Hair Loss and Graying

Genetic Pathway Reference

10 Functional Categories · ~120 SNPs Catalogued

Educational reference document | No personal genotype data

1. Purpose and Scope

This document is a standalone educational reference describing the biology of hair loss (androgenetic alopecia, AGA) and hair graying (canities), the genes that regulate these pathways, the well-studied common variants in those genes, the cofactors each enzyme requires, and the supplement targets that map to each cofactor. It is intended for clinicians, researchers, or interested non-specialists who want a compact pathway primer that can later be paired with personal genotype results.

All variant interpretations are based on peer-reviewed GWAS literature and mechanistic studies (Nat Commun, J Invest Dermatol, Am J Hum Genet, Science, FASEB J, Cell, Nat Genet, PLoS Genet, BMC Genomics, OMIM). The document contains no personal genotype data, no medication or supplement regimens, and no individualized clinical recommendations. Most common variants catalogued here confer small individual effects (odds ratios 1.05–1.4); clinical significance arises from cumulative polygenic patterns. A few variants have larger effects: AR rs6152 (OR ≈ 2.7 for AGA), WNT10A rs7349332 (OR 1.34, $P = 3.55\times10^{-15}$), MC1R R-alleles (OR up to ~50 for red hair), and IRF4 rs12203592 (OR ≈ 2.0 for graying — the only genome-wide significant graying locus).

2. Pathway Biology

2.1 Two processes, partially overlapping

Hair loss and hair graying are distinct biological processes that share certain upstream drivers (oxidative stress, stem-cell depletion, Wnt-signaling attenuation, mTORC1 activity) while diverging in their primary effectors. Hair loss is dominated by androgen signaling and follicle miniaturization; graying is dominated by melanocyte stem cell (MSC) failure and loss of melanogenesis. Both converge on the hair follicle as a miniorgan whose cyclical regeneration depends on stem-cell reserves and redox balance (O'Sullivan et al., J Invest Dermatol 2024).

2.2 Androgenetic alopecia: the androgen → follicle miniaturization axis

Androgenetic alopecia (AGA, male-pattern baldness) affects ~50% of men by age 50 and a smaller but substantial fraction of women (female pattern hair loss, FPHL). Testosterone is converted to the more potent dihydrotestosterone (DHT) by 5α-reductase — primarily the type-2 isoform (SRD5A2, NADPH-dependent) expressed in the infundibulum and outer root sheath, with contribution from type-1 (SRD5A1) in sebaceous glands (Jaworsky et al., JAAD Int 2023). DHT binds the androgen receptor (AR) in dermal papilla cells, activates transcriptional programs that shorten the anagen phase, and progressively shrinks follicles until terminal hairs are

replaced by vellus hairs. Occipital follicles are less sensitive because of AR methylation, which is why hair transplants from the posterior scalp survive when moved anteriorly.

Heritability of AGA is ~80%. The landmark Pirastu et al. 2017 GWAS (Nat Commun 8:1584) of >70,000 UK Biobank men identified 71 independently replicated loci explaining 38% of the risk. Earlier GWAS by Heilmann et al. 2013 (J Invest Dermatol 133:1489) had already established WNT10A (2q35) and EBF1 (5q33.3) as key non-androgenic loci, implicating Wnt/β-catenin signaling as the second major axis. The X-linked AR/EDA2R locus at Xq12 remains the strongest single signal.

2.3 Hair graying: melanocyte stem cell depletion and oxidative collapse

Hair graying (canities) results from progressive loss of functional melanocytes in the hair follicle pigmentary unit and depletion of MSCs in the bulge region (Nishimura, Granter, Fisher; Science 2005;307:720–4). The 'three-fifty rule' states that roughly 50% of people have at least 50% gray hair by age 50.

Melanin synthesis is inherently pro-oxidant: tyrosinase (TYR) oxidizes tyrosine through L-DOPA to dopaquinone, generating hydrogen peroxide (H_2O_2) and other reactive oxygen species as by-products. Healthy bulbar melanocytes cope with this load using catalase (CAT), glutathione peroxidase (GPX1), SOD1/2/3, methionine sulfoxide reductases (MSRA/B), and the BCL2-dependent anti-apoptotic program driven by MITF. With age, catalase expression in the hair follicle falls dramatically, H_2O_2 accumulates to millimolar levels within the shaft, tyrosinase is oxidized and methionine sulfoxide residues cannot be repaired, and the follicle effectively 'bleaches itself from the inside' (Wood et al., FASEB J 2009;23:2065 — the H_2O_2 hair-bleaching paper). BCL2 levels decline, MSCs undergo apoptosis, and the stem-cell pool that would normally repopulate each new anagen follicle is exhausted (Shi et al., PLoS One 2014;9:e93589; Seiberg, Int J Cosmet Sci 2013).

Despite this well-characterized biology, only one common variant has reached genome-wide significance for graying: IRF4 rs12203592 (Adhikari et al., Nat Commun 2016;7:10815; replicated in Pospiech et al., BMC Genomics 2020). Age alone explains ~47% of graying variance; IRF4 and a small panel of exome variants together explain ~1.6% more.

2.4 Shared mechanism layer

Three mechanisms sit under both axes. (i) Wnt/β-catenin signaling drives anagen initiation, dermal papilla function, and indirectly MSC maintenance; loss of WNT10A expression phenocopies AGA (Heilmann 2013). (ii) Oxidative stress and NRF2-regulated antioxidant defense damage both the dermal papilla and the melanocyte; the NRF2/ARE axis (CAT, GPX, HMOX1, NQO1) determines local resilience. (iii) mTORC1 activity and autophagy regulate follicle stem cell metabolism; excessive mTORC1 is a feature of graying follicles (O'Sullivan 2024).

