



Fagron NutriGen™

Doctor report



Fagron NutriGen

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SAMPLE REPORT



Fagron NutriGen

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Patient identification data

1



Patient's name and last name — DEMO 3 WOMAN Formulation
Date of Birth — 03-03-1996
Gender — Female
Sample type — Buccal mucosa
Sample code — NUT20548AA
Sample date — 13-03-2024
Report date — 03-01-2024

Doctor's name and last name — Nutrigen 3 Validation
Email address — nutrigen_3_validation@demo.com

LEGAL DISCLAIMER

Fagron Genomics, S.L.U. carries out genetic tests upon request by healthcare professionals, in relation to biological samples from patients obtained from a healthcare professional. Our tests do not replace a medical consultation, nor do they make up a diagnostic or treatment plan. They should 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,
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IVDD
Self-Declared
98/79/EC

REF FGMS-Nutri

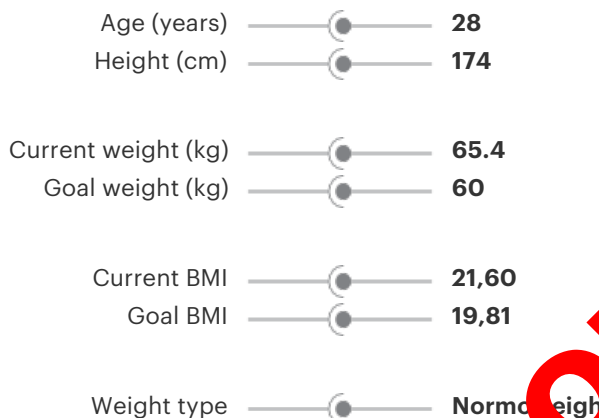
IVD In Vitro Diagnostic MedicalDevice

UDI 8437024682FGMS-NutriVC

Patient characteristics

2

Weight related variables



Physical exercise and metabolism related factors

Daily sport activity Ultra light

- Basal metabolism -



- Current DEE -









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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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.	35.71% 
	<ul style="list-style-type: none"> Genetic risk of overweight/obesity Risk of rebound weight gain Risk of increased BMI Basal metabolic rate (burn calories at rest) Weight loss capability during diet interventions 	<ul style="list-style-type: none"> MEDIUM-LOW RISK OF OVERWEIGHT/OBESITY ● HIGH REBOUND EFFECT ● MEDIUM-LOW RISK OF INCREASED BMI ● MEDIUM-LOW BURNER AT REST ● SLOW WEIGHT LOSS ● 	<ul style="list-style-type: none"> Pg. 31 Pg. 32 Pg. 33 Pg. 34 Pg. 35
3.2.	CATEGORY	DESCRIPTION	RESULTS
	 Behavioural genetics in food intake	Normal behavioural genetics related to food intake and satiety regulation. In case of excessive quantity or compulsive intake, strategies to reduce anxiety such as meditation or even clinical interventions should be considered.	83.03% 
	<ul style="list-style-type: none"> Appetite and anxiety risk Satiety: Feeling Full 	<ul style="list-style-type: none"> SLIGHTLY INCREASED APPETITE AND ANXIETY RISK ● NORMAL SATIETY ● 	<ul style="list-style-type: none"> Pg. 36 Pg. 37
3.3.	CATEGORY	DESCRIPTION	RESULTS
	 Flavour sensitivities	Moderately elevated for flavour sensitivity. Your body will crave more salt and sugar than average with associated cardiovascular and obesity risks. Try to incorporate other spices in your diet to reduce the risks.	33.22% 
	<ul style="list-style-type: none"> Bitter taste sensitivity Salt sensitivity Sweet flavour preference 	<ul style="list-style-type: none"> NORMAL BITTER TASTE SENSITIVITY ● HIGH SALT SENSITIVITY ● HIGHLY INCREASED SWEET PREFERENCE ● 	<ul style="list-style-type: none"> Pg. 38 Pg. 39 Pg. 40

INDICATIONS

■ Positive effect
 ■ Medium-positive effect
 ■ Medium-negative effect
 ■ Negative effect

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Genetic results overview

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3.4.	CATEGORY	DESCRIPTION	RESULTS
	 Fat metabolism	Moderate fat burning capacity. A healthy fat intake should not be a reason for being overweight.	51% 
	<ul style="list-style-type: none"> • Response to monounsaturated fats (MUFAs) VERY LOW MUFA METABOLISM ● Pg. 41 • Response to polyunsaturated fats (PUFAs) FAST PUFA METABOLISM ● Pg. 42 • Response to fat intake to improve the HDL levels MEDIUM-HIGH BENEFITS TO IMPROVE HDL ● Pg. 43 		
3.5.	CATEGORY	DESCRIPTION	RESULTS
	 Lipid metabolism	Highly affected lipid metabolism. Cholesterol and triglyceride levels may show irregular results in blood analyses. Specific LDL or HDL targets are recommended. High cardiovascular risk.	16.86% 
	<ul style="list-style-type: none"> • Predisposition to reduced HDL levels REDUCED HDL LEVELS ● Pg. 44 • Predisposition to increased levels of triglycerides HIGHLY INCREASED TRIGLYCERIDES ● Pg. 45 • Predisposition to increased oxidation of LDL HIGHLY INCREASED LDL OXIDATION ● Pg. 46 • Risk of increased cholesterol LDL levels INCREASED LDL LEVELS ● Pg. 47 • Risk of unbalanced Triglycerides/HDL ratio HIGHLY INCREASED TG/HDL RATIO ● Pg. 48 		
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.	31.58% 
	<ul style="list-style-type: none"> • Capability to digest starchy foods REDUCED STARCH DIGESTION ● Pg. 49 • Refined carbohydrate sensitivity NORMAL CARBOHYDRATE SENSITIVITY ● Pg. 50 • Carbohydrates and HDL levels predisposition HIGH RISK OF HDL DYSREGULATION ● Pg. 51 • Carbohydrates and LDL levels HIGH RISK OF LDL DYSREGULATION ● Pg. 52 		

