- Patient's name: Dan Lawrence
- Received in laboratory 12-09-2024
- Genetic results date 12-11-2024

Hair Loss Treatment Test

Results Report

Roots by GENETIC ARTS

Genetic report

METHODOLOGY AND LIMITATIONS DISCLAIMER:

Testing for genetic variation/mutation on listed genes was performed using Real-Time PCR with TaqMan® allele-specific probes on the QuantStudio 12K Flex. All genetic testing is performed by GX Sciences, LLC d/b/a Fagron Genomics US ("Fagron Genomics US") located at 807 Las Cimas Pkwy, Suite 145, Austin TX 78746. This test will not detect all the known alleles that result in altered or inactive tested genes. This test does not account for all individual variations in the individual tested. Test results do not rule out the possibility that this individual could be a carrier of other mutations/variations not detected by this gene mutation/variation panel. Rare mutations surrounding these alleles may also affect our detection of genetic variations. Thus, the interpretation is given as a probability. Therefore, this genetic information shall be interpreted in conjunction with other clinical findings and familial history for the administration of specific treatments. Patients should receive appropriate genetic counseling to explain the implications of these test results. The analytical and performance characteristics of this laboratory developed test (LDT) were determined by Fagron Genomics US's laboratory (Laboratory Director: James Jacobson, PhD) pursuant to Clinical Laboratory Improvement Amendments (CLIA) requirements (CLIA #: 45D2144988).

MEDICAL DISCLAIMER:

This test was developed and its performance characteristics determined by Fagron Genomics US. It has not been cleared or approved by the FDA. The laboratory is regulated under CLIA and qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. The Reference SNP Cluster IDs (rsIDs) for the alleles being tested were obtained from the Single Nucleotide Polymorphism Database (dbSNP). These products are not approved by the Food and Drug Administration and are not intended to diagnose, treat, cure or prevent disease. These recommendations are for report purposes only and an individual is not required to use such products. These are recommendations only and do not replace the advisement of your own healthcare practitioner.

LEGAL DISCLAIMER:

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I.
Patient identification data

1.
Patient identification data

Ordering physician	**Riley Greene**
Patient's name	**Dan Lawrence**
Gender	**Male**
Date of birth	**05-11-1974**
Sample type	**Buccal Swab**
Sample code	**TRI65317AA**
Collection date	**12-02-2024**
Received date	**12-09-2024**
Genetic results date	**12-11-2024**

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	53%
• Cetirizine Hcl	50%
• Latanoprost	44%

Antiandrogenic	
• Dutasteride	100%
• Finasteride	99%
• Spironolactone	100%
• Melatonin	75%

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

Immunomodulator	
• Tacrolimus	

Hair care supplements

Circulation	
• Arginine	57%
• Caffeine	25%

Collagen synthesis
• Cystine

Blocked — Recommended

Vitamin, mineral and antioxidant supplements

Vitamin deficiency	
• Vitamin B12 (Cyanocobalamin)	100%
• Vitamin B7 (Biotin)	67%
• Vitamin D	67%
• Vitamin E (Tocoferol)	67%
• Vitamin C (Ascorbic Acid)	
• Lysine	
• Vitamin B9 (Folate)	

Keratolytic	
• Tretinoin	50%

Minerals	
• Zinc gluconate	100%
• Zinc acetate	100%
• Iron sulfate	67%
• Magnesium Gluconate	

III.
Complete data

3.
Complete data
Data from the medical questionnaire

Patient demographics

Gender	**Male**
Age (years)	**50**
Family history of alopecia	**Siblings**

Hair loss data

Type of alopecia	**Androgenic alopecia**
Grade of alopecia	**Grade Vertex**

Norwood-Hamilton Scale

Clinical examination

Amount of hair loss	**Little bit**
Complaints associated with alopecia	**No**
Patchy alopecia	**No**

3.
Complete data
Pharmacogenetic results

1. Anti-alopecic drugs

Treatment efficacy with prostaglandin inhibitors

Prostaglandin D2			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
GPR44-1	rs545659	**CT**	Genetic result: Predisposition to slightly higher GPR44 mRNA stability. Interpretation: Higher expression of the Prostaglandin D2 receptor 2 (GPR44) receptor may lead to increased responsiveness to prostaglandin D2. Treatment recommendation: Consider treatment with Prostaglandin D2 inhibitors (e.g., Cetirizine, Prostaquinon) at standard doses.
GPR44-2	rs533116	**CT**	Genetic result: Predisposition to slightly higher GPR44 mRNA stability. Interpretation: Higher expression of the Prostaglandin D2 receptor 2 (GPR44) receptor may lead to increased responsiveness to prostaglandin D2. Treatment recommendation: Consider treatment with Prostaglandin D2 inhibitors (e.g., Cetirizine, Prostaquinon) at standard doses.

