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Role of skin microbiome and exosomes in Hair regeneration and disorders in diseases like Alopecia, Acne: A Comprehensive Review

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Abstract:

Hair disorders such as alopecia, acne, and seborrheic dermatitis affect millions worldwide, with significant psychosocial and clinical impact. Recent research highlights the importance of the skin microbiome and exosome-mediated cell communication in regulating hair follicle health, growth cycles, and immune balance. The skin microbiome forms a dynamic ecosystem that influences keratinocyte behavior, sebaceous activity, and local immune tone, while exosomes act as molecular couriers carrying growth factors, microRNAs, and cytokines that direct follicular regeneration and angiogenesis. Dysbiosis of the scalp microbiome can trigger inflammation, disrupt immune privilege, and contribute to hair loss, whereas targeted modulation of microbial diversity may restore homeostasis. Similarly, exosome-based therapies have shown potential to stimulate bulge stem cells, counteract follicular miniaturization, and promote hair regrowth. This review synthesizes current evidence on the microbiome–exosome crosstalk, explores its role in common follicular disorders, and discusses translational advances including microbiome restoration strategies, bioengineered exosomes, and combination therapies. Understanding these interconnected systems provides a foundation for precision scalp medicine and novel regenerative interventions.

Keywords: Skin microbiome, Exosomes, Hair regeneration, Alopecia, Seborrheic dermatitis

1. Introduction

Hair is more than a cosmetic attribute; it is tightly linked to identity, social belonging, and mental well-being. Disorders that affect hair and the surrounding skin such as alopecia, acne, and seborrheic dermatitis—therefore carry a clinical burden that spans physiology and psychology. Over the past decade, two lines of investigation have converged to reshape how we think about hair health: first, the realization that the scalp's microbial communities actively participate in cutaneous homeostasis rather than merely inhabit it; second, the discovery that exosomes—nanoscale extracellular vesicles loaded with proteins, lipids, and nucleic acids—serve as potent messengers within the hair follicle niche. Together, these advances suggest that hair growth and many common follicle-centered diseases are not just cell-autonomous problems but systems phenomena, where microbes, immune cells, keratinocytes, sebocytes, dermal papilla cells, and their vesicular cargo communicate continuously

across scales [1]. The skin microbiome of the scalp is a structured ecosystem comprising bacteria (for example Cutibacterium and Staphylococcus species), fungi (notably various Malassezia clades), mites, and viruses. Its composition is shaped by sebum output, sweat, hair density and curvature, personal hygiene, hormones, and environment. In steady state, this community contributes to barrier integrity, metabolizes lipids, trains immune tolerance, and competes with pathogens. When balance shifts—through antibiotics, stress, climate, cosmetics, or endocrine change—microbial metabolites and surface molecules can tip immune signaling toward inflammation, disturb keratinization, and alter the follicular microenvironment. Dysbiosis has been associated with conditions ranging from dandruff and seborrheic dermatitis to inflammatory forms of alopecia, while shifts in Cutibacterium and Staphylococcus proportions are repeatedly reported in acne-prone skin. These associations imply mechanisms: lipase activity that liberates irritating free fatty acids from sebum, biofilm formation that sustains low-grade inflammation, and pattern-recognition receptor activation (for instance TLR-mediated pathways) that fuels cytokine cascades [2].

Parallel to microbial ecology, exosome biology has reframed communication inside the hair unit. Exosomes originate from endosomal multivesicular bodies and carry curated cargo—microRNAs (miRNAs), mRNAs, enzymes, growth factors, and membrane proteins—that survive extracellular transit to reprogram recipient cells. Within the follicle, dermal papilla cells, hair follicle stem cells, outer root sheath keratinocytes, melanocytes, endothelial cells, immune cells, and sebocytes can all secrete or receive exosomes. Experimental work shows that exosomes modulate pathways central to follicle cycling and regeneration, including Wnt/β-catenin, Sonic hedgehog, BMP, and Notch networks. Importantly, exosome cargo such as miR-218, miR-21, or miR-125 family members has been implicated in stem cell activation, angiogenesis, and anti-apoptotic effects that favor anagen entry and maintenance. These vesicles can thus operate as precision packets that synchronize dermal and epithelial compartments during growth, rest, and regression [3]. The meeting point of microbiome and exosome biology is a fertile frontier. Microbial metabolites—including short-chain fatty acids, tryptophan-derived aryl hydrocarbon receptor (AHR) ligands, and diverse lipid species—can influence host exosome biogenesis, loading, and release. Conversely, host-derived exosomes can carry antimicrobial peptides, alarmins, and immunoregulatory miRNAs that reshape microbial composition and virulence behaviors such as quorum sensing. On the scalp, where sebum is abundant and follicle density is high, this two-way conversation may be especially consequential: sebaceous lipids feed microbes; microbes remodel those lipids; remodeled lipids and microbial signals tune sebocyte and keratinocyte exosome output; the resulting vesicles, in turn, recalibrate inflammation and stem-cell dynamics in the bulge niche [4]. Alopecia illustrates these interconnections. In androgenetic alopecia, follicle miniaturization stems from altered dermal papilla signaling, perifollicular fibrosis, microvascular changes, and androgen effects. Dysbiosis could amplify low-grade inflammation and oxidative stress, compounding signaling defects. In alopecia areata, an autoimmune process targets anagen follicles. Here, microbial cues may act as adjuvants that lower the threshold for immune activation in susceptible hosts, while

exosomes released by immune or follicular cells might propagate inflammatory programming or, conversely, serve as vehicles for tolerance-inducing cargo. Early preclinical studies suggest mesenchymal stromal cell–derived exosomes can tilt the balance toward regeneration by promoting angiogenesis and suppressing pro-inflammatory cytokines around the follicle bulb [5].