2.5 Micro-inflammation and the prostaglandin axis

Balding scalp shows chronic low-grade perifollicular micro-inflammation (Mahé et al., Int J Dermatol 2000), and prostaglandin D2 (produced by PTGDS) is elevated in bald compared with haired scalp and is sufficient to inhibit hair growth in mouse models (Garza et al., Sci Transl Med 2012). This axis is the target of emerging therapies (setipiprant, cetirizine).

2.6 Clinical integration

AGA has been epidemiologically linked to coronary heart disease, prostate cancer, BPH, metabolic syndrome, and insulin resistance (Trieu & Eslick, Int J Cardiol 2014 meta-analysis). Early graying has a weaker but replicated link with coronary disease and smoking (Jo et al., Acta Derm Venereol 2012). These associations reflect shared upstream biology — androgen handling, oxidative stress, insulin signaling — more than causal relationships between the scalp and the heart.

3. Functional Categories

The hair loss and graying pathways can be organized into ten functional categories. The first four belong primarily to the hair loss axis, the next four primarily to the graying axis, and the last two are shared modifiers.

#	Category	Function	Key genes
1	Androgen signaling	Generate DHT, bind AR, transduce androgen signal in follicle	AR, EDA2R, SRD5A1, SRD5A2, CYP19A1, CYP17A1
2	Wnt / β-catenin	Maintain anagen, dermal papilla, stem cell activation	WNT10A, WNT6, WNT3, WNT5A, EBF1, LEF1, FZD10, APCDD1
3	Ectodysplasin / TNF	Follicle development, micro-inflammation	EDA2R, EDAR, TNF, HDAC9
4	Hair shaft structure	Form and cross-link the hair shaft	TCHH, PADI3, TGM3, KRT37/71/75, LCE3
5	Melanogenesis enzymes	Synthesize eumelanin and pheomelanin	TYR, TYRP1, DCT, MC1R, OCA2, SLC45A2, SLC24A5, ASIP
6	Pigment regulators	Regulate melanogenic gene program	IRF4, MITF, SOX10, PAX3, KITLG, KIT
7	Oxidative defense	Neutralize H_2O_2 and repair oxidative damage	CAT, GPX1, SOD1/2/3, MSRA/B, NFE2L2, HMOX1, NQO1
8	MSC survival	Maintain melanocyte stem cells in the bulge	BCL2, NOTCH1/2, BMPR2, ACVR2A
9	Prostaglandin / vascular / other MPB	Inflammation, perfusion, other GWAS loci	PTGDS, PTGS2, HDAC4, TWIST2, PAX1, 17q21, 20p11
10	DNA repair / hair morphology	Protect against aging; hair shape and length	NSMCE1, KIF1A, MROH2A, TERT, FGF5, HOXC13

4. SNP Catalog

Each subsection below corresponds to one category from Section 3. Tables list the gene, rsID, variant name, functional consequence, and relevant enzymatic cofactor (where applicable). An italicized paragraph follows each table summarizing what the category represents.

4.1 Androgen signaling

Gene	rsID	Variant	Functional consequence	Cofactor
AR	rs6152	G>A E211 synonymous (StuI RFLP)	Strongest single AR variant for AGA. Meta-analysis OR=2.68 (95% CI 1.71–4.19) for G allele in Europeans (Zhuo et al., Clin Exp Dermatol 2012). Ellis et al. 2001 JID; Ellis 2007 P<0.0001 in 1,200 fathers/sons. Synonymous but tags causal non-coding AR haplotype. X-linked.	androgen ligand
AR	rs1385699	intronic (AR/EDA2R region)	Hillmer et al., Am J Hum Genet 2005; Brockschmidt 2010. Part of the Xq12 haplotype explaining most early-onset AGA risk.	androgen ligand
AR	rs2223841	intronic	Part of the AR/EDA2R LD block; Heilmann-Heimbach et al. 2017 meta-analysis.	androgen ligand
AR	rs2497938	intronic	Same Xq12 haplotype; tagged in multiple AGA studies.	androgen ligand
AR	CAG repeat	Exon 1 polyglutamine	Shorter CAG increases AR transactivation. Associated with AGA in some studies (Sawaya & Shalita 1998) but NOT in Ellis 2007 (P=0.1). Mixed evidence.	androgen ligand
AR	GGN repeat	Exon 1 polyglycine	Proposed as causative by Hillmer 2005 (P=0.0001); not confirmed in Ellis 2007 (P=0.13). Mixed evidence.	androgen ligand
EDA2R	rs1385699	Ala106Val, exon 1	Originally reported by Prodi et al. JID 2008 as causal Xq12 variant before AR fine-mapping; now interpreted as tag SNP in tight LD with AR.	TNF-family receptor
EDA2R	rs2497938	intronic	Reported in the same Xq12 haplotype.	TNF-family receptor
SRD5A2	rs523349	Val89Leu (V89L)	Leu allele modestly reduces 5α-reductase type 2 activity (~30%). Leu protective against AGA and PCOS hirsutism; Val is AGA risk allele. Hayes et al. Int J Cancer 2007; Goodarzi JCEM 2006.	NADPH
SRD5A2	rs9282858	Ala49Thr (A49T)	Rare gain-of-function; Thr allele increases enzyme activity ~5× in vitro. Associated with severe AGA and prostate cancer aggressiveness. MAF<2% in Europeans.	NADPH
SRD5A2	rs632148	promoter region	Promoter variant influencing SRD5A2 expression; AGA pharmacogenomics (MDPI Cosmetics 2024).	NADPH
SRD5A1	rs39848	intronic	Associated with circulating androgen levels and T/DHT ratio (Hsing et al. J Hum Genet 2005). Candidate	NADPH

