

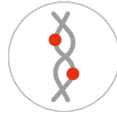
• Patient's name: Man Demo Patient  
• Sample code: TRI39339AA  
• Report date: 09-02-2023

---



# Fagron TrichoTest™

## Results report



## TrichoTest™ Genetic report

### LEGAL DISCLAIMER

Fagron Genomics, S.L.U carries out genetic tests upon request by healthcare professionals, in relation to biological samples from patients obtained by the healthcare professional. Our tests do not replace a medical consultation, nor do they make up a diagnostic or treatment, nor should they be interpreted this way. Only healthcare professionals can interpret the results of said tests, based on their knowledge of the clinical records of the patients and other relevant factors and, under their responsibility, give a diagnostic or prescribe treatment to the patient. We decline all responsibility derived from the use and interpretation of the results of our tests by the solicitant healthcare professional. Fagron Genomics, S.L.U expressly reserves any legal actions in case of an innapropriate, 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.





# I. Patient identification data

# 1.

## Patient identification data



Ordering physician —●— **DOCTOR DEMO**  
Contact —●— **manelfrances@gmail.com**  
Patient's name —●— **Man Demo Patient**  
Gender —●— **Male**  
Date of birth —●— **09-03-1975**  
Sample type —●— **Bucal swab**  
Sample code —●— **TRI39339AA**  
Sample date —●— **21-11-2022**  
Report date —●— **09-02-2023**



## II. Recommendation of the most suitable drugs and supplements

## 2. Recommendation of the most suitable drugs and supplements

The **genetic test** uses an automated qualitative pharmacogenetic algorithm that analyzes the patient's genetic data and combines this information with relevant patient history to recommend the most suitable active ingredients. Next, we show on a color scale which compounds the algorithm recommends the most. The transition from white to dark green indicates drugs from least recommended to most recommended. Medications blocked due to intolerances or contraindications are shown in red.

### Anti-alopecic drugs

Prostaglandins	
• Minoxidil	73%
• Latanoprost Fagron	67%
• Prostaquinon TM	67%

Antiandrogenic	
• Finasteride	86%
• Topical Saw Palmetto	53%
• Saw Palmetto	53%
• Ginseng	50%
• 17- $\alpha$ Estradiol	37%
• Melatonin	25%
• Dutasteride	

Anti-inflammatory	
• Clobetasol propionate	
• Triamcinolone acetonide	
• Hydrocortisone	
• Betamethasone dipropionate	
• Desonide	
• Fluocinolone acetonide	
• Prednicarbate	

Immunomodulator	
• Tacrolimus	

### Hair care supplements

Circulation	
• Arginine	67%
• Ginkgo biloba	67%
• Caffeine	50%
• L-Carnitine L-tartrate	50%
• CafeiSome TM	40%

Collagen synthesis	
• Oral SiliciuMax TM	
• Cystine	

Insulin-like growth factor increase	
• IGrantine-F1 TM	67%
• TrichoXidil	67%

Blocked



Recommended



## Vitamin, mineral and antioxidant supplements

Vitamin deficiency	
• Vitamin D	67%
• Vitamin B9 (Folate)	67%
• Vitamin E (Tocoferol)	67%
• Vitamin B7 (Biotin)	
• Retinol palmitate	
• Vitamin C (Ascorbic Acid)	
• Vitamin B12 (Cianocobalamin)	
• Vitamin C (Ascorbic Acid)	

Antioxidant
• Selenium yeast
• Resveratrol

Minerals	
• Iron sulfate	67%
• Magnesium Gluconate	67%
• Zinc gluconate	
• Zinc acetate	

## Recommendations for mesotherapy

The **genetic test** algorithm has selected the following active ingredients for use in mesotherapy. The doctor must prepare the prescription adapted to its preparation in pharmacy.

• Finasteride Liposomade 0,05%	86%
• Minoxidil Liposomade 0,25%	73%
• Latanoprost Liposomade 0,001%	67%
• Protasquinon Liposomade 0,4%	67%
• Dutasteride Liposomade 0,01%	
• Acid Retinoic 0,1%	

The amount and combination of active ingredients to be administered depends on medical criteria.