INDICATIONS

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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.	35.04% 
	<ul style="list-style-type: none"> • Risk of increased glucose levels in plasma after fasting • Risk of insulin resistance • Risk of Type-II diabetes 	<p>MEDIUM-HIGH RISK OF HIGH GLUCOSE LEVELS ● Pg. 53</p> <p>MEDIUM-HIGH INSULIN RESISTANCE ● Pg. 54</p> <p>HIGH DIABETES TYPE-II RISK ● Pg. 55</p>	
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.	12.72% 
	<ul style="list-style-type: none"> • Benefits from endurance exercise for improving HDL levels • Exercise to reduce body fat 	<p>VERY LOW BENEFIT FROM EXERCISE FOR IMPROVING HDL ● Pg. 56</p> <p>MEDIUM-LOW BENEFIT FROM EXERCISE TO REDUCE FAT ● Pg. 57</p>	
3.9.	CATEGORY	DESCRIPTION	RESULTS
	 Detoxification imbalances	Slightly reduced detoxification capacities. Try to decrease toxin exposure and intake.	71.19% 
	<ul style="list-style-type: none"> • Antioxidant capability 	<p>SLIGHTLY REDUCED ANTIOXIDANT CAPABILITY ● Pg. 58</p>	
3.10.	CATEGORY	DESCRIPTION	RESULTS
	 Intolerance	Please find below the different analysed categories related to intolerances and sensitivities.	
	<ul style="list-style-type: none"> • Lactose intolerance risk • Risk of celiac disease • Fructose intolerance risk • Caffeine metabolism • Alcohol metabolism 	<p>LOWER RISK OF LACTOSE INTOLERANCE ● Pg. 59</p> <p>MEDIUM-HIGH RISK OF CELIAC DISEASE ● Pg. 60</p> <p>LOWER RISK OF FRUCTOSE INTOLERANCE ● Pg. 62</p> <p>SLOW CAFFEINE METABOLIZER ● Pg. 63</p> <p>NORMAL ALCOHOL METABOLISM ● Pg. 64</p>	

INDICATIONS




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 ■ Medium-negative effect
 ■ Negative effect

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Genetic results overview

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3.11. Hormones

CATEGORY	DESCRIPTION
 Leptin <ul style="list-style-type: none"> • LEP 	<p>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.</p> <p>Predisposition to normal levels of leptin leading to normal satiety regulation.</p>
 Visfatin <ul style="list-style-type: none"> • NAMPT 	<p>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.</p> <p>High predisposition to increased levels of circulating visfatin augmenting considerably the risk of an altered inflammatory response.</p>
 Ghrelin <ul style="list-style-type: none"> • GHSR 	<p>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.</p> <p>Predisposition to normal ghrelin receptor (GHSR) expression. Circulating levels of this hormone and the sensation of hunger are increased.</p>
 Adiponectin <ul style="list-style-type: none"> • ADIPOQ • ADIPOQ 	<p>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.</p> <p>High predisposition to lower adiponectin plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.</p> <p>Increased predisposition to lower adiponectin plasma levels that leads to a slightly increased inflammation process, lipid abnormalities and insulin resistance.</p>

INDICATIONS


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Genetic results overview


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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 average levels of TNF-alpha. ●

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 intermediate levels of IL-6. Pro-inflammation. ●

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

• IL-10

Predisposition to intermediate levels of the anti-inflammatory cytokine IL-10. ●

SAMPLE REPORT

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	Normal vitamin A metabolism. Ensure daily recommended intake.	
Vitamin B6	High risk of vitamin B6 deficiency. Increase daily vitamin B6 intake. Supplementation should be evaluated.	
Vitamin B9	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.	
Vitamin B12	High risk of vitamin B12 deficiency. Increase daily vitamin B12 intake. Supplementation should be evaluated.	
Vitamin C	Medium risk of Vitamin C deficiency. Ensure daily recommended intake. Supplementation strategies might be of interest.	
Vitamin D	Low risk of Vitamin D deficiency. Ensure daily recommended intake.	
Vitamin E	High risk of Vitamin E deficiency. Ensure daily recommended intake. Supplementation strategies would be recommended.	

SAMPLE REPORT


INDICATIONS


■ Positive effect
 ■ Medium-positive effect
 ■ Medium-negative effect
 ■ Negative effect

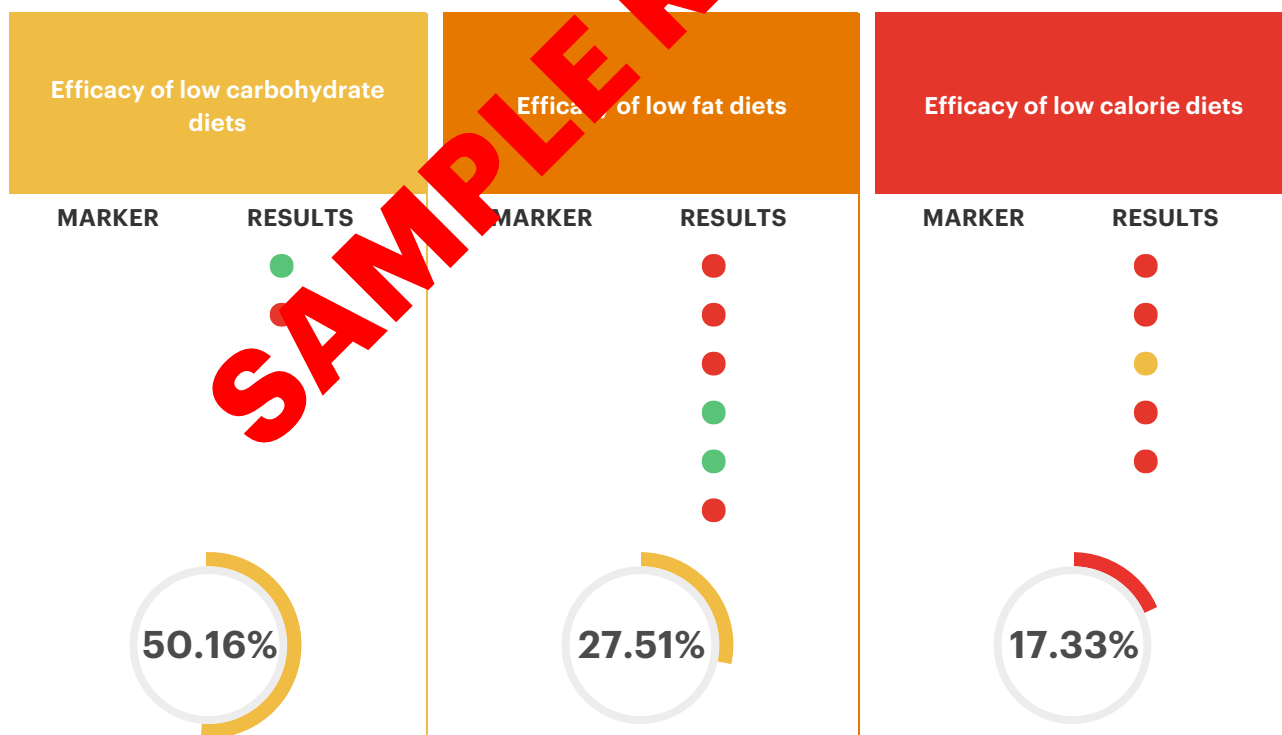
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Genetic results overview

