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Review Article

The role of epigenetics in the pathogenesis of cutaneous diseases: a comprehensive review

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ABSTRACT

Epigenetics has recently emerged as a highly innovative and dynamic area of study within dermatology. This field concentrates on modifications to gene expression that occur without altering the underlying DNA sequence. These altered expressions were initiated by environmental influences and lifestyle choices, and play a fundamental role in both the onset and progression of numerous dermatological disorders. This article aims to delineate the essential roles that epigenetic modifications play in driving the pathogenesis of diverse dermatological disorders, as well as to highlight their clinical applications within current skin research. A comprehensive review of recent literature was conducted to elucidate the role of epigenetic perturbations in driving skin disease pathogenesis, illustrate cutaneous responses to environmental stimuli, and evaluate therapeutic trials aimed at reversing these epigenetic shifts. This article comprehensively outlines the pivotal role of epigenetic modifications; specifically, DNA methylation, histone modification, and non-coding RNA pathways, in the pathogenesis of various dermatological disorders. Epigenetics offers a promising paradigm shift toward targeted interventions for traditionally imprecise dermatological treatments. The sheer multiplicity and redundancy of disease-associated epigenetic modifications frequently bottleneck therapeutic innovation. Overcoming this barrier requires a transition from symptomatic management to personalized medicine that can precisely reset disease-driving epigenetic switches.

Keywords: DNA methylation, Non-coding RNA, Histone chemical alterations, Skin diseases

INTRODUCTION

Epigenetics studies heritable, reversible gene expression patterns resulting from environment-driven changes in chromosomal function without altering the nuclear DNA sequence.[1] Epigenetic modifications exert a critical role in various physiological and pathological conditions.[2] Epigenetic modifications alter DNA accessibility through various mechanisms and influence the instructions encoded in DNA. Hypermethylation or hypomethylation of gene-regulatory DNA sequences results in gene silencing or active transcription, respectively. DNA methylation causes sustained changes in the transcription of specific genes.[3] Common techniques for profiling DNA methylation include whole-genome bisulfite sequencing, which has been used to study skin diseases such as psoriasis.[4]

Histone chemical modifications, including various post-translational modifications, are epigenetic regulatory mechanisms that influence cellular functions by altering chromatin packaging and accessibility, thereby shaping gene expression.[5] Epigenome sequencing technologies, such as chromatin immunoprecipitation and assay for transposase-accessible chromatin sequencing, are widely used to analyze histone-DNA interactions, identify open regions within chromatin, and study histone modifications.[6] Deregulated expression levels of microRNAs have been linked to environmental influences and complex diseases.[7] Long non-coding RNAs

(lncRNAs) are increasingly recognized as vital modulators of gene expression, executing fundamental functions across diverse biological processes and human pathologies.[8]

The immune responses of skin cells are largely mediated by the expression of recognition receptors that identify components of invading microorganisms, initiate immune cascades to combat these invaders, and interact with immune cells. Additionally, keratinocytes can express epigenetic regulators that shape their response to environmental cues.[9] Altered epigenetic regulators of the cutaneous immune response induce an imbalance in skin immunity and are fateful for the pathogenic mechanisms of chronic cutaneous immune disorders.[10] Epigenetic mechanisms are increasingly recognized as a key link between environmental exposures and the development of stable skin disease phenotypes. Notably, these mechanisms drive persistent inflammatory states and tissue disease memory, which endure even after clinical improvement is achieved.[11]

Since epigenetic modifications are modifiable and reversible, epigenetic modifier drugs that reverse epigenetic signatures are a new potential strategy for treating skin diseases.[12] Therefore, understanding epigenetic mechanisms in the skin might open avenues for developing novel therapeutic targets. This review aims to outline the fundamental roles of epigenetic modifications in driving the development of various skin disorders.

EPIGENETIC CHANGES IN CHRONIC INFLAMMATORY AND IMMUNE-RELATED SKIN DISEASES

Epigenetic regulation constitutes a molecular link between genetic susceptibility and the exposures to environmental cues. Consequently, it played a crucial role in either driving the pathogenesis or exacerbating the severity of diverse chronic inflammatory and immune related dermatoses.

