY	DESCRIPTION	RESULTS
Fat metabolism	Negative fat burning capacity. It would be recommended to decrease the general fat intake.	43.28%

• Response to monounsaturated fats (MUFAs)	VERY LOW MUFA METABOLISM	Pg. 39
• Response to polyunsaturated fats (PUFAs)	FAST PUFA METABOLISM	Pg. 40
• Response to fat intake to improve the HDL levels	MEDIUM-HIGH BENEFITS TO IMPROVE HDL	Pg. 41

3.5.

CATEGORY	DESCRIPTION	RESULTS
Lipid metabolism	Affected lipid metabolism. Cholesterol and triglyceride levels may show irregular results in blood analyses. Specific LDL or HDL treatments would be recommended. Increased cardiovascular risk.	38.95%

• Predisposition to reduced HDL levels	REDUCED HDL LEVELS	Pg. 42
• Predisposition to increased levels of triglycerides	HIGHLY INCREASED TRIGLYCERIDES	Pg. 43
• Predisposition to increased oxidation of LDL	SLIGHTLY INCREASED LDL OXIDATION	Pg. 44
• Risk of increased cholesterol LDL levels	INCREASED LDL LEVELS	Pg. 45
• Risk of unbalanced Triglycerides/HDL ratio	SLIGHLTLY INCREASED TG/HDL RATIO	Pg. 46

3.6.

CATEGORY	DESCRIPTION	RESULTS
Carbohydrate metabolism	Negative carbohydrate metabolism: Carbohydrate intake will lead to dysregulation in cholesterol levels and also to increased calorie and fat intake. Eliminating refined carbohydrates is urgent; move to wholegrain carbohydrates and reduce the quantity.	37.38%

• Capability to digest starchy food	REDUCED STARCH DIGESTION	Pg. 47
• Refined carbohydrate sensitivity	NORMAL CARBOHYDRATE SENSITIVITY	Pg. 48
• Carbohydrates and HDL levels predisposition	HIGH RISK OF HDL DYSREGULATION	Pg. 49
• Carbohydrates and LDL levels	HIGH RISK OF LDL DYSREGULATION	Pg. 50

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

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3.7.

CATEGORY	DESCRIPTION	RESULTS
Glucose metabolism	Medium-high dysregulation of glucose metabolism. Intake of refined sugar and carbohydrates will be dangerous. High risk of developing Type-II diabetes.	37.02%

• Risk of increased glucose levels in plasma after fasting	HIGH RISK OF HIGH GLUCOSE LEVELS	Pg. 51
• Risk of insulin resistance	MEDIUM-LOW INSULIN RESISTANCE	Pg. 52
• Risk of Type-II diabetes	MEDIUM-LOW DIABETES TYPE-II RISK	Pg. 53

3.8.

CATEGORY	DESCRIPTION	RESULTS
Efficacy of exercise	Very low efficacy of exercise to reduce body fat and regulate cholesterol levels. Intensive dietary interventions may be the best option.	12.22%

• Benefits from endurance exercise for improving HDL levels	VERY LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL	Pg. 54
• Exercise to reduce body fat	VERY LOW BENEFIT FROM EXERCISE TO REDUCE FAT	Pg. 55

3.9.

CATEGORY	DESCRIPTION	RESULTS
Detoxification imbalances	Average detoxification capacities.	79.81%

• Antioxidant capability	NORMAL ANTIOXIDANT CAPABILITY	Pg. 56

3.10.

CATEGORY	DESCRIPTION
Intolerance	Please find below the different analysed categories related to intolerances and sensitivities.

• Lactose intolerance risk	LOWER RISK OF LACTOSE INTOLERANCE	Pg. 57
• Risk of celiac disease	MEDIUM-HIGH RISK OF CELIAC DISEASE	Pg. 58
• Fructose intolerance risk	LOWER RISK OF FRUCTOSE INTOLERANCE	Pg. 60
• Caffeine metabolism	FAST CAFFEINE METABOLIZER	Pg. 61
• Alcohol metabolism	NORMAL ALCOHOL METABOLISM	Pg. 62

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

3

3.11. Hormones

CATEGORY	DESCRIPTION
Leptin	Leptin is a hormone which main function is sending a signal to the brain for food intake regulation. Leptin is commonly called the "satiety hormone". Low levels of leptin may imply problems of overeating and/or burning the stored fat. LEP-R is the gene coding for the cellular receptor of the leptin hormone. Its capability to bind leptin and start the cellular signalling is key for the satiety regulation function. Lower leptin binding capability may lead to high possibilities of leptin resistance, overeating and lower fat burning.
• LEP	**Predisposition to increased levels of leptin leading to leptin receptor saturation that may lead to increased risk of overeating and lower fat burning.**

CATEGORY	DESCRIPTION
Visfatin	Visfatin is an adipokine with an inflammatory and catabolic profile that has been associated with several metabolic risk factors, such as obesity, insulin resistance, and Type-II diabetes.
• NAMPT	**No predisposition to increased levels of circulating visfatin predisposing to normal inflammatory response.**

CATEGORY	DESCRIPTION
Ghrelin	Ghrelin is a hormone produced in the gut, often termed "the hunger hormone", since it causes an increase in appetite through its effect in the brain. Imbalances in ghrelin are associated with appetite increase, increased calorie consumption and fat storage.
• GHSR	**Predisposition to normal ghrelin receptor (GHSR) expression. Circulating levels of this hormone and the sensation of hunger are increased.**

CATEGORY	DESCRIPTION
Adiponectin	Adiponectin is a hormone that regulates glucose levels and fatty acid breakdown. Low levels of adiponectin are associated with inflammation, lipid abnormalities and insulin resistance.
• ADIPOQ	**High predisposition to lower adiponectin plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.**
• ADIPOQ	**High predisposition to lower adiponectin plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.**

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

3

3.12. Inflammation

CATEGORY	DESCRIPTION
TNF-α	TNF-α is a pro-inflammatory cytokine, strongly linked to many inflammatory conditions, expressed in, and secreted by adipose tissues. Increased levels are associated with obesity-induced inflammation, adiposity and insulin resistance.

• TNF-α	**Predisposition to moderately increased levels of TNF-alpha. Pro-inflammation tendency.**

CATEGORY	DESCRIPTION
IL-6	IL-6 is an interleukin with mainly pro-inflammatory functions and is commonly used as inflammatory marker. High levels of IL-6 are associated with obesity, insulin resistance and metabolic syndrome.

• IL-6	**Predisposition to highly increased levels of IL-6. Pro-inflammation.**

CATEGORY	DESCRIPTION
IL-10	IL-10 is a cytokine with potent anti-inflammatory properties.

• IL-10	**Predisposition to higher levels of the anti-inflammatory cytokine IL-10.**

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

3

3.13. Vitamins

Major genetic variations related to the metabolism of each vitamin are analysed. Possible deficiencies are determined so that our specialists are able to adapt your diet to improve your health and prevent putative diseases related to the lack of vitamins.

VITAMINS	DESCRIPTION	RESULTS
Vitamin A	Low risk of vitamin A deficiency. Ensure daily recommended intake or slightly increase it.	
Vitamin B^6	High risk of vitamin B6 deficiency. Increase daily vitamin B6 intake. Supplementation should be evaluated.	
Vitamin B^9	Normal folate metabolism. Ensure daily recommended intake.	
Vitamin B^{12}	High risk of vitamin B12 deficiency. Increase daily vitamin B12 intake. Supplementation should be evaluated.	
Vitamin C	Normal vitamin C metabolism and levels. Ensure daily recommended intake.	
Vitamin D	Low risk of Viamin D deficiency. Ensure daily recommended intake.	
Vitamin E	Medium risk of Vitamin E deficiency. Ensure daily recommended intake. Supplementation strategies might be of interest.	

INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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Genetic results overview

3

3.14.

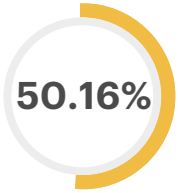
CATEGORY	DESCRIPTION
Minerals	Please find below the different analysed categories related to food supplementation needs.

3.15.

CATEGORY	DESCRIPTION
Effectiveness of diets	13 genetic variations related to the metabolism of various nutrients are analyzed in this section. This information allow us to develop a personalized plan aimed at improving your eating habits and exercise, that will help you achieve your weight goals, improve your muscle and bone mass, lower the fat mass and maintain a balanced and healthy diet.

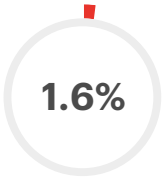
Efficacy of low carbohydrate diets

MARKER	RESULTS
KCTD10	
MMAB	



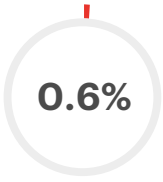
Efficacy of low calorie diets

MARKER	RESULTS
PPARG	
ADIPOQ	
LEPR	
ACSL5	



Efficacy of low fat diets

MARKER	RESULTS
PPARG	
GHSR	
APOA2	
SH2B1	
TCF7L2	
FTO	



INDICATIONS

Positive effect **Medium-positive effect** **Medium-negative effect** **Negative effect**

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4

Recommended nutritional plan

Recommended nutritional plan

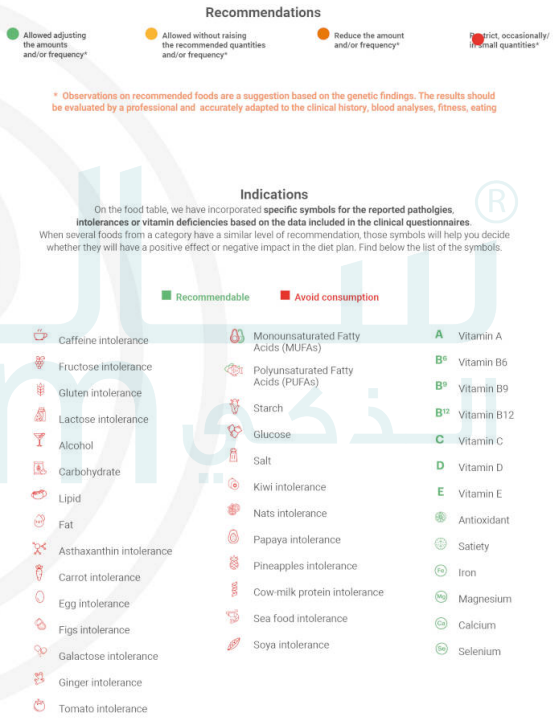
4

The most effective diet for your patient, after the genetic analysis, would be

LOW IN CARBOHYDRATES
INTEGRATED NUTRITIONAL PLAN

Check the FOOD LIST

recommended for you



5

Recommended supplements

Recommended supplements

5

The supplements recommended
to combat overweight and ageing are divided into 3 phases

1

DETOX

DETOX I
Detoxification
(oxidation)
30 days

- Vitamin B6 (Pyridoxine hydrochloride)
- Resveratrol
- Silybin®
- Pinus pinaster dry extract standardized
- Ubiqsome®
- Brocophanus®
- Glutathione (Reduced glutathione)
- Manganese
- Green tea dry extract (Camellia sinensis)
- Quercetin
- Selenium (Selenium yeast)
- Copper
- Nicotinamide (Niacinamida)
- Vitamin B2 (Riboflavine)

DETOX II
Detoxification
(conjugation)
30 days

- Citrimax®
- CitrusiM®
- Resveratrol
- Vitamin D3 (Cholecalciferol)
- Silybin®
- Taurine
- Brocophanus®
- Glutathione (Reduced glutathione)
- Green tea dry extract (Camellia sinensis)
- Spirulina
- Quercetin
- Vitamin B9 (Methylfolate)
- Glutamine (levoglutamine)
- Gotu kola dry extract (Centella asiatica)
- Omega 3
- Methionine

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Recommended supplements

5

The supplements recommended
to combat overweight and ageing are divided into 3 phases

2

INTESTINAL

INTESTINAL
Transportation
Excretion
30 days

- Bromelain
- Biointestil®
- Ginger dry extract

3

SUPPLEMENTATION

SUPPLEMENTATION
Prevention, maintaining optimal nutrition
3-6 months

- Citrimax®
- Vitamin B6 (Pyridoxine hydrochloride)
- Glucosamine sulfate
- CitrusiM®
- Resveratrol
- Vitamin D3 (Cholecalciferol)
- Vitamin K2
- Miodesin™
- SiliciuMax® powder
- Pinus pinaster dry extract standardized
- Ubiqsome®
- Taurine
- Horsetail dry extract (Equisetum arvense)
- Biotin
- Manganese
- Spirulina
- Ginseng dry extract (Panax ginseng)
- Quercetin
- Selenium (Selenium yeast)
- Mitocondrin®
- Vitamin B9 (Methylfolate)
- Vitamin B1 (Thiamine hydrochloride)
- Glutamine (levoglutamine)
- Gotu kola dry extract (Centella asiatica)
- Copper
- Nicotinamide (Niacinamida)
- Niacin (nicotinic acid)
- Omega 3
- Imuno TF®
- Methionine
- Ginger dry extract
- Vitamin B2 (Riboflavine)

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6

Recommended formulations

Recommended formulations

6

Detoxification
(oxidation)
30 days

Suggested formula: CAPSULE

Detox 1 capsule	
Silybin®	160 mg
Pinus pinaster dry extract standardized	40 mg
Ubiqsome®	150 mg
Manganese	2 mg

Daily dosage

Treatment for 30 days in accordance with the physician's decision.