3

3.14.	CATEGORY	DESCRIPTION
	 Minerals	Please find below the different analysed categories related to food supplementation needs.
	<ul style="list-style-type: none"> • Calcium malabsorption risk • Predisposition to dysregulated calcium levels • Risk of iron overload • Risk of low iron plasma levels • Predisposition to dysregulated magnesium levels • Predisposition to dysregulated selenium levels • Sodium sensitivity 	<p>LOW RISK OF CALCIUM MALABSORPTION ● Pg. 79</p> <p>INCREASED RISK OF DYSREGULATED PLASMA CALCIUM LEVELS ● Pg. 80</p> <p>LOW RISK OF HEMOCHROMATOSIS ● Pg. 81</p> <p>HIGH RISK OF DECREASED IRON LEVELS ● Pg. 82</p> <p>MEDIUM-LOW RISK OF DYSREGULATED MAGNESIUM LEVELS ● Pg. 83</p> <p>NO ADDITIONAL RISK OF DYSREGULATED SELENIUM LEVELS ● Pg. 84</p> <p>HIGH SODIUM SENSITIVITY ● Pg. 85</p>

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.



INDICATIONS

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 ■ Negative effect

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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

Daily food intake

Recommendations

- Allowed adjusting the amounts and/or frequency
- Allowed without reducing the recommended quantities and/or frequency
- Reduce the amount and/or frequency

Indications

On the food table, we have incorporated specific symbols for the intolerances or vitamin deficiencies based on the data included in the report. When several foods from a category have a similar level of recommendation, those whether they will have a positive effect or negative impact in the diet plan. Find out more.

Food list

Fruits and derivatives

FOOD CONTENT	FOOD	NUTRIENT CONTENT
🍏 🍏 🍏 C D	Custard apple	B* E B* 🍏 C D B* 🍏 🍏 🍏 B* 🍏
🍏 🍏 C D	Yellow plum, with skin	B* E B* 🍏 C D
🍏 🍏 C D	Skinned plum	B* E B* 🍏 C D B* 🍏 🍏 🍏 B* 🍏
🍏 🍏 C D	Plum	B* E B* 🍏 C D B* 🍏 🍏 🍏 B* 🍏
🍏 🍏 C D	Lime	B* E B* 🍏 C D
🍏 🍏 C D	Apple	B* E B* 🍏 C D B* 🍏 🍏 🍏 B* 🍏
🍏 🍏 C D	Peach	B* E B* 🍏 C D B* 🍏 🍏 🍏 B* 🍏
🍏 🍏 C D	Medlar, canned in juice	B* E B* 🍏 C D
🍏 🍏 C D	Medlar, without skin, frozen	B* E B* 🍏 C D E B*

Recommendations

- Allowed adjusting the amounts and/or frequency
- Allowed without reducing the recommended quantities and/or frequency
- Reduce the amount and/or frequency
- Restrict, occasionally in small quantities

5

Recommended supplements

SAMPLE REPORT

CGATCGATCGATCGATCGATCGTATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTG
GACCGA C CAGTCATCCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGATCGATCGATCGA
ATCGATCGATCGATCGATCGACGTACTGATCGA TCGATCGAATCGATCGATCGATCGACGTACTGATCGA
ATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGAT
ATCGATCGATCGATCGATCGATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGAT
ATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGAT

Recommended supplements

5

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

1

DETOX

DETOX I
Detoxification
(oxidation)
30 days

DETOX II
Detoxification
(conjugation)
30 days

- Vitamin B6 (Pyridoxine hydrochloride)
- Silybin®
- Silymarin
- Vitamin E
- Ubiqsome®
- Brocophanus®
- Vitamin B2 (Riboflavine)
- Zinc gluconate
- Ubiquinol
- Pinus pinaster dry extract standardized

- Magnesium
- Vitamin B9 (Methylolate)
- Silybin®
- Silymarin

SAMPLE REPORT

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Recommended supplements

5

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



INTESTINAL

INTESTINAL
Transportation
Excretion
30 days

- Biointestil®
- Lactobacillus acidophilus
- Lactobacillus plantarum
- Lactobacillus salivarius



SUPPLEMENTATION

SUPPLEMENTATION
Prevention, maintaining
optimal nutrition
3-6 months

- Magnesium
- Vitamin B6 (Pyridoxine hydrochloride)
- Ginseng dry extract (Panax ginseng)
- Glutamine (levoglutamine)

SAMPLE REPORT

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Recommended formulations

6



Detoxification
(oxidation)
30 days

Suggested formula: **CAPSULE**

Detox 1 capsule	
	240 mg
	17 mg
	26 mg
	150 mg
	40 mg
	4 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:

My Amazing Clinic
Awesome Street, 123
08452, Gotham

Signature:

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Recommended formulations

6



Detoxification
(conjugation)
30 days



In this phase, we propose one formulation in capsules and another in sachet. Please prescribe only one option, avoiding concurrent use of both.