Acne provides a complementary use case. Excess sebum, altered lipid composition, follicular plugging, Cutibacterium overgrowth, and inflammation define the disease. Microbial lipases increase free fatty acids, which irritate the follicular wall and skew immune responses. Keratinocytes and sebocytes under inflammatory pressure change their exosome output; those vesicles can carry IL-1 pathway mediators, matrix-modifying enzymes, and miRNAs that foster hyperkeratinization and persistent inflammation. Targeting either side of this loop—by rebalancing microbial communities or intercepting pathogenic exosomal signals—could attenuate lesion formation and accelerate resolution [6]. Seborrheic dermatitis and dandruff bring Malassezia into focus. These lipophilic yeasts rely on sebaceous lipids, producing metabolites that can compromise barrier function and trigger inflammation in predisposed individuals. Host cells sensing these metabolites may adjust exosome cargo toward cytokine release and epidermal turnover. Clinical improvements seen with antifungals highlight the microbial lever, but residual scaling and itch suggest parallel host-signaling circuits remain active. Here, exosome-based diagnostics might reveal persistent inflammatory signatures, and exosome-informed therapeutics (for example, vesicles enriched for anti-inflammatory miRNAs) could complement antimicrobial regimens [7].

From a systems perspective, hair growth depends on nutrient supply, vascular support, mitochondrial health, and neuroendocrine cues. Psychosocial stress, sleep loss, micronutrient insufficiency, and pollution can all perturb this system. Each stressor leaves molecular fingerprints—oxidative adducts, altered lipidomes, skewed cytokine milieus—that influence both the microbiome and the vesicular language of the skin. For instance, particulate matter can change sebum chemistry, thereby selecting for different microbial consortia, while reactive oxygen species modulate exosome biogenesis pathways. This dynamic context helps explain inter-individual variability and fluctuating disease courses [8]. Translational opportunities follow naturally. Microbiome-targeted strategies include probiotics, prebiotics, synbiotics, bacteriotherapy, phage therapy, and postbiotic metabolites. Delivery matters: shampoos, leave-on tonics, occlusive masks, and microneedle-assisted application can differentially reach follicles and sebaceous ducts. On the exosome side, allogeneic or autologous vesicles derived from mesenchymal stromal cells, dermal papilla cells, or induced pluripotent stem cell derivatives are being explored. Formulation science—hydrogels, lipid nanoparticles, and adhesive patches—aims to stabilize vesicles, protect cargo, and enhance follicular penetration. Combining both levers could inaugurate a “precision scalp medicine,” where a patient’s microbial profile and vesicle signatures guide tailored interventions [9].

However, moving from concept to clinic requires methodological rigor. Microbiome studies vary in sampling (swab vs. tape-strip vs. follicular casts), sequencing (16S/ITS vs. shotgun metagenomics), and analytical pipelines, making cross-study comparisons difficult. For exosomes, isolation methods (ultracentrifugation, size-

exclusion chromatography, polymer precipitation, immuno-capture, microfluidics) and characterization standards differ, influencing purity and reported effects. Batch-to-batch variability, storage conditions, and donor heterogeneity complicate reproducibility. Harmonized protocols, orthogonal validation, and transparent reporting are prerequisites for credible biomarkers and interventions [10]. Safety and regulation present additional hurdles. Microbiome modulation must avoid opportunistic infections, resistance selection, or unintended ecological shifts. Exosome therapeutics raise questions about immunogenicity, oncogenic cargo, biodistribution, and long-term persistence. Manufacturing at scale demands closed-system bioprocessing, potency assays, stability testing, and release criteria aligned with evolving regulatory frameworks for biologics and advanced therapies. Ethical considerations also surface: equitable access to personalized treatments, responsible claims in cosmetic markets, and robust informed consent for donor-derived products [11]. Clinical trial design needs to catch up with mechanistic insight. Endpoints that capture patient-centered outcomes (shedding reduction, anagen/telogen ratio, lesion counts, pruritus, quality of life) should sit alongside molecular readouts such as microbial diversity indices, metabolomics of sebum, and exosomal miRNA panels. Adaptive trials and platform designs could accelerate comparison of combined microbiome–exosome strategies against standard care. Real-world evidence from dermatology practices can complement randomized data, especially for chronic conditions with fluctuating severity and high placebo response rates [12].