Latanoprost			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
PTGFR-1	rs6686438	**GG**	Genetic Result: This prostaglandin F receptor (PTGFR) variant may be associated with significantly lower probability of treatment efficacy with latanoprost (prostaglandin analog). Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment recommendation: Consider the use of concomitant or alternative therapies.
PTGFR-2	rs1328441	**CT**	Genetic Result: This prostaglandin F receptor (PTGFR) variant may be associated with an intermediate probability of treatment efficacy with latanoprost (prostaglandin analog). Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment recommendation: Consider treatment with Latanoprost at standard or higher doses.
PTGFR-3	rs10782665	**GT**	Genetic Result: This prostaglandin F receptor (PTGFR) variant may be associated with an intermediate probability of treatment efficacy with latanoprost (prostaglandin analog). Interpretation: Prostaglandin F receptor (PTGFR) variants are related with Latanoprost treatment efficacy (prostaglandin analog) . Treatment recommendation: Consider treatment with Latanoprost at standard or higher doses.

Treatment efficacy with minoxidil

Minoxidil			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
PTGES2	rs13283456	**CT**	Genetic Result: Predisposition toward slightly reduced PGE2 levels. Interpretation: Lower PTGES2 activity may result in significantly lower PGE2 levels which is related to alopecia development. Minoxidil promotes hair growth by increasing the production of PGE2 via PTGES2. Treatment recommendation: Consider oral, topical, or combination minoxidil treatment at standard doses to stimulate PGE2 production.
SULT1A1	rs1042028	**AG**	Genetic result: Predisposition to slightly reduced SULT1A1 activity and therefore less conversion of minoxidil into its active metabolite minoxidil sulfate. Interpretation: Minoxidil Sulfotransferase Enzyme (SULT1A1) variants predict response to minoxidil treatment. Treatment recommendation(s): Consider increasing the dosage or frequency of administration of oral or topical minoxidil. Consider combining oral and topical minoxidil treatments concurrently. Consider the use of a SULTA1A enzyme activity enhancing agent (e.g. tretinoin) compounded with topical minoxidil.

Treatment efficacy with glucocorticoid anti-inflammatories

Glucocorticoides			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
GR-alpha	rs6198	**CT**	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 recommendation: 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)**	**Patient genotype**	**Pharmacogenetic result**
CYP19A1	rs2470152	**AA**	Genetic result: Predisposition to significantly reduced CYP19A1 expression. Interpretation: Lower Aromatase (CYP19A1) activity may be associated with reduced conversion of testosterone into estrogens and higher conversion into DHT (a known hair growth inhibitor). Treatment recommendation: Consider treatment with 17-α Estradiol (an aromatase inducer) at higher doses. Consider adjuvant anti-androgen therapies and additional modalities.

Dutasteride			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
SRD5A1	rs39848	**CC**	Genetic result: Predisposition significantly increased SRD5A1 activity. Interpretation: Steroid 5α-Reductase 1 (SRD5A1) variants are associated with increased SRD5A1 activity leading to increased DHT levels and hair growth inhibition. Treatment/dosage: Treatment with Dutasteride at higher doses is recommended.

Finasteride			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
SRD5A2	rs523349	**CG**	Genetic result: Predisposition to slightly increased SRD5A2 activity. Interpretation: Steroid 5α-Reductase 2 (SRD5A2) variants are associated with increased SRD5A2 activity leading to increased DHT levels and hair growth inhibition. Treatment recommendation: Consider treatment with traditional doses of oral finasteride. Consider topical finasteride. Consider adding additional anti-androgen therapies or other modalities (e.g., pharmaceutical, phototherapy, nutraceutical, regenerative, etc.).

2. Hair care supplements

Vasodilatation and blood circulation

Circulation stimulators			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
ACE	rs4343	**AG**	Genetic result: Predisposition to slightly increased ACE activity. Interpretation: Increased Angiotensin-converting enzyme (ACE) activity may be associated with increased plasma levels of Angiotensin II, an extremely potent vasoconstrictor. Treatment recommendation: Consider normal doses of circulation stimulators such as Minoxidil, caffeine, Ginkgo Biloba, Ginseng or Arginine.

Collagen synthesis

Hair strengthening supplements			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
COL1A1	rs1800012	**CC**	Genetic result: Predisposition to normal COL1A1 expression. Interpretation: Increased COL1A1 expression is correlated with androgenetic alopecia and may be associated with a higher degree of collagen molecule instability due to an altered ratio of collagen a1/a2. Treatment recommendation: While hair strengthening composites may be of benefit, the SNP analysis does not indicate a relative indication nor contraindication for their use.

Reduction of IGF-1 levels

Hair strengthening supplements			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
IGF1R	rs2229765	**AA**	Genetic result: Predisposition to 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 recommendation: A treatment with Igrantine-F1 and TrichoXidil (IGF-1 inducers) at high doses would be recommended.