Suggested formula: **CAPSULE**

Detox 2 capsule

200 mg

500 mg

150 mg

400 mg

2000 UI

75 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:

My Amazing Clinic
Awesome Street, 123
08452, Gotham

Signature:

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Recommended formulations

6



Detoxification
(conjugation)
30 days

! In this phase, we propose one formulation in capsules and another in sachet. Please prescribe only one option, avoiding concurrent use of both.

Suggested formula: **SACHET**

Detox 2 sachet

200 mg

400 mg

2000 UI

300 mg

150 mg

150 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:

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Awesome Street, 123
08452, Gotham

Signature:

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Recommended formulations

6



Transportation
Excretion
30 days

Suggested formula: **CAPSULE**

Intestinal capsule	
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
	1 x10 ⁹ UFC
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:

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Signature:

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Recommended formulations

6



Prevention, maintaining
optimal nutrition
3-6 months



In this phase, we propose one formulation in capsules and another in sachet. Please prescribe only one option, avoiding concurrent use of both.

Suggested formula: **CAPSULE**

Supplementation capsule

400 mg

96 mg

5 mg

700 mcg

260 mg

200 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:

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Signature:

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Recommended formulations

6



Prevention, maintaining
optimal nutrition
3-6 months



In this phase, we propose one formulation in capsules and another in sachet. Please prescribe only one option, avoiding concurrent use of both.

Suggested formula: **SACHET**

Supplementation sachet

400 mg

96 mg

5 mg

700 mcg

260 mg

500 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:

My Amazing Clinic
Awesome Street, 123
08452, Gotham

Signature:

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Annex to the formulation

6

Composites daily dosage recommendation

Composite Name	Min	Max	Unit	Composite Name	Min	Max	Unit
	125	250	mg		1 x10 ⁸	1 x10 ⁹	UFC
	100	600	mg		1 x10 ⁸	1 x10 ⁹	UFC
	500	750	mg		1 x10 ⁸	1 x10 ⁹	UFC
	4	18	mg		1 x10 ⁸	1 x10 ⁹	UFC
	6	8	mg		100	1200	mg
	125	3000	mg		10	20	mg
	1 x10 ⁸	1 x10 ⁹	UFC		5	15	mg
	1 x10 ⁸	1 x10 ⁹	UFC		250	400	mg
	1 x10 ⁸	1 x10 ⁹	UFC		1	5	mg
	1 x10 ⁸	1 x10 ⁹	UFC		500	1000	mg
	160	240	mg		100	200	mg
	300	600	mg		1	50	mg
	40	300	mcg		1	500	mg
	500	1000	mg		2	300	mg
	100	200	mg		40	120	mg
	100	500	mg		250	500	mg
	500	3000	mg		3	24	g
	250	500	mg		250	1000	mg
	250	1000	g		50	500	mg
	500	1500	g		400	1600	mg
	300	5	mg		50	200	mcg
	0.5	2	mg		150	600	mg
	25	100	mg		80	360	mg
	50	100	mg		80	200	mg
	75	250	mg		100	300	mg
	250	750	mg		100	300	mg
	100	200	mg		100	1050	mg
	500	1500	mg		2	100	mg
	3000	10000	mg		250	1000	mcg
	200	3000	mg		1	30	mg
	1000	4000	mg		10	50	mg
	50	750	mg		1	100	mg
	500	1500	mg		0.25	5	mg
	5	7	g		80	1000	mg
	75	150	mg		600	5000	UI
	25	100	mg		11	400	mg
	200	400	mg		65	320	mcg
	500	1000	mg		2	2	mg
	6	40	g		6	50	mg
	100	900	mg				

SAMPLE REPORT

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Complete genetic results

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 - 7.1.2. Risk of rebound weight gain
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 - 7.13.7. Vitamin E
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 - 7.14.3. Risk of iron overload
 - 7.14.4. Risk of low iron plasma levels
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 - 7.14.6. Predisposition to dysregulated selenium levels
 - 7.14.7. Sodium sensitivity
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 - 7.15.1. Efficacy of low calorie diets
 - 7.15.2. Efficacy of low carbohydrate diets
 - 7.15.3. Efficacy of low fat diets

CGATCGATCGATCGATCGATCGGTATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTG
GACCGA C CAGTCATCCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGATCGATCGATCGA
ATCGATCGATCGATCGATCGACGTACTGATCGAATCGATCGATCGATCGACGTACTGATCGA
ATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGAT
ATCGATCGATCGATCGATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGAT
ATCGACGTACTGATCGATCGATCGAATCGATCGATCGATCGACGTACTGATCGATCGATCGAGTACTGAT

Complete genetic results

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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.
		GG	HIGH	High predisposition to obesity, insulin resistance.
		TT	LOW	Normal risk of obesity.
		GG	LOW	Normal risk of obesity.
		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.

SAMPLE REPORT

* 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
		GG	HIGH	Predisposition to regain weight after weight loss interventions.

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 capacity 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.

SAMPLE REPORT

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Complete genetic results

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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
		GG	LOW	Normal risk of increased BMI.
		TT	LOW	Normal risk of increased BMI.
		AA	HIGH	High risk of increased BMI, increased waist circumference, and type 2 diabetes.

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.

SAMPLE REPORT

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Complete genetic results

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7.1 Morphological genetics for weight control



7.1.4. Basal metabolic rate (burn calories at rest)

RESULT	ABOUT
--------	-------

MEDIUM-LOW BURNER AT REST

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

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		TT	HIGH	Predisposition to decreased resting metabolic rate.
		AA	LOW	Predisposition to normal 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

SAMPLE REPORT

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Complete genetic results

7

7.1 Morphological genetics for weight control



7.1.5. Weight loss capability during diet interventions

RESULT	ABOUT
<p>SLOW WEIGHT LOSS</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		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.