Diagnostics may evolve in tandem. Noninvasive scalp sampling coupled with rapid sequencing and vesicle profiling could provide actionable phenotypes at point of care: “inflammatory-dysbiosis-dominant” versus “signal-deficit-dominant,” for example. Such labels could guide whether to start with antimicrobial/postbiotic regimens, exosome-based pro-regenerative therapy, or a hybrid. Machine learning models that integrate clinical images, trichoscopy, microbiome compositions, lipidomics, and exosome cargo maps may predict responders, optimize dosing intervals, and flag early relapse [13]. Beyond single diseases, the microbiome–exosome framework may clarify overlaps. Patients with seborrheic dermatitis often have acne, and individuals with androgenetic alopecia can show micro-inflammation and barrier dysfunction resembling eczematous patterns. Mapping shared exosomal signatures (e.g., pro-inflammatory miRNAs and matrix-remodeling proteins) and shared microbial motifs (e.g., Malassezia-dominated communities in oily zones) could reveal common nodes to target. Lifestyle interventions—stress reduction, sleep optimization, balanced nutrition—might then be coupled with topical precision tools to push the scalp ecosystem toward resilience [14]. Equity and accessibility should remain central. Many affected individuals seek over-the-counter solutions; thus, translational science must inform safe, affordable formulations that do not depend on specialized equipment. Educational outreach that demystifies the role of microbes and vesicles—avoiding both “kill all germs” and “exosomes cure everything” extremes—can empower self-care while steering expectations realistically. Partnerships between academia, clinics, and industry will be crucial for transparent data sharing, rigorous product claims, and sustained innovation [15].

The aim of this paper is to synthesize current evidence on how the skin microbiome and exosomes influence hair follicle biology; to explain their roles in disorders including alopecia, acne, and seborrheic dermatitis; to evaluate therapeutic strategies that target microbes, exosomes, or both; and to highlight methodological, safety, and regulatory gaps that must be addressed to translate this emerging science into precise, effective, and equitable care.

2. Overview of Hair Biology and Disorders

Hair is a unique integumentary structure with both protective and psychosocial functions. Each hair follicle is a complex mini-organ embedded in the dermis, undergoing lifelong cyclical remodeling. The follicle comprises the dermal papilla, matrix cells, inner and outer root sheath, sebaceous gland, and arrector pili muscle. The dermal papilla serves as the signaling hub, controlling proliferation and differentiation of the matrix keratinocytes. Hair growth follows a repeating cycle with three major stages: anagen (active growth), catagen (regression), and telogen (resting), followed by shedding in exogen and re-entry into a new anagen phase [16]. This cycle is under the influence of multiple regulatory axes, including hormonal, immune, neural, and vascular systems. The anagen phase in scalp hair can last two to six years, allowing for significant length, whereas catagen is short, lasting only a few weeks, characterized by apoptosis-driven regression. Telogen lasts several months, after which synchronized shedding occurs. Disruption in the length or quality of any of these phases is often the first step in hair disorders.

The scalp microenvironment plays a crucial role in follicular health. Adequate vascularization supplies oxygen and nutrients, while innervation contributes neuropeptides and neurotransmitters that modulate follicular activity. Sebaceous glands produce sebum, which lubricates hair shafts, maintains barrier function, and creates a nutrient-rich habitat for commensal microbes. Mechanical forces, ultraviolet radiation, and environmental toxins can affect keratinocyte turnover and dermal papilla activity, influencing overall hair density and quality [17].

Hair disorders can be broadly classified into non-scarring (reversible) and scarring (cicatricial) types. Non-scarring alopecias include androgenetic alopecia, telogen effluvium, alopecia areata, and traction alopecia. These conditions preserve follicular stem cells, allowing regrowth if triggers are addressed. Androgenetic alopecia is driven by follicular miniaturization under androgenic influence, shortening the anagen phase and producing thinner, vellus-like hairs. Telogen effluvium occurs when stressors such as illness, hormonal fluctuations, or drugs shift a large number of follicles into telogen prematurely, resulting in diffuse shedding. Alopecia areata is an autoimmune process where autoreactive T cells attack the hair bulb, producing patchy loss. Traction alopecia results from chronic mechanical stress such as tight hairstyles [18].

Scarring alopecias involve irreversible destruction of the follicular epithelium, replaced by fibrous tissue. Examples include lichen planopilaris, discoid lupus erythematosus of the scalp, and folliculitis decalvans. Early detection is critical because timely intervention can prevent progression. Inflammatory infiltrates, keratinocyte

apoptosis, and perifollicular fibrosis are pathological hallmarks. These conditions are less common but pose a greater challenge because regenerative options are limited once the follicle is lost [19].

Acne vulgaris is another common follicular disorder, primarily affecting sebaceous gland-rich areas including the scalp, face, chest, and back. The pathogenesis involves four interlinked factors: increased sebum production, follicular hyperkeratinization, colonization by lipophilic bacteria, and inflammation. Although not always classified under "hair disorders," acne involves the pilosebaceous unit and can indirectly influence hair quality through inflammation and altered sebum composition. Nodulocystic acne and scalp folliculitis can lead to secondary scarring alopecia in severe or untreated cases. Acne also serves as a model for understanding microbe–host interactions in pilosebaceous homeostasis [20].