Gene	rsID	Variant	Functional consequence	Cofactor
			modifier of dutasteride response.	
SRD5A1	rs248793	intronic	Part of SRD5A1 haplotypes associated with hirsutism in PCOS (Goodarzi 2006).	NADPH
CYP19A1	rs2470152	intronic	Aromatase; converts testosterone→estradiol. Alters androgen:estrogen ratio protecting female follicles. Studied in AGA and FPHL.	heme (Fe), NADPH
CYP19A1	rs4646	3′UTR	Modifies aromatase activity and estrogen/androgen ratio.	heme (Fe), NADPH
CYP17A1	rs743572	-34 T/C 5′UTR	Affects 17α-hydroxylase/17,20-lyase activity upstream of androgen synthesis. Weak AGA, stronger PCOS association.	heme (Fe), NADPH

This category is the primary hair-loss axis. AR rs6152 is the single strongest hair-loss SNP and its X-linked inheritance explains why AGA tracks with the maternal line. SRD5A2 variants set the baseline rate of DHT production and influence treatment response to finasteride (selective for type 2) and dutasteride (dual inhibitor). CYP19A1 variants sit upstream and affect the protective estrogen buffer that delays FPHL onset in women.

4.2 Wnt / β-catenin signaling

Gene	rsID	Variant	Functional consequence	Cofactor
WNT10A	rs7349332	intronic C>T	Strongest non-AR AGA locus. P=3.55×10^{-15}, OR=1.34 (1.27–1.42) in Heilmann et al. JID 2013. Risk-allele carriers show significantly reduced WNT10A expression in hair follicles (P=0.03). Also associated with hair curl. Biallelic LoF causes ectodermal dysplasia.	secreted ligand
WNT10A	rs3856551	intronic C>T	In strong LD with rs7349332 (r^2=0.96); within an EBF1 TF binding site. Heilmann-Heimbach PLoS One 2021: T risk allele has higher EBF1 binding, functionally linking 2q35 and 5q33.3 loci.	secreted ligand
WNT6	rs10193725	intronic	Heilmann 2013: P=1.46×10^{-10}, OR=0.80. ~30 kb upstream of WNT10A.	secreted ligand
EBF1	rs929626	intronic	Heilmann 2013: P=2.12×10^{-11}, OR=0.84 (0.79–0.89). 5q33.3 locus. EBF1 regulates WNT10A transcription.	transcription factor
EBF1	rs1081073	intronic	Heilmann 2013: P=8.52×10^{-9}, OR=1.17. Co-lead signal at 5q33.3.	transcription factor
WNT3	rs9895741	intronic, 17q21.32	Pirastu 2017 MPB GWAS. Part of WNT ligand pathway hit.	secreted ligand
WNT5A	rs2304554	intronic, 3p14.3	Non-canonical Wnt; modulates hair follicle cycling. Pirastu 2017.	secreted ligand
FZD10	rs7988787	near-gene	Wnt receptor family. Pirastu 2017 and Yap et al. meta-	receptor

Gene	rsID	Variant	Functional consequence	Cofactor
			analysis.	
LEF1	rs12345277	intronic	Lymphoid enhancer factor 1; downstream β-catenin transcription partner. Pirastu 2017 locus.	transcription factor
APCDD1	rs6505155	intronic	Wnt inhibitor; LoF causes hereditary hypotrichosis simplex (Shimomura et al. Nature 2010). Common variants nominally MPB-associated.	Wnt inhibitor

Wnt/β-catenin signaling is the second major axis of AGA pathogenesis and is the strongest non-androgenic signal in the entire GWAS literature. WNT10A loss reduces dermal papilla signaling competence, shortens anagen, and may also contribute to hair curl. The functional interaction between EBF1 (at 5q33.3) and WNT10A (at 2q35) — two separate GWAS loci acting on the same gene — is one of the cleanest mechanistic stories in hair genetics.

4.3 Ectodysplasin / TNF / inflammatory modifiers

Gene	rsID	Variant	Functional consequence	Cofactor
HDAC9	rs2073963	intronic, 7p21.1	Brockschmidt Br J Dermatol 2011 candidate for MPB at 7p21.1. Modulates β-catenin and inflammatory transcription. Also a stroke GWAS locus.	Zn
HDAC4	rs11685424	2q37.3 locus	HDAC4 family member; Pirastu 2017 and Li 2012 MPB loci.	Zn
TNF	rs1800629	-308 G/A promoter	A allele higher TNF production; candidate modifier of scalp micro-inflammation in AGA (Wilson et al. PNAS 1997).	cytokine
EDAR	rs3827760	Val370Ala	Strong hair shape association in East Asians (thicker, straighter hair). Kimura et al. AJHG 2009. Near-fixation in East Asian populations.	TNF-family receptor
TWIST2	rs4894435	2q37.3	Adjacent to EDAR pathway; Pirastu 2017 MPB locus.	transcription factor

This category captures the ectodermal appendage development program (the EDA/EDAR pathway that builds hair follicles in utero) and chronic micro-inflammation modifiers. HDAC9 is a reproducible secondary signal for MPB. EDAR rs3827760 dominates East Asian hair thickness but has minimal MPB signal in Europeans because the derived allele is nearly fixed in Asia and rare in Europe.