Psoriasis

The transcriptomic remodeling characteristic of psoriasis is driven by a combination of transcription factors and epigenetic mechanisms. Together, these elements control the expression of disease-associated genes through altered DNA methylation, non-coding RNA pathways, shifts in chromatin accessibility, and direct transcriptional activation or repression.[13]

In psoriatic skin, decreased expression of the ten-eleven translocation family of enzymes, which regulates DNA methylation, dysregulates epithelial barrier molecules, particularly filaggrin (FLG).[14] Elevated levels of tripartite motif 27 and activation of the interleukin (IL)-6/signal transducer and activator of transcription 3 (STAT3) signaling pathway trigger keratinocyte cytopathy by inducing N6-adenosine methylation (m6A) in mRNA.[15] Downregulated phosphoglycerate dehydrogenase in psoriatic lesions leads to increased nuclear factor (NF)-κB activation and overexpression of IL-6 in keratinocytes by inhibiting DNA methylation at the promoter region and increased binding of myocyte enhancer factor 2A.[16] Exposure to air pollutants alters CpG site methylation at the *ZMIZ1* locus to influence psoriasis risk, exhibiting a dose-response relationship that depends on specific *ZMIZ1* genotypes. This environmental link to skin psoriasis is further driven by an immune cascade, where the *ZMIZ1* genetic variation-associated cytokines IL-15, IL-19, and IL-22 act as significant mediators.[17]

Acetylation and methylation of histones H3 and H4 may be associated with psoriasis severity and serve as therapeutic response markers.[18] Dysregulated microRNAs (miR) in keratocytes and immune cells contribute to the onset and progression of psoriasis. Specific microRNAs promote inflammatory signaling and Th17 responses, while others are anti-inflammatory regulators. Thus, estimating levels of circulating and extracellular vesicle-associated miRNAs can serve as non-invasive biomarkers that reflect disease severity and therapeutic response.[19] Overexpression of tumor necrosis factor (TNF)-α in human neutrophils induces the release of miR-1290, which promotes the proliferation of neutrophil differentiation and keratinocyte. Downregulated expression levels of miR-181a/b-5p negatively regulated keratinocyte proliferation by targeting the Maternal Embryonic Leucine Zipper Kinase.[20] Long non-coding RNA genetic variants play a major role in determining psoriasis risk and how different populations respond to therapy. For instance, Single-nucleotide polymorphisms are associated with psoriasis susceptibility and treatment outcomes in a population-specific manner, making them promising candidates for disease biomarkers.[21]

The PCR-Restriction fragment length polymorphism (RFLP) technique detected a significantly high predominance of the A/A genotype of the vitamin-D receptor gene polymorphism in psoriasis patients and of A/a genotype in healthy subjects, with significant over-representation of the A allele in psoriatic patients.[22] Integrative therapeutic strategies that combine vitamin D supplementation, targeted microbiota interventions, and miRNA-based therapies show strong promise for the management of concomitant inflammatory skin disorders and osteoporosis.[23]

Atopic dermatitis

As a chronic inflammatory skin condition, atopic dermatitis (AD) is fundamentally driven by three interconnected factors: a compromised skin barrier, a dysregulated immune response, and an imbalance in the skin's microbial ecosystem.[24]

Epigenetic regulation may play a pivotal role in the development of AD. MicroRNA-939 is highly upregulated in AD lesions and contributes to cutaneous colonization by

S. aureus and to exacerbation of AD-like inflammation by inducing matrix metalloproteinases.[25] MicroRNA-143, by modulating IL-13 signaling, might counteract allergic inflammation by adjusting cutaneous immune responses in parallel with IgE levels in AD. MicroRNAs-124 and 151a, acting through the NF-κB signaling pathway and modulation of the IL-12 receptor β2, respectively, have anti-inflammatory effects. MicroRNA-10a regulates keratinocyte proliferation and differentiation, while miR-29b regulates apoptosis in these cells.[26] microRNA-711 and miR-203b-3p were upregulated in certain clinical models of itching.[27]

DNA methylation-based analyses in a pediatric AD cohort detected acceleration of the epigenetic and biological age, but not telomere length.[28] Interferon (IFN)-γ and IL-5 genes were hypermethylated in breastfed AD infants, while they showed decreased methylation in milk formula infants. Additionally, higher methylation of IL-4 was positively correlated with the number of allergic family members and the age of the AD infant, while IL-10 and Forkhead box P3 (FOXP3) methylation were negatively correlated with the AD infant's age.[29] The major allele of rs7872806 increases AD risk by 2.64-fold, and this is associated with increased methylation at cg13920460 site and under-expression of tight junction protein-2 with increased trans-epidermal water loss.[30]

