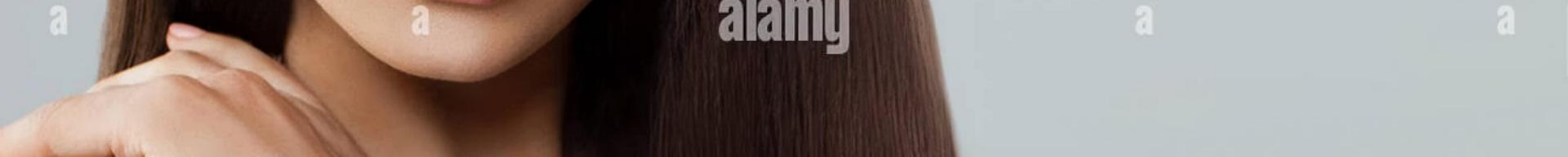


Aging in Skin and Hair



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Aging in Skin and Hair: Key Mechanisms

Skin and hair aging result from a synergy of intrinsic factors (genetic, chronological decline) and extrinsic elements (environment, lifestyle). Below are the principal mechanisms contributing to visible and functional changes.

- Intrinsic Aging**
Genetically programmed decline in cellular function over time leads to natural aging of skin and hair.
- Extrinsic Factors**
Environmental stressors like UV rays, pollution, and smoking accelerate skin and hair aging beyond genetic programming.
- Oxidative Stress**
Excessive free radicals cause cellular and molecular damage, driving visible signs of aging.
- Glycation**
Sugar molecules bind to collagen, that decrease skin elasticity and vitality.
- Photoaging**
Chronic sun exposure leads to wrinkles, spots, and loss of skin tone due to cumulative UV damage.
- Hormonal Effects**
Hormonal changes alter hair density and skin structure, contributing to visible aging.

Biochemistry of Reactive Oxygen Species



Types of ROS

Reactive Oxygen Species (ROS): Superoxide (O_2^-), hydroxyl radicals (OH^\cdot), hydrogen peroxide (H_2O_2)



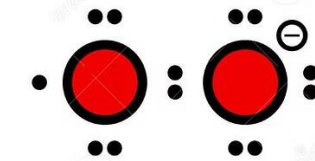
Exogenous Sources

UV radiation, pollution, cigarette smoke

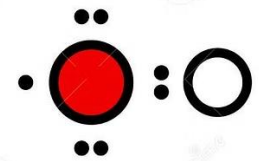


Endogenous Sources

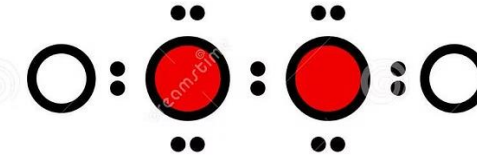
Mitochondrial electron transport chain.



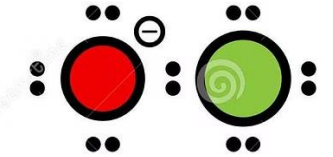
superoxide anion



hydroxyl radical



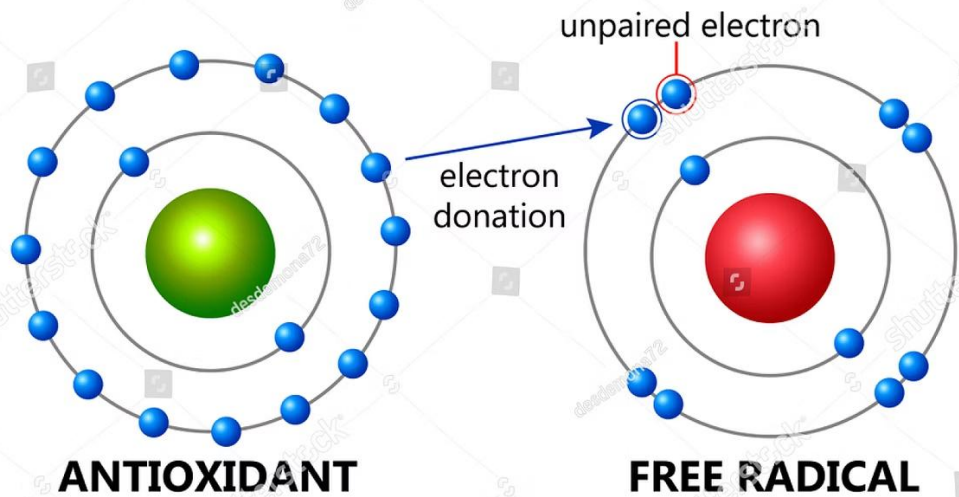
hydrogen peroxide



hypochlorite anion

Antioxidant Defense Systems

How antioxidants reduce free radicals



chemically reactive unpaired electron + electron donation:
stable electron pair is formed, free radical is neutralised