Seborrheic dermatitis and dandruff represent another spectrum of scalp disorders closely tied to the hair microenvironment. These conditions are characterized by excessive sebum, overgrowth of lipophilic yeasts, and abnormal epidermal turnover, resulting in flaking, erythema, and pruritus. While primarily affecting the scalp skin, seborrheic dermatitis can cause secondary shedding by creating an inflamed, less hospitable niche for follicles. Chronic cases often involve dysregulated immune responses to commensal organisms, supporting the idea that microbial balance is key to maintaining hair and scalp health [21].

Psychological and systemic effects of hair disorders are significant. Hair loss is associated with decreased self-esteem, social withdrawal, and even clinical depression. In adolescents and young adults, acne and seborrheic dermatitis can lead to stigmatization and body image concerns. Systemic conditions such as thyroid dysfunction, anemia, polycystic ovary syndrome, and chronic infections can manifest with hair changes, making hair loss a valuable marker of underlying disease. Understanding hair biology therefore has implications not only for cosmetic concerns but also for systemic health screening [22].

Recent advances in molecular biology have refined our understanding of hair disorders. Dysregulation of growth factor signaling, altered Wnt/β-catenin pathway activity, abnormal immune privilege collapse, oxidative stress, and mitochondrial dysfunction are increasingly recognized as drivers of alopecia. Similarly, the role of skin microbiome diversity and microbial metabolites has emerged as a key determinant of scalp health, influencing both immune tone and sebaceous activity. These findings have shifted therapeutic approaches toward targeted interventions—ranging from anti-androgens, cytokine inhibitors, and platelet-rich plasma to microbiome-modulating agents and regenerative medicine techniques such as stem cell or exosome therapy. This integrative understanding positions hair disorders as multifactorial conditions requiring combined dermatological, immunological, and even psychological management strategies [23].

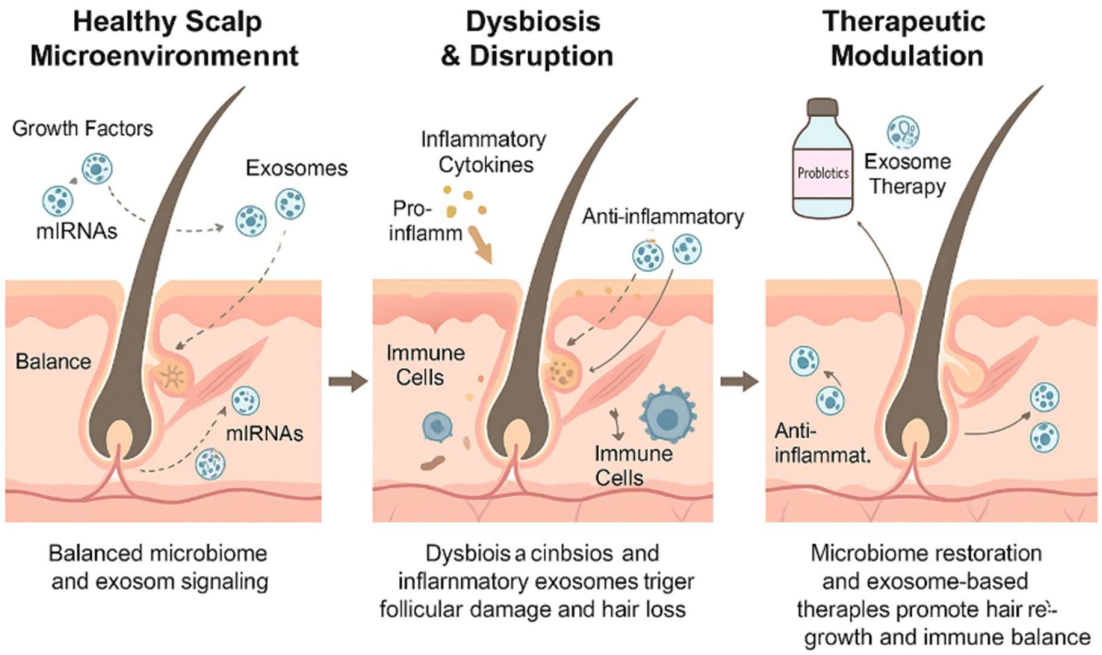

Figure 1: Schematic Representation of Microbiome–Exosome Crosstalk in Scalp Health, Dysbiosis, and Therapeutic Modulation

3. Composition and Function of the Skin Microbiome

The skin microbiome is a dynamic and diverse ecosystem that covers the entire integumentary surface, including the scalp, face, and hair follicles. It is composed of bacteria, fungi, viruses, archaea, and microscopic mites, all of which coexist with host tissues in a finely tuned equilibrium. On the scalp specifically, the microbiome is shaped by sebaceous gland activity, hair density, moisture level, pH, and exposure to cosmetic products. The primary bacterial phyla present include Actinobacteria, Firmicutes, Proteobacteria, and Bacteroidetes, with Cutibacterium, Staphylococcus, and Corynebacterium being dominant genera. These organisms form a symbiotic relationship with the host, engaging in nutrient exchange and immune training. Fungi, most notably Malassezia species, constitute an important proportion of the scalp microbiota, metabolizing sebum lipids and influencing skin barrier physiology [24].