In AD lesions, caspase-1-upregulated gasdermin D (GSDMD) suppresses human keratinocyte differentiation. Mechanistically, GSDMD reduces *FLG* expression by decreasing histone acetylation at the *FLG* promoter via histone deacetylase 1 (HDAC1). Furthermore, GSDMD prevents the degradation of HDAC1 by disrupting its interaction with Potassium Channel Tetramerization Domain Containing-6.[31] Filaggrin levels tended to decrease with increased HDAC-3 and HDAC-6 on IL-4/13 and particulate matter-10 cotreatment than when each was given individually in the AD-like in-vivo model. However, treatment with trichostatin A ameliorates these effects on FLG expression, indicating its potential as a novel therapeutic approach for AD.[32] Histone deacetylase inhibitors alleviate pruritus in AD.[27]

Vitiligo

Vitiligo, an acquired chronic disorder of depigmentation, stems from the loss or impaired function of melanocytes. The underlying pathogenesis involves a complex interplay of four key elements: genetic predisposition, environmental influences, epigenetic regulation, and a dysregulated autoimmune response.[33,34]

Overexpression of miR-21-5p reduces melanin content in melanocytes, suppresses tyrosinase activity, and downregulates expression of the melanogenesis-related gene and its protein in melanocytes by directly targeting the special AT-rich sequence-binding protein-1.[35] Upregulation of miR-125b-5p downregulates expression of the microphthalmia-associated transcription factor, thereby affecting vitiligo melanocyte biological behavior and melanogenesis.[36] Overexpressed levels of miR-1469 increased the proliferation and viability of natural killer cells and their secretion capacity of IFN-γ.[37] Moreover, miR-200c, which enhances the expression of melanogenesis-related genes by suppressing SRY-Box Transcription Factor 1 to activate β-catenin, was downregulated.[38]

Oxidative stress is a key driver of vitiligo pathogenesis through inducing hypermethylation of the ubiquitin-specific peptidase gene 18 promoter, which increases the expression of the interferon-stimulated gene 15 in keratinocytes and melanocytes, leading CD8+ T cells to produce IFN-γ, the main pathogenic cytokine in vitiligo.[39] Also, Oxidative stress induces hypermethylated-triggered downregulation of annexin A2 receptor, a crucial gene in cell apoptosis, leading to melanocyte apoptosis, and, through inhibiting the secretion of stem-cell factor from cutaneous cells, causes impaired survival of melanocytes.[40] Overexpression of miR-493-3p increases dopamine secretion, leading to overproduction of reactive oxygen species, which dysregulates melanocyte function, induces melanocyte apoptosis, and decreases melanocyte proliferation and melanin synthesis by targeting the heterogeneous nuclear ribonucleoprotein-U, which binds to and regulates catechol-O-methyltransferase.[41] Additionally, hypomethylated perforin promoter induces its overexpression, leading to activation of CD8+ T cells, which destroy melanocytes.[42]

Cutaneous lupus erythematosus

Systemic lupus erythematosus (SLE) is a complex and heterogeneous autoimmune disease arising from the interplay between genetic predisposition and environmental factors, which collectively contribute to its pathogenesis. Environmental exposures throughout the lifespan are increasingly recognized as potential contributors to the pathogenesis of SLE, partly through their effects on epigenetic regulation and cellular function. Hypomethylation of the miR-17-92 cluster, through activating T cells, is an epigenetic locus for lupus. Moreover, direct interrelation between cutaneous lupus erythematosus (CLE) activity and the expression levels of miR-19b1 and 18a was evident.[43]

Sustained type I interferon (IFN) upregulation in keratinocytes (KCs) is a feature of both lesional and non-lesional lupus skin. This IFN-rich microenvironment alters cellular death responses in the epidermis and heightens inflammatory reactions following ultraviolet light exposure.[44]

Patients with SLE demonstrate high expression of the *OAS1*, *IFI27*, and *IFI44L* genes, driven by an epigenetic profile of elevated H3K4me3 trimethylation and reduced promoter DNA methylation. At the *IFI44L* locus in particular, this active state is further marked by increased

H3K27ac modifications, greater chromatin accessibility, and heightened chromatin interactions.[45]