Signature of the prescribing physician:

Dr

Physician Registration

Date of prescription

Address:

Signature:

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Recommended formulations

6

Detoxification
(conjugation)
30 days

Suggested formula: **CAPSULE**

Detox 2 capsule	
Vitamin D3 (Cholecalciferol)	2000 UI
Taurine	150 mg
Brocophanus®	100 mg
Spirulina	250 mg

Daily dosage

Treatment for 30 days in accordance with the physician's decision.

Signature of the prescribing physician:

Dr
Physician Registration
Date of prescription

Address:

Signature:

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Recommended formulations

6

Transportation
Excretion
30 days

Suggested formula: CAPSULE

Intestinal capsule	
Bromelain	150 mg
Biointestil®	300 mg

Daily dosage

Treatment for 30 days in accordance with the physician's decision.

Signature of the prescribing physician:

Dr

Physician Registration

Date of prescription

Address:

Signature:

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Recommended formulations

6

Prevention, maintaining optimal nutrition
3-6 months

Suggested formula: CAPSULE

Supplementation capsule	
Citrimax®	500 mg
Vitamin B6 (Pyridoxine hydrochloride)	45 mg
Glucosamine sulfate	600 mg
Resveratrol	208 mg

Daily dosage

Treatment for 3-6 months in accordance with the physician's decision.

Signature of the prescribing physician:

Dr
Physician Registration
Date of prescription

Address:

Signature:

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Annex to the formulation

6

Composites daily dosage recommendation

Composite Name	Min	Max	Unit
Biointestil®	300	600	mg
Biotin	40	300	mcg
Brocophanus®	100	200	mg
Bromelain	100	500	mg
Citrimax®	500	1500	mg
CitrusiM®	300	500	mg
Copper	0.5	2	mg
Ginger dry extract	250	750	mg
Ginseng dry extract (Panax ginseng)	100	200	mg
Glucosamine sulfate	500	1500	mg
Glutamine (levoglutamine)	3000	10000	mg
Glutathione (Reduced glutathione)	200	3000	mg
Gotu kola dry extract (Centella asiatica)	50	750	mg
Green tea dry extract (Camellia sinensis)	250	500	mg
Horsetail dry extract (Equisetum arvense)	100	900	mg
Imuno TF®	25	100	mg
Manganese	1	5	mg
Methionine	500	1000	mg
Miodesin™	250	1000	mg

Composite Name	Min	Max	Unit
Mitocondrin®	100	200	mg
Niacin (nicotinic acid)	1	50	mg
Nicotinamide (Niacinamida)	1	500	mg
Omega 3	0.5	3000	mg
Pinus pinaster dry extract standardized	40	120	mg
Quercetin	250	1000	mg
Resveratrol	50	500	mg
Selenium (Selenium yeast)	50	200	mcg
SiliciuMax® powder	150	600	mg
Silybin®	80	360	mg
Spirulina	500	4000	mg
Taurine	100	600	mg
Ubiqsome®	100	300	mg
Vitamin B1 (Thiamine hydrochloride)	2	100	mg
Vitamin B2 (Riboflavine)	1	30	mg
Vitamin B6 (Pyridoxine hydrochloride)	1	100	mg
Vitamin B9 (Methylfolate)	0.25	5	mg
Vitamin D3 (Cholecalciferol)	600	5000	UI
Vitamin K2	65	320	mcg

Annex to the formulation

6

Fagron Brands Glossary

Product Name	Description
Biointestil®	Palmarosa Essential Oil and Ginger Rhizoma Fiber. Standardized at a minimum of 14% Geraniol and 0.4% 6-Gingerol.
Brocophanus®	Brassica Oleracea and Raphanus sativus nigra standardized in sulforafano (1%) and mirosinase (0.5%).
Citrimax®	Garcinia cambogia extract rich in a salt of HCA (hydroxycitric acid) bound to calcium and potassium, which gives it greater solubility and bioavailability.
CitrusiM®	Extract from the fruit of Citrus sinensis L. Osbeck Moro variety (blood orange), containing a high concentration (minimum 3%) of anthocyanins (C3G) and flavones.
Imuno TF®	Transfer factors isolated
Miodesin™	Phytoactive composed of Uncaria tomentosa, Endopleura uchi and Haematococcus algae.
Mitocondrin®	Angelica Keiskei (Ashitaba) and Panax Ginseng (Red Ginseng).
SiliciuMax® powder	Stabilized orthosylic acid in maltodextrin, which inhibits its polymerization, increasing its bioavailability.
Silybin®	Silybum Marianum extract (L.) standardized in Silibina.
Ubiqsome®	Coenzyme Q10 Phytosome.

7

Complete genetic results

7.1. Morphological genetics for weight control
- 7.1.1. Genetic risk of overweight/obesity
- 7.1.2. Risk of rebound weight gain
- 7.1.3. Risk of increased BMI
- 7.1.4. Basal metabolic rate (burn calories at rest)
- 7.1.5. Weight loss capability during diet interventions

7.2. Behavioural genetics in food intake
- 7.2.1. Appetite and anxiety risk
- 7.2.2. Satiety: Feeling Full

7.3. Flavour sensitivities
- 7.3.1. Bitter taste sensitivity
- 7.3.2. Salt sensitivity
- 7.3.3. Sweet flavour preference

7.4. Fat metabolism
- 7.4.1. Response to monounsaturated fats (MUFAs)
- 7.4.2. Response to polyunsaturated fats (PUFAs)
- 7.4.3. Response to fat intake to improve the HDL levels

7.5. Lipid metabolism
- 7.5.1. Predisposition to reduced HDL levels
- 7.5.2. Predisposition to increased levels of triglycerides
- 7.5.3. Predisposition to increased oxidation of LDL
- 7.5.4. Risk of increased cholesterol LDL levels
- 7.5.5. Risk of unbalanced Triglycerides/HDL ratio

7.6. Carbohydrate metabolism
- 7.6.1. Capability to digest starchy food
- 7.6.2. Refined carbohydrate sensitivity
- 7.6.3. Carbohydrates and HDL levels predisposition
- 7.6.4. Carbohydrates and LDL levels

7.7. Glucose metabolism
- 7.7.1. Risk of increased glucose levels in plasma after fasting
- 7.7.2. Risk of insulin resistance
- 7.7.3. Risk of Type-II diabetes

7.8. Efficacy of exercise
- 7.8.1. Benefits from endurance exercise for improving HDL levels
- 7.8.2. Exercise to reduce body fat

7.9. Detoxification imbalances
- 7.9.1. Antioxidant capability

7.10. Intolerance
- 7.10.1. Lactose intolerance risk
- 7.10.2. Risk of celiac disease
- 7.10.3. Fructose intolerance risk
- 7.10.4. Caffeine metabolism
- 7.10.5. Alcohol metabolism

7.11. Hormones
- 7.11.1. Leptin
- 7.11.2. Visfatin
- 7.11.3. Ghrelin
- 7.11.4. Adiponectin

7.12. Inflammation
- 7.12.1. TNF-α
- 7.12.2. IL-6
- 7.12.3. IL-10

7.13. Vitamins
- 7.13.1. Vitamin A
- 7.13.2. Vitamin B6
- 7.13.3. Vitamin B9 (folate)
- 7.13.4. Vitamin B12
- 7.13.5. Vitamin C
- 7.13.6. Vitamin D
- 7.13.7. Vitamin E

7.14. Minerals
- 7.14.1. Calcium malabsorption risk
- 7.14.2. Predisposition to dysregulated calcium levels
- 7.14.3. Risk of iron overload
- 7.14.4. Risk of low iron plasma levels
- 7.14.5. Predisposition to dysregulated magnesium levels
- 7.14.6. Predisposition to dysregulated selenium levels
- 7.14.7. Sodium sensitivity

7.15. Effectiveness of diets
- 7.15.1. Efficacy of low calorie diets
- 7.15.2. Efficacy of low carbohydrate diets
- 7.15.3. Efficacy of low fat diets

Complete genetic results

7

7. 1 Morphological genetics for weight control

7. 1. 1. Genetic risk of overweight/obesity

RESULT	ABOUT
MEDIUM-LOW RISK OF OVERWEIGHT/OBESITY	Key genetic predisposition genes to obesity and weight gain are analysed. Obesity is influenced by the interplay between external factors (such as diet and/or physical activity) and is highly linked to individual genetics. Genetics highly determine how the body processes or metabolizes fats and/or nutrients. Therefore, understanding our own genetics is important to control obesity and as a key weight reduction tool.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
MC4R	rs2229616	CC	**HIGH**	Higher risk of obesity. High predisposition to increased glycosylated hemoglobin (increased risk of type 2 diabetes) and decreased HDL-cholesterol levels.
SH2B1	rs7498665	AA	LOW	Normal risk of obesity.
FTO	rs9939609	TT	LOW	Normal risk of obesity.
FTO	rs1121980	GG	LOW	Normal risk of obesity.
MC4R	rs17700633	GG	LOW	Normal risk of obesity.

INDICATIONS

LOW RISK OF OVERWEIGHT/OBESITY
Reduced risk of obesity due to inherited genetic factors.

MEDIUM-LOW RISK OF OVERWEIGHT/OBESITY
Medium-low risk of obesity due to inherited genetic factors.

MEDIUM-HIGH RISK OF OVERWEIGHT/OBESITY
Medium-high risk of obesity due to inherited genetic factors. Other factors such as intake due to anxiety or low satiety may explain excess weight.

HIGH RISK OF OVERWEIGHT/OBESITY
High risk of obesity due to inherited genetic factors. Other factors such as intake due to anxiety or low satiety may explain excess weight.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 1 Morphological genetics for weight control

7. 1. 2. Risk of rebound weight gain

RESULT	ABOUT
HIGH REBOUND EFFECT	Individuals with certain genetic variants of the ADIPOQ gene were found to be more susceptible to regain weight after weight loss interventions (rebound effect).

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ADIPOQ	rs17300539	GG	**HIGH**	Predisposition to regain weight after dieting.

INDICATIONS

LOW REBOUND EFFECT
Low risk of rebound weight after diet interventions. Normal weight loss capacity.

MEDIUM-LOW REBOUND EFFECT
Medium-low risk of rebound weight after diet interventions. Normal weight loss capacity.

MEDIUM-HIGH REBOUND EFFECT
Medium-high risk of rebound weight after diet interventions. Lower weight loss capability than normal during interventions.

HIGH REBOUND EFFECT
High risk of rebound weight after diet interventions. Lower weight loss capability than normal during interventions. It will require an extra effort to loose weight and keep it off afterwards.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 1 Morphological genetics for weight control

7. 1. 3. Risk of increased BMI

RESULT	ABOUT
MEDIUM-LOW RISK OF INCREASED BMI	The predisposition to increase waist circumference and body mass index (BMI) is analyzed. BMI is used to determine whether an individual is in a healthy weight range for the correspondent height. It is useful to consider BMI alongside waist circumference, as waist measurement helps to assess risk by measuring the amount of fat carried around the middle.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
MC4R	rs12970134	GA	MEDIUM	Increased risk of increased BMI, increased waist circumference, and insulin resistance.
MC4R	rs17782313	CT	MEDIUM	Increased risk of increased BMI, increased waist circumference, and insulin resistance.
SH2B1	rs4788102	GG	LOW	Normal risk of increased BMI.