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IMAGEID: 1290409063
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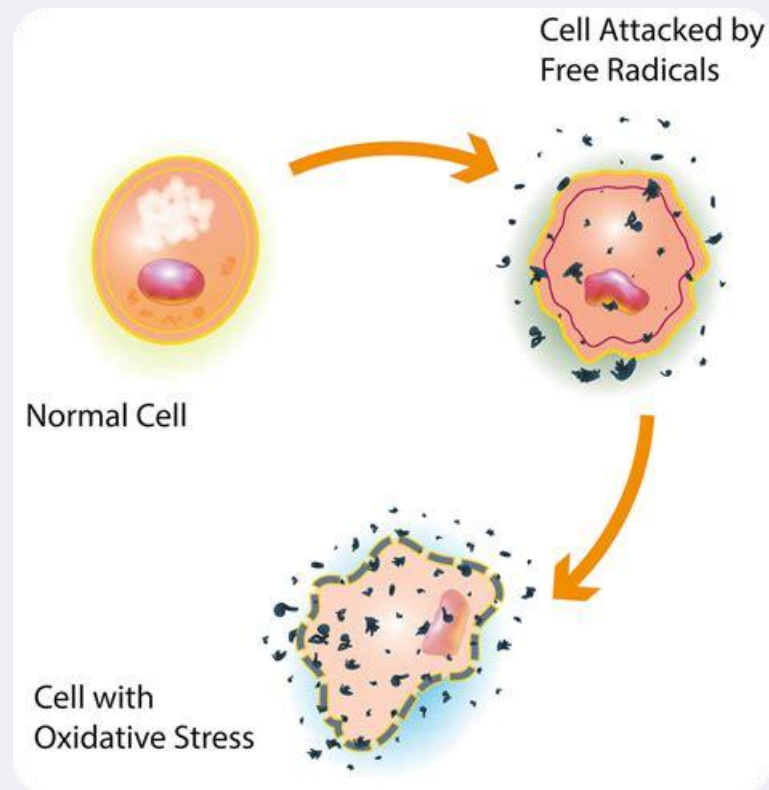
Enzymatic Antioxidants

- Superoxide dismutase (SOD)
- Catalase
- Glutathione peroxidase

Non-enzymatic Antioxidants

- Vitamin C
- Vitamin E
- Glutathione
- Coenzyme Q10

Oxidative Damage to Skin



Collagen Degradation

ROS activate matrix metalloproteinases (MMPs: MMP-1, MMP-3)



Lipid Peroxidation

Damage to cell membranes → inflammation (release of prostaglandins, cytokines)

3

DNA Damage

mutations, impaired repair (↓ BRCA1)

HAIR DAMAGE TYPES



healthy



raised cuticle



split ends



breakage

Oxidative Damage to Hair



Melanocyte Apoptosis

ROS \uparrow melanocyte stem cell depletion \rightarrow graying



Keratin Damage

Disulfide bond oxidation \rightarrow weak hair

Photoaging: UV Radiation Types



UVA (320–400 nm)