The microbial composition of the scalp is not static but varies by anatomical site, age, hormonal status, and environmental conditions. Sebaceous areas, such as the scalp, favor lipophilic organisms because of the abundance of triglycerides and wax esters in sebum. Moist areas such as skin folds have more diverse populations including Gram-negative organisms, while dry sites harbor distinct communities with lower biomass. Microbial diversity is crucial because a balanced ecosystem can prevent overgrowth of opportunistic pathogens by competitive exclusion and production of antimicrobial peptides. When this balance is disturbed—a state known as dysbiosis—opportunistic species can expand, triggering inflammation or altering barrier function. Dysbiosis

has been linked to conditions such as seborrheic dermatitis, acne, atopic dermatitis, and scalp folliculitis, all of which may indirectly influence hair growth and shedding [25].

The microbiome is not merely a passive resident; it actively participates in skin homeostasis. Commensal bacteria metabolize sebum components such as triglycerides into free fatty acids, which acidify the skin surface and inhibit the growth of pathogenic bacteria. Certain microbes produce bacteriocins and other antimicrobial substances that selectively suppress competitors, maintaining ecological balance. Others interact with keratinocytes and immune cells through pattern recognition receptors such as Toll-like receptors, fine-tuning the immune tone of the skin. Low-level microbial stimulation is essential for immune education, leading to tolerance rather than chronic inflammation. This controlled dialogue helps preserve the immune privilege of the hair follicle, a state in which immune surveillance is dampened to protect the growing follicle from immune attack [26].

Microbiome-host interactions also involve metabolic and structural contributions to barrier integrity. Bacterial metabolites like short-chain fatty acids can enhance tight junction expression, improving skin cohesion and reducing transepidermal water loss. Certain microbial products influence keratinocyte differentiation, affecting desquamation and the rate of hair shaft emergence from follicles. Sebaceous gland activity, in turn, shapes microbial communities by providing lipids that serve as energy sources. A healthy microbiome thus establishes a mutually reinforcing loop: sebum feeds commensals, commensals produce protective metabolites, and the scalp barrier remains intact. Disturbance at any point—excessive sebum production, changes in lipid composition, or external insults like detergents—can shift this balance, enabling pathogenic biofilm formation and inflammation [27].

Fungal members of the microbiome play a critical role, especially on the scalp. Malassezia species are lipophilic yeasts that hydrolyze sebum triglycerides and release free fatty acids, some of which are irritant and can trigger inflammatory responses in susceptible individuals. The density and species distribution of Malassezia have been correlated with conditions such as dandruff and seborrheic dermatitis, where scaling, itching, and erythema are prominent features. However, Malassezia is not always pathogenic; in balanced numbers, it contributes to lipid metabolism and helps shape the immune environment. Dysregulated host responses to commensal fungi rather than mere overgrowth are believed to drive pathology, underscoring the complexity of host–microbe interplay. Other fungi, including Candida and filamentous species, may occasionally be present and become pathogenic under immunocompromised conditions [28].

Viruses and bacteriophages are also part of the cutaneous microbiome, though less studied. Phages can modulate bacterial populations by lysing specific strains, indirectly influencing inflammatory potential. Viral nucleic acids may be sensed by pattern recognition receptors, influencing innate immune responses. Although their precise role in hair biology remains poorly characterized, it is likely that viral communities participate in shaping microbial ecology and immune vigilance. Similarly, Demodex mites inhabit pilosebaceous units and may carry bacteria on their surface, acting as vectors that influence follicular immunity. Their overpopulation has been associated with

inflammatory disorders such as rosacea and could contribute to perifollicular inflammation on the scalp in certain settings [29].

Functionally, the skin microbiome is increasingly recognized as an organ-like system, contributing to metabolic, immunological, and structural functions. It assists in xenobiotic metabolism, degrades environmental pollutants, and produces signaling molecules that can reach systemic circulation. In the context of hair regeneration, the microbiome may influence key pathways such as Wnt/β-catenin and aryl hydrocarbon receptor signaling, both of which are involved in stem cell activation and follicle cycling. Microbial products can act as ligands or cofactors in these pathways, either promoting anagen entry or perpetuating telogen arrest depending on the balance. Understanding these mechanisms has opened avenues for targeted modulation through probiotics, postbiotics, and prebiotic formulations designed to restore microbial diversity and function. Such strategies aim not merely to eradicate pathogens but to reestablish a balanced ecosystem that supports hair follicle vitality [30].

Microbiome Dysbiosis and Hair Disorders

Microbiome dysbiosis refers to an imbalance in the composition, diversity, or functional activity of microbial communities, resulting in disruption of the homeostatic relationship between host and commensals. On the scalp and hair-bearing skin, this imbalance can manifest as shifts in dominant bacterial or fungal species, reduction in diversity, emergence of pathogenic strains, or altered microbial metabolite production. Dysbiosis is now recognized as a key contributor to several common hair and scalp disorders, including alopecia, seborrheic dermatitis, folliculitis, and acneiform eruptions. Rather than a single pathogenic organism, these disorders often represent a complex network of microbial and host factors that collectively tilt the follicular microenvironment toward inflammation, immune dysregulation, or abnormal keratinization [31].