INDICATIONS

LOW RISK OF INCREASED BMI
Reduced risk of increased BMI, waist circumference and insulin resistance due to genetics.

MEDIUM-LOW RISK OF INCREASED BMI
Medium-low risk of increased BMI, waist circumference and insulin resistance due to genetics.

MEDIUM-HIGH RISK OF INCREASED BMI
Medium-high risk of increased BMI, waist circumference and insulin resistance due to genetics.

HIGH RISK OF INCREASED BMI
High risk of increased BMI, waist circumference and insulin resistance due to genetics.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 1 Morphological genetics for weight control

7. 1. 4. Basal metabolic rate (burn calories at rest)

RESULT	ABOUT
LOW BURNER AT REST	The predisposition to an increase/decrease in energy expenditure while resting is analysed. Some people have a higher tendency then others to expend less energy when not performing any physical activity, which supports weight gain.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
FABP2	rs1799883	CT	**HIGH**	Predisposition to decreased resting metabolic rate.
LEPR	rs2025804	GG	**HIGH**	Predisposition to decreased resting metabolic rate.

INDICATIONS

HIGH BURNER AT REST
High energy/calorie burning capacity at rest.

MEDIUM-HIGH BURNER AT REST
Medium-high capacity to burn energy/calories at rest.

MEDIUM-LOW BURNER AT REST
Medium-low capacity of energy/calorie burning at rest.

LOW BURNER AT REST
Low energy/calorie burning capacity at rest.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 1 Morphological genetics for weight control

7. 1. 5. Weight loss capability during diet interventions

RESULT	ABOUT
SLOW WEIGHT LOSS	The predisposition to an increase/decrease in weight loss during diet interventions is analysed. Some people have a higher tendency than others to lose weight when they follow a diet intervention. Lower capabilities will imply a longer time to accomplish the goals and would require a stricter intervention.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ACSL5	rs2419621	CC	HIGH	Predisposition to slow diet-induced weight loss.

INDICATIONS

RAPID WEIGHT LOSS
Diet interventions should be successful due to a higher capability to reduce weight while on diet.

NORMAL WEIGHT LOSS
Diet interventions should be successful due to a normal capability to reduce weight while on diet. However it may take a minimum of 3-6 months to be effective.

SLIGHTLY SLOW WEIGHT LOSS
Standard diet interventions could not be successful due to a low capability to reduce weight while on diet. Specialized treatments would be recommended.

SLOW WEIGHT LOSS
Diet interventions should contain a complete approach for the patient, both nutritional and psychological, due to the lower capability to reduce weight while on diet. Specialised treatments will be recommended.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 2 Behavioural genetics in food intake

7. 2. 1. Appetite and anxiety risk

RESULT	ABOUT
INCREASED APPETITE AND ANXIETY RISK	Genetic variations affecting appetite and anxiety related to eating are analysed. Appetite is a phenomenon created by our nervous system which results in a desire to eat, either by necessity or by pleasure, and in which external factors (such as odors, flavours, appearance and presentation of food) are involved. It has been seen in numerous studies that the appetite or desire to eat can also have genetic causes that can determine inhibition of intake or reduced feeling of being full. Anxiety related to food intake can be caused by periods of stress, but it has also been seen that there is an important genetic component that makes us more prone to anxiety and translates into compulsive eating more easily. The main parameters related to genetic predisposition to deregulated levels of appetite and anxiety in food intake, increased risk of obesity, increased food intake and reduced fullness are analysed below. Knowing how these genetic processes affect your diet allows proper handling of meals.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
COMT	rs4680	AG	MEDIUM	Increased risk of overeating.
NMB	rs1051168	GG	LOW	Normal risk of eating disinhibition.
DRD2	rs1800497	AG	**HIGH**	Predisposition to emotional eating and obesity.
MC4R	rs2229616	CC	**HIGH**	Predisposition to binge eating.
DRD2	rs6277	AA	**HIGH**	Predisposition to binge eating.

INDICATIONS

NORMAL APPETTITE AND ANXIETY RISK
Normal or well-balanced regulation of appetite and eating-related anxiety.

SLIGHTLY INCREASED APPETITE AND ANXIETY RISK
Medium-low dysregulation of the appetite, leading to some levels of anxiety affecting food intake.

INCREASED APPETITE AND ANXIETY RISK
Medium-high dysregulation of the appetite, leading to elevated levels of anxiety affecting food intake. Appetite suppressants may be helpful.

HIGHLY INCREASED APPETITE AND ANXIETY RISK
High dysregulation of the appetite, leading to high levels of anxiety affecting food intake. Appetite suppressants may be required and possibly anxiolytic prescription upon medical decision.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 2 Behavioural genetics in food intake

7. 2. 2. Satiety: Feeling Full

RESULT	ABOUT
NORMAL SATIETY	The perception of feeling full and satisfied after food intake is different within individuals. This is particularly important as the longer it takes to reach this feeling, the more food intake will occur, contributing to weight gain.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
FTO	rs9939609	TT	LOW	Predisposition to normal satiety.

INDICATIONS

NORMAL SATIETY
Normal perception of satiety after eating, activated after 15-20 minutes of the start of the meal.

SLIGHTLY LOWER SATIETY
Slighlty reduced perception of satiety after eating a meal. Try to eat slower to allow the satiety center to be activated.

LOWER SATIETY
Reduced perception of satiety after eating a meal. Eat slower to allow the satiety center to be activated.

VERY LOW SATIETY
Very low perception of satiety after eating a meal. Eat very slow to allow the satiety center to be activated. Incorporate satiating food in your daily diet.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 3 Flavour sensitivities

7. 3. 1. Bitter taste sensitivity

RESULT	ABOUT
NORMAL BITTER TASTE SENSITIVITY	Sensitivity to bitter flavours is deeply linked to genetics. A high sensitivity to bitter flavours is usually linked to increased salt consumption. Therefore there is a higher predisposition to cardiovascular risks when extra salt is consumed intending to mask the bitter flavours.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
TAS2R38	rs1726866	AG	LOW	Predispositon to normal sensitivity to bitter taste.
TAS2R38	rs713598	CG	LOW	Predispositon to normal sensitivity to bitter taste.

INDICATIONS

NORMAL BITTER TASTE SENSITIVITY
Normal or decreased sensitivity to bitter flavours. No extra salt should be consumed for this reason.

SLIGHTLY INCREASED BITTER TASTE SENSITIVITY
Slightly increased sensitivity to bitter flavours. No extra salt should be consumed for this reason.

INCREASED BITTER TASTE SENSITIVITY
Increased sensitivity to bitter flavours. Try to minimize bitter-tasting food, since it may lead to an increased consumption of salt.

HIGHLY INCREASED TASTE SENSITIVITY
High sensitivity to bitter flavours. Try to avoid bitter-tasting food, since it may lead to an increased consumption of salt.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 3 Flavour sensitivities

7. 3. 2. Salt sensitivity

RESULT	ABOUT
LOW SALT SENSITIVITY	Salt sensitivity is defined as a physiological trait by which blood pressure shows changes parallel to changes in salt intake. In many individuals, when salt intake increases, the excess amount is excreted by the way of kidney or sweat. However, there are some individuals where this mechanism is faulty and increased salt is retained and manifests as high blood pressure.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ACE	rs4343	AA	LOW	Predisposition to normal salt sensitivity.

INDICATIONS

LOW SALT SENSITIVITY
Normal salt sensitivity: no increased blood pressure risk due to salt consumption.

MEDIUM-LOW SALT SENSITIVITY
Slightly increased salt sensitivity: moderately increased blood pressure risk due to salt consumption.

MEDIUM-HIGH SALT SENSITIVITY
Increased salt sensitivity: increased blood pressure risk due to salt consumption. Reduce current salt consumption, if daily intake is high.

HIGH SALT SENSITIVITY
High salt sensitivity: high blood pressure risk due to salt consumption. Reduce current salt consumption, if daily intake is high.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 3 Flavour sensitivities

7. 3. 3. Sweet flavour preference

RESULT	ABOUT
NORMAL SWEET PREFERENCE	Increased desire to eat sweet food due to an incapacity of tasting sweet flavours.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
SLC2A2	rs5400	GG	LOW	No predisposition for preferring sugar-containing foods.

INDICATIONS

NORMAL SWEET PREFERENCE
Normal taste of sweet flavour. No excess sugar intake should be required.

SLIGHTLY INCREASED SWEET PREFERENCE
Slight incapacity to taste sweet flavours. This will lead to an increase in sugar consumption and obesity risk.

INCREASED SWEET PREFERENCE
Incapacity to taste sweet flavours. This will lead to an increase in the sugar consumption and obesity risk. Consider using artificial sweeteners in your diet.

HIGHLY INCREASED SWEET PREFERENCE
Major incapacity to taste sweet flavours. This will lead to an increase in the sugar consumption and obesity risk. Consider using artificial sweeteners in your diet.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 4 Fat metabolism

7. 4. 1. Response to monounsaturated fats (MUFAs)

RESULT	ABOUT
VERY LOW MUFA METABOLISM	The predisposition to a higher/lower capacity to metabolize monounsaturated fatty acids (MUFAs) is analysed. MUFAs are a class of fatty acids found in foods such as olive oil, nuts and avocados. The beneficial effects of MUFAs on cardiovascular disease risk and blood lipid profiles have been extensively studied: dietary MUFAs decrease oxidized LDL, LDL cholesterol, total cholesterol, and triglyceride concentrations, without the concomitant decrease in HDL typically seen with low-fat diets.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ADIPOQ	rs17300539	GG	**HIGH**	No predisposition to reduce BMI and decrease obesity risk in response to monounsaturated fatty acids (MUFA) intake.

INDICATIONS

FAST MUFA METABOLISM
Normal capability of burning monounsaturated fat (MUFA). Increased capability to intake and metabolize MUFA with low weight gain.

MEDIUM MUFA METABOLISM
Medium capability of burning monounsaturated fat (MUFA). MUFA intake may lead to low weight gain unless a high-fat diet is followed.

LOW MUFA METABOLISM
Low capability of burning monounsaturated fat (MUFA). Direct correlation of high-MUFA intake and weight gain due to fat accumulation.

VERY LOW MUFA METABOLISM
Very low capability of burning monounsaturated fat (MUFA). Direct correlation on high-MUFA intake and weight gain due to fat accumulation.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 4 Fat metabolism

7. 4. 2. Response to polyunsaturated fats (PUFAs)

RESULT	ABOUT
FAST PUFA METABOLISM	The predisposition to a higher/lower capacity to metabolize polyunsaturated fatty acids (PUFA) and to improve the lipidic profile (decreased LDL-levels) with PUFA intake is analysed. Polyunsaturated fatty acids are necessary to build cell membranes and nerve coverings as well as for proper blood clotting, muscle movement and inflammation. There are two main types of polyunsaturated fats: omega-3 fatty acids and omega-6 fatty acids. Both types provide health benefits.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	LOW	Predisposition to improve lipid profile (LDL and total cholesterols) and reduce BMI in response to a PUFA-rich diet.
FADS1	rs174547	CT	MEDIUM	Age-related predisposition to slightly reduced PUFA biosynthetic capacity and lower plasma omega 3 concentration.

INDICATIONS

FAST PUFA METABOLISM
Normal capability of burning polyunsaturated fat (PUFA). Increased capability to intake and metabolize PUFA with low weight gain. Improved lipidic profiles with PUFA intake.

MEDIUM PUFA METABOLISM
Medium capability of burning polyunsaturated fat (PUFA). PUFA intake may lead to low weight gain unless a high-fat diet is followed. Improved lipidic profiles with PUFA intake.

LOW PUFA METABOLISM
Low capability of burning polyunsaturated fat (PUFA). Direct correlation of high-PUFA intake and weight gain due to fat accumulation.

VERY LOW PUFA METABOLISM
Very low capability of burning polyunsaturated fat (PUFA). Direct correlation of high-PUFA intake and weight gain due to fat accumulation.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 4 Fat metabolism

7. 4. 3. Response to fat intake to improve the HDL levels

RESULT	ABOUT
MEDIUM-HIGH BENEFITS TO IMPROVE HDL	The predisposition to have increased or reduced levels of HDL is analyzed according to the genetic situation of liver lipases. With this category, we understand if a low fat diet is a good strategy to regulate cholesterol levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
LIPC	rs1800588	CT	MEDIUM	Slight predisposition to improve HDL cholesterol levels in response to low fat diet.