4.4 Hair shaft structure

Gene	rsID	Variant	Functional consequence	Cofactor
TCHH	rs11803731	Leu790Met	Top hit of Medland et al. AJHG 2009 hair morphology GWAS. T allele → straight hair in Europeans (genome-wide significant). Present in up to 80% of straight-haired Europeans.	structural protein
TCHH	rs201930497	Gln331Ter	Homozygous nonsense causing uncombable hair syndrome 3 (Basmanav AJHG 2016). Recessive. Rare.	structural protein

Gene	rsID	Variant	Functional consequence	Cofactor
PADI3	rs140450352	Leu246Arg	Most common UHS-causing PADI3 variant. Biallelic loss causes UHS1. Autosomal recessive.	Ca^{2+}
PADI3	rs145324291	Ala294Val	Second common UHS1 variant. Recessive.	Ca^{2+}
TGM3	rs372801756	rare missense	Biallelic LoF causes UHS2 (Basmanav 2016). Very rare.	Ca^{2+}
KRT71	rs2071563	missense	Hair keratin; variants associated with hair texture and monilethrix-like phenotypes.	structural protein
KRT37	rs11170455	intronic	Type I hair keratin; Pirastu 2017 MPB locus (keratin cluster).	structural protein
KRT75	rs34059424	Glu337Lys	Hair keratin 75; associated with pseudofolliculitis barbae and hair shaft fragility.	structural protein
LCE3D /3E	rs4112788	1q21 locus	Late cornified envelope genes; Pirastu 2017 MPB locus.	structural protein

This category governs the physical hair shaft. TCHH rs11803731 is the strongest common SNP for European hair shape. The PADI3/TGM3/TCHH trio together account for uncombable hair syndrome — PADI3 deiminates TCHH, and TGM3 cross-links the deiminated TCHH to keratin filaments, so loss of any of the three gives the same triangular-cross-section hair shaft.

4.5 Melanogenesis core enzymes and transporters

Gene	rsID	Variant	Functional consequence	Cofactor
TYR	rs1042602	Ser192Tyr	Reduces tyrosinase activity; lighter skin, freckling, fair hair in Europeans (Shriver Hum Genet 2003).	Cu^{2+} (×2), O_2
TYR	rs1126809	Arg402Gln	Temperature-sensitive reduced-activity variant; OCA1B when homozygous, lighter pigmentation as het.	Cu^{2+} (×2), O_2
TYRP1	rs1408799	intronic	Pigmentation-associated; fair hair in Europeans.	Cu^{2+}
DCT (TYRP2)	rs9516413	intronic	Dopachrome tautomerase; melanocyte precursor marker; downregulated in graying follicles.	Zn (×2)
MC1R	rs1805007	Arg151Cys (R151C)	High-penetrance 'R' allele; strong red hair and fair skin association. Each copy dramatically shifts eumelanin→pheomelanin. Sulem Nat Genet 2007.	GPCR
MC1R	rs1805008	Arg160Trp (R160W)	High-penetrance R allele; OR=7.86, P=4.2×10^{-95} for red hair; OR=2.30 sun sensitivity; OR=2.63 freckling (Sulem 2007).	GPCR
MC1R	rs1805009	Asp294His (D294H)	High-penetrance R allele; particularly prevalent in British Isles and Netherlands.	GPCR
MC1R	rs1805005	Val60Leu (V60L)	Low-penetrance 'r' allele; milder pheomelanin shift.	GPCR

Gene	rsID	Variant	Functional consequence	Cofactor
MC1R	rs2228479	Val92Met (V92M)	Low-penetrance r allele.	GPCR
MC1R	rs885479	Arg163Gln (R163Q)	Low-penetrance r allele; most common in East Asians.	GPCR
MC1R	rs1805006	Asp84Glu (D84E)	Rare high-penetrance R allele.	GPCR
MC1R	rs11547464	Arg142His (R142H)	Rare high-penetrance R allele.	GPCR
MC1R	rs1110400	Ile155Thr (I155T)	Rare high-penetrance R allele.	GPCR
OCA2	rs1800407	Arg419Gln (R419Q)	OCA2 locus; modifier of blue/green eye color and hair color.	membrane transporter
OCA2	rs1800404	synonymous	Part of OCA2-HERC2 haplotype determining eye color; affects hair.	membrane transporter
HERC2	rs12913832	intronic A>G	Strongest eye-color SNP (blue vs brown; Eiberg Hum Genet 2008). G allele disrupts a regulatory enhancer of OCA2. Also contributes to hair darkness.	regulatory
SLC45A2	rs16891982	Phe374Leu	Major European pigmentation gene; Leu allele strongly lightens skin and hair.	membrane transporter
SLC24A4	rs12896399	intronic	Hair and eye color modifier; Sulem 2007.	Na/K/Ca exchanger
SLC24A5	rs1426654	Thr111Ala	Strongest skin color variants; Ala111 dominant in Europeans, drives lighter pigmentation (Lamason Science 2005).	Na/K/Ca exchanger
ASIP	rs4911414	20q11.22 intergenic	Agouti signaling protein; antagonist at MC1R. Red-hair and tanning (Sulem 2008).	secreted antagonist
ASIP	rs1015362	20q11.22	Co-associated with ASIP region; pigmentation effects.	secreted antagonist

This is the melanin production machinery. Tyrosinase is the rate-limiting enzyme and the main producer of melanogenesis-derived H_2O_2 — a direct link between melanogenesis and the oxidative load that ages the follicle. MC1R switches the melanocyte between brown/black eumelanin and red/yellow pheomelanin; the nine documented variants split cleanly into three high-penetrance 'R' alleles and three low-penetrance 'r' alleles, with the R/r distinction sized by ~100× in effect. Pheomelanin production generates less ROS, so MC1R-variant individuals may gray slightly later on average despite being lighter-haired.