Penetrates dermis → ROS generation, lipid peroxidation



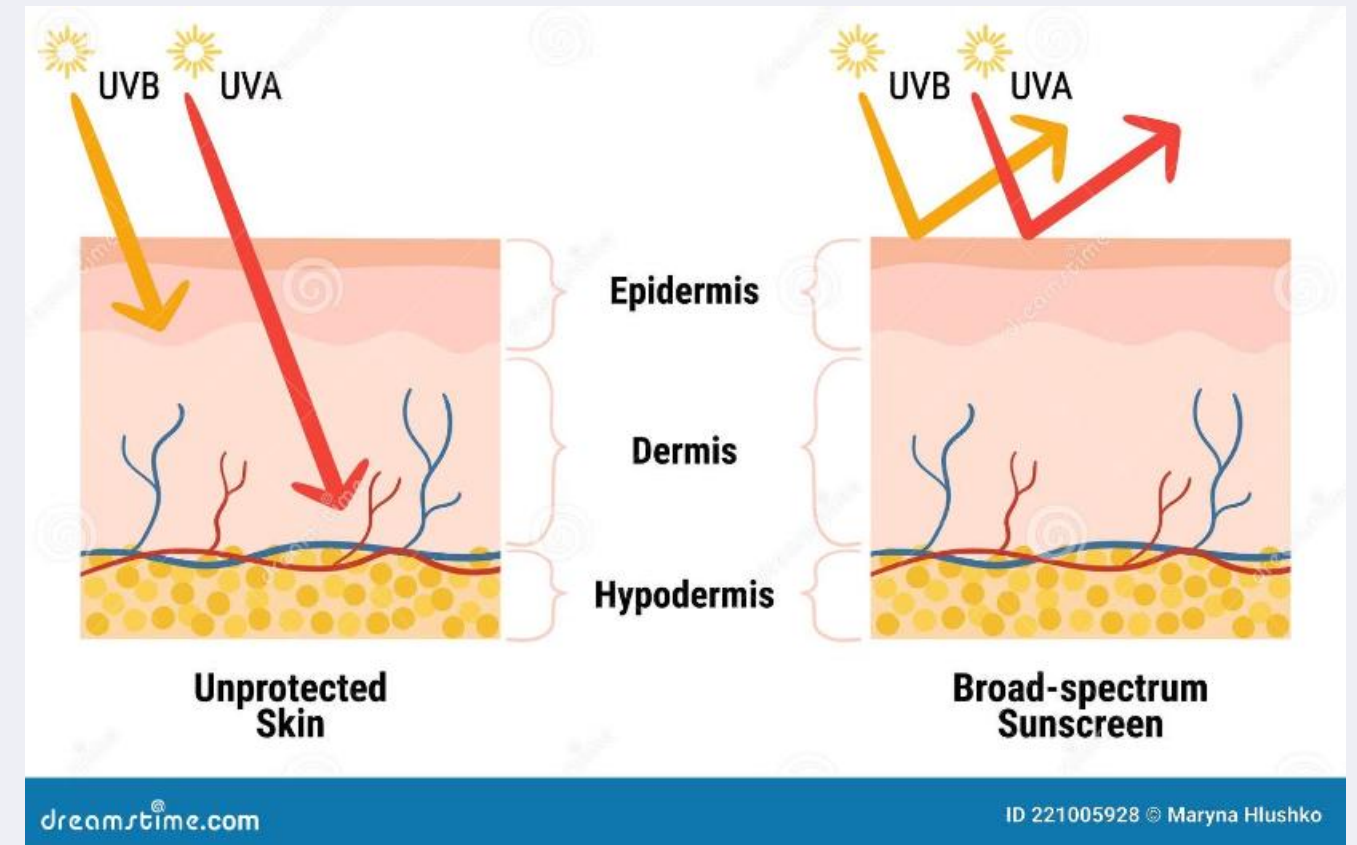
UVB (290–320 nm)