Blocked



Recommended







### III. Formulas for personalized treatment

### 3. Formulas for personalized treatments

The pharmacogenetic algorithm has selected a series of formulations for topical, oral use or capillary mesotherapy for the care and hygiene of your patient's scalp. These personalized formulations have been selected taking into account the genetics, the type of alopecia, and the relevant history of the patient.

#### Topical treatment

Formula	
Minoxidil	6 %
Finasteride	0.93 %
Arginine	1 %
IGrantine-F1 TM	0.42 %
<b>TrichoSol</b>	<b>100ml</b>

**Posology**  
Apply at night before bedtime. Leave the solution on your scalp for as long as possible. Wash your scalp the next day.

Signature of the prescribing physician	
<b>Dr</b>	
<b>Physician registration No.</b>	
<b>Date</b>	

Address	Signature
<b>My Demo Clinic</b> Fantastic street, 123 08766, Best City +34 666 777 555	

• Patient name: **Man Demo Patient**

• Patient ID: **12345678Z**

• Date of Birth: **09-03-1975**

• Sample code: **TRI39339AA**

• Sample date: **21-11-2022**

• Date of the results: **09-02-2023**

## Oral treatment

### Formula

Iron sulfate	33 mg
Saw Palmetto	202 mg
Caffeine	23 mg

### Posology

1 capsule per day, 90 capsules for 3 months

### Signature of the prescribing physician

**Dr**

**Physician registration No.**

**Date**

**Address**

**My Demo Clinic**  
Fantastic street, 123  
08766, Best City  
+34 666 777 555

**Signature**

## Scalp care and hygiene

### Topical treatment

#### Formula

Prostaquinon TM	2 %
Topical Saw Palmetto	2 %
Ginkgo biloba	2 %
<b>TrichoOil</b>	<b>30ml</b>

#### Posology

1-2 times / week, massage for 3-5 minutes and leave it on for 10 min before washing your hair.

#### Signature of the prescribing physician

**Dr**

**Physician registration No.**

**Date**

**Address**

**My Demo Clinic**  
Fantastic street, 123  
08766, Best City  
+34 666 777 555

**Signature**

• Patient name: **Man Demo Patient**

• Patient ID: **12345678Z**

• Date of Birth: **09-03-1975**

• Sample code: **TRI39339AA**

• Sample date: **21-11-2022**

• Date of the results: **09-02-2023**

## Scalp care and hygiene

### Topical treatment

#### Formula

Ginseng 2 %

Arginine 1 %

Vitamin E (Tocoferol) 3 %

**TrichoWash 250ml**

#### Posology

Massage for 2 minutes and rinse

#### Signature of the prescribing physician

**Dr**

**Physician registration No.**

**Date**

**Address**

**My Demo Clinic**  
Fantastic street, 123  
08766, Best City  
+34 666 777 555

**Signature**

• Patient name: **Man Demo Patient**

• Patient ID: **12345678Z**

• Date of Birth: **09-03-1975**

• Sample code: **TRI39339AA**

• Sample date: **21-11-2022**

• Date of the results: **09-02-2023**

## Scalp care and hygiene

### Topical treatment

#### Formula

Ginseng 2 %

Arginine 1 %

Vitamin E (Tocoferol) 3 %

**TrichoCond 250ml**

#### Posology

After washing your hair, apply the conditioner and leave it on for 2-3 minutes before rinse.

#### Signature of the prescribing physician

**Dr**

**Physician registration No.**

**Date**

**Address**

**My Demo Clinic**  
Fantastic street, 123  
08766, Best City  
+34 666 777 555

**Signature**

• Patient name: **Man Demo Patient**

• Patient ID: **12345678Z**

• Date of Birth: **09-03-1975**

• Sample code: **TRI39339AA**

• Sample date: **21-11-2022**

• Date of the results: **09-02-2023**

## Scalp care and hygiene

### Topical treatment

#### Formula

Arginine	1 %
Vitamin E (Tocoferol)	3 %
Ginkgo biloba	2 %
<b>TrichoSerum</b>	<b>50ml</b>

#### Posology

After washing your hair, apply on wet hair.