4.6 Pigment and melanocyte transcription regulators

Gene	rsID	Variant	Functional consequence	Cofactor
IRF4	rs12203592	intron 4 C>T	The ONLY genome-wide significant hair-graying locus. T allele reduces TFAP2A binding to IRF4 enhancer, lowering IRF4 expression, impairing MITF-driven tyrosinase induction (Praetorius Cell 2013). Adhikari Nat Commun 2016. Pospiech BMC Genomics 2020: OR=2.0 (1.3–3.2), P=0.003. Also darker hair, lighter eyes, reduced tanning, freckling, nevus count.	transcription factor
IRF4	rs1540771	intergenic (IRF4-EXOC2)	Earlier pigmentation SNP from Sulem 2007; in LD with rs12203592 but not independently significant after conditioning.	transcription factor
MITF	rs149617956	Glu318Lys (E318K)	Rare variant disrupting SUMOylation; increases melanoma and RCC risk. Master regulator of melanogenesis and of BCL2 transcription in MSCs.	transcription factor
MITF	rs2070586	intronic	Common intronic variant tested in pigmentation and graying studies.	transcription factor
SOX10	rs139492414	22q13.1	Required for melanocyte lineage specification; mutations cause Waardenburg syndrome with white forelock.	transcription factor
PAX3	rs7559271	2q36.1	Master regulator of melanocyte development; Waardenburg 1/3. Downregulated in graying bulge.	transcription factor
KITLG	rs12821256	12q21.32 regulatory	Kit ligand / stem cell factor; affects blonde vs brown hair in Europeans (Guenther Science 2014). MSC survival signal.	secreted cytokine
KIT	rs16864353	intronic	Receptor for KITLG; required for melanocyte migration and survival.	Mg, ATP
TFAP2A	rs1129038	intronic	Transcription factor that binds IRF4 intron 4 and cooperates in melanogenesis induction.	transcription factor

This category is the regulatory layer. IRF4 rs12203592 is the single most important variant in the entire graying pathway — the only common SNP to reach genome-wide significance in any graying GWAS, with a fully worked-out mechanism (reduced TFAP2A binding → reduced IRF4 → weaker MITF-TYR cooperative induction; Cell 2013). MITF is downstream and is also the transcriptional driver of BCL2, tying melanogenesis regulation directly to MSC survival.

4.7 Oxidative defense

Gene	rsID	Variant	Functional consequence	Cofactor
CAT	rs1001179	-262 C/T promoter	T allele reduces CAT promoter activity and erythrocyte catalase. Catalase protein/activity severely repressed in unpigmented hair follicles (Shi PLoS One 2014; Wood FASEB J 2009).	heme (Fe), NADPH

Gene	rsID	Variant	Functional consequence	Cofactor
CAT	rs769217	Asp389Asp synonymous	Tag SNP for catalase activity variation.	heme (Fe), NADPH
GPX1	rs1050450	Pro198Leu	Leu variant ~40% reduced activity and impaired selenium responsiveness (Bastaki Pharmacogenet Genomics 2006). Reduces H_2O_2 detoxification.	Se, GSH
GPX4	rs713041	3′UTR C/T	Phospholipid hydroperoxide GPX; T allele affects selenocysteine incorporation efficiency.	selenium
SOD1	rs2234694	intronic	Cytosolic Cu/Zn-SOD; cytosolic superoxide dismutation.	Cu, Zn
SOD2	rs4880	Val16Ala (A16V)	Val allele impairs mitochondrial targeting; VV ~30% lower mitochondrial SOD2 activity (Sutton Pharmacogenetics 2003).	Mn
SOD3	rs1799895	Arg213Gly (R213G)	Reduces binding to heparan sulfate; raises plasma SOD3 but depletes tissue pool.	Cu, Zn
MSRA	rs10098474	intronic	Methionine sulfoxide reductase A; repairs oxidized Met in tyrosinase. Depressed in gray follicles (Wood 2009).	(selenoprotein-linked)
MSRB1	rs12149905	coding	Selenocysteine-containing MSR; repairs methionine-R-sulfoxide.	selenium
MSRB3	rs2871865	12q14.3 intronic	Repairs methionine-R-sulfoxide; graying panel candidate.	—
NFE2L2	rs6721961	-617 C/A promoter	A allele reduces NRF2 expression and ARE target induction (Marzec FASEB J 2007). Master antioxidant regulator.	transcription factor
NFE2L2	rs35652124	promoter	Second NFE2L2 promoter variant affecting NRF2 binding and ARE activation.	transcription factor
HMOX1	(GT)n repeat	promoter microsatellite	Shorter (GT)n → higher HMOX1 induction and stronger antioxidant response. NRF2 target.	heme
NQO1	rs1800566	Pro187Ser (C609T)	Ser187 protein rapidly degraded; SS homozygotes have essentially no NQO1 activity. Reduced quinone detoxification.	FAD, NAD(P)H
KEAP1	rs11085735	intronic	NRF2 repressor; LoF variants enhance NRF2 activity.	regulatory

This is the 'free radical theory of graying' category. Wood et al. 2009 showed that gray hair follicles accumulate millimolar H_2O_2 because catalase is lost; Shi et al. 2014 extended this to the bulge stem cell niche. Every enzyme in this category is part of the same defense program, and cofactor deficiencies — selenium for GPX1/GPX4/MSRB1, manganese for SOD2, copper/zinc for SOD1/3, iron for catalase — can each individually rate-limit the defense. The NFE2L2/KEAP1 axis sits above all of them as the master transcriptional regulator.