INDICATIONS

HIGH BENEFITS TO IMPROVE HDL
A low fat diet will be of great help in increasing HDL levels.

MEDIUM-HIGH BENEFITS TO IMPROVE HDL
A low fat diet will be a good support to increase HDL levels.

MEDIUM-LOW BENEFITS TO IMPROVE HDL
Low fat diet will not be enough to increase HDL levels.

VERY LOW BENEFITS TO IMPROVE HDL
Low fat diet will not be enough to increase HDL levels.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 5 Lipid metabolism

7. 5. 1. Predisposition to reduced HDL levels

RESULT	ABOUT
REDUCED HDL LEVELS	Although environmental factors play a role, variation in HDL levels are at least 50% genetically determined. In this category the main genes involved in the predisposition to higher or lower HDL levels are analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
APOA5	rs662799	AA	LOW	Predisposition to normal levels of HDL cholesterol.
CETP	rs5883	CC	**HIGH**	Predisposition to decreased HDL cholesterol levels.

INDICATIONS

NORMAL HDL LEVELS
Normal regulation of HDL levels. No increased risk of cardiovascular risk.

SLIGHTLY DECREASED HDL LEVELS
Slightly lower HDL levels leading to increased cardiovascular risk.

REDUCED HDL LEVELS
Lower HDL levels leading to increased cardiovascular risk.

HIGLY REDUCED HDL LEVELS
Very low HDL levels leading to increased cardiovascular risk.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 5 Lipid metabolism

7. 5. 2. Predisposition to increased levels of triglycerides

RESULT	ABOUT
HIGHLY INCREASED TRIGLYCERIDES	Triglycerides are a type of fat (lipid) found in your blood. When you eat, your body converts any calories it doesn't need to use right away into triglycerides. The triglycerides are stored in your fat cells. Later, hormones release triglycerides for energy between meals. If you regularly eat more calories than you burn, particularly from high-carbohydrate foods, you may have high triglycerides (hypertriglyceridemia). In this category we analyse the genes related to the predisposition of having increased levels of triglycerides.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	**HIGH**	Predisposition to increased levels of triglycerides.

INDICATIONS

TRIGLYCERIDES NOT INCREASED
No predisposition to increased triglyceride levels.

SLIGHTLY INCREASED TRIGLYCERIDES
Slight predisposition to increased triglyceride levels.

INCREASED TRIGLYCERIDES
Medium-high predisposition to increased triglyceride levels.

HIGHLY INCREASED TRIGLYCERIDES
High predisposition to increased triglyceride levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 5 Lipid metabolism

7. 5. 3. Predisposition to increased oxidation of LDL

RESULT	ABOUT
SLIGHTLY INCREASED LDL OXIDATION	Oxidized low-density lipoprotein (LDL) is a harmful type of cholesterol that is produced in your body when normal LDL cholesterol is damaged by chemical interactions with free radicals. These, and a related series of inflammatory responses can result in atherosclerosis, which is the hardening of the arteries. The resulting decrease in blood flow in your arteries increases your chances of having a heart attack or a stroke. You can produce high levels of oxidized LDL if you have excessive free radical formation or simply high LDL cholesterol levels. In this category, the genes related to an increased predisposition to oxidize LDL are analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
APOB	rs676210	AG	MEDIUM	Predisposition to increased LDL oxidation.

INDICATIONS

NOT INCREASED LDL OXIDATION
Normal LDL oxidation.

SLIGHTLY INCREASED LDL OXIDATION
Moderate increase in the LDL oxidation. Increased risk of atherosclerosis.

INCREASED LDL OXIDATION
Increased LDL oxidation. Increased risk of atherosclerosis. Strategies for reducing LDL levels would be recommended.

HIGHLY INCREASED LDL OXIDATION
Higly increased LDL oxidation and risk of atherosclerosis. Intense strategies for reducing LDL levels should be inititated.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 5 Lipid metabolism

7. 5. 4. Risk of increased cholesterol LDL levels

RESULT	ABOUT
INCREASED LDL LEVELS	Low-density lipoprotein (LDL) is one of the five major groups of lipoprotein which transport all fat molecules around the body in extracellular water. LDL delivers fat molecules to cells. LDL can contribute to atherosclerosis if it is oxidized within the walls of arteries. In this category, the genes related to the risk of having increased cholesterol LDL levels in your body are analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
CELSR2	rs12740374	GT	MEDIUM	Increased predisposition to lower LDL cholesterol levels.
HNF1A	rs2650000	AA	**HIGH**	Predisposition to increased LDL cholesterol levels.
LDLR	rs6511720	GG	**HIGH**	High risk of increased LDL cholesterol levels.
ABCG8	rs6544713	CC	LOW	High risk of increased LDL cholesterol levels.

INDICATIONS

NOT INCREASED LDL LEVELS
Lower risk of high LDL levels

SLIGHTLY INCREASED LDL LEVELS
Moderate risk of high LDL levels

INCREASED LDL LEVELS
High risk of high LDL levels.

HIGHLY INCREASED LDL LEVELS
Very high risk of high LDL levels.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 5 Lipid metabolism

7. 5. 5. Risk of unbalanced Triglycerides/HDL ratio

RESULT	ABOUT
SLIGHLTLY INCREASED TG/HDL RATIO	The predisposition to an unbalanced Triglyceride/HDL cholesterol (TG/HDL-C) ratio is analysed. High TG/HDL ratio has been identified as a risk factor for cardiovascular (CV) diseases.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
HMGCR	rs3846663	CT	MEDIUM	Predisposition to slightly higher triglyceride (TG) levels, and increased TG/HDL cholesterol ratio.

INDICATIONS

NORMAL TG/HDL RATIO
Not associated with increased TG/HDL ratio.

SLIGHLTLY INCREASED TG/HDL RATIO
Slightly associated with increased TG/HDL ratio.

INCREASED TG/HDL RATIO
Increased TG/HDL ratio leads to a highly increased risk of cardiovascular pathologies. Risk of insulin insensitivity.

HIGHLY INCREASED TG/HDL RATIO
A very high TG/HDL ratio leads to a highly increased risk of cardiovascular pathologies. Risk of insulin insensitivity.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 6 Carbohydrate metabolism

7. 6. 1. Capability to digest starchy food

RESULT	ABOUT
REDUCED STARCH DIGESTION	The capability to break down starch from food is analysed. Amylase is an enzyme that catalyzes the hydrolysis of starch into sugars. Amylase is present in the saliva of humans and some other mammals, where it begins the chemical process of digestion. When starch is not properly processed, its consumption must be reduced in a diet plan.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
AMY1-AMY2	rs11577390	CC	HIGH	No predisposition to increased expression of the amylase gene.
AMY1	rs4244372	TT	LOW	Predisposition to increased expression of the amylase gene which is likely to enable more efficient starch digestion.

INDICATIONS

INCREASED STARCH DIGESTION
Increased capability to digest starch from food due to an increase in the expression and the activity of amylase enzyme. It is known that reducing calories will be beneficial.

MEDIUM STARCH DIGESTION
Moderate capability to digest starch from food due to an increase in the expression and the activity of amylase enzyme. It is known that reducing calories will be beneficial.

REDUCED STARCH DIGESTION
Reduced capability to digest starch in food due to a decrease in amylase enzyme activity. It would be beneficial to decrease starch intake.

HIGHLY REDUCED STARCH DIGESTION
Highly reduced capability to digest starch in food due to a decrease in amylase enzyme activity. It would be beneficial to decrease starch intake.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 6 Carbohydrate metabolism

7. 6. 2. Refined carbohydrate sensitivity

RESULT	ABOUT
NORMAL CARBOHYDRATE SENSITIVITY	Carbohydrate consumption initially produces a slight euphoria, followed by a sugar low, this is then replaced by tiredness. This adverse feeling causes a desire to snack more, perpetuating this unhealthy cycle, without ever feeling satisfied. In carbohydrates sensitive people the carbohydrate-insulin-serotonin connection has malfunctioned, or become desensitised and the amount of calories extracted by the consumption of refined carbohydrates is higher than average, also due to a continuous increase of its consumption.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
FABP2	rs1799883	CT	LOW	Predisposition to normal sensitivity to refined carbohydrates.

INDICATIONS

NORMAL CARBOHYDRATE SENSITIVITY
Normal calorie extraction from carbohydrate consumption.

MEDIUM CARBOHYDRATE SENSITIVITY
Moderate calorie extraction from carbohydrate consumption. Medium risk of weight gain.

HIGH CARBOHYDRATE SENSITIVITY
Increased calorie extraction from carbohydrate consumption. Higher risk of weight gain.

VERY HIGH CARBOHYDRATE SENSITIVITY
Highly increased calorie extraction from carbohydrate consumption. Very high risk of weight gain.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 6 Carbohydrate metabolism

7. 6. 3. Carbohydrates and HDL levels predisposition

RESULT	ABOUT
HIGH RISK OF HDL DYSREGULATION	Carbohydrate intake has an implication on the regulation of cholesterol levels. We analyse the predisposition to increase or decrease the HDL cholesterol levels depending on carbohydrate intake.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
KCTD10	rs10850219	GG	HIGH	Predisposition to reduce HDL cholesterol levels in response to a carbohydrate-rich diet.

INDICATIONS

LOW RISK OF HDL DYSREGULATION
High carbohydrate consumption will not lead to a cholesterol dysregulation.

MEDIUM-LOW RISK OF HDL DYSREGULATION
High carbohydrate consumption may lead to slightly increased LDL and decreased HDL levels.

MEDIUM-HIGH RISK OF HDL DYSREGULATION
High carbohydrate consumption will lead to increased LDL and decreased HDL levels.

HIGH RISK OF HDL DYSREGULATION
High carbohydrate consumption will lead to highly increased LDL and decreased HDL levels.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 6 Carbohydrate metabolism

7. 6. 4. Carbohydrates and LDL levels

RESULT	ABOUT
HIGH RISK OF LDL DYSREGULATION	Effect of carbohydrate intake in the regulation of cholesterol levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
MMAB	rs2241201	GG	**HIGH**	High risk to increase LDL-cholesterol levels and decrease HDL-cholesterol levels in response to high intake of carbohydrates.

INDICATIONS

LOW RISK OF LDL DYSREGULATION
High carbohydrate consumption will not lead to cholesterol dysregulation.

MEDIUM-LOW RISK OF LDL DYSREGULATION
High carbohydrate consumption will lead to very slight increased LDL and decreased HDL levels.

MEDIUM-HIGH RISK OF LDL DYSREGULATION
High carbohydrate consumption will lead to increased LDL and decreased HDL levels.

HIGH RISK OF LDL DYSREGULATION
High carbohydrate consumption will lead to highly increased LDL and decreased HDL levels.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 7 Glucose metabolism

7. 7. 1. Risk of increased glucose levels in plasma after fasting

RESULT	ABOUT
HIGH RISK OF HIGH GLUCOSE LEVELS	Fasting blood sugar levels give vital clues about how a person's body is managing blood sugar. Blood sugar tends to peak about an hour after eating and declines after that. High fasting blood sugar levels point to insulin resistance or diabetes. In this category, the genes related to the predisposition to an increased level of glucose after fasting are analysed, helping to understand how the body manages sugar.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PLIN1	rs2289487	CC	**HIGH**	High risk of increased plasma glucose levels after fasting.
GHSR	rs490683	GG	**HIGH**	High risk of increased plasma glucose levels after fasting.

INDICATIONS

LOW RISK OF HIGH GLUCOSE LEVELS
Normal fasting plasma glucose levels. No increased risk of Type-II diabetes.

MEDIUM-LOW RISK OF HIGH GLUCOSE LEVELS
Normal or slightly increased fasting plasma glucose levels. No increased risk of Type-II diabetes.

MEDIUM-HIGH RISK OF HIGH GLUCOSE LEVELS
Increased fasting plasma glucose levels. Increased risk of Type-II diabetes.