#### Signature of the prescribing physician

**Dr**

**Physician registration No.**

**Date**

**Address**

**My Demo Clinic**  
Fantastic street, 123  
08766, Best City  
+34 666 777 555

**Signature**



## IV. Complete data



## 4.

### Complete data Data from the medical questionnaire

#### Patient demographics

Gender  Male

Age (years)  47

Height (cm)  168

Weight (kg)  68

BMI  24.09

Family history of alopecia  Parents

#### Hair loss data

Type of alopecia  Androgenic alopecia

Grade of alopecia  Grade II

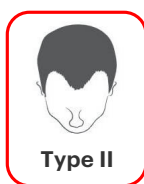
Time elapsed since start of hair loss  -1

Prescription of testosterone derivatives  No

#### Norwood-Hamilton Scale



Type I



Type II



Type III



Vertex



Type IV



Type V



Type VI



Type VII

#### Clinical examination

Amount of hair loss  Nothing

Complaints associated with alopecia  No

Patchy alopecia  No

Current anti-alopecia treatment  No

Previous anti-alopecia treatment  No

## 4.

### Complete data Pharmacogenetic results

#### 1. Anti-alopecic drugs

##### Treatment efficacy with prostaglandin inhibitors

Prostaglandin D2				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
GPR44-1	rs545659 (A>G)	G	GA	Genetic result: Predisposition to slightly higher GPR44 mRNA stability. Interpretation: Prostaglandin D2 receptor 2 (GPR44 or CRTH2) variants are associated with an increased GPR44 mRNA stability leading to an increased responsiveness to prostaglandin D2 and hair follicle regression. Treatment/dosage: Treatment with prostaglandin D2 inhibitors (Cetirizine and/or Prostaquinon) at normal doses would be highly recommended.
GPR44-2	rs533116 (G>A)	A	GA	Genetic result: Predisposition to slightly higher GPR44 mRNA stability. Interpretation: Prostaglandin D2 receptor 2 (GPR44 or CRTH2) variants are associated with an increased GPR44 mRNA stability leading to higher responsiveness to prostaglandin D2 and hair follicle regression. Treatment/dosage: Treatment with prostaglandin D2 inhibitors (Cetirizine and/or Prostaquinon) at normal doses would be highly recommended.

Latanoprost				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
PTGFR-1	rs6686438 (T>G)	G	GG	Genetic result: Increased likelihood of not having a positive response to Latanoprost. Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment/dosage: Treatment with Latanoprost at normal doses is not recommended.
PTGFR-2	rs1328441 (G>A)	A	GG	Genetic result: High likelihood of having a positive response to Latanoprost. Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment/dosage: Treatment with latanoprost at normal doses is highly recommended.
PTGFR-3	rs10782665 (T>G)	G	TT	Genetic result: High likelihood of having a positive response to Latanoprost. Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment/dosage: Treatment with latanoprost at normal doses is highly recommended.

### Treatment efficacy with minoxidil

Minoxidil				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
PTGES2	rs13283456 (C>T)	T	CC	Genetic result: Predisposition to normal PGE2 levels. Interpretation: Prostaglandin E synthase 2 (PTGES2) variants are associated with lower prostaglandin E2 production (hair growth promoter). Treatment/dosage: SNP analysis does not indicate a necessity to treat with Minoxidil.
SULT1A1	rs9282861 (C>T)	T	CC	Genetic result: Predisposition to normal SULT1A activity. Interpretation: Minoxidil Sulfotransferase Enzyme (SULT1A1) variants predict response to minoxidil treatment. Treatment/dosage: Minoxidil at normal doses would be highly recommended.

### Treatment efficacy with glucocorticoid anti-inflammatories

Glucocorticoides				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
GR-alpha	rs6198 (A>G)	G	GA	Genetic result: Predisposition to moderate resistance to glucocorticoid anti-inflammatory treatments. Interpretation: Glucocorticoid Receptor (GR or NR3C1) variants are associated with resistance or sensitivity to corticosteroids. Treatment/dosage: If glucocorticoid anti-inflammatory treatment is used, doses should be slightly increased or an alternative treatment with non-glucocorticoid anti-inflammatory drugs should be chosen.