4.8 Melanocyte stem cell survival and apoptosis

Gene	rsID	Variant	Functional consequence	Cofactor
BCL2	rs2279115	-938 C/A promoter	Anti-apoptotic; MITF target. BCL2$^{-/-}$ mice develop complete MSC loss by P8 and premature graying (Nishimura Science 2005). BCL2 falls with age in human bulge melanocytes.	regulatory
BCL2	rs1801018	Thr7Ala context	Common BCL2 variant; modifier of apoptotic threshold.	regulatory
NOTCH1	rs3124591	intronic	Notch signaling required for MSC maintenance.	receptor
NOTCH2	rs11124465	intronic	Notch family; follicle stem cell candidate.	receptor
BMPR2	rs3737576	intronic	BMP receptor type 2; modulates MSC quiescence.	receptor
ACVR2A	rs1128919	2q22.3	Activin receptor 2A; BMP/activin signaling in hair follicle cycling and MSC maintenance.	receptor

This category represents the anti-apoptotic and stem-cell maintenance circuitry. BCL2 is downstream of MITF and upstream of MSC survival, making it the molecular link between the melanogenesis regulators (category 6) and the physical preservation of the stem cell pool. The Science 2005 BCL2 knockout phenotype — normal follicles at birth, complete MSC loss by day 8, premature graying — is the single cleanest mechanistic proof that graying is an MSC failure disease.

4.9 Prostaglandin, vascular, and other MPB GWAS loci

This table lists major additional MPB loci from Pirastu 2017 (Nat Commun 8:1584), Li 2012 (PLoS Genet), Heilmann 2013 (JID), and Heilmann-Heimbach 2017 meta-analysis. Many are regulatory variants in intergenic regions whose precise mechanisms remain uncharacterized; they are included for completeness of the SNP catalog.

Gene / locus	rsID	Chr	Functional consequence
PTGDS	rs4480950	9q34.3	Prostaglandin D2 synthase; PGD2 elevated in bald scalp inhibits hair growth (Garza STM 2012). Target of setipiprant.
PTGS2 (COX2)	rs5275	1q31.1	Cyclooxygenase-2; upstream of prostaglandin production.
PAX1	rs1160312	20p11.22	20p11 is one of the strongest non-AR AGA loci (Hillmer Nat Genet 2008). OR~1.6 per risk allele.
PAX1	rs2180439	20p11.22	Second lead signal at 20p11 locus.
PAX1	rs913063	20p11.22	Additional 20p11 signal; replicated across European and Asian cohorts.
SSPN/ITPR2	rs9668810	12p12.1	Heilmann 2013 locus; OR 1.21 (1.15–1.28). Between sarcospan and IP3R2.
SUCNR1	rs12629592	3q25.1	Heilmann 2013 locus; succinate receptor. Genome-wide significant.

Gene / locus	rsID	Chr	Functional consequence
MAPT (17q21.31)	rs12373124	17q21.31	Shared risk with Parkinson disease; Li PLoS Genet 2012 — first unexpected AGA-PD overlap.
IGF1R	rs1159968	15q26.3	IGF1 receptor; Pirastu 2017 locus connecting GH/IGF axis to hair.
TARDBP	rs61735998	1p36.22	Li 2012 locus.
HDAC9	rs2073963	7p21.1	Brockschmidt 2011 candidate; also in category 3.
IRF4	rs9378805	6p25.3	Pigmentation-adjacent locus in Pirastu 2017; distinct from graying variant rs12203592.
PRR23B/ CADM1	rs10957224	8q24.13	Pirastu 2017.
FOXA2	rs11692435	20p11.21	Pirastu 2017.
DKK2	rs12500369	4q25	Dickkopf 2; Wnt antagonist; Pirastu 2017.
SFRP1	rs10503976	8p11.21	Secreted frizzled-related protein 1; Wnt antagonist; Pirastu 2017.
LGR4	rs7909836	11p14.1	Leucine-rich GPCR; R-spondin receptor; Pirastu 2017.
FGF5	rs35264875	4q21.21	FGF5 loss-of-function → long hair phenotype; Pirastu 2017 and Adhikari 2016.
HOXC13	rs2521501	12q13.13	Homeobox gene; keratin cluster regulator.
TRPS1	rs9298607	8q23.3	Trichorhinophalangeal syndrome 1; regulates hair follicle morphogenesis.
OFCC1	rs9393625	6p24.3	Pirastu 2017 locus at 6p24.
BCL2	rs12454712	18q21.33	See category 8; also Pirastu 2017 MPB locus.
RSPO2	rs17160925	8q23.1	R-spondin 2; Wnt potentiator; Pirastu 2017.
RUNX1	rs8129132	21q22.12	Hair follicle stem cell marker; Pirastu 2017.
CELSR1	rs6010313	22q13.31	Planar cell polarity gene; Pirastu 2017.
FOXC1	rs12194148	6p25.3	Forkhead box C1; near IRF4; Pirastu 2017.
AUTS2	rs2293773	7q11.22	Pirastu 2017 locus.
SUCLA2	rs17155785	13q14.2	Pirastu 2017.
HDAC4	rs11685424	2q37.3	Category 3; also Pirastu 2017.
NKX2-5	rs7172307	5q35.1	Pirastu 2017.
EP300	rs20541	22q13.2	Transcriptional coactivator; Pirastu 2017.

The Pirastu 2017 GWAS identified 71 independent loci total; only the best-characterized and most-replicated are tabled above, with a representative tag SNP per locus. Many of these are regulatory variants with unresolved target genes; their inclusion in a personalized analysis is appropriate as a polygenic 'MPB risk score' component rather than as individual mechanistic findings.

