SCIENTIFIC BASE: THE IMPACT OF GENETICS ON HAIR LOSS, AND PRECISION TREATMENT

Roots
by GENETIC ARTS

DNA & HAIR LOSS

Unlocking answers to hair loss

Roots by Genetic Arts hair loss test is a pharmacogenetic test that determines whether person having androgenetic alopecia (balding), alopecia areata or telogen effluvium is likely to be responsive to administration of specific hair loss treatments.

In it's current version, the test analyses 16 genetic variations in 13 genes, 7 pathways and is used as biomarkers for predicting patient response prior to therapy.

Type and grade of alopecia, current medication, pathologies, intolerance, allergy, physiological and emotional stress are taken into consideration through a questionnaire. Our proprietary algorithm combines genetic data with relevant patient's anamnesis and possible contraindications to select the most appropriate vehicles and active pharmaceutical ingredients.

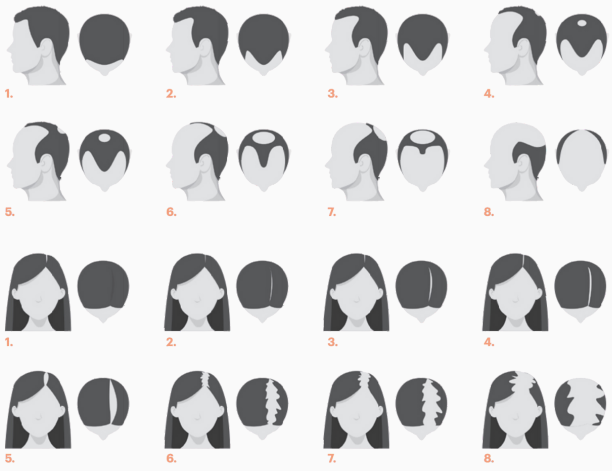
Hair loss background

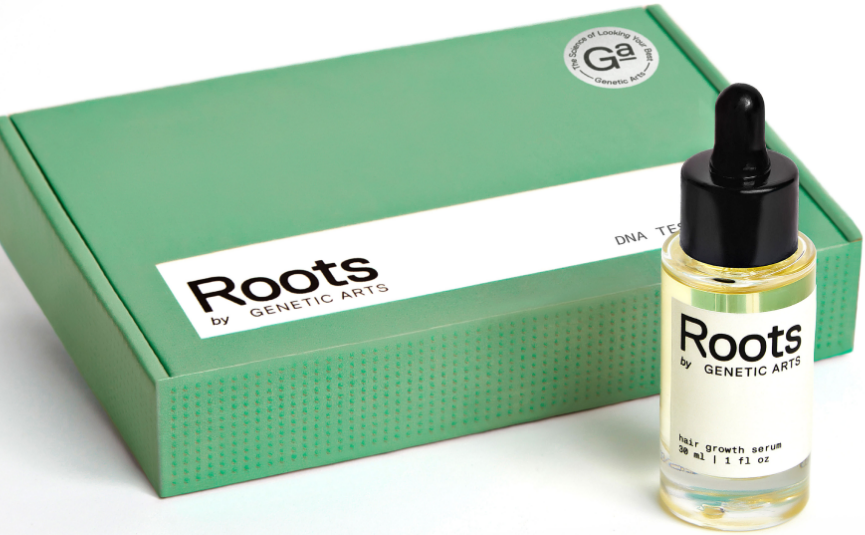
Hair is considered an essential part of most people's identity and for many hair loss or irregular hair growth can have a significant impact on their psychological health. A hair growth cycle consists of growth (ana-gen), involution (catagen), and rest (telogen). Most people lose 50 to 100 hairs per day as part of this nat-ural cycle. Alopecia is an abnormal hair loss that can occur anywhere on the body, but most commonly affects the scalp and is caused by an interruption in the hair growth cycle, whether due to age, autoimmune conditions, or stress. Common types of scalp alopecia include **androgenetic alopecia**, **alopecia areata,** and a thinning of hair known as **telogen effluvium**.

Androgenetic Alopecia (AGA)

30% of men will have AGA by the age of 30, up to 50% by the age of 50, and 80% by the age of 70.[1]

Androgenetic Alopecia (AGA), also known as pattern hair loss, pattern alopecia or common baldness, is the most common type of progressive hair loss in both men and women. Although AGA is regarded more as a physiological condition than a disease, the psychological impact of hair loss can be profound and there is a significant association between AGA and life-threatening conditions, such as coronary artery disease and prostate cancer.