### Treatment efficacy with antiandrogenics

17-α estradiol				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
CYP19A1	rs2470152 (C>T)	T	TC	Genetic result: Predisposition to reduced CYP19A1 activity. Interpretation: Aromatase (CYP19A1) variants are associated to low conversion of testosterone in estrogens and to high conversion into DHT (hair growth inhibitor). Treatment/dosage: Treatment with topical 17-α Estradiol (aromatase inducer) at normal doses is recommended.

Dutasteride				
Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
SRD5A1	rs39848 (T>C)	C	TT	Genetic result: Predisposition to normal SRD5A1 activity. Interpretation: Steroid 5α-Reductase 1 (SRD5A1) variants are associated with reduced SRD5A1 activity leading to increased DHT levels and hair growth inhibition. Treatment/dosage: SNP analysis does not indicate a necessity to treat with dutasteride.

**Finasteride**

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
SRD5A2	rs523349 (C>G)	G	<b>CG</b>	Genetic result: Predisposition to increased SRD5A2 activity leading to increased levels of DHT Interpretation: Steroid 5α-Reductase 2 (SRD5A2) variants are associated with increased SRD5A2 activity leading to increased DHT levels and hair growth inhibition. Treatment/dosage: Treatment with Finasteride at normal doses is recommended.

**2. Hair care supplements**

**Vasodilatation and blood circulation**

**Circulation stimulators**

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
ACE	rs4343 (A>G)	G	<b>AG</b>	Genetic result: Predisposition to an increased Angiotensin conversion activity. Interpretation: Angiotensin-converting enzyme (ACE) variants are associated with increased plasma levels of angiotensin 2, an extremely potent vasoconstrictor. Treatment/dosage: Normal doses of circulation stimulators are recommended, such as Minoxidil, caffeine, Ginkgo biloba, Ginseng or Arginine.

**Collagen synthesis**

**Hair strengthening supplements**

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
COL1A1	rs1800012 (G>T)	T	<b>GG</b>	Genetic result: Predisposition to normal collagen stability. Interpretation: Collagen, type I, alpha 1 (COL1A1) variants are associated with collagen instability. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with hair strengthening composites.

**Reduction of IGF-1 levels**

**Hair strengthening supplements**

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
IGF1R	rs2229765 (G>A)	A	<b>AG</b>	Genetic result: Predisposition to moderately reduced IGF-1 levels. Interpretation: Insulin-like growth factor-I (IGF-I) variants are associated with lower plasma IGF-1 levels leading to hair loss. Treatment/dosage: A treatment with Igrantine-F1 and TrichoXidil (IGF-1 inducers) at normal doses would be recommended.

### 3. Vitamin, mineral and antioxidant supplements

#### Vitamins

##### Vitamin A

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
CRABP2	rs12724719 (G>A)	A	<b>GG</b>	Genetic result: Predisposition to normal retinoic acid intracellular transport. Interpretation: Cellular retinoic acid-binding protein 2 (CRABP2) variants are associated with lower retinoic acid (vitamin A) intracellular transport. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with vitamin A.

##### Vitamin B7

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
BTD	rs13078881 (G>C)	C	<b>GG</b>	Genetic result: Predisposition to normal biotinidase activity. Interpretation: Biotinidase (BTD) variants are associated with low biotin (vitamin B7) uptake from the diet. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with vitamin B.

##### Vitamin C

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
SLC23A1	rs33972313 (C>T)	T	<b>CC</b>	Genetic result: Predisposition to higher vitamin C serum level. Interpretation: Solute carrier family 23 member 1 (SLC23A1) variants are associated with lower serum concentration of vitamin C. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with vitamin C.

##### Vitamin B9

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
MTHFR	rs1801133 (G>A)	A	<b>GA</b>	Genetic result: Increased predisposition to folate deficiency. Interpretation: Methylene tetrahydrofolate reductase (MTHFR) variants are associated with risk of folate deficiency. Treatment/dosage: Folate supplementation should be considered.

• Patient name: **Man Demo Patient**

• Patient ID: **12345678Z**

• Date of Birth: **09-03-1975**

• Sample code: **TRI39339AA**

• Sample date: **21-11-2022**

• Date of the results: **09-02-2023**

#### Vitamin D

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
GC	rs2282679 (T>G)	G	<b>GT</b>	Genetic result: Predisposition to slightly lower vitamin D serum level. Interpretation: Vitamin D-binding protein (GC or DBP) variants are associated with lower vitamin D serum level. Treatment/dosage: Supplementation should be considered.