Epidermal damage → sunburn, DNA mutations

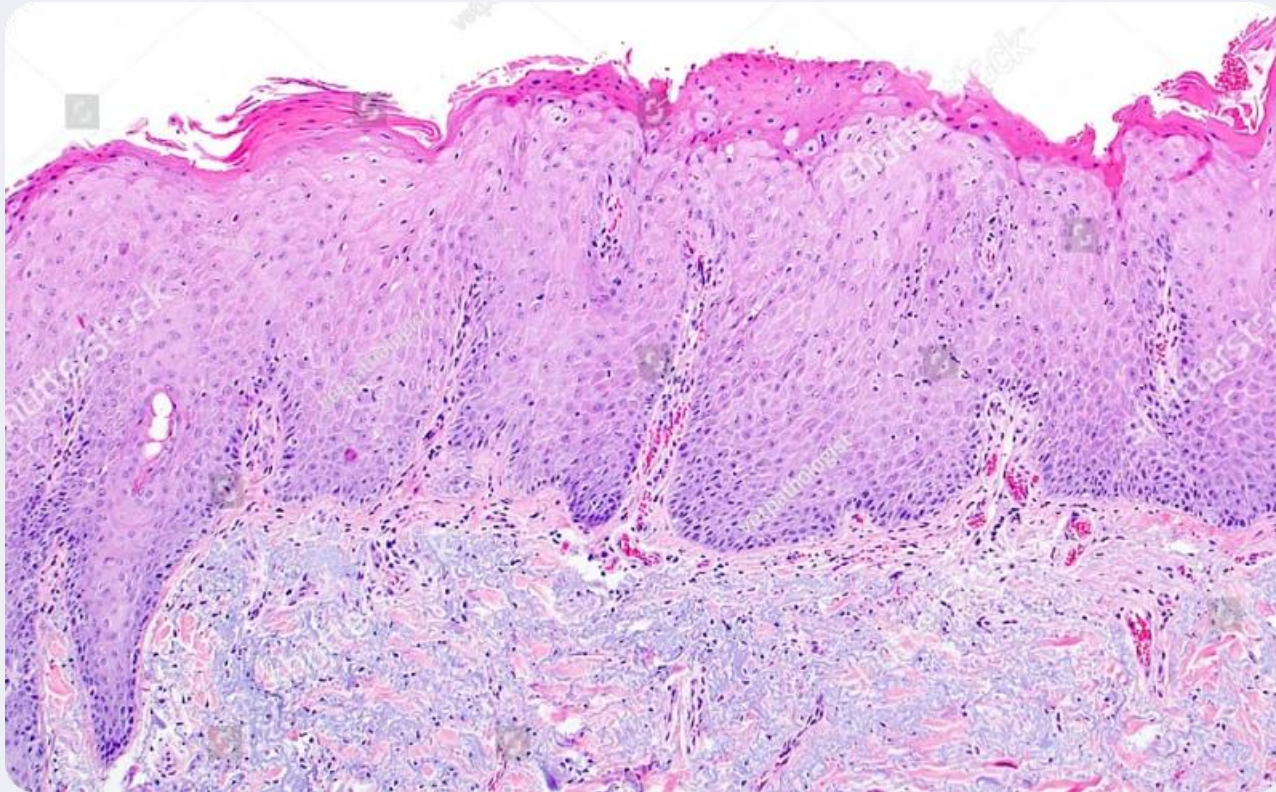


Molecular Mechanisms

DNA Damage, Melanocyte Stimulation



Clinical Features of Photoaging



Skin Changes

- Wrinkles (dynamic → static)
- Solar elastosis (yellow, thickened dermis)
- Telangiectasias (broken capillaries)
- Actinic keratosis (pre-cancerous lesions)



Hair Changes

- UV-induced melanin degradation → fading, dryness
- Cuticle damage → split ends

Photoaging vs. Intrinsic Aging

Feature	Intrinsic Aging	Photoaging
Skin Thickness	Thinning epidermis/dermis	Thickened, leathery texture
Pigmentation	Uniform pallor	Mottled dyspigmentation
Wrinkles	Fine lines	Deep, coarse wrinkles



Clinical Interventions for Oxidative Stress

(4) Topical Antioxidants

Vitamin C (L-ascorbic acid): Boosts collagen, regenerates vitamin E

Niacinamide (vitamin B3): ↑ reduces ROS

Oral Supplements

Polyphenols (green tea)