Epigenetic regulation of macrophage polarization dynamics by lncRNA emerging as a critical contributor to SLE pathogenesis.[46] The lncRNA nuclear paraspeckle assembly transcript 1 (NEAT1) influences gene expression through epigenetic, transcriptional, and post-transcriptional mechanisms. It is upregulated in various skin disorders, including SLE, AD, and melanoma, and functions as a competitive endogenous RNA by targeting microRNAs and regulating multiple immune cells.[47]

PRECANCEROUS AND CANCEROUS DERMATOLOGICAL DISORDERS

Lichen planus

MicroRNA-27b was significantly downregulated, particularly in atrophic-erosive oral LP (OLP), not in reticular OLP.[48] MicroRNA-562 and 203, which are the target miRNAs of IL-22, were significantly downregulated and overexpressed, respectively, in OLP patients compared with normal controls.[49] Overexpression of miR-21 and downregulation of miR-125a in the saliva of OLP patients indicate poor prognosis, whereas absence of significant alterations in the expression levels of miR-31 and miR-200a may suggest the absence of malignant transformation.[50] The prevalence of the C>T allele at rs16906252, which is associated with methylation of the O16-methylguanine-DNA methyltransferase (MGMT) promoter, was 10 times higher in OLP than in controls.[51] The expression levels of Yes-Associated Protein were significantly upregulated in oral and cutaneous LP and showed significantly higher expression levels in poorly and moderately differentiated squamous cell carcinoma (SCC) than in well-differentiated SCC.[52] ZNF582 methylation was significantly decreased in OLP lesions compared to lesions showing dysplastic features and oral SCC (OSCC), so it may be a potential biomarker for distinguishing OLP from SCC.[53] A significant causal relationship between intrinsic epigenetic age acceleration and oral diseases, especially oral lichen planus (LP), was detected.[54]

Lichenoid dermatosis

Methylated p16, death-associated protein kinase 1 (DAPK1), and MGMT genes were observed in 12.7% of potentially malignant oral disorders and oral cancer lesions. Methylated p16 and DAPK1 were found in 13.23%, methylated p16 and MGMT in 12.12%, and methylated DAPK1 and MGMT were found in 15.27% of oral cancer lesions.[55] The methylation patterns reveal distinct tiers among the tissues: E-cadherin was hypermethylated in OLP, SCC, and radicular cysts relative to non-inflamed gingiva. However, when comparing these conditions directly, DNA hypermethylation was confirmed to be significantly higher in OLP and SCC than in either the non-inflamed gingiva or radicular cysts. Hypermethylation of $p16^{ink4a}$ was observed only in SCC.[56] Multiple aberrantly expressed microRNAs were identified in oral LD (OLD), especially miR-146a, which was suggested to have a possible role in OLD etiopathogenesis.[57]

Cutaneous malignant lesions

DNA methylation at regulatory regions between undifferentiated and differentiated keratinocytes defined AK/cutaneous SCC (CSCC) subtypes with epidermal stem cell- or keratinocyte-like features.[58] An increased percentage of mothers against decapentaplegic homolog-4 gene methylation, with decreased expression of its mRNA, was detected in patients with basal cell carcinoma, CSCC, and basosquamous carcinoma compared with healthy tissue.[59] The upregulated expression of miR-199a-3p in CSCC induced decreased phosphorylated focal adhesion kinase activity with subsequent downregulation of epithelial-to-mesenchymal transition marker genes.[60] The alleles of the programmed death (PD) gene have contradictory effects on the risk of metastatic melanoma (MM). PD-1.7 C allele, particularly the C/C genotype, exhibits a higher risk of MM in the codominant and allelic models, while the PD-1.5 T allele, via the promotion of anti-tumor immunity, reduced the risk of MM in both codominant and dominant models through increasing the activity of the PD1-positive effector T cell.[61] In undifferentiated CSCC, the histone variant H2A.J is highly expressed in the nucleus. This variant promotes cancerous transformation and makes the tumor resistant to radiation by turning on the WNT16 signaling pathway. Because of these distinct mechanics, tracking H2A.J-associated signatures offers a promising way to improve risk stratification, allowing clinicians to identify aggressive CSCCs that could benefit from higher doses of radiation therapy.[62] Research highlights distinct epigenetic profiles for two skin cancers. In CSCC tissues and cell lines, epigenetic upregulation leads to the overexpression of *IDO1*, *IFI6*, and *OAS2*, which functionally regulate tumor cell proliferation, migration, and invasion.[63] Meanwhile, a global loss of the H3K27me3 histone marker is highly specific to Merkel cell carcinoma, where H3K27me3 labeling is low but still retained indicated significantly longer survival outcomes.[64]