#### Vitamin B12

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
FUT2	rs602662 (A>G)	G	<b>AA</b>	Genetic result: Predisposition to higher vitamin B12 serum level. Interpretation: Galactoside 2-alpha-L-fucosyltransferase 2 (FUT2) variants are associated lower vitamin B12 serum level. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with vitamin B12.

#### Vitamin E

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
ZPR1	rs964184 (G>C)	C	<b>CG</b>	Genetic result: Predisposition to slightly lower serum tocopherol levels. Interpretation: Zinc Finger Protein ZPR1 variants are associated with low serum alpha-tocopherol (vitamin E) levels. Treatment/dosage: Vitamin E supplementation should be considered.

### Antioxidants

#### Antioxidants

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
NQO1	rs1800566 (G>A)	A	<b>GG</b>	Genetic result: Predisposition to normal NQO1 enzyme activity. Interpretation: NAD(P)H dehydrogenase [quinone] 1 (NQO1) variants are associated with lower NQO1 enzyme activity and may have less effective protection against oxidative stress. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with antioxidants.

## Minerals

### Magnesium

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
MUC1	rs4072037 (T>C)	C	CT	Genetic result: Predisposition to intermediate magnesium serum level. Interpretation: Mucin 1, cell surface associated (MUC1) variants are associated with lower magnesium serum level. Treatment/dosage: Magnesium supplementation should be considered.

### Zinc sulfate

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
SLC30A3	rs11126936 (T>G)	G	GT	Genetic result: Predisposition to higher serum zinc level. Interpretation: Solute carrier family 30 member 3 (SLC30A3) variants are associated with lower zinc blood level. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with Zinc Sulphate.

### Iron

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
TMPRSS6	rs855791 (G>A)	A	TC	Genetic result: Predisposition to slightly reduced serum levels of transferrin and iron. Interpretation: Transmembrane protease, serine 6 (TMPRSS6 or matriptase-2) variants are associated with decreased serum levels of transferrin and iron. Treatment/dosage: Supplementation should be considered.

### Selenium

Gene	SNP (transition)	Activating allele	Patient genotype	Pharmacogenetic result
DMGDH	rs921943 (T>C)	C	CT	Genetic result: Predisposition to higher selenium serum level. Interpretation: Dimethylglycine dehydrogenase (DMGDH) variants are associated with low selenium serum level. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with selenium.



## V. Methodology



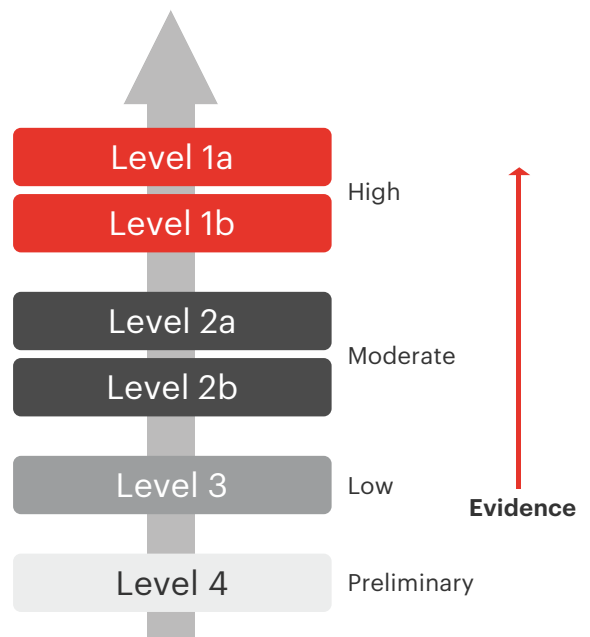
## 5. Methodology

### How were the genetic variants studied selected and evaluated?

The **genetic test** was developed by a multidisciplinary team of medical doctors, pharmacists, geneticists, and programmers, following the 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. Relevant patient’s anamnesis (intolerances, diseases, medication, blood pressure, among others) that can affect recommendations was taken into account through medical questionnaires elaborated by health professionals.

- **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 variants from level 1a to 2b were selected.