In androgenetic alopecia, there is growing evidence that microinflammation around the follicular bulge contributes to progressive miniaturization. Perifollicular infiltrates consisting of lymphocytes and macrophages have been observed, along with mild fibrosis. Dysbiosis may act as a trigger for this chronic low-grade inflammatory state. Increased colonization by certain Cutibacterium species and reduced levels of beneficial commensals can result in higher levels of lipase activity, liberating pro-inflammatory free fatty acids from sebum triglycerides. These metabolites can stimulate Toll-like receptor pathways, leading to cytokine release that damages the perifollicular extracellular matrix. This hostile microenvironment may shorten anagen duration and push follicles prematurely into catagen, culminating in thinner hair shafts over successive cycles [32].

Alopecia areata represents a different but equally intriguing model of dysbiosis. This autoimmune condition is characterized by the collapse of hair follicle immune privilege, allowing cytotoxic T cells to attack anagen follicles. While genetic susceptibility is well established, environmental triggers are thought to precipitate disease onset. Dysbiotic shifts in scalp or gut microbiota may prime systemic immune responses, producing molecular patterns that cross-react with follicular antigens. Microbial components such as lipopolysaccharides, peptidoglycans, and fungal cell wall glucans can heighten Th1 and Th17 responses, amplifying inflammation.

Additionally, dysbiosis may deplete regulatory taxa that normally promote tolerance, thereby impairing the restoration of immune privilege. Clinical observations of spontaneous remission and relapse suggest that restoring microbial balance could be a therapeutic strategy for maintaining remission [33].

Seborrheic dermatitis and dandruff are among the best-studied scalp disorders linked to dysbiosis. These conditions feature increased density of Malassezia species, particularly those that secrete lipases capable of hydrolyzing sebum into irritating free fatty acids. While Malassezia is a commensal organism, an overabundance coupled with altered host immune sensitivity can initiate inflammation, erythema, and scaling. The resulting increase in epidermal turnover produces characteristic flakes and can disturb the follicular ostia, sometimes leading to hair shedding. Dysbiosis in this context is not limited to fungal overgrowth; bacterial populations can also shift, reducing protective commensals and favoring opportunistic staphylococcal strains that release pro-inflammatory toxins. Such synergistic dysbiosis intensifies scalp irritation and pruritus, further damaging the follicular environment through scratching and barrier breakdown [34].

Folliculitis decalvans and dissecting cellulitis are chronic, scarring follicular disorders strongly associated with bacterial dysbiosis. Biofilm-forming Staphylococcus aureus has been implicated in sustaining persistent inflammation and neutrophil recruitment. Biofilms protect bacteria from immune clearance and antibiotic therapy, creating a reservoir that drives recurrent pustules and sinus tract formation. The prolonged neutrophilic assault leads to follicular destruction and replacement with fibrotic tissue, resulting in permanent hair loss. Understanding the microbial ecology of these conditions has highlighted the importance of targeting biofilm disruption and restoring commensal balance as part of long-term management, beyond mere suppression of acute flares [35].

Acne and scalp folliculitis are also shaped by microbial imbalance. In acne, dysbiosis is marked by overrepresentation of certain virulent Cutibacterium acnes phylotypes that produce pro-inflammatory porphyrins and extracellular enzymes. These virulent strains outcompete health-associated phylotypes, resulting in an inflammatory cascade that recruits neutrophils and promotes follicular rupture. Inflammatory mediators then diffuse to adjacent follicles, perpetuating lesion formation. On the scalp, chronic folliculitis can present with pustules and papules that damage hair shafts and cause temporary shedding. Left untreated, repeated episodes may lead to scarring alopecia. Therapeutic approaches that modulate microbial composition, such as narrow-spectrum antibiotics, probiotics, and bacteriophage-based therapies, are being explored to selectively suppress pathogenic phylotypes while preserving protective commensals [36].

Importantly, dysbiosis is not merely a local phenomenon. The gut–skin axis highlights how intestinal microbial imbalance can influence systemic inflammation and skin immune responses. Increased intestinal permeability, altered metabolite production (such as short-chain fatty acids and tryptophan derivatives), and systemic endotoxemia can prime the immune system, lowering the threshold for cutaneous inflammation. This systemic contribution may explain why some patients with alopecia areata, seborrheic dermatitis, or acne benefit from

dietary modulation and gut-targeted interventions. It also suggests that microbiome restoration may need to occur at multiple sites—scalp, skin, and gut—to achieve durable improvement. Future studies using longitudinal multi-omics profiling will likely clarify whether dysbiosis is a cause, a consequence, or both, in the pathophysiology of hair disorders [37].