EPIGENETIC CHANGES IN ALOPECIA

Alopecia areata

Nineteen miRNAs were significantly downregulated in AA, nine of which were validated for both mild and severe forms, with potential applications for both diagnosis and therapy.[65] Upregulation of miR-199a-3p suppressed hair growth by upregulating immune-related genes and downregulating hair follicle development-related genes, primarily via the protein tyrosine phosphatase receptor type F/β-catenin axis.[66] MiR-101 and nuclear-enriched abundant transcript-1 were downregulated in AA patients.[67] The binding of miR-365-1 to the promoter

region of its target gene DAP3 (Death Associated Protein 3) reduced the expression of the apoptotic protein in the hair follicle stem cells (HFSCs); however, hypermethylation of miR-365-1 led to its downregulation and increased expression of apoptotic proteins and death of HFSCs.[68] The direct impact of hsa-miR-193a-5p on the inositol-pentakisphosphate 2-kinase gene is involved in the development of AA.[69] MicroRNA-200c-3p also affects the epidermal growth factor receptor, and so has a remarkable role in AA development.[70] The significant upregulation of microRNAs 19b-3p, 182-5p, and 155-5p in AA patients suggests they could serve as valuable clinical tools. Specifically, these microRNAs show strong potential for diagnosing AA, tracking its progression, and guiding the development of targeted therapies.[71] Expression levels of miR-29a and lncRNA H19 were upregulated in AA patients compared to controls.[72] Researchers developed an injectable hydrogel responsive to reactive oxygen species to provide targeted, long-term delivery of miR-665. In human skin cells, the delivered miR-665 blocks STAT3 phosphorylation, reversing the inhibitory effects of IFN-γ on the proliferation and migration of keratinocytes and dermal papilla cells. When compared to controls, this hydrogel platform drastically enhances hair regeneration, repairs follicle structures, and mitigates T-cell infiltration.[73]

The rs11073001 and rs17875491 Silent information regulator-1, a type III histone deacetylase, may play a role in the pathogenesis of AA by promoting the production of IFN-γ, TNF-α, CXCL9, and CXCL10 in hair follicle outer root sheath cells.[74] Single Nucleotide Polymorphisms located in the exon and promoter regions of the IL-16 gene were more frequently distributed in AA patients than controls.[75]

Androgenic alopecia

Nine target miRNAs and two target lncRNAs were differentially expressed in hair follicle cycling, and downregulation of GSPT1 expression is possibly associated with AGA progression.[76] Overexpression of the lncRNA AC010789.1 promoted the proliferation and differentiation of HFSCs through downregulating miR-21-5p and tissue growth factor-β1 expression but upregulating the Wnt/β-catenin signaling pathway.[77] Downregulation of lncRNA RP11-818O24.3 in HFSCs affects its proliferation and differentiation through downregulation of phosphatidylinositol 3-kinase-Akt (PI3K/AKT) pathway and the fibroblast growth factor-2 in HFSCs.[78] Retinoic acid promotes hair growth by stimulating stem cells via Wnt/β-catenin signaling and accelerating the transition from a dormant to an activated state.[79] Exosome therapy shows great potential as a minimally invasive treatment for hair loss by shifting hair follicles out of the resting (telogen) phase and into the active growth (anagen) phase. Preclinical studies show this is achieved because exosomes boost dermal papilla cell proliferation and activate critical molecular pathways, namely Wnt/β-catenin, VEGF, and PI3K/AKT.[80] Hair follicle vitality is closely tied to systemic signaling: the gut microbiota shapes host immunity, metabolism, and the cutaneous microbiome, which collectively boost the activity of follicular stem cells. Working alongside this system, exosome-transported miRNAs target specific suppressor molecules to reverse follicular miniaturization and manage the hair growth cycle. Harnessing these dual pathways by targeting the "microbiota-miRNA axis" offers an innovative approach to treating AGA.[81]