Sun Protection

Sunscreen

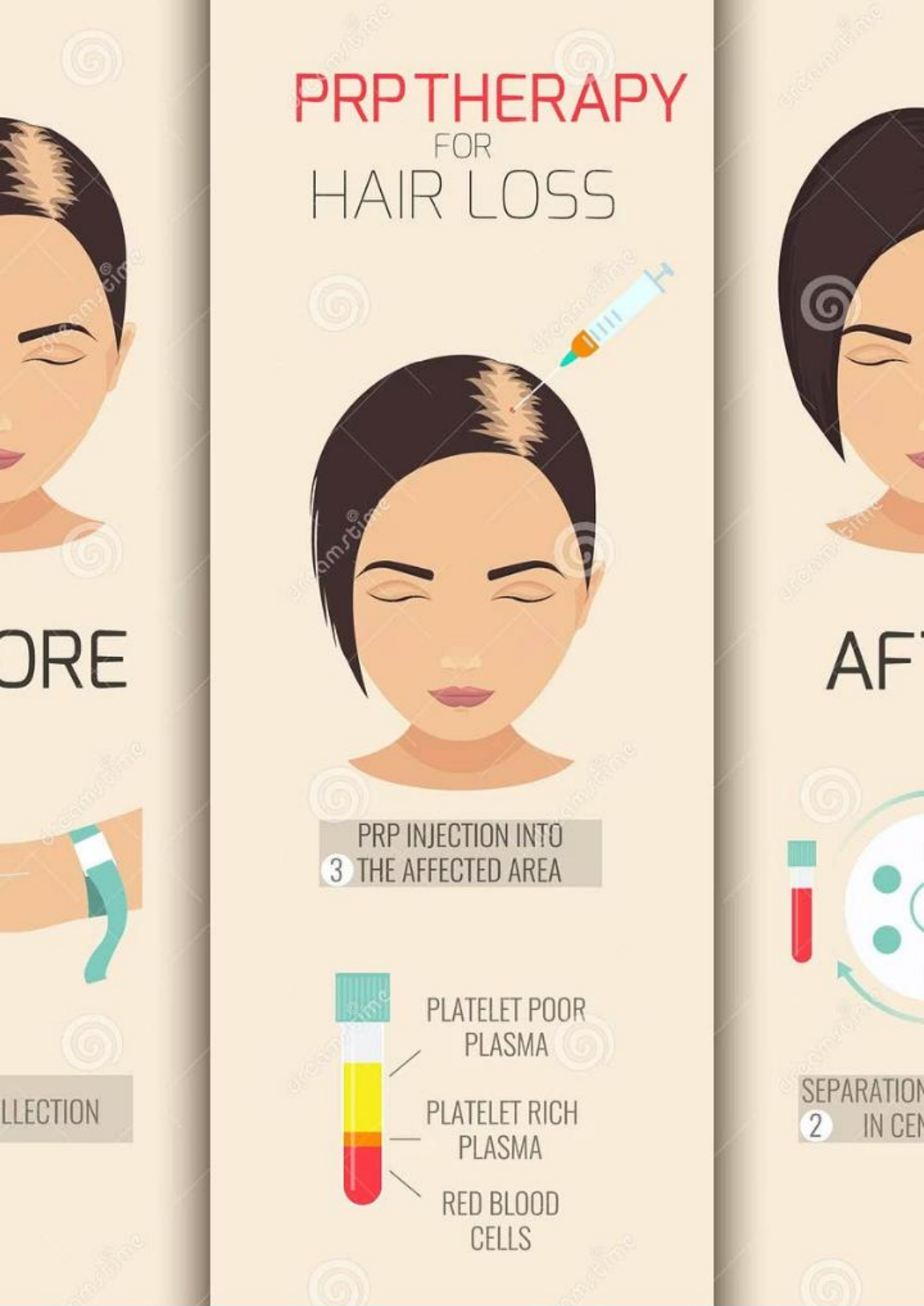
Broad-spectrum (UVA/UVB) with zinc oxide/titanium dioxide