Incidence and prevalence of AGA depend on age and race. Based on the little prevalence data available, we know that up 30% of Caucasian men will have AGA by the age of 30, up to 50% by the age of 50, and 80% by the age of 70[1]. Other ethnic groups are less affected than Caucasians. AGA is known to be androgen-dependent and higher levels of serum testosterone are associated with increased risk. Thinning of the hair usually begins between the ages of 12 and 40 years in both sexes, and its natural course may be either slowly or rapidly progressing[2]. Factors that may accelerate progression include androgen metabolism, inflammatory scalp disorders, and lifestyle.

Alopecia treatment options vary, including acceptance of the condition or shaving one's head. Common medical treatments for Androgenetic Alopecia (AGA) involve inhibiting Dihydrotestosterone (DHT) synthesis through medications like finasteride and dutasteride, as well as using prostaglandin analogues such as bimatoprost or latanoprost to counter the effects of androgen receptor activation. Minoxidil is another medication that modifies hair cycle dynamics. The FDA-approved treatments for AGA are minoxidil and finasteride. Minoxidil is available in topical solutions for men and women, while finasteride is specifically for men and prevents the miniaturization of hair follicles by inhibiting 5-alphareductase (5AR) enzyme. Dutasteride, an alternative to finasteride, inhibits both types of 5AR and is more effective in suppressing DHT levels. Combination therapies with minoxidil, finasteride, or dutasteride have shown positive results in treating hair loss.

Responses to finasteride may vary, and while it stops hair loss in the majority of men, only around 66% experience moderate hair regrowth, **while 11% achieve marked regrowth.** [3,53]

Prostaglandins, such as PGD2, PGE2, and PGF2a, play a role in regulating the hair cycle, and their manipulation through medications like latanoprost or cetirizine has shown promising effects. Saw Palmetto extract inhibits both types of 5AR and activates estrogen receptors, aiding in hair follicle health. Melatonin-alcohol solution and ginseng intake have also been associated with increased hair growth. Nutritional deficiencies may contribute to alopecia, but further research is needed to fully understand their benefits.

Alopecia areata (AA)

Alopecia areata (AA) Alopecia areata (AA), also known as patchy baldness or spot baldness, is a common form of immune-mediated alopecia in which an autoimmune attack on the proximal hair follicle (results in non-scarring hair loss ranging in presentation from circular patches on the scalp to total scalp or full-body hair loss. AA has a lifetime prevalence of approximately 2%.[24]

Men and women are equally affected, with onset of symptoms occurring most commonly before age 30. The condition has a hereditary component 20% of patients possess at least one first-degree relative with AA.

AA is commonly perceived as a cosmetic, rather than medical, concern. However, substantial evidence exists describing the negative impact on quality of life, as the disease affects patients personally, socially, financially, and physically. The unpredictable course of the disease also makes it a mental struggle and AA patients are more often associated with depression and anxiety compared to the healthy population.

Furthermore, AA is associated with other concurrent diseases (comorbidities) including several autoimmune diseases including thyroid disease (hyperthyroidism, hypothyroidism, goiter ant thyroiditis), lupus erythematosus, vitiligo, psoriasis, rheumatoid arthritis and inflammatory bowel disease.[24-25]

Telogen effluvium (TE)

Telogen effluvium (TE) is a form of temporary hair loss, characterized by the thinning or shedding of hair, that usually happens after stress, a shock, or a traumatic event. There is usually a trigger that occurs 2 - 4 months before onset of hair loss. The cause may be endocrine, in the event of childbirth and hyper/hypothyroidism; nutritional, encompassing crash diets and vitamin A excess; drug-related, most notably anticoagulants and b-blockers; and stress.