### How was this test performed?

DNA was extracted from the buccal swab sample 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 carried out using a custom-designed microfluidic card to measure for the chemiluminescent detection of each of them using TaqMan® technology. TaqMan® technology for genotyping testing is proven and widely used in clinical and research settings. The sensitivity (detection limit) of this study is 99%.

## genetic test algorithm

The **genetic test** qualitative pharmacogenetic algorithm analyzes single nucleotide polymorphisms (SNPs) associated with metabolic pathways involved in alopecia predisposition and treatment and combines this data with relevant patient history to predict treatment responses and recommends the most appropriate active ingredients.

The **genetic test** is an in vitro diagnostic medical device developed by **Fagron Genomics** and marketed under the CE-IVD mark in conformity with European Directive 98/79/EC and the transitional provisions (article 130) of European Regulation 2017/746.



**Fagron Genomics S.L.U.,**

SRN: ES-MF-000001092

C/ de les Cosidores, 150

08226 Terrassa, Barcelona (Spain)

## What are the limits of this report?

Each genetic marker tested is just one factor that predicts the likelihood of a particular outcome. However, the lifestyle, diet, and environment to which the patient is exposed may impact the expected outcomes. These external factors cannot be taken into account in this report.

The information in this report is not used to diagnose genetic diseases or abnormalities, as it does not predict the risk and likelihood of certain genetic outcomes. It is also not intended to diagnose or cure any disease. The **genetic test** is intended to assist health professionals in making patient-specific care decisions regarding the treatment or prevention of androgenetic alopecia, areata alopecia, and telogen effluvium.

Our clinical laboratory has standard and effective procedures to protect against technical and operational problems. However, problems may occur in the shipment to the laboratory or in the handling of the sample, including, but not limited to, damage to the sample, mislabeling, and loss or delay in receiving the sample. In such cases, the medical laboratory may need to request a new sample.

As with all medical laboratory tests, there is a small chance that the laboratory may provide inaccurate information.

It is the responsibility of the professional who requests a test from us to guarantee the interested party appropriate genetic counseling in accordance with Law 14/2007, of July 3, on Biomedical Research.

**Fagron Genomics S.L.U.** declines all responsibility derived from the use and interpretation of the results of our tests by the requesting health professional.

**Fagron Genomics S.L.U.** does not access data identifying the patient from whom the sample comes, so it is also the responsibility of the requesting professional to comply with the applicable data protection regulations.