3. Vitamin, mineral and antioxidant supplements

Vitamins

Vitamin A			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
CRABP2	rs12724719	**AG**	Genetic Result: This CRABP2 variant is associated with slightly lower retinoic acid (vitamin A) intracellular transport. Interpretation: Cellular retinoic acid-binding protein 2 (CRABP2) variants are associated with lower retinoic acid (vitamin A) intracellular transport. Treatment recommendation: Consider nutritional supplementation with normal doses of vitamin A.

Vitamin B7			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
BTD	rs13078881	**CG**	Genetic Result: This BTD variant suggests a predisposition to slightly reduced Biotidinase activity. Interpretation: Reduced biotidinase activity may result in low biotin levels due to significantly lower biotin (vitamin B7) uptake from the gut. Treatment recommendation: Consider supplementation with normal doses of B vitamins.

Vitamin C			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
SLC23A1	rs33972313	**CC**	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. Test for serum levels of vitamin C.

Vitamin B9			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
MTHFR	rs1801133	**GG**	Genetic result: No predisposition to folate deficiency. Interpretation: Methylene tetrahydrofolate reductase (MTHFR) variants are associated with risk of folate deficiency. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with folate. Test serum levels of folate prior to supplementation.

Vitamin D			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
GC	rs2282679	**GT**	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.Test serum levels of vitamin D prior to supplementation.

Vitamin B12			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
FUT2	rs602662	**AG**	Genetic result: Predisposition to low vitamin B12 serum level. Interpretation: Galactoside 2-alpha-L-fucosyltransferase 2 (FUT2) variants are associated lower vitamin B12 serum level. Treatment/dosage: Supplementation with vitamin B12 is highly recommended.

Vitamin E			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
ZPR1	rs964184	**CG**	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. Test serum levels of vitamin E prior to supplementation.

Antioxidants

Antioxidants			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
NQO1	rs1800566	**GG**	Genetic result: Predisposition to normal NQO1 enzyme activity. Interpretation: NAD(P)H dehydrogenase [quinone] 1 (NQQ1) 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. Test serum levels of selenium prior to supplementation.

Minerals

Magnesium			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
MUC1	rs4072037	**TT**	Genetic result: Predisposition to higher magnesium serum level. Interpretation: Mucin 1, cell surface associated (MUC1) variants are associated with lower magnesium serum level. Treatment/dosage: SNP analysis does not indicate the necessity to supplement with magnesium. Test serum levels of magnesium prior to supplementation.

Zinc sulfate			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
SLC30A3	rs11126936	**GG**	Genetic result: Predisposition to lower serum zinc level. Interpretation: Solute carrier family 30 member 3 (SLC30A3) variants are associated with lower serum zinc level. Treatment/dosage: Supplementation with Zinc sulfate would be highly recommended. Test serum levels of zinc prior to supplementation.

Iron			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
TMPRSS6	rs855791	**AG**	Genetic result: Predisposition to slightly reduced serum levels of tranferrin 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. Test serum levels of iron prior to supplementation.

Selenium			
Gene	**SNP (transition)**	**Patient genotype**	**Pharmacogenetic result**
DMGDH	rs921943	**CC**	Genetic result: Predisposition to lower selenium serum level. Interpretation: Dimethylglycine dehydrogenase (DMGDH) variants are associated with low selenium serum level. Treatment/dosage: Selenium supplementation should be considered. Test serum levels of selenium prior to supplementation.

IV.
Methodology

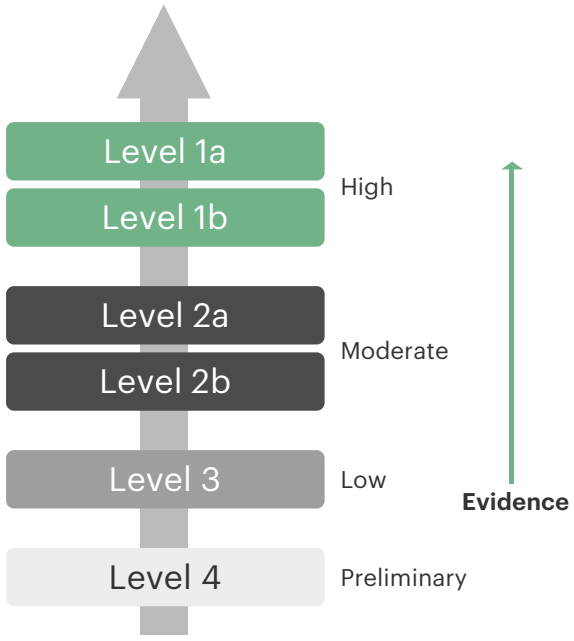
4. Methodology

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 algoritm

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 underthe CE-IVD mark in conformity with European Directive 98/79/EC and the transitional provisions (article 130) of European Regulation 2017/746.

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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.

V.
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 17a-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 5a-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 CO2 extract (SPSE) for 5a-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 α and β 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 Investig Dermatol. Symp. Proc. 10: 209-211.

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