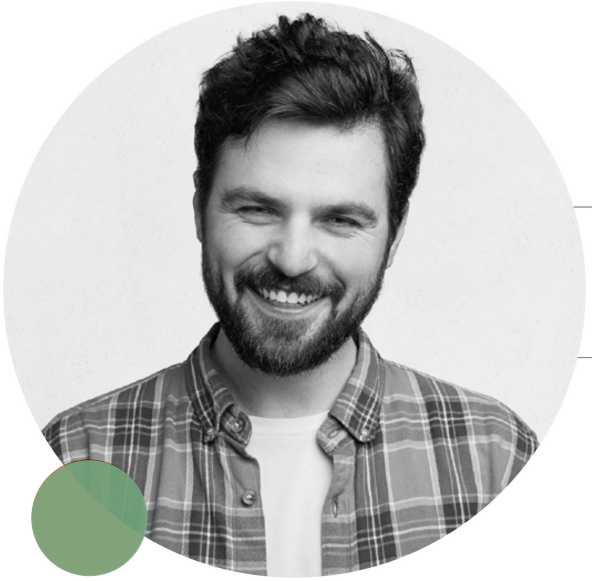
TE can occur in people of any age, any gender, and any racial background. The exact prevalence of TE is not known, but it is considered to be quite common.[50] Studies have reported TE incidence in children to be around 2.7%.[51] A large percentage of adults experience an episode of TE at some point. TE can occur in either sex, though women have a greater tendency to experience this condition because of postpartum hormonal changes. Also, women are more disturbed by hair shedding than men and are therefore more likely to seek medical attention.

Unmet customer needs

The efficacy of the most recognized alopecia treatments is not absolute, and it requires at least a 4-to-6 month trial before noticing improvement and must be used indefinitely to maintain a response.

Although topical **minoxidil** exhibits a good safety profile, its efficacy in the overall population remains **relatively low at 30 - 40%**. While **finasteride** arrests hair loss in over 87% of men, **only 11% achieve marked hair regrowth.**[3,53]

Due to the significant time commitment and low response rate, biomarkers for predicting patient response prior to therapy are advantageous. Numerous polymorphisms defining potential response to alopecia treatments have been defined, but not until Roots by Genetic Arts has a there been a DNA test for predicting patient response prior to therapy.



Genetic Factors
Roots by GA analyzes the most relevant genetic variations for personalizing alopecia treatment.

Patient anamnesis
Current medication, pathologies, intolerance, allergy, physiological and emotional stress are also taken into consideration through a questionnaire.

Roots by Genetic Arts hair loss test is a pharmacogenetic test that determines whether a male or female subject having androgenetic alopecia, alopecia areata or telogen effluvium is likely to be responsive to administration of specific hair loss treatments. In its current version, Roots by Genetic Arts analyses 16 genetic variations in 13 genes, used as biomarkers for predicting patient response prior to therapy.

The 7 Biomarkers, Genes and APIs analyzed by Roots by GA are summarized below:

Prostaglandin metabolism

Although topical minoxidil exhibits a good safety profile, its efficacy in the overall population remains relatively low at 30-40%.[54] To observe significant improvement in hair growth, minoxidil is typically used daily for a period of at least 3-4 months Minoxidil is converted to its active form (minoxidil sulfate) by the hair sulfotransferase enzyme (SULT1A1).[54] Since minoxidil response correlates with SULT1A1 enzyme activity, rs9282861 SULT1A1 variant analysis (level 1B) could be used to predict minoxidil response.[57-58] If the patient is identified as "responder", minoxidil and agents known to increase sulfotransferase, and to enhance the regrowth effect of topical Minoxidil could be recommended.[59] The analysis of the rs13283456 PTGES2 (Prostaglandin E synthase 2) analysis (level 1B) provides an indication whether a patient is likely to have low prostaglandin E2 production in the hair follicle that needs to be stimulated by minoxidil therapy.[60,61-63]

The proposed mechanism behind the hair-growth-stimulating effect of minoxidil is through its stimulating effect of prostaglandin E2 (PGE2) synthesis.[64] Prostaglandins (PGs) are potent proinflammatory mediators and play an important role in modulating inflammatory and allergic immune responses. The role of prostaglandins in regulating hair growth and their dysregulation in AGA has been documented in the literature. For example, although PGE2 and PGF2a have been shown to stimulate hair lengthening in mice,[65] PGD2 and its receptor GPR44 (also known as CRTH2) have been implicated as negative regulators[66] of hair growth. GPR44 expression is elevated in bald scalp compared to haired scalp of men with AGA.[69]

Blocking the GPR44 receptor promotes hair shaft growth in cultured human hair follicles.[69] GPR44 rs545659 G polymorphism, resulting in an increased GPR44 mRNA stability,[70] is associated with asthma and allergic sensitization.[70-74] Similarly, rs533116 GPR44 A polymorphism results in a higher level of GPR44 mRNA and is associated with asthma.[75]