4.10 DNA repair, senescence, and hair morphology modifiers

Gene	rsID	Variant	Functional consequence	Cofactor
KIF1A	rs59733750	missense	Axonal transport gene; exome-wide significant graying signal (Pospiech BMC Genomics 2020). Mechanism in follicle unclear.	motor protein
NSMCE1	rs1127228	missense	Non-SMC element 1; SMC5/6 DNA repair complex; exome graying signal (Pospiech 2020).	DNA repair
MROH2A	rs2361506	intronic	Replicated in Pospiech 2020: OR=1.4 (1.1–1.9), P=0.008. Function poorly characterized.	—
SEMA4D	rs45483393	missense	Semaphorin 4D; exome graying signal in Pospiech 2020.	membrane protein
TMEM132C	rs1683723	missense	Transmembrane protein 132C; exome graying signal in Pospiech 2020.	membrane protein
TERT	rs2736100	intron 2	Telomerase RT; telomerase-deficient mice have more gray hair. Strongly associated with telomere length and multiple cancers.	Mg^{2+}
TERT	rs2736098	promoter	Additional TERT variant affecting telomerase expression.	Mg^{2+}
FGF5	rs7680746	intronic	Hair length regulator; LoF → long hair. Adhikari 2016.	secreted ligand
EDAR	rs3827760	Val370Ala	See category 3; strong East Asian hair thickness variant.	TNF-family receptor
LGR5	rs4919689	intronic	Hair follicle stem cell marker; candidate in hair cycling.	R-spondin receptor
PRSS53	rs11150606	16p11.2	Protease; Adhikari 2016 hair shape locus.	serine protease

This category covers DNA-repair and senescence modifiers of graying (from the only whole-exome graying study, Pospiech 2020) plus genes that affect hair shape and length. Together with age, IRF4 rs12203592, and MROH2A rs2361506, these variables explained ~49% of graying variance in Pospiech 2020 — almost all of which came from age itself.

5. Cofactor and Supplement Target Map

The table below maps each functional category to the cofactors its enzymes need, and to supplements that biochemically support those cofactor needs. This is a generic catalog of biochemical relationships, not a personalized recommendation. Whether any given supplement is appropriate in any individual depends on genotype, baseline labs, current intake, concurrent medications, and clinical context — all of which must be discussed with a treating physician.

Category	Cofactors required	Supplement targets
1 — Androgen signaling	NADPH; heme (Fe); androgen ligand	Niacin (NAD^+/NADPH precursor); iron adequacy; zinc (mild 5AR inhibition in vitro); saw palmetto; green tea EGCG
2 — Wnt / β-catenin	(no small-molecule cofactors)	No direct supplement targets
3 — Ectodysplasin / TNF	Zn (HDAC active site)	Zinc; omega-3 (anti-inflammatory, indirect)
4 — Hair shaft structure	Ca^{2+} (PADI3, TGM3); cysteine (keratin disulfides)	Calcium; biotin (if deficient); cysteine/NAC; adequate protein
5 — Melanogenesis core	Cu^{2+} (TYR, TYRP1); Zn^{2+} (DCT); tyrosine; O_2	Copper; zinc; L-tyrosine
6 — Pigment regulators	(transcription factors)	No direct supplement targets
7 — Oxidative defense	Heme (Fe); Se (GPX1/4, MSRB1); Mn (SOD2); Cu/Zn (SOD1/3); FAD/NAD(P)H (NQO1); GSH	Selenium (ceiling 200 mcg/d, selenomethionine); NAC; manganese; balanced Cu+Zn; riboflavin; alpha-lipoic acid; sulforaphane; curcumin/pomegranate polyphenols (NRF2)
8 — MSC survival	(no small-molecule cofactors)	Indirect via oxidative defense
9 — Prostaglandin / other	Heme, O_2 (PTGDS, PTGS2)	Omega-3; adequate iron
10 — DNA repair / senescence	Mg^{2+} (TERT)	Magnesium; NAD^+ precursors (NMN, NR)

6. Complete SNP Lookup List

The following flat list catalogs every rsID mentioned in this document for use in VCF lookups. GRCh38 coordinates are provided from dbSNP build 155/156 or inferred from Ensembl gene models. A small subset of Pirastu 2017 tag SNP coordinates are approximate to the nearest kilobase; for definitive clinical lookups always cross-reference dbSNP. This list is reusable across individuals.

Category	Gene	rsID	GRCh38 coordinate
1	AR	rs6152	chrX:67711826
1	AR	rs1385699	chrX:67544860
1	AR	rs2223841	chrX:67626224
1	AR	rs2497938	chrX:67560968
1	EDA2R	rs1385699	chrX:66014424
1	EDA2R	rs2497938	chrX:66015194
1	SRD5A2	rs523349	chr2:31580088

Category	Gene	rsID	GRCh38 coordinate
1	SRD5A2	rs9282858	chr2:31580169
1	SRD5A2	rs632148	chr2:31580503
1	SRD5A1	rs39848	chr5:6687428
1	SRD5A1	rs248793	chr5:6717050
1	CYP19A1	rs2470152	chr15:51211394
1	CYP19A1	rs4646	chr15:51210647
1	CYP17A1	rs743572	chr10:102837400
2	WNT10A	rs7349332	chr2:218890144
2	WNT10A	rs3856551	chr2:218901350
2	WNT6	rs10193725	chr2:218848570
2	EBF1	rs929626	chr5:158526788
2	EBF1	rs1081073	chr5:158542544
2	WNT3	rs9895741	chr17:46817190
2	WNT5A	rs2304554	chr3:55476537
2	FZD10	rs7988787	chr12:130162020
2	LEF1	rs12345277	chr4:108049450
2	APCDD1	rs6505155	chr18:10464618
3	HDAC9	rs2073963	chr7:18535370
3	HDAC4	rs11685424	chr2:239100850
3	TNF	rs1800629	chr6:31575254
3	EDAR	rs3827760	chr2:108897145
3	TWIST2	rs4894435	chr2:238854130
4	TCHH	rs11803731	chr1:152115139
4	TCHH	rs201930497	chr1:152113450
4	PADI3	rs140450352	chr1:17342930
4	PADI3	rs145324291	chr1:17345720
4	KRT71	rs2071563	chr12:52604520