SAMPLE REPORT

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Complete genetic results

7

7. 2 Behavioural genetics in food intake



7. 2. 1. Appetite and anxiety risk

RESULT	ABOUT
--------	-------

SLIGHTLY 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
		GG	HIGH	High risk of overeating and bulimic nervosa associated with an increased risk of type 2 diabetes and impaired glucose tolerance.
		GG	LOW	Normal risk of eating disorders.
		GG	LOW	No predisposition to emotional eating.
		CC	HIGH	Predisposition to binge eating.
		GG	LOW	No predisposition to binge eating.

INDICATIONS

NORMAL APPETITE AND ANXIETY RISK

Normal or well-balanced regulation of appetite and eating-related anxiety.

SLIGHTLY INCREASED APPETITE AND ANXIETY RISK

Medium 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.



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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
		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

Slightly 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. try to 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.

SAMPLE REPORT

* 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
<p>●</p> <p>NORMAL BITTER TASTE SENSITIVITY</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GA	LOW	Predispositon to normal sensitivity to bitter taste.
		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, as 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.

SAMPLE REPORT

* 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
--------	-------

HIGH 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
		GG	HIGH	High risk of increased salt sensitivity and salt sensitive hypertension.

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.

SAMPLE REPORT

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Complete genetic results

7

7. 3 Flavour sensitivities



7. 3. 3. Sweet flavour preference

RESULT	ABOUT
<p>HIGHLY INCREASED SWEET PREFERENCE</p>	Increased desire to eat sweet food due to an incapacity of tasting sweet flavours.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GA	HIGH	Predisposition to increased preference for 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.

SAMPLE REPORT

* 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
<p>VERY LOW MUFA METABOLISM</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		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.

SAMPLE REPORT

* 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
<p>FAST PUFA METABOLISM</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		CC	LOW	Predisposition to improve lipid profile (LDL and total cholesterols) and reduce BMI in response to a PUFA-rich diet.
		TT	LOW	Predisposition to normal PUFA biosynthetic capacity.

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.

SAMPLE REPORT

* 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
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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
		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.

SAMPLE REPORT

* 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
		AA	LOW	Predisposition to normal levels of HDL cholesterol.
		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

Low hdl levels leading to increased cardiovascular risk.



HIGLY REDUCED HDL LEVELS

Very low hdl levels leading to increased cardiovascular risk.

SAMPLE REPORT

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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
		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

SAMPLE REPORT

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Complete genetic results

7

7. 5 Lipid metabolism



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

RESULT	ABOUT
<p>HIGHLY INCREASED LDL OXIDATION</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GG	HIGH	High predisposition to increased LDL oxidation.

INDICATIONS

<p>NOT INCREASED LDL OXIDATION</p> <p>Normal ldl oxidation.</p>	<p>SLIGHTLY INCREASED LDL OXIDATION</p> <p>Moderate increase in the ldl oxidation. increased risk of atherosclerosis.</p>	<p>INCREASED LDL OXIDATION</p> <p>Increased ldl oxidation. increased risk of atherosclerosis. strategies for reducing ldl levels would be recommended.</p>	<p>HIGHLY INCREASED LDL OXIDATION</p> <p>Higly increased ldl oxidation and risk of atherosclerosis. intense strategies for reducing ldl levels should be initiated.</p>
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SAMPLE REPORT

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Complete genetic results

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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
		GG	HIGH	No predisposition to lower LDL cholesterol levels.
		AC	MEDIUM	Predisposition to slightly increased LDL cholesterol levels.
		GG	HIGH	High risk of increased LDL cholesterol levels.
		TC	MEDIUM	Predisposition to slightly 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.

SAMPLE REPORT

* 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
--------	-------

HIGHLY 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
		TT	HIGH	Predisposition to higher triglyceride (TG) levels, and higher TG/HDL cholesterol ratio.

INDICATIONS



NORMAL TG/HDL RATIO

Not associated with increased tg/hdl ratio.



SLIGHTLY INCREASED TG/HDL RATIO

Slightly associated with increased tg/hdl ratio.



INCREASED TG/HDL RATIO

Increased triglyceride levels 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.

SAMPLE REPORT

* 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
		CC	HIGH	No predisposition to increased expression of the amylase gene.
		TA	MEDIUM	Predisposition to slightly 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.

SAMPLE REPORT

* These recommendations are based only on 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
<p>NORMAL CARBOHYDRATE SENSITIVITY</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		TT	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.

SAMPLE REPORT

* 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
		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.

SAMPLE REPORT

* 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
		CG	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.

SAMPLE REPORT

* 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
--------	-------

MEDIUM-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
		TT	LOW	Predisposition to normal plasma glucose levels after fasting.
		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.

SAMPLE REPORT

* 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-HIGH 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
		CC	HIGH	High predisposition to insulin resistance.
		GG	HIGH	High predisposition to insulin resistance.
		TT	HIGH	High predisposition to insulin resistance.
		TT	LOW	No predisposition to insulin resistance.
		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

SAMPLE REPORT

* 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
--------	-------

HIGH 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
		CC	HIGH	Increased risk of diabetes type 2.
		TT	LOW	Normal risk of type 2 diabetes.
		TT	HIGH	High risk of diabetes type 2.
		TT	LOW	Normal risk of diabetes type 2.
		GG	LOW	No predisposition to obesity and type 2 diabetes.
		TT	HIGH	High risk of type 2 diabetes.
		CC	HIGH	Increased risk of type 2 diabetes.
		AA	HIGH	High risk of type 2 diabetes.
		AG	HIGH	Increased risk of type 2 diabetes.
		CC	HIGH	Increased risk of type 2 diabetes.