Higher GPR44 expression levels and increased responsiveness to its PGD2 ligand may be the biological basis for the association of this receptor with inflammatory and allergic immune responses.[70,76] rs545659 GPR44 polymorphism (level 1B) in combination with rs533116 (level 1B) provides information to healthcare providers about whether a patient is likely to benefit from treatments reducing prostaglandin D2 (PGD2) levels: cetirizine and prostaquinon, a phytocomplex derived from Nigella sativa seeds essential oil. Topical cetirizine is known for its anti-inflammatory properties and its ability to decrease PGD2 production.[9] It has been shown to significantly improve the initial framework of AGA with an increase in total hair density, terminal hair density, and diameter variation, TE Patients treated with a scalp lotion containing 0.5% N. sativa seeds essential oil showed a significant increment of hair density and hair thickness. Thymoquinone is the most prominent constituent of Nigella sativa seeds essential oil[81] and has been shown to inhibit prostaglandin PGD2 production.[79-82]

PGF2a analog latanoprost is FDA–approved and routinely used clinically to enhance hair growth of human eyelashes.[83] Latanoprost significantly increases the capillary density of bald patients.[8,84-85] PTGFR encodes the prostaglandin F receptor that binds to and mediates the biological actions of PGF2a. PTGFR polymorphisms are associated with positive and negative responses to latanoprost.[86-88] rs10782665 PTGFR allele variant T (level 2A) is related to a high probability of treatment efficacy with Latanoprost.[88] Patients with the G have an increased likelihood of not having a positive response to Latanoprost.[88] Similarly, rs6686438 PTGFR allele variant T (level 2A) and rs1328441 PTGFR allele variant G (level 2A) are related with a higher efficacy in treatments with Latanoprost.[88] Analysis of these three PTGFR polymorphisms provides an indication to healthcare providers whether a patient has a high likelihood of having a positive response to Latanoprost or an increased likelihood of not responding to Latanoprost treatment, a quite expensive treatment option.

Inflammation

Intralesional corticosteroids are widely used in the treatment of alopecia areata.[89] Time to clinical improvement with triamcinolone acetonide, the most commonly used intralesional corticosteroid for AA ranges from 2 to 6 weeks. Topical corticosteroids applied to the scalp are less effective than steroid injections but might offer benefit in approximately 30% to 50% of AA patients.[90] Initial signs of improvement can take anywhere from 6 weeks to 3 months (up to 6 months in some). GR, also known as NR3C1, is the receptor to which cortisol and other glucocorticoids bind.[91] Several GR variants lead to altered sensibility of GR to glucocorticoids and are associated with resistance or sensitivity to corticosteroids.[92-93] rs6198 GR (Glucocorticoid Receptor) polymorphism (level 1B) is associated with resistance or sensitivity to corticosteroids and provides a valuable information about response to corticosteroids.[94-96] If the patient is identified as potentially resistant to corticosteroid, alternative treatment with tacrolimus, ginseng or melatonin. Tacrolimus is an immunosuppressive drug used in the treatment of alopecia areata to reduce inflammation.[45-47] Ginseng intake can improve blood vessel health via modulation of vasodilation, oxidation stress, and pro-inflammatory cytokines.[19-20] Topical treatment with 1 ml of a 0.1% melatonin-alcohol solution in women with AGA and diffuse alopecia resulted in a significant increase in detectable anagen hairs in the occipital and frontal areas after six months compared with placebo.[15-17]

Androgenic effect – DHT metabolism

To observe significant improvement in hair growth, oral finasteride must be used daily for a period of at least 3 - 6 months. The overall effect of hair growth resulting of 5 years treatment with finasteride 1 mg is a stabilization of hair loss in 87% of patients, in whom only around 11% noticed an important hair regrowth.[53] Finasteride competitively and specifically inhibits the SR5DA2 enzyme,[97-99] one of the two forms of steroid 5a-reductase responsible for the conversion of cortisol to dihydrocortisol and of testosterone to the more potent dihydrotestosterone (DHT). rs523349 SRD5A2 polymorphism (level 1B) associated with active and less active forms of the SRD5A2[100-102] provides an indication about potential response to Finasteride treatment.