## VI. References

## References

1. Cranwell and Sinclair. Male Androgenetic Alopecia. [Updated 2016 Feb 29]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000
2. Price (2003) Androgenetic Alopecia in Women, Journal of Investigative Dermatology Symposium Proceedings, Volume 8, Issue 1: 24-273
3. York et al. (2020). Treatment review for male pattern hair-loss. Expert Opinion on Pharmacotherapy 21: 603-612.
4. Shanshanwal et al. (2017) Superiority of dutasteride over finasteride in hair regrowth and reversal of miniaturization in men with androgenetic alopecia: A randomized controlled open-label, evaluator-blinded study. Indian J. Dermatol. Venereol. Leprol. 83: 47–54.
5. Tsunemi et al. (2014) Long-term safety and efficacy of dutasteride in the treatment of male patients with androgenetic alopecia. J. Dermatol. 43: 1051–1058.
6. Abdel-Raouf et al. (2021) A novel topical combination of minoxidil and spironolactone for androgenetic alopecia: Clinical, histopathological, and physicochemical study. Dermatol Ther. 34: e14678.
7. Rossi et al. (2020) Efficacy of Topical Finasteride 0.5% vs 17 $\alpha$ -Estradiol 0.05% in the Treatment of Postmenopausal Female Pattern Hair Loss: A Retrospective, Single-Blind Study of 119 Patients. Dermatol. Pract. Concept. 10: e2020039.
8. Blume-Peytavi et al. (2012) A randomized double-blind placebo-controlled pilot study to assess the efficacy of a 24-week topical treatment by latanoprost 0.1% on hair growth and pigmentation in healthy volunteers with androgenetic alopecia. J. Am. Acad. Dermatol. 66, 794–800.
9. Charlesworth et al. (1989) Effect of cetirizine on mast cell-mediator release and cellular traffic during the cutaneous late-phase reaction. J. Allergy Clin. Immunol. 83, 905–912.
10. Rossi et al. (2018) A preliminary study on topical cetirizine in the therapeutic management of androgenetic alopecia. J. Dermatolog. Treat. 29, 149–151.
11. Iehlé et al. (1995) Human prostatic steroid 5 $\alpha$ -reductase isoforms-A comparative study of selective inhibitors. J. Steroid Biochem. Mol. Biol. 54: 273–279.
12. Pais et al. (2016) Determination of the potency of a novel saw palmetto supercritical CO<sub>2</sub> extract (SPSE) for 5 $\alpha$ -reductase isoform II inhibition using a cell-free in vitro test system. Res. Reports Urol. 8: 41–49.
13. Wessagowit et al. (2016) Treatment of male androgenetic alopecia with topical products containing Serenoa repens extract. Australas. J. Dermatol. 57: e76–e82.
14. Rossi et al. (2016) Aromatase inhibitors induce ‘male pattern hair loss’ in women? Ann. Oncol. 24: 1710–1711.
15. Fischer et al. (2004) Melatonin increases anagen hair rate in women with androgenetic alopecia or diffuse alopecia: Results of a pilot randomized controlled trial. Br. J. Dermatol. 150: 341–345
16. Park et al. (2015) Red ginseng extract promotes the hair growth in cultured human hair follicles. J. Med. Food 18: 354–362.
17. Almohanna et al. (2019) The Role of Vitamins and Minerals in Hair Loss: A Review. Dermatol. Ther. (Heidelb). 9, 51–70.
18. Pratt et al. (2017) Alopecia areata. Nat Rev Dis Primers 3: 17011.
19. Qi and Garza (2014) An overview of alopecias. Cold Spring Harb. Perspect. Med. 4: 1–14.
20. Pourang and Mesinkovska (2020) New and Emerging Therapies for Alopecia Areata. Drugs. 80: 635-646.
21. Kumaresan (2020) Intralesional steroids for alopecia areata. Int J Trichol2 :63-65.
22. Lenane et al. (2014) Clobetasol propionate, 0.05%, vs hydrocortisone, 1%, for alopecia areata in children: A randomized clinical trial. JAMA Dermatology 150: 47– 50.
23. Tosti, et al. (2006) Efficacy and safety of a new clobetasol propionate 0.05% foam in alopecia areata: A randomized, double-blind placebo-controlled trial. J. Eur. Acad. Dermatology Venereol. 20, 1243–1247.
24. Tosti, et al. (2003) Clobetasol propionate 0.05% under occlusion in the treatment of alopecia totalis/universalis. J. Am. Acad. Dermatol. 49: 96–98.