INDICATIONS



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



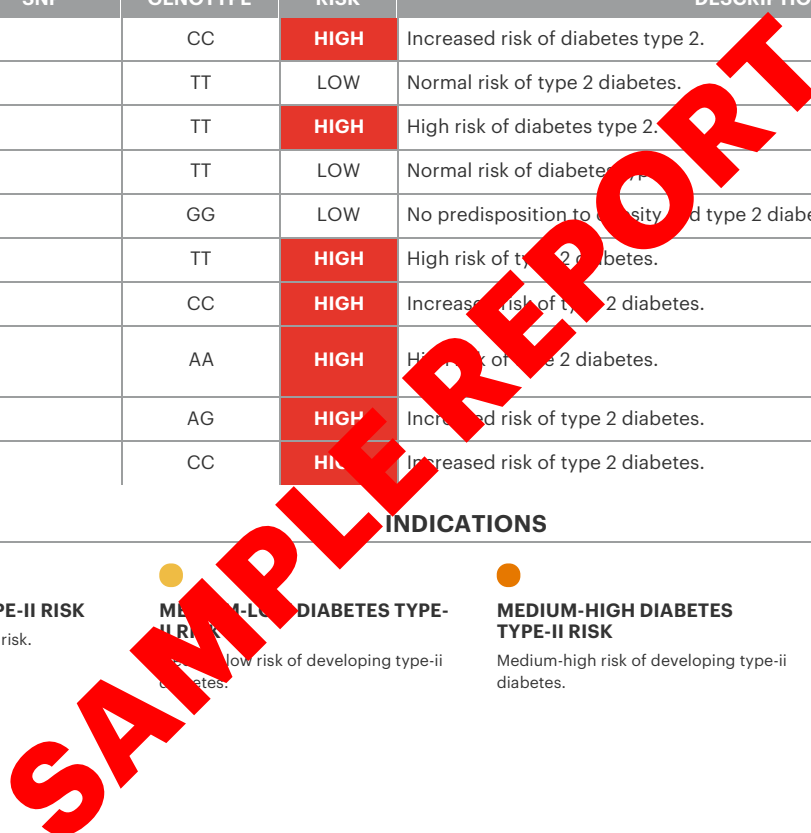
MEDIUM-LOW DIABETES TYPE-II RISK
Low risk of developing type-ii diabetes.



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
<p>VERY LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL</p>	<p>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.</p>

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

INDICATIONS

<p>HIGH BENEFITS FROM EXERCISE FOR IMPROVING HDL</p> <p>Exercise will be strongly beneficial for cholesterol regulation (hdl increase).</p>	<p>MEDIUM-HIGH BENEFITS FROM EXERCISE FOR IMPROVING HDL</p> <p>Exercise will be beneficial for cholesterol regulation (hdl increase).</p>	<p>MEDIUM-LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL</p> <p>Exercise will not be enough for cholesterol regulation.</p>	<p>VERY LOW BENEFITS FROM EXERCISE FOR IMPROVING HDL</p> <p>Exercise alone will not be enough for cholesterol regulation.</p>
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SAMPLE REPORT

* 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		
<p>MEDIUM-LOW BENEFIT FROM EXERCISE TO REDUCE FAT</p>		<p>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.</p>		
GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		TT	HIGH	No predisposition to lose fat during physical exercise.
		GG	HIGH	Predisposition to not lose fat during physical exercise.
		CT	MEDIUM	Slight predisposition to benefit from physical exercise to increase HDL cholesterol levels.
		GG	LOW	Normal predisposition to exercise-induced fat loss.

INDICATION



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).

SAMPLE REPORT

* 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
--------	-------

**SLIGHTLY REDUCED
 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
		GG	LOW	Predisposition to normal hydrogen peroxide detoxification.
		GG	LOW	Predisposition to normal NQO1 activity.
		GG	LOW	Predisposition to normal COMT activity.
		GG	HIGH	Predisposition to reduced hydrogen peroxide detoxification and increased oxidative damage.
		CC	LOW	Predisposition to normal CYP1B1 enzyme activity.
		TT	LOW	Predisposition to normal CYP1A1 enzyme activity.
		GG	HIGH	Predisposition to reduced GSTP1 activity leading to low xenobiotic detoxification and increased susceptibility to oxidative stress.

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.

SAMPLE REPORT

* 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
		CT	LOW	Normal predisposition to lactose tolerance.
		GA	LOW	Normal predisposition to lactose tolerance.

INDICATIONS



LOWER RISK OF LACTOSE INTOLERANCE

Lower risk of lactose intolerance.



SLIGHTLY INCREASED RISK OF LACTOSE INTOLERANCE

Slightly increased risk of lactose intolerance. Lower capability to digest lactose. Rather reduce the lactose intake.



MEDIUM RISK OF LACTOSE INTOLERANCE

Medium 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.

SAMPLE REPORT



If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance is advised 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 on 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
<p>MEDIUM-HIGH RISK OF CELIAC DISEASE</p>	<p>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.</p>

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

* 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. 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
		CC	LOW	No predisposition to develop hereditary fructose intolerance.
		GG	LOW	No predisposition to develop hereditary fructose intolerance.

INDICATIONS



LOWER RISK OF FRUCTOSE INTOLERANCE

Lower risk of fructose intolerance.



SLIGHTLY INCREASED RISK OF FRUCTOSE INTOLERANCE

Slightly increased risk of fructose intolerance. lower capability to digest fructose. rather reduce the fructose intake.



MEDIUM RISK OF FRUCTOSE INTOLERANCE

Medium 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.

SAMPLE REPORT



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

* 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. 4. Caffeine metabolism

RESULT	ABOUT
--------	-------

SLOW 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
		CC	HIGH	Predisposition to slow caffeine metabolism.
		CA	HIGH	Predisposition to slow 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 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.

SAMPLE REPORT



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

* 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. 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
		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 risk of alcohol toxicity due to low metabolism.



SLOW ALCOHOL METABOLISM

High risk of alcohol toxicity due to slow metabolism.

SAMPLE REPORT



If your patient suffers from these symptoms and/or has a medium or high risk of developing intolerance it 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

* 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. 1. 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
		GG	LOW	Predisposition to normal levels of leptin leading to normal satiety regulation.

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

SAMPLE REPORT

* 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. Adiponectin

RESULT	ABOUT
--------	-------

MEDIUM-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
		AA	HIGH	High predisposition to increased levels of circulating visfatin augmenting considerably the risk of an altered inflammatory response.