If the patient is identified as a potential “responder”, finasteride and agents known to enhance treatment effect could be recommended: Zinc sulphate is a natural 5a-reductase inhibitor[103-106] shown to be effective in hair loss treatment.[107-108] Caffeine has vasodilator effects.[109-110] Ginseng intake can improve blood vessel health via modulation of vasodilation, oxidation stress, and pro-inflammatory cytokines.[18-19] The ginsenosides have also been discovered to promote hair growth through a mechanism similar to that of minoxidil.[20-22] Extract of Serenoa repens, commonly known as saw palmetto, is a botanical extract with antiandrogenic properties[11-12] used to treat AGA.[13-14] Dutasteride inhibits both forms of the steroid 5a-reductase.[97,111] rs39848 SRD5A1 variant A (level 2A) is associated with a tendency to hirsutism in PCOS[112] and to lower levels of cortisol and higher levels of testosterone indicating a reduced enzyme activity.[113] rs39848 polymorphism provides an indication about potential response to dutasteride treatment and dosages to use. If the patient is identified as a potential “responder”, dutasteride and agents known to enhance treatment effect could be recommended. CYP19A1 gene encodes the aromatase, responsible for the final step of the biosynthesis of estradiol and estrone. A decreased activity of aromatase leads to a decreased conversion of testosterone in estrogens and a higher conversion into DHT and results in hair loss.[14,114-115] rs2470152 CYP19A1 allele variant G (level 1B) is associated to increased circulating estrogen levels and E2/T (estradiol/testosterone) ratios, a marker of aromatase activity.[116-120] rs2470152 polymorphism provides an indication to healthcare providers about potential response to 17-Estradiol treatments (stimulation of aromatase activity) and complementary (spironolactone) or alternative (ginseng, melatonin) treatments. Topical applications of lotions containing 17-estradiol have been shown to be effective to treat AGA.[121] Spironolactone is currently being used in dermatology as an antiandrogen[122-128] for the treatment of acne, diffuse hair loss in females, and hirsutism.[129] Topical treatment of spironolactone allows high penetration of the drug to the active site with the advantage of minimizing unwanted adverse effects[130] and has been shown to be effective in AGA treatment.[6]

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Vasodilatation and blood circulation

Growing evidence supports the hypothesis that the hair follicle cycling is associated with the remodelling of skin vascularization and perfusion.[131-132] Angiotensin-converting enzyme (ACE) is a membrane-bound, zinc-dependent dipeptidase that catalyses the conversion of angiotensin I to the physiologically active Angiotensin II, an extremely potent vasoconstrictor.[133] The ACE Insertion (I)/Deletion (D) Polymorphism affects ACE activity: Patients with allele D present increased ACE activity compared to patients carrying I.[134] Rs4343 . Rs4343 A . Rs4343A<G polymorphism is in near perfect linkage disequilibrium with ACE I/D in Europeans (A and G alleles marking I and D alleles, respectively).[135] ACE polymorphism may influence numerous disparate conditions or phenotypes, including hypertension, Alzheimer and Coronary Artery Disease. rs4343 ACE polymorphism (level 1B) provides an indication to healthcare providers about the predisposition to increased levels of angiotensin II,[134-137] a potent vasoconstrictor,[133] and the possibility to treat with vasodilators commonly used for hair loss treatment: minoxidil,[138] L-arginine,[139-141] Caffeine,[109-110] Ginkgo biloba,[142] and Ginseng.[19-21]

Collagen synthesis

Type I collagen is the most abundant collagen of the human body, representing up 75 - 90% of the collagen found in the skin, hair, nails, organs, bone, and ligaments. Type I collagen molecule is a heterotrimer consisting of two a1(I) and one a2(I) chains, which are encoded for by the COL1A1 and COL1A2 genes, respectively. Col1A1 is over-expressed in situations of androgenic alopecia.[168] COL1A1 rs1800012 G>T (level 2A) results in increased COL1A1 gene expression[143-144] and has been associated with increased risk of osteoporosis[145] and of acute musculoskeletal soft tissue injuries.[146] The rs1800012 T allele is also associated with a higher 1 (I) to 2 (I) collagen protein ratio, which reflects the increased ratio of COL1A1 mRNA relative to COL1A2 and may result in an instability of collagen molecules.[143] If rs1800012(T) allele is detected, the Roots by GA algorithm suggests the inclusion in the formula of commonly used composites associated with hair strengthening: cystine, silicon, adenosine. Cystine is crucial for hair strength, with its levels considered a surrogate measure for hair fragility.[147] Reduced levels of cystine have been associated with genetic disorders characterized by significantly in hair that is fragile, brittle, and fails to grow long.[148-149] Many nutraceuticals contain cysteine rather than cystine, as it is better absorbed than any other cysteine product. Oral administration of L-cystine, alone or in combination with other active ingredients, increases hair density and anagen rate.[150-155] Silicon prevents the loss of hair tensile strength suggesting that it has a structural effect on hair fibers.[156] Adenosine improves hair loss by stimulating hair growth and by thickening hair shafts. Finally, methylsulfonylmethane (MSM) is a natural and highly absorbable source of sulfur necessary for the formation of keratin in nails, skin, and hair. MSM is a supplement recognized as safe (GRAS status) by the FDA for which small-scale studies have suggested benefits, particularly for treatment of skin, nail and hair conditions.[162-163]