4. Exosomes: Structure, Biogenesis, and Function

Exosomes are nanoscale extracellular vesicles that serve as vital mediators of intercellular communication, influencing a wide range of physiological and pathological processes. They typically range from 30 to 150 nanometers in diameter and are characterized by their lipid bilayer structure, which encapsulates bioactive cargo including proteins, lipids, messenger RNAs, microRNAs, and other non-coding RNAs. Unlike apoptotic bodies or larger microvesicles, exosomes originate through a highly regulated endosomal pathway and are released into the extracellular environment as part of normal cell communication. Their small size allows them to circulate in biological fluids such as plasma, saliva, cerebrospinal fluid, and sebum, making them ideal carriers of molecular signals over both short and long distances [38].

The biogenesis of exosomes begins with inward budding of the plasma membrane to form early endosomes. These early endosomes undergo maturation, producing late endosomes that accumulate intraluminal vesicles (ILVs) through inward budding of their limiting membrane. The multivesicular bodies (MVBs) thus formed can follow one of two fates: fusion with lysosomes for degradation or fusion with the plasma membrane to release ILVs as exosomes. This process is orchestrated by a series of molecular complexes including the endosomal sorting complexes required for transport (ESCRT) machinery, as well as ESCRT-independent mechanisms involving tetraspanins, ceramide-rich microdomains, and lipid raft-associated proteins. The cargo loading into ILVs is not random but highly selective, allowing the cell to package specific proteins, nucleic acids, and lipids according to its physiological state or stress conditions [39].

Exosomal cargo composition reflects the phenotype and status of the parent cell. Proteomic studies reveal enrichment of heat shock proteins, tetraspanins (CD9, CD63, CD81), integrins, and cytoskeletal components that facilitate vesicle stability and uptake. Lipid analysis shows high levels of sphingomyelin, cholesterol, and phosphatidylserine, contributing to membrane rigidity and protection of contents against enzymatic degradation. On the nucleic acid side, exosomes frequently carry microRNAs that regulate gene expression post-transcriptionally by targeting messenger RNAs in recipient cells. Because these vesicles are resistant to nuclease activity and physical stress, they can deliver intact regulatory signals to distant targets, making them effective conveyors of information within tissue microenvironments such as the hair follicle niche [40].

Once released, exosomes interact with recipient cells through several mechanisms. They can fuse directly with the plasma membrane, delivering their contents into the cytosol; they can be internalized through endocytosis or micropinocytosis; or they can bind to surface receptors to trigger intracellular signaling cascades without cargo transfer. These interactions can activate multiple pathways relevant to hair follicle biology, including Wnt/β-

catenin for stem cell activation, PI3K/Akt for survival signaling, and VEGF-mediated angiogenesis. In the immune system, exosomes can present antigens, deliver cytokines, or transport immune-regulatory microRNAs, thereby shaping local inflammatory tone. Their ability to cross biological barriers and circulate systemically also raises the possibility of endocrine-like effects, linking distant tissues through vesicular communication [41].

In the context of hair and scalp health, exosomes play a particularly crucial role in maintaining the regenerative capacity of the follicle. Dermal papilla cells secrete exosomes that carry growth factors, extracellular matrix proteins, and stimulatory microRNAs, which activate bulge stem cells and outer root sheath keratinocytes to initiate the anagen phase. Similarly, exosomes from mesenchymal stem cells have been shown to promote neovascularization and supply pro-survival cues to follicular cells, sustaining a robust growth phase. Keratinocyte- and melanocyte-derived exosomes contribute to pigmentation and structural integrity by transferring melanosomes and structural proteins. This orchestration of signals ensures that follicle cycling is synchronized and that hair shafts grow with appropriate thickness and pigmentation [42].

Exosomes also function as carriers of stress and damage signals. Under inflammatory or oxidative stress conditions, keratinocytes and sebocytes release exosomes enriched in damage-associated molecular patterns (DAMPs) and pro-inflammatory mediators. These vesicles can recruit immune cells, amplify cytokine cascades, and exacerbate local tissue damage. In chronic inflammatory scalp conditions, sustained release of such pro-inflammatory exosomes may contribute to microenvironmental deterioration, perifollicular fibrosis, and eventual hair loss. Conversely, exosomes with anti-inflammatory cargo—such as specific microRNAs or suppressor proteins—can help restore immune privilege around the follicle and mitigate excessive immune responses. Thus, exosomes have a dual potential, either aggravating or resolving inflammation depending on their source and context [43].

Beyond local tissue effects, exosomes represent a promising class of biomarkers and therapeutic tools. Because their molecular content mirrors the parent cell's state, analyzing exosomal cargo from scalp sebum, blood, or follicular secretions can provide early diagnostic clues about impending hair cycle disruptions or inflammatory flare-ups. Therapeutically, bioengineered exosomes can be loaded with specific microRNAs, small molecules, or proteins to target signaling pathways implicated in alopecia or seborrheic dermatitis. Advances in nanotechnology are optimizing delivery systems to ensure these vesicles reach the follicular bulge or dermal papilla with high efficiency. The relative biocompatibility, stability, and low immunogenicity of exosomes make them attractive candidates for regenerative medicine, including hair restoration therapies [44].