EPIGENETIC CHANGES IN ACNE

Significantly higher serum levels of histone deacetylase 1, a zinc-dependent enzyme that removes acetyl groups from histones and provides epigenetic regulation of gene expression, were found in blood samples from AA and acne vulgaris (AV) patients compared with control samples, with higher levels in AA patients' samples.[82] Trait loci analysis identified 7 unique CpGs and 22 unique SNP-CpG pairs that reached the expression threshold in patients with acne, especially severe acne.[83] Gene-set enrichment analysis revealed negative enrichment of keratinization in acne-prone non-lesional (NL) skin. Mammalian target of rapamycin inhibitors was able to restore NL acne skin to a healthier state.[84] Genome-wide DNA methylation and gene expression analysis identified DNA methylation at 23 differentially methylated sites in severe acne and detected higher expression of the differentially methylated poly (ADP-Ribose) polymerase family member 8 and mitogen-activated protein kinase-activated protein kinase 2 genes in severe acne than in healthy controls.[85] A genome-wide association study detected four novel loci among the 23 mapped risk loci, with the strongest associations on chromosomes 11, 17, and 22. Gene prioritization ascribed these loci to fatty acid synthase, desaturase-2, zinc, and ring finger-3 genes.[85] Epigenetic profiling highlights two distinct mechanisms in skin pathology and homeostasis. First, hypermethylation at the *cg18095732* CpG site induces the upregulation of *ZDHHC20* (a palmitoyltransferase gene), which is strongly associated with a reduced risk of developing acne.[86] Second, spatial mapping shows that the loss of UTX (*KDM6A*), an X-linked histone demethylase and master enhancer regulator, dysregulates differentiation within the hair follicles, sebaceous glands, and epidermis. Because this loss triggers genome-wide depletion of H3K27ac but leaves H3K27me3 largely unaltered, it reveals that UTX acts in a mostly non-catalytic manner to preserve skin homeostasis.[87]

EPIGENETIC CHANGES IN MISCELLANEOUS DERMATOLOGICAL DISORDERS

Intracellular signaling plays a critical role in sheet integrity; specifically, the histone deacetylase 1/lysine demethylase 1A axis signals downstream to activate the extracellular signal-regulated kinase pathway. This activation ultimately drives the loss of cellular cohesion and promotes the apoptosis characteristic of pemphigus vulgaris.[88] In patients with hidradenitis suppurativa, there

is a significant overexpression of three specific long non-coding RNAs: lncRNA-TINCR, lncRNA-RBM5-AS1, and lncRNA-MRPL23-AS1.[89] Mutations in the FLG gene (FLG R501X and 2282del4) were detected in AD and Ichthyosis Vulgaris (IV) patients with a heterozygous (AT) genotype, respectively, and combined mutations were detected in the AD and IV groups with a heterozygous (AT) genotype.[90] Whole-exome sequencing identified two novel FLG gene variants, c.3320del and c.4909del, so nine variants of the FLG gene were associated with the symmetrical acral keratoderma.[91] Deposition of extracellular matrix in the keloid lesions was modulated by upregulated expression levels of the lymphocyte-specific protein-1 pseudogene 5, through repressing the expression levels of CCAAT enhancer binding protein-α, which is an antifibrotic gene.[92] Chronic arsenic exposure accelerated the skin and blood epigenetic, biological, and mitotic ages in arsenic-exposed adults with skin lesions compared to individuals who were arsenic-exposed but lesion-free and healthy arsenic-naïve individuals.[93]

CONCLUSION

Epigenetics examines how genes and environmental cues interact, with particular attention to conditions where treatment can be frustratingly imprecise. This field offers more promising, targeted therapeutic strategies. However, this review illustrated the multiplicity of epigenetic modifications underlying a specific dermatological disease. This redundancy limits the development of novel therapeutics. To overcome this, we must move beyond merely managing symptoms and instead deepen our research into personalized treatments capable of modifying the specific epigenetic switches that dictate disease progression. Achieving this objective requires active, interdisciplinary collaboration among molecular biologists, dermatologists, and pharmaceutical researchers. Furthermore, recognizing how heavily the environment influences our epigenetic machinery underscores the vital role of lifestyle medicine. This includes patient education focused on maintaining a balanced diet, implementing proper skincare routines to prevent secondary bacterial infections, and consciously avoiding exposure to substances that trigger these adverse epigenetic cascades.

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