Insulin-like growth factor-I

Insulin-like growth factor-I (IGF-I) is a growth factor critically involved in promoting hair growth by regulating cellular proliferation and migration during the development of hair follicles.[164-165] Low circulating IGF-1 level is associated with hair loss.[166-167] To exert its biological effects, IGF-I must activate cells by binding to specific cell-surface receptors.[168] The type I IGF receptor (IGF1R) is the only IGF receptor to have IGF-mediated signalling functions.[169] rs2229765 IGF1R polymorphism (level 1B) provides an indication to healthcare providers about potential Insulin-like growth factor-I (IGF-I) levels and the need to treat with API increasing IGF-1 levels (Igrantine-F1 and TrichoXidil): Patients carrying at least one A allele have lower free plasma IGF-1 levels.[170-172] Cepharanthine is a natural product extracted from Stephania cepharantha with antiinflammatory properties. It is an approved drug used for more than 70 years in Japan to treat a variety of acute and chronic diseases.[173] Cepharanthine stimulates hair growth by increasing the production of IGF-1.[174] Igrantine-F1 (Fagron) is a substance isolated from Stephania cepharantha's dry extract containing at least 98% cepharanthin. TrichoXidil™ is a phytocomplex with specific fractions of vegetable oils. Treatment with TrichoXidil ™ in the vehicle TrichoSol ™ promoted a reduction in 90 days of 37% of follicles in telogen phase and an increase of 29% of follicles in anagen phase. It significantly increased expression of growth factors KGF, IGF-1 and VEGF compared to controls (a Fagron clinical study).

Metabolism of vitamins and minerals

Deficiency of essential nutrients and vitamins may represent a modifiable risk factor associated with the development, prevention, and treatment of alopecia.[23, 175]

Vitamin A deficiency results in ichthyosis-like skin changes and sometimes causes telogen effluvium and the fragility of the hair. Vitamin C intake is crucial in patients with hair loss associated with iron deficiency. Vitamin A and its derivatives (retinoids) are critically important in the development and maintenance of multiple epithelial tissues, including skin, hair, and sebaceous glands, as shown by the detrimental effects of either vitamin A deficiency or toxicity.[176,177] CRABP2, a cytosolic protein, moves to the nucleus upon binding of retinoic acid (RA) and thus is responsible for RA intracellular transport.[178-180] rs12724719 CRABP2 AA (level 1B) is associated with higher RA concentration in blood and lower intracellular transport.[181-182] rs12724719 CRABP2 polymorphism provides an indication to healthcare providers about predisposition to inefficient intracellular transport of retinoic acid and the need to supplement with vitamin A derivatives, tocopherol and zinc. Retinol is one of the two forms of vitamin A available in the human diet. It has been suggested that vitamin A regulates both the hair cycle and immune response to alter the progression of AA.[176,183] Tretinoin, also known as all-trans retinoic acid, is a vitamin A derivative known to increase the percutaneous absorption of minoxidil and, therefore, to enhance the response of AGA to minoxidil.[184-185] The combined preparation minoxidil/tretinoin has been shown to be as safe as conventional minoxidil.[59,186] Tocopherol exerts an antioxidant action via the prevention of lipid peroxidation, similar to retinoic acid.[187] Supplementation with vitamin E has been shown to be beneficial for hair conditions.[188]