HIGH RISK OF HIGH GLUCOSE LEVELS
High risk of Increased fasting plasma glucose levels. Increased risk of Type-II diabetes.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 7 Glucose metabolism

7. 7. 2. Risk of insulin resistance

RESULT	ABOUT
MEDIUM-LOW INSULIN RESISTANCE	Insulin resistance (also called metabolic syndrome) is when cells in your muscles, fat, and liver don't respond well to insulin and can't use glucose from your blood for energy. To make up for it, your pancreas makes more insulin. Over time, your blood sugar levels go up. Insulin resistance syndrome includes a group of problems like obesity, high blood pressure, high cholesterol, and Type-II diabetes. In this category the genetic predisposition towards a higher risk of insulin resistance is analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	**HIGH**	High predisposition to insulin resistance.
ADIPOQ	rs17300539	GG	**HIGH**	High predisposition to insulin resistance.
TCF7L2	rs7903146	CC	LOW	No predisposition to insulin resistance.
FTO	rs9939609	TT	LOW	No predisposition to insulin resistance.
FTO	rs1121980	GG	LOW	No predisposition to insulin resistance.

INDICATIONS

LOW INSULIN RESISTANCE
Low inherited risk of insulin resistance

MEDIUM-LOW INSULIN RESISTANCE
Medium-low inherited risk of insulin resistance

MEDIUM-HIGH INSULIN RESISTANCE
Medium-high inherited risk of insulin resistance

HIGH INSULIN RESISTANCE
High inherited risk of insulin resistance

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 7 Glucose metabolism

7. 7. 3. Risk of Type-II diabetes

RESULT	ABOUT
MEDIUM-LOW DIABETES TYPE-II RISK	Type-II diabetes mellitus (T2DM) is caused by complex interplay between multiple genetic and environmental factors. In this category, a complete analysis of the main genetic variants related to an increase in the risk of developing Type-II diabetes is analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	**HIGH**	Increased risk of diabetes type 2.
PLIN1	rs2289487	CC	**HIGH**	Increased risk of type 2 diabetes.
TCF7L2	rs7903146	CC	LOW	Normal risk of diabetes type 2.
FTO	rs9939609	TT	LOW	Normal risk of diabetes type 2.
MC4R	rs17700633	GG	LOW	No predisposition to obesity and type 2 diabetes.
CDKN2A/B	rs10811661	CT	**HIGH**	High risk of type 2 diabetes.
KCNQ1	rs2237892	CC	**HIGH**	Increased risk of type 2 diabetes.
CDKN2A, CDKN2B	rs2383208	AG	MEDIUM	Increased risk of type 2 diabetes.
CDKAL1	rs7756992	AA	LOW	Normal risk of type 2 diabetes.
TCF7L2	rs7901695	TT	LOW	Normal risk of type 2 diabetes.

INDICATIONS

LOW DIABETES TYPE-II RISK
Normal diabetes type-II risk.

MEDIUM-LOW DIABETES TYPE-II RISK
Medium-low risk of developing type-II diabetes.

MEDIUM-HIGH DIABETES TYPE-II RISK
Medium-high risk of developing type-II diabetes.

HIGH DIABETES TYPE-II RISK
High risk of developing type-II diabetes.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 8 Efficacy of exercise

7. 8. 1. Benefits from endurance exercise for improving HDL levels

RESULT	ABOUT
VERY LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL	The predisposition to improving the HDL cholesterol levels via exercising is analysed. The expected efficacy of exercise on cholesterol regulation differs between individuals and is highly dependant on your genetics.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARD	rs2016520	TT	HIGH	No predisposition to increase HDL cholesterol levels in response to endurance exercise.

INDICATIONS

HIGH BENEFITS FROM EXERCISE FOR IMPROVING HDL
Exercise will be strongly beneficial for cholesterol regulation (HDL increase).

MEDIUM-HIGH BENEFITS FROM EXERCISE FOR IMPROVING HDL
Exercise will be beneficial for cholesterol regulation (HDL increase).

MEDIUM-LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL
Exercise alone will not be enough for cholesterol regulation.

VERY LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL
Exercise alone will not be enough for cholesterol regulation.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 8 Efficacy of exercise

7. 8. 2. Exercise to reduce body fat

RESULT	ABOUT
VERY LOW BENEFIT FROM EXERCISE TO REDUCE FAT	The efficacy of physical activity to reduce body fat is different among all of us and the cause is mainly genetic. This is the reason why some people, even exercising daily tend to lose less weight than others exercising a couple of times a week. In this category, the genes related to the efficacy of exercise to reduce body fat are analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
FTO	rs9939609	TT	**HIGH**	No predisposition to lose fat during physical exercise.
FTO	rs1121980	GG	**HIGH**	Predisposition to not lose fat during physical exercise.
LIPC	rs1800588	CT	MEDIUM	Slight predisposition to benefit from physical exercise to increase HDL cholesterol levels.
LEP	rs7799039	AG	MEDIUM	Predisposition to medium response to exercise-induced fat loss.

INDICATIONS

HIGH BENEFIT FROM EXERCISE TO REDUCE FAT
An exercise strategy will be a very good option for weight loss. Exercise 3-4 times per week at medium-high intensity will be beneficial for slimming. Introduce also some diet restrictions.

MEDIUM-HIGH BENEFIT FROM EXERCISE TO REDUCE FAT
An exercise strategy may be a good option for weight loss. Exercise 2-3 times per week at medium-high intensity will be beneficial for slimming. Also introduce some diet restrictions.

MEDIUM-LOW BENEFIT FROM EXERCISE TO REDUCE FAT
An exercise strategy may not be the best option for weight loss. Rather introduce diet restrictions and institute healthy sport-related habits (walking, swimming at low intensity).

VERY LOW BENEFIT FROM EXERCISE TO REDUCE FAT
An exercise strategy may not be the best option for weight loss. Rather introduce diet restrictions and institute healthy sport-related habits (walking, swimming at low intensity).

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 9 Detoxification imbalances

7. 9. 1. Antioxidant capability

RESULT	ABOUT
NORMAL ANTIOXIDANT CAPABILITY	The balance between production and clearance of reactive oxygen species (ROS) is essential for cell survival. Antioxidant cellular systems evolved to maintain a redox homeostasis under different physiological and pathological conditions. Therefore, understanding the status of the antioxidant mechanisms is a key factor for health improvement. The main genes involved in the human antioxidant capability are analysed in this category, allowing us to understand whether we need extra help via specific supplementation or if our internal antioxidant mechanisms work properly.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
GPX1	rs1050450	GG	LOW	Predisposition to normal hydrogen peroxide detoxification.
NQO1	rs1800566	AG	MEDIUM	Predisposition to reduced NQO1 activity resulting in less effective protection against oxidative stress.
COMT	rs4680	AG	MEDIUM	Predisposition to slightly reduced COMT enzyme activity resulting in a less efficient inactivation of neurotransmitters and catecholestrogens.
SOD2	rs4880	AG	LOW	Predisposition to normal hydrogen peroxide detoxification.
CYP1B1	rs1056836	CG	MEDIUM	Predisposition to increased CYP1B1 activity which could result in an increased accumulation of carcinogenic products.
CYP1A1	rs1048943	TT	LOW	Predisposition to normal CYP1A1 enzyme activity.
GSTP1	rs1695	AA	LOW	Predisposition to normal GSTP1 activity.

INDICATIONS

NORMAL ANTIOXIDANT CAPABILITY
Normal capacity of metabolizing free radicals and cellular toxins.

SLIGHTLY REDUCED ANTIOXIDANT CAPABILITY
Slightly reduced capability of metabolizing free radicals and cellular toxins.

REDUCED ANTIOXIDANT CAPABILITY
Reduced capability of metabolizing free radicals and cellular toxins. Increased risk of cellular damage. Prescribe supplementation as suggested at gene level.

LOW ANTIOXIDANT CAPABILITY
Low capability of metabolizing free radicals and cellular toxins. High risk of cellular damage. Prescribe supplementation as suggested at gene level.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 10 Intolerance

7. 10. 1. Lactose intolerance risk

RESULT	ABOUT
LOWER RISK OF LACTOSE INTOLERANCE	Lactose intolerance means that there are insufficient lactase enzymes to break down all the consumed lactose in the intestine. Partially digested or undigested lactose passes into the large intestine and that causes symptoms such as pain, abdominal bloating and diarrhea.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
MCM6	rs182549	TT	LOW	Normal predisposition to lactose tolerance.
MCM6	rs4988235	AA	LOW	Normal predisposition to lactose tolerance.

INDICATIONS

LOWER RISK OF LACTOSE INTOLERANCE
Lower risk of lactose intolerance.

SLIGHTLY INCREASED RISK LACTOSE INTOLERANCE
Slightly increased risk of lactose intolerance. Lower capability to digest lactose. Rather reduce the lactose intake.

MEDIUM-HIGH RISK LACTOSE INTOLERANCE
Medium-high risk of lactose intolerance. Lower capability to digest lactose. Rather reduce or avoid lactose-rich food.

LACTOSE INTOLERANCE
Lactose intolerance. Move to a lactose-free diet.

If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advisable to eliminate as much dairy products from their diet as possible.

Major intestinal symptoms following ingestion of dairy products.

- ▶ Nausea
- ▶ Abdominal pain
- ▶ Spasms
- ▶ Swelling and abdominal bloating
- ▶ Abdominal gases and flatulence
- ▶ Acidic diarrhea
- ▶ Vomiting

Other nonspecific symptoms due to an alteration of the intestinal mucosa.

- ▶ Abatement
- ▶ Tiredness
- ▶ Extremities pain
- ▶ Skin problems
- ▶ Reduced mental concentration
- ▶ Nervousness
- ▶ Sleep Disorders

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 10 Intolerance

7. 10. 2. Risk of celiac disease

RESULT	ABOUT
MEDIUM-HIGH RISK OF CELIAC DISEASE	Celiac disease is an autoimmune disorder that occurs in genetically predisposed people where the ingestion of gluten leads to damage in the small intestine and causes digestive problems such as malabsorption of nutrients, abdominal pain or diarrhea. There are different risk haplotypes for celiac disease, the most prevalent is the haplotype HLA-DQ2.5 that covers 90% of celiac disease patients. However, there are other haplotypes (HLA-DQ2.2, HLA-DQ8) which account for 10% of cases and increase the risk of suffering celiac disease. This test determines whether or not an at-risk individual carries this additional risk.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
HLA-DQ2.5	rs2187668	CT	MEDIUM	Increased risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.
HLA-DQ7.5	rs4639334	GA	MEDIUM	Slightly increased risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.
HLA-DQ2.2	rs2395182	TT	MEDIUM	Slightly increased risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.
HLA-DQ2.2	rs4713586	AA	MEDIUM	Slightly increased risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.
HLA-DQ8	rs7454108	TT	LOW	Normal risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.
HLA-DQ2.2	rs7775228	TT	LOW	Normal risk of celiac disease which should be analysed in combination with the other SNPs tested in HLA-DQ region. See the haplotypes in the table below.

INDICATIONS

NO ADDITIONAL RISK OF CELIAC DISEASE
No additional risk of celiac disease

LOW RISK OF CELIAC DISEASE
Carrier of celiac disease risk variant. Try to reduce the gluten intake (consult your specialist before making any dietary changes).

MEDIUM-HIGH RISK OF CELIAC DISEASE
Carrier of celiac disease risk variants. Try to reduce or avoid gluten-containing food (consult your specialist before making any dietary changes).