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

SAMPLE REPORT

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Complete genetic results

7

7. 11 Hormones



7. 11. 3. Leptin

RESULT	ABOUT
--------	-------

LOW 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
		GG	HIGH	Predisposition to normal ghrelin receptor (GHSR) expression. Circulating levels of this hormone and the sensation of hunger are increased.

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

SAMPLE REPORT

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Complete genetic results

7

7. 11 Hormones



7. 11. 4. Visfatin

RESULT	ABOUT
<p>HIGH RISK OF INCREASED VISFATIN LEVELS</p>	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GG	HIGH	High predisposition to lower adiponection plasma levels that leads to an increased inflammation process, lipid abnormalities and insulin resistance.
		TG	MEDIUM	Increased predisposition to lower adiponection plasma levels that leads to a slightly increased inflammation process, lipid abnormalities and insulin resistance.

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

SAMPLE REPORT

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Complete genetic results

7

7. 12 Inflammation



7. 12. 1. IL-10

RESULT	ABOUT
<p>MEDIUM-LOW RISK OF DECREASED ANTIINFLAMMATORY CYTOKINE IL-10 LEVELS</p>	<p>IL-10 is a cytokine with potent anti-inflammatory properties.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GG	LOW	Predisposition to average levels of TNF alpha.

INDICATIONS

- LOW RISK OF DECREASED ANTIINFLAMMATORY CYTOKINE IL-10 LEVELS**
 Low risk of decreased antiinflammatory cytokine il-10 levels
- MEDIUM-LOW RISK OF DECREASED ANTIINFLAMMATORY CYTOKINE IL-10 LEVELS**
 Medium-low risk of decreased antiinflammatory cytokine il-10 levels
- MEDIUM-HIGH RISK OF DECREASED ANTIINFLAMMATORY CYTOKINE IL-10 LEVELS**
 Medium-high risk of decreased antiinflammatory cytokine il-10 levels
- HIGH RISK OF DECREASED ANTIINFLAMMATORY CYTOKINE IL-10 LEVELS**
 High risk of decreased antiinflammatory cytokine il-10 levels

SAMPLE REPORT

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Complete genetic results

7

7. 12 Inflammation



7. 12. 2. TNF- α

RESULT	ABOUT
--------	-------

LOW RISK OF DYSREGULATED 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
		CG	MEDIUM	Predisposition to intermediate levels of IL-6. Pro-inflammation.

INDICATIONS

LOW RISK OF DYSREGULATED TNF- α LEVELS.
Low risk of dysregulated tnf- α 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

SAMPLE REPORT

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Complete genetic results

7

7. 12 Inflammation



7. 12. 3. IL-6

RESULT	ABOUT
--------	-------

MEDIUM-LOW 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
		TC	MEDIUM	Predisposition to intermediate levels of the anti-inflammatory cytokine IL-10.

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

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

A

7. 13. 1. Vitamin A

RESULT	ABOUT
--------	-------

LOW RISK OF VITAMIN A DEFICIENCY

Inherited risk of vitamin A metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		AT	MEDIUM	Increased predisposition to reduced vitamin A conversion and increased fasting β-carotene concentrations.
		CC	LOW	Normal risk of vitamin A deficiency.

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 if necessary, 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.

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

B6

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
		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 if needed, 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.

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

B9

7. 13. 3. Vitamin B9 (folate)

RESULT	ABOUT
--------	-------

HIGH RISK OF VITAMIN B9 (Folate) DEFICIENCY

Inherited risk of vitamin B9 (folate) metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		AA	HIGH	High risk of low serum levels of folate.

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.

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

B12

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
		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-high risk of vitamin b12 deficiency. ensure daily recommended intake and if needed, 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.

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

C

7. 13. 5. Vitamin C

RESULT	ABOUT
--------	-------

MEDIUM-HIGH RISK OF VITAMIN C DEFICIENCY

Inherited risk of vitamin C metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GG	HIGH	High risk of reduced circulating concentrations of vitamin C.
		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 vitamin 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 recommended.



HIGH RISK OF VITAMIN C DEFICIENCY

High risk of vitamin c deficiency. ensure daily recommended intake. supplementation strategies would be recommended.

SAMPLE REPORT

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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
		TT	LOW	Normal risk of vitamin D deficiency.
		GG	HIGH	High risk of low serum levels of vitamin D.
		GT	MEDIUM	Increased risk of lower serum levels of vitamin D.
		GG	LOW	Normal risk of vitamin D deficiency.
		GA	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 vitamin 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.

SAMPLE REPORT

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Complete genetic results

7

7. 13 Vitamins

E

7. 13. 7. Vitamin E

RESULT	ABOUT
--------	-------

HIGH RISK OF VITAMIN E DEFICIENCY

Inherited risk of vitamin E metabolism deficiency or low plasma levels.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		CC	HIGH	High risk of low plasma levels of alpha-tocopherol (Vitamin E).
		CC	HIGH	High risk of low plasma levels of alpha-tocopherol (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 vitamin 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 recommended.



HIGH RISK OF VITAMIN E DEFICIENCY

High risk of vitamin e deficiency. ensure daily recommended intake. supplementation strategies would be recommended.

SAMPLE REPORT

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Complete genetic results

7

7. 14 Minerals

Ca

7. 14. 1. Calcium malabsorption risk

RESULT	ABOUT
 LOW RISK OF CALCIUM MALABSORPTION	<p>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.</p>

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GA	MEDIUM	Predisposition to slightly reduced vitamin D levels and calcium absorption.
		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.

SAMPLE REPORT

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Complete genetic results

7

7. 14 Minerals



7. 14. 2. Predisposition to dysregulated calcium levels

RESULT	ABOUT
--------	-------

INCREASED 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
		CG	MEDIUM	Predisposition to slightly increased serum levels of calcium.
		AA	HIGH	Predisposition to reduced serum calcium levels and bone mineral density.
		AG	MEDIUM	Predisposition to slightly increased serum calcium levels.
		GT	MEDIUM	Predisposition to slightly increased serum calcium levels.
		GA	MEDIUM	Predisposition to slightly reduced serum calcium levels.
		CC	HIGH	Predisposition to reduced 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.