25. Ucak et al. (2012) The comparison of treatment with clobetasol propionate 0.05% and topical pimecrolimus 1% treatment in the treatment of alopecia areata. *J. Dermatolog. Treat.* 23: 410–420.
26. Lalosevic et al. (2019) Combined intravenous pulse and topical corticosteroid therapy for severe alopecia areata in children: Comparison of two regimens. *Dermatol. Ther.* 32: 1–9.
27. Lalosevic et al. (2015) Combined oral pulse and topical corticosteroid therapy for severe alopecia areata in children: A long-term follow-up study. *Dermatol. Ther.* 28, 309–317 (2015).
28. Jung et al. (2017) Comparison of the topical FK506 and clobetasol propionate as first-line therapy in the treatment of early alopecia areata. *Int. J. Dermatol.* 56, 1487–1488.
29. Callender et al. (2020) Safety and Efficacy of Clobetasol Propionate 0.05% Emollient Foam for the Treatment of Central Centrifugal Cicatricial Alopecia. *J Drugs Dermatol.* 19: 719-724.
30. Gasic et al. (2018) Pharmacogenomic markers of glucocorticoid response in the initial phase of remission induction therapy in childhood acute lymphoblastic leukemia. *Radiol. Oncol.* 52: 296–306.
31. Rodrigues et al. (2017) Decreased comfort food intake and allostatic load in adolescents carrying the A3669G variant of the glucocorticoid receptor gene. *Appetite* 116: 21–28.
32. Schaaf et al. (2002) AUUUA motifs in the 3'UTR of human glucocorticoid receptor  $\alpha$  and  $\beta$  mRNA destabilize mRNA and decrease receptor protein expression. *Steroids* 67: 627–636.
33. Van Rossum et al. (2004) Polymorphisms in the glucocorticoid receptor gene and their associations with metabolic parameters and body composition. *Recent Prog. Horm. Res.* 59: 333-357.
34. Van den Akker et al. (2008) Glucocorticoid Receptor Gene and Risk of Cardiovascular Disease. *Arch. Intern. Med.* 168: 33-39.
35. Gupta and Charrette (2014) The efficacy and safety of 5 alpha-reductase inhibitors in androgenetic alopecia: a network meta-analysis and benefit-risk assessment of finasteride and dutasteride. *J Dermatolog. Treat.* 25: 156-161.
36. Stoner (1990) The clinical development of a 5 alpha-reductase inhibitor, finasteride. *J Steroid Biochem. Mol. Biol.* 20: 37: 375-378.
37. Bayne et al. (1999) Immunohistochemical localization of types 1 and 2 5alpha-reductase in human scalp. *Br. J. Dermatol.* 141: 481-491.
38. Hsing et al. (2001) Polymorphic markers in the SRD5A2 gene and prostate cancer risk: a population-based case-control study. *Cancer Epidemiol. Biomarkers Prev.* 10: 1077-1082.
39. Allen et al. (2001) The association between polymorphisms in the CYP17 and 5alpha-reductase (SRD5A2) genes and serum androgen concentrations in men. *Cancer Epidemiol. Biomarkers Prev.* 10: 185-189.
40. Van Gils et al. (2003) The V89L Polymorphism in the 5-a-Reductase Type 2 Gene and Risk of Breast Cancer. *Cancer Epidemiol. Biomarkers Prev.* 12: 1194–1199.
41. Leake et al. (2017) The effect of zinc on the 5a-reduction of testosterone by the hyperplastic human prostate gland. *J. Steroid Biochem.* 20: 651–655.
42. Wickett et al. (2007) Effect of oral intake of choline-stabilized orthosilicic acid on hair tensile strength and morphology in women with fine hair. *Arch. Dermatol. Res.* 299: 499–505.
43. Oura et al. (2008) Adenosine increases anagen hair growth and thick hairs in Japanese women with female pattern hair loss: A pilot, double-blind, randomized, placebo-controlled trial. *J. Dermatol.* 35: 763–767.
44. Faghihi et al. (2013) Comparison of the efficacy of topical minoxidil 5% and adenosine 0.75% solutions on male androgenetic alopecia and measuring patient satisfaction rate. *Acta Dermatovenerologica Croat.* 21: 155–159.
45. Iwabuchi et al. (2016) Topical adenosine increases the proportion of thick hair in Caucasian men with androgenetic alopecia. *J. Dermatol.* 43: 567–570.
46. Watanabe et al. (2015) Topical adenosine increases thick hair ratio in Japanese men with androgenetic alopecia. *Int. J. Cosmet. Sci.* 37: 579-587.
47. Manabe et al. (2018) Guidelines for the diagnosis and treatment of male-pattern and female-pattern hair loss, 2017 version. *J. Dermatol.* 45: 1031–1043.
48. Muizzuddin et al. (2020) Beauty from within: Oral administration of a sulfur-containing supplement methylsulfonylmethane improves signs of skin ageing. *Int. J. Vitam. Nutr. Res.* 1–10 .

49. Shanmugam et al. (2020) The effect of methylsulfonylmethane on hair growth promotion of magnesium ascorbyl phosphate for the treatment of alopecia. *Biomol. Ther.* 17: 241-248.

50. Weger and Schlake (2005) Igf-I signalling controls the hair growth cycle and the differentiation of hair shafts. *J. Invest. Dermatol.* 125: 873-882.

51. Itami and Inui (2005) Role of androgen in mesenchymal epithelial interactions in human hair follicle. *J. Invest. Dermatol. Symp. Proc.* 10: 209-211.

**Together**  
we create the future  
of personalized medicine.



**Fagron Genomics, S.L.U.**  
C/ de les Cosidores, 150  
08226 Terrassa  
Barcelona (Spain)

[www.fagrongenomics.com](http://www.fagrongenomics.com)