HIGHER RISK OF CELIAC DISEASE
The genetic test indicates a high risk of developing celiac disease. Before initiating any dietary changes, consult your specialist for further analysis.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 10 Intolerance

7. 10. 2. Risk of celiac disease

HAPLOTYPE	HAPLOTYPE RESULT	HAPLOTYPE SNP DESCRIPTION	HAPLOTYPE RISK
DQ2.5/DQ2.5	Absent	DQ2.5/DQ2.5 = rs2187668 (TT)	HIGH
DQ2.5/DQ2.2	Absent	DQ2.5 = rs2187668 (T) & DQ2.2=rs2395182 (T) + rs7775228 (C) + rs4713586 (A)	HIGH
DQ2.2/DQ2.2	Absent	DQ2.2/DQ2.2=rs2395182 (TT) + rs7775228 (CC) + rs4713586 (AA)	MEDIUM
DQ2.5	Present	DQ2.5 = rs2187668 (T)	MEDIUM
DQ2.5/DQ8	Absent	DQ2.5= rs2187668 (T) & DQ8= rs7454108 (C)	MEDIUM
DQ2.5/DQ7.5	Present	DQ2.5= rs2187668 (T) & DQ7.5=rs4639334 (A)	MEDIUM
DQ2.2	Absent	DQ2.2=rs2395182 (T) + rs7775228 (C) + rs4713586 (A)	MEDIUM
DQ2.2/DQ8	Absent	DQ2.2 =rs2395182 (T) + rs7775228 (C) + rs4713586 (A) & DQ8= rs7454108 (C)	MEDIUM
DQ2.2/DQ7.5	Absent	DQ2.2 = rs2395182 (T) + rs7775228 (C) + rs4713586 (A) & DQ7.5=rs4639334 (A)	MEDIUM
DQ8/DQ8	Absent	DQ8/DQ8= rs7454108 (CC)	MEDIUM
DQ8/DQ7.5	Absent	DQ8= rs7454108 (C) & DQ7.5=rs4639334 (A)	MEDIUM
DQ8	Absent	DQ8= rs7454108 (C)	MEDIUM
DQ7.5/DQ7.5	Absent	DQ7.5/DQ7.5=rs4639334 (AA)	LOW
DQ7.5	Present	DQ7.5=rs4639334 (A)	LOW

If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advisable to eliminate as much gluten products from their diet as possible

Major intestinal symptoms
following ingestion of gluten products.

- Bloating
- Abdominal pain
- Skin problems
- Diarrhea, constipation and smelly feces
- Headaches
- Feeling tired
- Unexplained weight loss

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Complete genetic results

7

7. 10 Intolerance

7. 10. 3. Fructose intolerance risk

RESULT	ABOUT
LOWER RISK OF FRUCTOSE INTOLERANCE	Fructose malabsorption, or dietary fructose intolerance, occurs when cells on the surface of the intestines aren't able to break down fructose efficiently. Fructose is a simple sugar, known as a monosaccharide, that comes mostly from fruit and some vegetables. It's also found in honey, agave nectar, and many processed foods that contain added sugars. Symptoms of fructose malabsorption/intolerance include nausea, abdominal pain, diarrhea, vomiting, chronic fatigue, among others.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ALDOB	rs1800546	CC	LOW	No predisposition to develop hereditary fructose intolerance.
ALDOB	rs76917243	GG	LOW	No predisposition to develop hereditary fructose intolerance.

INDICATIONS

LOWER RISK OF FRUCTOSE INTOLERANCE
Lower risk of fructose intolerance.

SLIGHTLY INCREASED RISK FRUCTOSE INTOLERANCE
Slightly increased risk of fructose intolerance. Lower capability to digest fructose. Rather reduce the fructose intake.

MEDIUM-HIGH RISK FRUCTOSE INTOLERANCE
Medium-high risk of fructose intolerance. Lower capability to digest fructose. Rather reduce or avoid fructose-rich food.

HIGH RISK FRUCTOSE INTOLERANCE
Fructose intolerance. Move to a fructose-free diet.

If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advisable to eliminate as much fructose products from their diet as possible

Major intestinal symptoms
following ingestion of fructose products.

- Nausea
- Bloating
- Abdominal pain
- Vomiting
- Diarrhea, constipation and smelly feces
- Chronic fatigue
- Malabsorption of certain nutrients, such as iron

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Complete genetic results

7

7. 10 Intolerance

7. 10. 4. Caffeine metabolism

RESULT	ABOUT
FAST CAFFEINE METABOLIZER	Metabolism of caffeine. Slower metabolism implies that caffeine will take longer to be degraded and therefore its effects will be more noticeable. However there is a risk of feeling anxious due to excessive consumption. On the other hand, faster metabolism implies that the patient will tend to increase consumption to get the same stimulating effects, since caffeine will be rapidly degraded.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
CYP1A1	rs2470893	TT	LOW	Predisposition to fast caffeine metabolism.
CYP1A2	rs762551	AA	LOW	Predisposition to fast caffeine metabolism.

INDICATIONS

FAST CAFFEINE METABOLIZER
Fast speed of caffeine metabolism and increased desire to drink coffee in order to feel the benefits.

INTERMEDIATE-FAST CAFFEINE METABOLIZER
Intermediate speed of caffeine metabolism.

SLOW-INTERMEDIATE CAFFEINE METABOLIZER
Slow caffeine metabolism speed: caffeine will last longer in the body. Be careful with excess caffeine.

SLOW CAFFEINE METABOLIZER
Very slow caffeine metabolism speed: caffeine will last longer in the body. Be careful with excess caffeine.

If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advisable to eliminate as much caffeine products from their diet as possible.

Major intestinal symptoms
following ingestion of caffeine products.

- Headaches
- Acing heartbeat
- Jitters
- Nervousness or anxiousness
- Restlessness
- Insomnia

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Complete genetic results

7

7. 10 Intolerance

7. 10. 5. Alcohol metabolism

RESULT	ABOUT
NORMAL ALCOHOL METABOLISM	People of certain genetic type may experience symptoms like redness or flushing of the face and neck after consuming alcohol. These reactions can result from variants in the ALDH2 gene which is involved in breaking down alcohol.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ALDH2	rs671	GG	LOW	Predisposition to normal alcohol metabolism.

INDICATIONS

NORMAL ALCOHOL METABOLISM
Normal risk of alcohol toxicity due to a normal metabolism.

NORMAL-INTERMEDIATE ALCOHOL METABOLISM
Moderate risk of alcohol toxicity due to a slightly slower metabolism.

INTERMEDIATE-SLOW ALCOHOL METABOLISM
Medium-high risk of alcohol toxicity due to slow metabolism.

SLOW ALCOHOL METABOLISM
High risk of alcohol toxicity due to slow metabolism.

If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advisable to eliminate as much alcohol products from their diet as possible.

Major intestinal symptoms
following ingestion of alcohol products.

- Vomiting
- Facial redness (flushing)
- Red, itchy skin bumps (hives)
- Worsening of pre-existing asthma
- Runny or stuffy nose
- Low blood pressure
- Nausea and vomiting

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Complete genetic results

7

7. 11 Hormones

7. 11. 1. Leptin

RESULT	ABOUT
HIGH RISK OF DECREASED LEPTIN LEVELS	Leptin is a hormone which main function is sending a signal to the brain for food intake regulation. Leptin is commonly called the "satiety hormone". Low levels of leptin may imply problems of overeating and/or burning the stored fat. LEP-R is the gene coding for the cellular receptor of the leptin hormone. Its capability to bind leptin and start the cellular signalling is key for the satiety regulation function. Lower leptin binding capability may lead to high possibilities of leptin resistance, overeating and lower fat burning.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
LEP	rs7799039	AG	**HIGH**	Predisposition to increased levels of leptin leading to leptin receptor saturation that may lead to increased risk of overeating and lower fat burning.

INDICATIONS

LOW RISK OF DECREASED LEPTIN LEVELS
Low Risk Of Decreased Leptin Levels

MEDIUM-LOW RISK OF DECREASED LEPTIN LEVELS
Medium-Low Risk Of Decreased Leptin Levels

MEDIUM-HIGH RISK OF DECREASED LEPTIN LEVELS
Medium-High Risk Of Decreased Leptin Levels

HIGH RISK OF DECREASED LEPTIN LEVELS
High Risk Of Decreased Leptin Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 11 Hormones

7. 11. 2. Visfatin

RESULT	ABOUT
LOW RISK OF INCREASED VISFATIN LEVELS	Visfatin is an adipokine with an inflammatory and catabolic profile that has been associated with several metabolic risk factors, such as obesity, insulin resistance, and Type-II diabetes.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
NAMPT	rs9770242	CC	LOW	No predisposition to increased levels of circulating visfatin predisposing to normal inflammatory response.

INDICATIONS

LOW RISK OF INCREASED VISFATIN LEVELS
Low Risk Of Increased Visfatin Levels

MEDIUM-LOW RISK OF INCREASED VISFATIN LEVELS
Medium-Low Risk Of Increased Visfatin Levels

MEDIUM-HIGH RISK OF INCREASED VISFATIN LEVELS
Medium-High Risk Of Increased Visfatin Levels

HIGH RISK OF INCREASED VISFATIN LEVELS
High Risk Of Increased Visfatin Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 11 Hormones

7. 11. 3. Ghrelin

RESULT	ABOUT
HIGH GHRELIN RECEPTOR (GHSR) EXPRESSION	Ghrelin is a hormone produced in the gut, often termed "the hunger hormone", since it causes an increase in appetite through its effect in the brain. Imbalances in ghrelin are associated with appetite increase, increased calorie consumption and fat storage.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
GHSR	rs490683	GG	**HIGH**	Predisposition to normal ghrelin receptor (GHSR) expression. Circulating levels of this hormone and the sensation of hunger are increased.

INDICATIONS

LOW GHRELIN RECEPTOR (GHSR) EXPRESSION
Low Ghrelin Receptor (Ghsr) Expression

LOW-INTERMEDIATE GHRELIN RECEPTOR (GHSR) EXPRESSION
Low-intermediate ghrelin receptor (ghsr) expression

INTERMEDIATE- HIGH GHRELIN RECEPTOR (GHSR) EXPRESSION
Intermediate- High Ghrelin Receptor (Ghsr) Expression

HIGH GHRELIN RECEPTOR (GHSR) EXPRESSION
High Ghrelin Receptor (GHSR) Expression

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 11 Hormones

7. 11. 4. Adiponectin

RESULT	ABOUT
HIGH RISK OF DECREASED ADIPONECTIN LEVELS	Adiponectin is a hormone that regulates glucose levels and fatty acid breakdown. Low levels of adiponectin are associated with inflammation, lipid abnormalities and insulin resistance.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ADIPOQ	rs1501299	GG	**HIGH**	High predisposition to lower adiponectin plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.
ADIPOQ	rs2241766	TT	**HIGH**	High predisposition to lower adiponectin plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.

INDICATIONS

LOW RISK OF DECREASED ADIPONECTIN LEVELS
Low Risk Of Decreased Adiponectin Levels

MEDIUM-LOW RISK OF DECREASED ADIPONECTIN LEVELS
Medium-Low Risk Of Decreased Adiponectin Levels

MEDIUM-HIGH RISK OF DECREASED ADIPONECTIN LEVELS
Medium-High Risk Of Decreased Adiponectin Levels

HIGH RISK OF DECREASED ADIPONECTIN LEVELS
High Risk Of Decreased Adiponectin Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 12 Inflammation

7. 12. 1. TNF-α

RESULT	ABOUT
MEDIUM-LOW RISK OF INCREASED TNF-α LEVELS	TNF-α is a pro-inflammatory cytokine, strongly linked to many inflammatory conditions, expressed in, and secreted by adipose tissues. Increased levels are associated with obesity-induced inflammation, adiposity and insulin resistance.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
TNF-α	rs1800629	AG	MEDIUM	Predisposition to moderately increased levels of TNF-alpha. Pro-inflammation tendency.

INDICATIONS

LOW RISK OF DYSREGULATED TNF-α LEVELS.
Low Risk Of Dysregulated Tnf-A Levels.

MEDIUM-LOW RISK OF INCREASED TNF-α LEVELS
Medium-Low Risk Of Increased TNF-α Levels

MEDIUM-HIGH RISK OF INCREASED TNF-α LEVELS
Medium-High Risk Of Increased TNF-α Levels

HIGH RISK OF INCREASED TNF-α LEVELS
High Risk Of Increased TNF-α Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 12 Inflammation

7. 12. 2. IL-6

RESULT	ABOUT
HIGH RISK OF INCREASED IL-6 LEVELS	IL-6 is an interleukin with mainly pro-inflammatory functions and is commonly used as inflammatory marker. High levels of IL-6 are associated with obesity, insulin resistance and metabolic syndrome.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
IL-6	rs1800795	GG	**HIGH**	Predisposition to highly increased levels of IL-6. Pro-inflammation.