SAMPLE REPORT

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Complete genetic results

7

7. 14 Minerals

Fe

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
		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 OF HEMATOCHROMATOSIS

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.

SAMPLE REPORT

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Complete genetic results

7

7. 14 Minerals

Fe

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

RESULT	ABOUT
--------	-------

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
		AA	HIGH	Predisposition to increased serum ferritin and reduced serum iron levels.
		GA	MEDIUM	Predisposition to slightly reduced iron levels.
		TT	HIGH	Predisposition to increased transferrin 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

General 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.

SAMPLE REPORT

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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
		AG	MEDIUM	Predisposition to slightly higher serum magnesium levels.
		TT	HIGH	Predisposition to lower serum magnesium levels.
		AA	LOW	Predisposition to normal serum magnesium levels.
		CC	HIGH	Predisposition to lower serum magnesium levels.
		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.

SAMPLE REPORT

* 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

Se

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
		CC	LOW	Predisposition to normal selenium levels.
		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 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.

SAMPLE REPORT

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Complete genetic results

7

7. 14 Minerals



7. 14. 7. Sodium sensitivity

RESULT	ABOUT
--------	-------

HIGH SODIUM SENSITIVITY

Inherited risk of dietary salt-induced blood pressure.

GENE	SNP	GENOTYPE	RISK	DESCRIPTION
		GG	HIGH	High risk of increased sodium sensitivity and sodium sensitive hypertension.

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

Moderately increased 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.

SAMPLE REPORT

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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
		CC	HIGH	No predisposition to weight loss induced by a low calorie diet.
		GG	HIGH	No predisposition to weight loss induced by a low calorie diet.
		TC	MEDIUM	Increased predisposition to weight loss induced by a low calorie diet.
		CC	HIGH	No predisposition to weight loss induced by a low calorie diet.
		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.

SAMPLE REPORT

* 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
		GG	LOW	Predisposition to weight loss induced by a low carbohydrate diet.
		CG	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 options for weight loss. try to gradually 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.

SAMPLE REPORT

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Complete genetic results

7

7. 15 Effectiveness of diets



7. 15. 3. Efficacy of low fat diets

RESULT	ABOUT
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MEDIUM-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
		CC	HIGH	No predisposition to weight loss induced by a low fat diet.
		GG	HIGH	No predisposition to weight loss induced by a low fat diet. Also applicable after gastric bypass.
		AA	HIGH	No predisposition to weight loss induced by a low fat diet.
		GG	LOW	Predisposition to weight loss induced by a low fat diet.
		TT	LOW	Predisposition to weight loss induced by a low fat diet.
		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

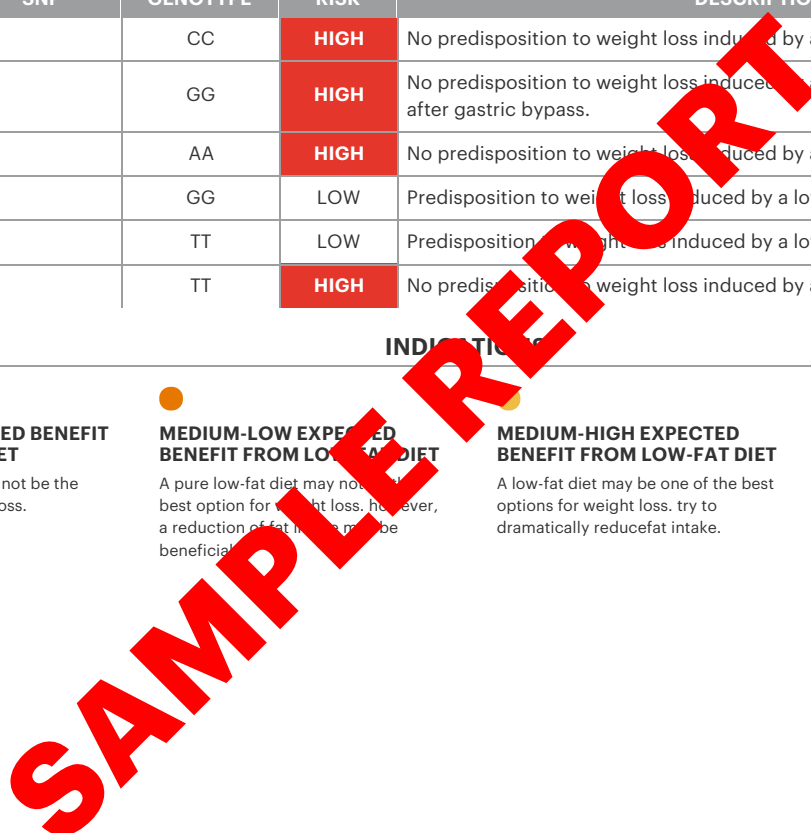
A pure low-fat diet may not be the best option for weight loss, however, a reduction of fat intake may be beneficial.

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 reduce fat 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.



* 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.

Methodology

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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.

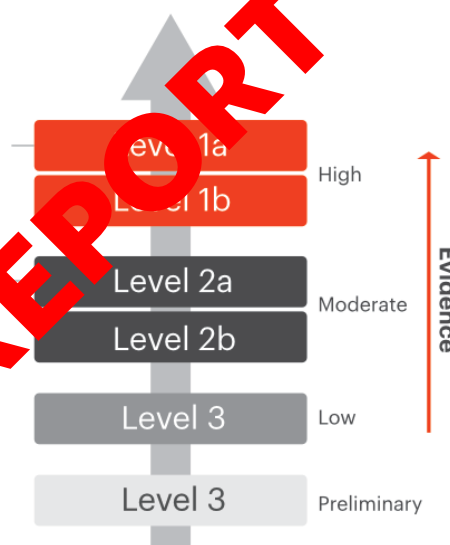
Level 2A: Annotation for a variant that qualifies for level 2B where the variant is within a Very Important known gene, so functional significance is more likely.

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 NutriGen report reference page.

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SAMPLE REPORT

Together
we create the future
of personalizing medicine.