Concomitant vitamin A and Zinc supplementation have been used for many years in the treatment of infammatory skin diseases[194] based on the observation that Zinc and vitamin A work in synergy for many functions in the body.[195] Zinc sulphate is also a natural 5α-reductase inhibitor[103,105] that has been shown to be an effective treatment option for hair loss treatment.[104-105]

Biotin (vitamin B7) is an important cofactor that contributes to the normal functioning of enzymes responsible for carboxylation. BTD encodes the Biotinidase enzyme that allows the body to use and to recycle biotin. Deficit of biotinidase leads to low biotin levels, which can cause hair loss skin rashes and brittle nails.[23] rs13078881 (also known as Asp444His or D444H) C allele in the Biotinidase (BTD) gene causes partial biotinidase deficiency.[189] rs13078881 BTD polymorphism (level 1B) provides an indication to healthcare providers about potential biotin (vitamin B7) deficiency[190] and the need to supplement with biotin and other forms or vitamin B. Biotin supplementation has been shown to be beneficial to hair improvement in cases of inherited biotin deficiency. Topical biotin (see biotin) is often included in hair loss treatment formulas.[191-192]

Clincial Experience and Validation

Extensive post-market surveillance data gathered over more than three years of clinical experience have been used to assess the safety of Roots by Genetics Arts process. The data includes information from **over 13,400 patients** who have been managed by health professionals using the test. Customer **satisfaction has been high, with a rating of 4.5 out of 5**, and no clinically relevant incidents have been reported.

The accumulated post-market data indicates that Roots by Genetics Arts does not pose an increased risk to the user. User experience reports have also been positive. Dr. Kuka and Dr. Epstein, renowned experts in regenerative medicine and hair transplantation, shared their opinion on the test.[195] They highlighted that patients appreciate having a definitive answer about which treatment is more likely to be effective for their hair loss, avoiding the expenses and waiting time for ineffective treatments.[195] Patients who have tried other treatments in the past or experienced side effects are particularly motivated to find a suitable solution. The genetic profile provided by Roots by Genetics Arts instills confidence in the proposed therapy.

Roots by Genetics Arts considers around 70 active pharmaceutical ingredients

(APIs) that can be compounded for the treatment of hair loss. These APIs possess anti-inflammatory, anti-androgen, or anti-oxidant properties. Some are used to improve absorption, while others are included to facilitate the delivery of the compounded lotion. Vitamins, minerals, and zinc sulfate also play a significant role in hair metabolism. The medication dosage varies depending on factors such as sulfotransferase activity. The test helps avoid unnecessary treatments by identifying redundant therapies based on the patient's genetic profile, reducing the risk of side effects without affecting the efficacy of the treatment.

6 benefits of testing

Roots by Genetic Arts is the first genetic test on the market to predict response to alopecia treatments.

01 **Personalizing formulations and dosages**

Roots by GA analyses 16 polymorphisms associated with treatment efficacy and combines this information with relevant clinical data to select the most effective and safest active pharmaceutical ingredients. Roots by GA can include over 70 active pharmaceutical ingredients commonly used in clinical practice. Dosages have been prepared by industrial pharmacists and reviewed by dermatologists.

02 **Minimizing the risks of intolerance or contraindications**

The patient questionnaire of Roots by GA has been elaborated by dermatologists and pharmacists to minimize risks of intolerance or contraindications.

03 **Test accuracy**

A retrospective case study showed that the test was accurate when selecting or discouraging the use of topical minoxidil, and when selecting dutasteride over finasteride. Roots by GA also asserted predicting corticosteroid response and suggesting alternative treatment when a risk of hypertension was detected.

04 **Satisfaction**

The high degree of customer satisfaction, and the very low level of clinically relevant complaints indicate that the clinical base of Roots by GA is well-founded, and outputs (genotype descriptions and personalized treatments) well accepted by healthcare professionals and patients.

05

Saving time and money

Most alopecia treatment should be maintained at least 4-to-6 months before noticing improvement. Roots by GA saves patients time and money avoiding treatments with a low response rate.

06

Clinical safety and performance

The analysis of risk management, scientific literature review, clinical experience, and postmarket experience with the evaluated test confirms the clinical safety and performance of Roots by GA. The test shows conformity by achieving the intended performance under normal conditions of use when weighting known or foreseeable risks and adverse events against the benefits of the intended performance.

Hair loss is optional, with the only solution optimized for YOU.

Avoid the trial-and-error approach, and get to the root of your hair growth.

RootsbyGA.com

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