Behavioral

Avoid peak sun hours (10 AM–4 PM), hats/UV-protective clothing





Hair-Specific Therapies and Emerging Treatments

Hair-Specific Therapies

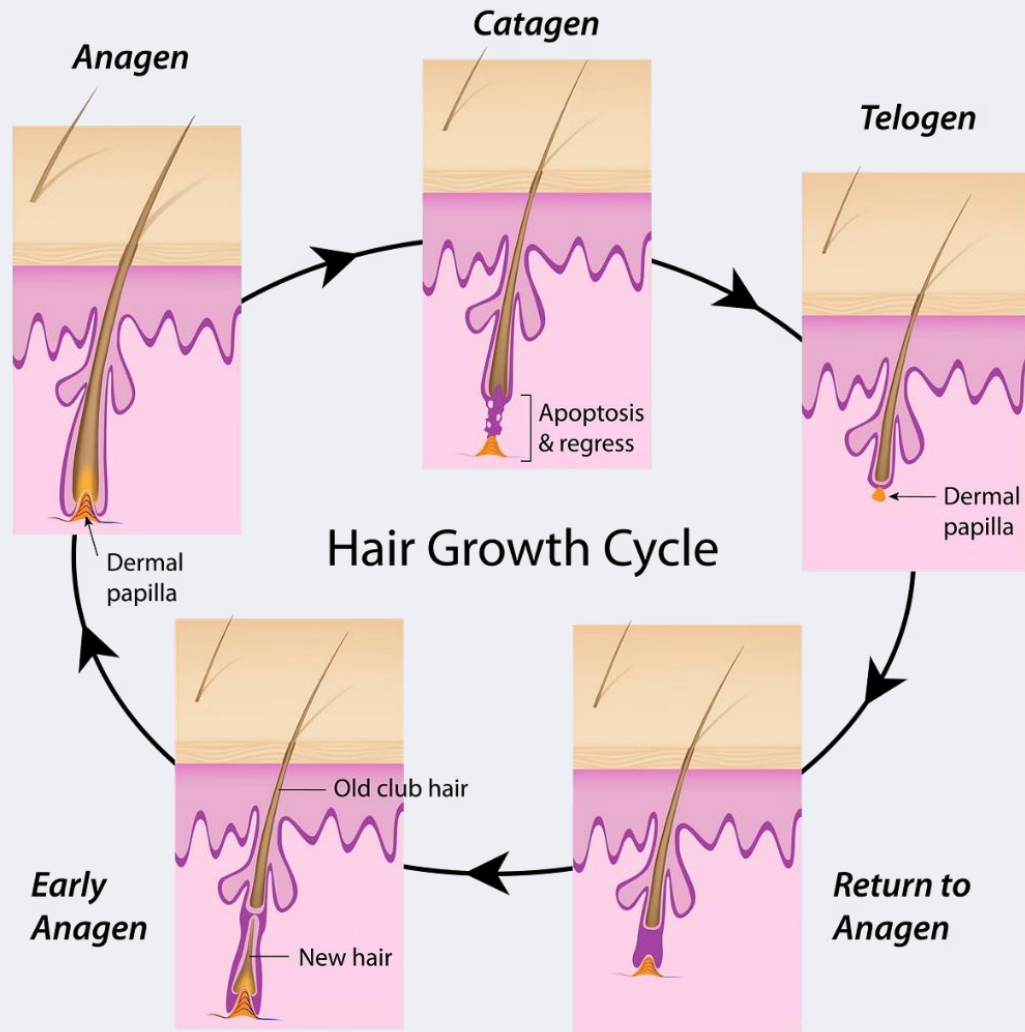
- Topical Minoxidil: ↑ Follicle blood flow
- Antioxidant Shampoos: Caffeine, rosemary oil (↓ oxidative stress)

Emerging Therapies

PRP , MESOTHERAPY , EXSOSOM

- Stem Cell Therapy: Follicle stem cell activation (experimental)

Hair Growth Cycle and Aging Effects



Anagen (2–6 years)

Aging effect: Duration shortens → hair becomes shorter and finer

Molecular drivers: Reduced Wnt/ β -catenin signaling and BMP inhibition



Catagen (2–3 weeks)

Apoptosis-driven regression; accelerated by oxidative stress



Telogen (3–4 months)

Aging effect: Prolonged telogen → increased shedding (e.g., "senescent alopecia")

Key mediators: Increased TGF- β , FGF5



Clinical Management and Future Directions

Hair Thinning Therapies

First-line: Minoxidil, Finasteride, Spironolactone (women)

Advanced: Platelet-Rich Plasma (PRP), Low-Level Laser Therapy (LLLT)

Gray Hair Management

Cosmetic: Temporary dyes (ammonia-free for sensitive scalps)

Emerging: Topical antioxidants, stem cell injections, oral L-Tyrosine

Holistic Approaches

Diet: Iron, zinc, biotin, vitamin D, antioxidant-rich foods

Lifestyle: Smoking cessation, stress management

Future Directions

Stem cell therapy, JAK/STAT inhibitors, CRISPR gene editing