INDICATIONS

LOW RISK OF INCREASED IL-6 LEVELS
Low Risk Of Increased Il-6 Levels

MEDIUM-LOW RISK OF INCREASED IL-6 LEVELS
Medium-Low Risk Of Increased Il-6 Levels

MEDIUM-HIGH RISK OF INCREASED IL-6 LEVELS
Medium-High Risk Of Increased Il-6 Levels

HIGH RISK OF INCREASED IL-6 LEVELS
High Risk Of Increased Il-6 Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 12 Inflammation

7. 12. 3. IL-10

RESULT	ABOUT
LOW RISK OF DECREASED ANTIINFLAMATORY CYTOKINE IL-10 LEVELS	IL-10 is a cytokine with potent anti-inflammatory properties.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
IL-10	rs1800896	CC	LOW	Predisposition to higher levels of the anti-inflammatory cytokine IL-10.

INDICATIONS

LOW RISK OF DECREASED ANTIINFLAMATORY CYTOKINE IL-10 LEVELS
Low Risk Of Decreased Antiinflamatory Cytokine Il-10 Levels

MEDIUM-LOW RISK OF DECREASED ANTIINFLAMATORY CYTOKINE IL-10 LEVELS
Medium-Low Risk Of Decreased Antiinflamatory Cytokine Il-10 Levels

MEDIUM-HIGH RISK OF DECREASED ANTIINFLAMATORY CYTOKINE IL-10 LEVELS
Medium-High Risk Of Decreased Antiinflamatory Cytokine Il-10 Levels

HIGH RISK OF DECREASED ANTIINFLAMATORY CYTOKINE IL-10 LEVELS
High Risk Of Decreased Antiinflamatory Cytokine Il-10 Levels

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 13 Vitamins

A

7. 13. 1. Vitamin A

RESULT	ABOUT
MEDIUM-LOW RISK OF VITAMIN A DEFICIENCY	Inherited risk of vitamin A metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
BCMO1	rs12934922	AT	MEDIUM	Increased predisposition to reduced provitamin A conversion and increased fasting β-carotene concentrations.
BCMO1	rs7501331	CT	MEDIUM	Increased predisposition to reduced provitamin A conversion.

INDICATIONS

LOW RISK OF VITAMIN A DEFICIENCY
Normal vitamin A metabolism. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN A DEFICIENCY
Low risk of vitamin A deficiency. Ensure daily recommended intake or slightly increase it.

MEDIUM-HIGH RISK OF VITAMIN A DEFICIENCY
Medium risk of vitamin A deficiency. Ensure daily recommended intake and increase it. Supplementation should be evaluated.

HIGH RISK OF VITAMIN A DEFICIENCY
High risk of vitamin A deficiency. Increase daily vitamin A intake. Supplementation should be evaluated.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 13 Vitamins

7. 13. 2. Vitamin B6

RESULT	ABOUT
HIGH RISK OF VITAMIN B6 DEFICIENCY	Inherited risk of vitamin B6 metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
NBPF3	rs4654748	CC	HIGH	High risk of low plasma vitamin B6 concentrations.

INDICATIONS

LOW RISK OF VITAMIN B6 DEFICIENCY
Normal vitamin B6 metabolism. Ensure daily recommended intake.

MODERATE RISK OF VITAMIN B6 DEFICIENCY
Little predisposition to a vitamin B6 deficiency. Make sure that the recommended daily intake is met.

MEDIUM-HIGH RISK OF VITAMIN B6 DEFICIENCY
Medium risk of vitamin B6 deficiency. Ensure daily recommended intake and increase it. Supplementation should be evaluated.

HIGH RISK OF VITAMIN B6 DEFICIENCY
High risk of vitamin B6 deficiency. Increase daily vitamin B6 intake. Supplementation should be evaluated.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 13 Vitamins

7. 13. 3. Vitamin B9 (folate)

RESULT	ABOUT
LOW RISK OF VITAMIN B9 (Folate) DEFICIENCY	Inherited risk of vitamin B9 (folate) metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
MTHFR	rs1801133	GG	LOW	Normal risk of folate deficiency.

INDICATIONS

LOW RISK OF VITAMIN B9 (Folate) DEFICIENCY
Normal folate metabolism. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN B9 (Folate) DEFICIENCY
Low risk of folate deficiency. Ensure daily recommended intake.

MEDIUM-HIGH RISK OF VITAMIN B9 (Folate) DEFICIENCY
Medium risk of folate deficiency. Ensure daily recommended intake. It is recommended to supplement with L-methylfolate due to a lower capability to activate folate. It also impacts lower B12 levels when low levels of folate are active.

HIGH RISK OF VITAMIN B9 (Folate) DEFICIENCY
High risk of folate deficiency. Ensure daily recommended intake. Highly recommended to supplement with L-methylfolate due to a almost null capability to activate folate. It also impacts lower B12 levels when low levels of folate are active.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 13 Vitamins

7. 13. 4. Vitamin B12

RESULT	ABOUT
HIGH RISK OF VITAMIN B12 DEFICIENCY	Inherited risk of vitamin B12 metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
FUT2	rs602662	GG	**HIGH**	High risk of vitamin B12 deficiency.

INDICATIONS

LOW RISK OF VITAMIN B12 DEFICIENCY
Normal vitamin B12 metabolism. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN B12 DEFICIENCY
Low risk of vitamin B12 deficiency. Ensure daily recommended intake.

MEDIUM-HIGH RISK OF VITAMIN B12 DEFICIENCY
Medium risk of vitamin B12 deficiency. Ensure daily recommended intake and increase it. Supplementation should be evaluated.

HIGH RISK OF VITAMIN B12 DEFICIENCY
High risk of vitamin B12 deficiency. Increase daily vitamin B12 intake. Supplementation should be evaluated.

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Complete genetic results

7

7. 13 Vitamins

C

7. 13. 5. Vitamin C

RESULT	ABOUT
LOW RISK OF VITAMIN C DEFICIENCY	Inherited risk of vitamin C metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
SLC23A2	rs1279683	GA	LOW	Normal risk of vitamin C deficiency.
SLC23A1	rs33972313	CC	LOW	Normal risk of vitamin C deficiency.

INDICATIONS

LOW RISK OF VITAMIN C DEFICIENCY
Normal vitamin C metabolism and levels. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN C DEFICIENCY
Low risk of Viamin C deficiency. Ensure daily recommended intake.

MEDIUM-HIGH RISK OF VITAMIN C DEFICIENCY
Medium risk of Vitamin C deficiency. Ensure daily recommended intake. Supplementation strategies might be of interest.

HIGH RISK OF VITAMIN C DEFICIENCY
High risk of Vitamin C deficiency. Ensure daily recommended intake. Supplementation strategies would be recommended.

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Complete genetic results

7

7. 13 Vitamins

D

7. 13. 6. Vitamin D

RESULT	ABOUT
MEDIUM-LOW RISK OF VITAMIN D DEFICIENCY	Inherited risk of vitamin D metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
GC	rs2282679	TT	LOW	Normal risk of vitamin D deficiency.
CYP2R1	rs10741657	AG	**HIGH**	High risk of low serum levels of vitamin D.
NADSYN1	rs12785878	GT	MEDIUM	Increased risk of lower serum levels of vitamin D.
CYP2R1	rs2060793	AG	MEDIUM	Increased risk of lower serum levels of vitamin D.
NADSYN1	rs3829251	AG	MEDIUM	Increased risk of lower serum levels of vitamin D.

INDICATIONS

LOW RISK OF VITAMIN D DEFICIENCY
Normal vitamin D metabolism and levels. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN D DEFICIENCY
Low risk of Viamin D deficiency. Ensure daily recommended intake.

MEDIUM-HIGH RISK OF VITAMIN D DEFICIENCY
Medium risk of Vitamin D deficiency. Ensure daily recommended intake. Supplementation strategies might be of interest.

HIGH RISK OF VITAMIN D DEFICIENCY
High risk of Vitamin D deficiency. Ensure daily recommended intake. Supplementation strategies would be recommended.

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Complete genetic results

7

7. 13 Vitamins

E

7. 13. 7. Vitamin E

RESULT	ABOUT
MEDIUM-HIGH RISK OF VITAMIN E DEFICIENCY	Inherited risk of vitamin E metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
INTERGENIC	rs12272004	CC	**HIGH**	High risk of low plasma levels of alpha-tocoferol (Vitamin E).
ZPR1	rs964184	CG	MEDIUM	Increased risk of lower plasma levels of alpha-tocoferol (Vitamin E).

INDICATIONS

LOW RISK OF VITAMIN E DEFICIENCY
Normal vitamin E metabolism and levels. Ensure daily recommended intake.

MEDIUM-LOW RISK OF VITAMIN E DEFICIENCY
Low risk of Viamin E deficiency. Ensure daily recommended intake.

MEDIUM-HIGH RISK OF VITAMIN E DEFICIENCY
Medium risk of Vitamin E deficiency. Ensure daily recommended intake. Supplementation strategies might be of interest.

HIGH RISK OF VITAMIN E DEFICIENCY
High risk of Vitamin E deficiency. Ensure daily recommended intake. Supplementation strategies would be recommended.

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Complete genetic results

7

7. 14 Minerals

7. 14. 1. Calcium malabsorption risk

RESULT	ABOUT
LOW RISK OF CALCIUM MALABSORPTION	Calcium dissolves in the stomach and is absorbed through the lining of the small intestine into the blood stream. Once in the blood stream, calcium builds bone, regulates the expansion and contraction of the blood vessels, and performs other important functions. Common factors for calcium malabsorption are a diet high in phytic acid (present in wholegrains), high levels of sodium intake, smoking and also genetic factors related to Vitamin D. In this category, the genetic factors related to a predisposition to calcium malabsorption due to lower levels of 25(OH) D (Vitamin D) are analysed. Therefore, a high risk of malabsorption would require an increase in vitamin D consumption or even controlled supplementation.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
CYP2R1	rs10766197	GG	LOW	Predisposition to normal vitamin D levels and calcium absorption.
GC	rs2282679	TT	LOW	Predisposition to normal vitamin D levels and calcium absorption.

INDICATIONS

LOW RISK OF CALCIUM MALABSORPTION
Low inherited risk of calcium malabsorption.

MEDIUM-LOW RISK OF CALCIUM MALABSORPTION
Medium-low inherited risk of calcium malabsorption.

MEDIUM-HIGH RISK OF CALCIUM MALABSORPTION
Medium-high inherited risk of calcium malabsorption.

HIGH RISK OF CALCIUM MALABSORPTION
High inherited risk of calcium malabsorption.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 14 Minerals

7. 14. 2. Predisposition to dysregulated calcium levels

RESULT	ABOUT
NO ADDITIONAL RISK OF DYSREGULATED PLASMA CALCIUM LEVELS	The predisposition to low or high levels of plasma calcium are analyzed in this category. A predisposition to high levels of calcium and increased absorption would be a warning against calcium supplementation due to the potential increased risk of vascular calcification.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
DGKD	rs1550532	CG	MEDIUM	Predisposition to slightly increased serum levels of calcium.
CYP24A1	rs1570669	AG	MEDIUM	Predisposition to slightly reduced serum calcium levels and bone mineral density.
CASR	rs17251221	AA	LOW	Predisposition to normal serum calcium levels.
CASR	rs1801725	GG	LOW	Predisposition to normal serum calcium levels.
CARS	rs7481584	GG	LOW	Predisposition to normal serum calcium levels
GCKR	rs780094	TT	LOW	Predisposition to normal serum calcium levels

INDICATIONS

NO ADDITIONAL RISK OF DYSREGULATED PLASMA CALCIUM LEVELS
No additional risk of dysregulated plasma calcium levels.

SLIGHTLY INCREASED RISK OF DYSREGULATED PLASMA CALCIUM LEVELS
Slightly increased risk of dysregulated plasma calcium levels.

INCREASED RISK OF DYSREGULATED PLASMA CALCIUM LEVELS
Increased risk of dysregulated plasma calcium levels.

HIGHER RISK OF DYSREGULATED PLASMA CALCIUM LEVELS
High risk of dysregulated plasma calcium levels.

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Complete genetic results

7

7. 14 Minerals

7. 14. 3. Risk of iron overload

RESULT	ABOUT
LOW RISK OF HEMOCHROMATOSIS	Iron overload is defined as excess stores of iron in the body. Excess iron is deposited in organs throughout the body. The most notable organs with iron deposition are the liver, heart, and endocrine glands. Resulting symptoms and diseases are related to specific organ damage. In this category, the genetic risk of iron overload on high intake is analysed.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
HFE	rs1800562	GG	LOW	Predisposition to normal absorption of dietary iron.