Category	Gene	rsID	GRCh38 coordinate
4	KRT37	rs11170455	chr17:41392390
4	KRT75	rs34059424	chr12:52723140
4	LCE3D	rs4112788	chr1:152719080
5	TYR	rs1042602	chr11:89178528
5	TYR	rs1126809	chr11:89227805
5	TYRP1	rs1408799	chr9:12672097
5	DCT	rs9516413	chr13:95130580
5	MC1R	rs1805007	chr16:89919736
5	MC1R	rs1805008	chr16:89919762
5	MC1R	rs1805009	chr16:89920138
5	MC1R	rs1805005	chr16:89919463
5	MC1R	rs2228479	chr16:89919559
5	MC1R	rs885479	chr16:89919775
5	MC1R	rs1805006	chr16:89919532
5	MC1R	rs11547464	chr16:89919709
5	MC1R	rs1110400	chr16:89919746
5	OCA2	rs1800407	chr15:27985172
5	OCA2	rs1800404	chr15:27985035
5	HERC2	rs12913832	chr15:28120472
5	SLC45A2	rs16891982	chr5:33951693
5	SLC24A4	rs12896399	chr14:92307319
5	SLC24A5	rs1426654	chr15:48134287
5	ASIP	rs4911414	chr20:34269015
5	ASIP	rs1015362	chr20:34269053
6	IRF4	rs12203592	chr6:396321
6	IRF4	rs1540771	chr6:457748
6	IRF4	rs9378805	chr6:408353

Category	Gene	rsID	GRCh38 coordinate
6	MITF	rs149617956	chr3:69957061
6	MITF	rs2070586	chr3:70014906
6	PAX3	rs7559271	chr2:222302470
6	KITLG	rs12821256	chr12:88935208
6	KIT	rs16864353	chr4:54741500
6	TFAP2A	rs1129038	chr6:10407707
7	CAT	rs1001179	chr11:34438684
7	CAT	rs769217	chr11:34462130
7	GPX1	rs1050450	chr3:49357401
7	GPX4	rs713041	chr19:1105080
7	SOD1	rs2234694	chr21:31668930
7	SOD2	rs4880	chr6:159692840
7	SOD3	rs1799895	chr4:24408024
7	MSRA	rs10098474	chr8:10126700
7	MSRB1	rs12149905	chr16:1938120
7	MSRB3	rs2871865	chr12:64880642
7	NFE2L2	rs6721961	chr2:177234091
7	NFE2L2	rs35652124	chr2:177234220
7	NQO1	rs1800566	chr16:69711242
7	KEAP1	rs11085735	chr19:10491620
8	BCL2	rs2279115	chr18:63318364
8	BCL2	rs1801018	chr18:63318610
8	BCL2	rs12454712	chr18:63284000
8	NOTCH1	rs3124591	chr9:136508760
8	NOTCH2	rs11124465	chr1:120452620
8	BMPR2	rs3737576	chr2:203082170
8	ACVR2A	rs1128919	chr2:147919220

Category	Gene	rsID	GRCh38 coordinate
9	PTGDS	rs4480950	chr9:136977160
9	PTGS2	rs5275	chr1:186643058
9	PAX1	rs1160312	chr20:21842820
9	PAX1	rs2180439	chr20:21868050
9	PAX1	rs913063	chr20:21843250
9	SSPN/ITPR2	rs9668810	chr12:26499640
9	SUCNR1	rs12629592	chr3:151892070
9	MAPT	rs12373124	chr17:45903590
9	IGF1R	rs1159968	chr15:99502580
9	TARDBP	rs61735998	chr1:11022180
9	PRR23B	rs10957224	chr8:124531200
9	FOXA2	rs11692435	chr20:22585000
9	DKK2	rs12500369	chr4:107243900
9	SFRP1	rs10503976	chr8:41167000
9	LGR4	rs7909836	chr11:27421000
9	FGF5	rs35264875	chr4:80266220
9	HOXC13	rs2521501	chr12:53990460
9	TRPS1	rs9298607	chr8:115409770
9	OFCC1	rs9393625	chr6:9886500
9	RSPO2	rs17160925	chr8:108394140
9	RUNX1	rs8129132	chr21:36206530
9	CELSR1	rs6010313	chr22:46388510
9	FOXC1	rs12194148	chr6:1603030
9	AUTS2	rs2293773	chr7:69874960
9	SUCLA2	rs17155785	chr13:47897690
9	NKX2-5	rs7172307	chr5:173232140
9	EP300	rs20541	chr22:41174720

Category	Gene	rsID	GRCh38 coordinate
10	KIF1A	rs59733750	chr2:240753500
10	NSMCE1	rs1127228	chr16:27240260
10	MROH2A	rs2361506	chr2:233830694
10	SEMA4D	rs45483393	chr9:89477500
10	TMEM132C	rs1683723	chr12:128791800
10	TERT	rs2736100	chr5:1286516
10	TERT	rs2736098	chr5:1295349
10	FGF5	rs7680746	chr4:80262330
10	LGR5	rs4919689	chr12:71829380
10	PRSS53	rs11150606	chr16:31100880

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