INDICATIONS

LOW RISK OF HEMOCHROMATOSIS
No additional risk of iron overload.

MEDIUM-LOW RISK OF HEMATOCHROMATOSIS
Some risk of having increased iron absorption on high iron intake. Avoid iron excess.

MEDIUM-HIGH RISK O FHEMATOCHROMATOSIS
Medium risk of having increased iron absorption on high iron intake. Avoid iron excess and/or supplements.

HIGH RISK OF HEMATOCHROMATOSIS
High risk of having increased iron absorption on high iron intake. Avoid iron excess and/or supplements.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 14 Minerals

7. 14. 4. Risk of low iron plasma levels

RESULT	ABOUT
MEDIUM-HIGH RISK OF DECREASED IRON LEVELS	Low iron levels may lead to anemia. In this category, the genetic risk of low transference of iron into the body is analysed. When your body has a predisposition to low iron levels, it will be necessary to ensure a diet with proper levels of iron.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
TF	rs3811647	AA	**HIGH**	Predisposition to increased serum ferretin and reduced serum iron levels.
TMPRSS6	rs4820268	AA	LOW	Predisposition to normal serum iron levels.
TF	rs8177253	TT	**HIGH**	Predisposition to increased total iron binding capacity.

INDICATIONS

LOW RISK OF DECREASED IRON LEVELS
No additional inherited risk of low iron levels.

MEDIUM-LOW RISK OF DECREASED IRON LEVELS
Some risk of having lower iron transference, only when iron intake is low. Ensure dietary daily recommended intake.

MEDIUM-HIGH RISK OF DECREASED IRON LEVELS
Moderate risk of having lower iron transference, only when iron intake is low. In that case, supplementation would be recommended.

HIGH RISK OF DECREASED IRON LEVELS
High risk of having lower iron transference, only when iron intake is low. In that case, supplementation would be recommended.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 14 Minerals

7. 14. 5. Predisposition to dysregulated magnesium levels

RESULT	ABOUT
MEDIUM-LOW RISK OF DYSREGULATED MAGNESIUM LEVELS	Inherited risk of low magnesium plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
CASR	rs17251221	AA	LOW	Predisposition to normal serum magnesium levels.
TRPM6	rs11144134	TT	HIGH	Predisposition to lower serum magnesium levels.
SHROOM3	rs13146355	AG	MEDIUM	Predisposition to slightly lower serum magnesium levels.
DCDC5	rs3925584	TT	LOW	Predisposition to normal serum magnesium levels.
MUC1	rs4072037	TT	LOW	Predisposition to normal magnesium levels.

INDICATIONS

NO ADDITIONAL RISK OF DYSREGULATED MAGNESIUM LEVELS
No additional risk of dysregulated plasma magnesium levels.

MEDIUM-LOW RISK OF DYSREGULATED MAGNESIUM LEVELS
Some risk of dysregulated plasma magnesium levels.

MEDIUM-HIGH RISK OF DYSREGULATED MAGNESIUM LEVELS
Medium risk of dysregulated plasma magnesium levels.

HIGH RISK OF DYSREGULATED MAGNESIUM LEVELS
High risk of dysregulated plasma magnesium levels.

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Complete genetic results

7

7. 14 Minerals

7. 14. 6. Predisposition to dysregulated selenium levels

RESULT	ABOUT
NO ADDITIONAL RISK OF DYSREGULATED SELENIUM LEVELS	Selenium is an essential mineral and micronutrient. It is fundamental to human health and found in many foods. It is found in meat, grain cereals, egg yolk, milk, brazil nuts, mushrooms, garlic and seafood (hence, selenium levels are high in populations with high intake of seafood). Understanding the predisposition to low or high selenium levels will help for ensuring the proper selenium daily intake.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
AGA	rs1395479	AC	MEDIUM	Predisposition to slightly increased serum levels of selenium.
SLC39A11	rs891684	GG	LOW	Predisposition to normal serum selenium levels.

INDICATIONS

NO ADDITIONAL RISK OF DYSREGULATED SELENIUM LEVELS
No additional risk of dysregulated plasma selenium levels.

MEDIUM-LOW RISK OF DYSREGULATED SELENIUM LEVELS
Some risk of dysregulated plasma selenium levels.

MEDIUM-HIGH RISK OF DYSREGULATED SELENIUM LEVELS
Medium risk of dysregulated plasma selenium levels.

HIGH RISK OF DYSREGULATED SELENIUM LEVELS
High risk of dysregulated plasma selenium levels.

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Complete genetic results

7

7. 14 Minerals

7. 14. 7. Sodium sensitivity

RESULT	ABOUT
LOW SODIUM SENSITIVITY	Inherited risk of dietary salt-induced blood pressure.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
ACE	rs4343	AA	LOW	Predisposition to normal sodium sensitivity.

INDICATIONS

LOW SODIUM SENSITIVITY
Normal sodium sensitivity: no increased blood pressure risk due to salt consumption.

MEDIUM-LOW SODIUM SENSITIVITY
Slightly increased sodium sensitivity: moderately increased blood pressure risk due to salt consumption.

MEDIUM-HIGH SODIUM SENSITIVITY
Moderate sodium sensitivity: increased blood pressure risk due to salt consumption. Reduce current salt consumption, if daily intake is high.

HIGH SODIUM SENSITIVITY
High sodium sensitivity: high blood pressure risk due to salt consumption. Reduce current salt consumption, if daily intake is high.

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Complete genetic results

7

7. 15 Effectiveness of diets

7. 15. 1. Efficacy of low calorie diets

RESULT	ABOUT
VERY LOW EXPECTED BENEFIT FROM LOW-CALORIE DIET	A complete set of genes related to the expected efficacy of a low-calorie diet is analysed in this category.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	**HIGH**	No predisposition to weight loss induced by a low calorie diet.
ADIPOQ	rs17300539	GG	**HIGH**	No predisposition to weight loss induced by a low calorie diet.
LEPR	rs1805134	TT	**HIGH**	No predisposition to weight loss induced by a low calorie diet.
ACSL5	rs2419621	CC	**HIGH**	No predisposition to weight loss induced by a low calorie diet.

INDICATIONS

VERY LOW EXPECTED BENEFIT FROM LOW-CALORIE DIET
A pure low-calorie diet may not be the best option for weight loss.

MEDIUM-LOW EXPECTED BENEFIT FROM LOW-CALORIE DIET
A pure low-calorie diet may not be the best option for weight loss. However, a reduction in calorie intake may be beneficial.

MEDIUM-HIGH EXPECTED BENEFIT FROM LOW-CALORIE DIET
A low-calorie diet may be one of the best options for weight loss. Try to dramatically reduce calorie intake.

HIGH EXPECTED BENEFIT FROM LOW-CALORIE DIET
High expected efficacy of a low-calorie diet. It is strongly recommended to follow it.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 15 Effectiveness of diets

7. 15. 2. Efficacy of low carbohydrate diets

RESULT	ABOUT
MEDIUM-HIGH EXPECTED BENEFIT FROM LOW-CARBOHYDRATE DIET	A complete set of genes related to the expected efficacy of a low-carbohydrate diet is analysed in this category.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
KCTD10	rs10850219	GG	LOW	Predisposition to weight loss induced by a low carbohydrate diet.
MMAB	rs2241201	GG	**HIGH**	No predisposition to weight loss induced by a low carbohydrate diet.

INDICATIONS

VERY LOW EXPECTED BENEFIT FROM LOW-CARBOHYDRATE DIET
A pure low-carbohydrate diet may not be the best option for weight loss.

MEDIUM-LOW EXPECTED BENEFIT FROM LOW-CARBOHYDRATES DIET
A pure low-carbohydrate diet may not be the best option for weight loss. However, a reduction in carbohydrate intake may be beneficial.

MEDIUM-HIGH EXPECTED BENEFIT FROM LOW-CARBOHYDRATE DIET
A low-carbohydrate diet may be one of the best option for weight loss. Try to dramatically reduce carbohydrate intake.

HIGH EXPECTED BENEFIT FROM LOW-CARBOHYDRATE DIET
High expected efficacy of a low-carbohydrate diet. It is strongly recommended to follow it.

* These recommendations are based only in the analysis of your genetic test. Always seek the advice of your physician or other qualified health specialist before proceeding with any nutritional or dietary modifications.

Complete genetic results

7

7. 15 Effectiveness of diets

7. 15. 3. Efficacy of low fat diets

RESULT	ABOUT
VERY LOW EXPECTED BENEFIT FROM LOW-FAT DIET	A complete set of genes related to the expected efficacy of a low-fat diet is analysed in this category.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
PPARG	rs1801282	CC	HIGH	No predisposition to weight loss induced by a low fat diet.
GHSR	rs490683	GG	HIGH	No predisposition to weight loss induced by a low fat diet. Also applicable after gastric bypass.
APOA2	rs5082	AA	HIGH	No predisposition to weight loss induced by a low fat diet.
SH2B1	rs7498665	AA	HIGH	No predisposition to weight loss induced by a low fat diet.
TCF7L2	rs7903146	CC	HIGH	No predisposition to weight loss induced by a low fat diet.
FTO	rs9939609	TT	HIGH	No predisposition to weight loss induced by a low fat diet.

INDICATIONS

VERY LOW EXPECTED BENEFIT FROM LOW-FAT DIET
A pure low-fat diet may not be the best option for weight loss.

MEDIUM-LOW EXPECTED BENEFIT FROM LOW-FAT DIET
A pure low-fat diet may not be the best option for weight loss. However, a reduction of fat intake may be beneficial.

MEDIUM-HIGH EXPECTED BENEFIT FROM LOW-FAT DIET
A low-fat diet may be one of the best options for weight loss. Try to dramatically reducefat intake.

HIGH EXPECTED BENEFIT FROM LOW-FAT DIET
The expected efficacy of a low-fat diet is high. It is strongly recommended to follow it.

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Methodology

8

How were the genetic variants selected and evaluated?

This test was developed by a multidisciplinary team of medical doctors, geneticists, and programmers, following highest quality standards. In particular, an expert team specialized in the curation of genetic variants reviewed each variant to ensure that selection, interpretation and impact of variants in the algorithms are based on the highest scientific evidence.

The following selection criteria were applied for classifying genetic variants:

Level 1A: Annotation for a variant in medical society-endorsed or implemented in a major health system.

Level 1B: Annotation for a variant where the preponderance of evidence shows an association. The association must be replicated in more than one cohort with significant p-values, and preferably will have a strong effect size.

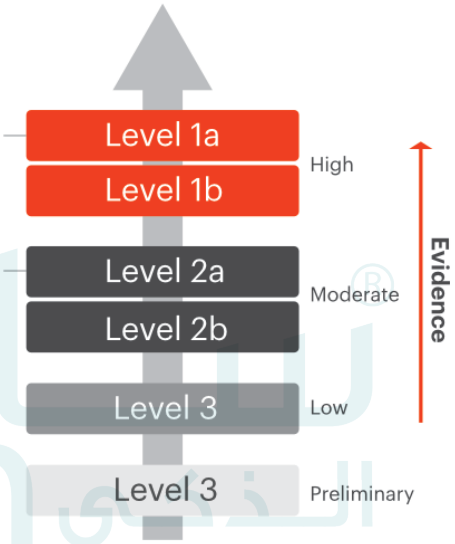
Level 2A: Annotation for a variant that qualifies for level 2B where the variant is within a Very Important known gene, so functional significance is more likely.

Level 2B: Annotation for a variant with moderate evidence of an association. The association must be replicated but there may be some studies that do not show statistical significance, and/or the effect size may be small.

Level 3: Annotation for a variant based on a single significant (not yet replicated) study or annotation for a variant evaluated in multiple studies but lacking clear evidence of an association.

Level 4: Annotation based on a case report, non-significant study or in vitro, molecular or functional assay evidence only.

Only genetic variants from level 1A to 2A were selected.



How was it analyzed?

The DNA was extracted from the buccal swab sample you provided and was analyzed by our clinical analysis laboratory. DNA was extracted using the KingFisher Flex® robotic extraction system (Thermo Fisher Scientific). The study of the genetic variants was performed by NGS (Next Generation Sequencing) using the Ion GeneStudio S5 system (Thermo Fisher Scientific).

References

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References

Scan the QR code to access our
Smart DNA - Nutrition Package report reference page.

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