Role of Exosomes in Hair Regeneration

Exosomes have emerged as critical regulators of hair follicle regeneration due to their ability to transfer bioactive molecules that influence cell proliferation, differentiation, and survival. The hair follicle is a regenerative mini-organ that depends on precise crosstalk between dermal papilla cells, follicular stem cells, keratinocytes, and surrounding immune cells. Exosomes act as the messengers of this communication network, coordinating signals

that determine the initiation, progression, and termination of the hair cycle. In the anagen phase, exosomes derived from dermal papilla cells deliver Wnt ligands, fibroblast growth factors, and microRNAs that activate the canonical Wnt/β-catenin pathway in bulge stem cells, leading to their proliferation and differentiation into matrix keratinocytes [45].

One of the most significant contributions of exosomes is their ability to create a regenerative microenvironment. They promote angiogenesis by carrying vascular endothelial growth factor (VEGF) and other pro-angiogenic mediators, ensuring that the growing follicle receives adequate oxygen and nutrients. Enhanced microvascular density around the follicle correlates with robust anagen maintenance and increased hair shaft thickness. Exosomes also contain extracellular matrix proteins and metalloproteinases that remodel the perifollicular matrix, allowing the follicle to expand and push the hair shaft outward. This combination of stem cell activation and tissue remodeling is central to successful hair regeneration [46].

Stem cell–derived exosomes, particularly those from mesenchymal stromal cells, have demonstrated regenerative potential beyond natural cycling. These vesicles are enriched in anti-apoptotic proteins and immunomodulatory microRNAs that protect follicular keratinocytes from programmed cell death during stress conditions. They can shift the local immune balance toward a tolerogenic state by increasing regulatory T-cell activity and suppressing pro-inflammatory cytokines. This immunomodulation is especially relevant in inflammatory alopecias where immune attack against the follicle disrupts hair growth. By restoring immune privilege and dampening inflammation, exosomes help follicles re-enter the growth phase [47].

Another important role of exosomes is in reversing miniaturization in androgen-sensitive follicles. Miniaturization involves a progressive reduction in dermal papilla size and signaling capacity, resulting in thin, vellus-like hairs. Exosomes enriched with growth factors and microRNAs can counteract this process by stimulating dermal papilla cell proliferation, restoring their ability to secrete anagen-inducing signals. This approach offers a biological alternative to pharmacological treatments such as anti-androgens and vasodilators, potentially with fewer systemic side effects [48].

Exosome-based therapies are also being investigated as adjuncts to conventional hair restoration techniques. When combined with microneedling or follicular unit transplantation, exosomes may enhance graft survival, reduce postoperative inflammation, and accelerate hair regrowth. Their ability to penetrate follicular openings and deliver regenerative cargo makes them suitable for topical application as well, opening avenues for non-invasive treatments. Early clinical data suggest that patients receiving exosome-based formulations show improved hair density and shaft diameter compared to baseline [49].

Beyond direct follicular effects, exosomes may influence the sebaceous gland and surrounding skin, improving the overall scalp environment. By modulating sebocyte activity and normalizing sebum composition, they can reduce conditions that promote microbial overgrowth and inflammation. A healthier scalp environment supports

sustained follicular regeneration and reduces recurrence of shedding episodes. This systems-level impact highlights that exosomes act not only at the follicle but also at the ecosystem level [50].

The potential of exosomes in regenerative dermatology lies in their versatility. They can be harvested from autologous or allogeneic sources, engineered to carry specific cargo, and delivered via minimally invasive routes. However, their efficacy depends on dosage, timing, and stability of preparations. Standardization of isolation techniques, storage protocols, and potency assays is necessary to ensure reproducibility across studies. Regulatory approval will also depend on demonstrating safety and long-term outcomes [51].

Overall, exosomes represent a paradigm shift in hair regeneration research. Their ability to integrate molecular signals, restore immune balance, and promote angiogenesis positions them as a promising frontier for treating various alopecias and scalp disorders. By leveraging their natural role as intercellular couriers, future therapies may achieve sustained and targeted hair regrowth without systemic exposure to drugs. Continued exploration of exosomal cargo, delivery mechanisms, and combination strategies is likely to redefine approaches to hair restoration in the coming years [52].

Microbiome–Exosome Crosstalk

The concept of microbiome–exosome crosstalk has recently gained attention as a unifying framework for understanding the scalp as an integrated ecosystem. Both microbial communities and host-derived exosomes contribute to the regulation of cutaneous homeostasis, immune balance, and follicular health. Rather than functioning as independent systems, these two signaling networks are intertwined, forming a bidirectional communication loop that continuously adapts to environmental, hormonal, and metabolic stimuli. This interaction influences not only the state of the skin barrier but also the behavior of hair follicle stem cells and dermal papilla cells, thereby shaping hair growth patterns and disease susceptibility [53].

Microbes residing on the scalp generate a wide range of metabolites and structural molecules that can act as potent regulators of host cell physiology. Short-chain fatty acids such as acetate and butyrate have anti-inflammatory effects, modulating keratinocyte gene expression and tightening epithelial junctions. Other microbial by-products, including tryptophan derivatives, activate aryl hydrocarbon receptor signaling, which is known to influence keratinocyte differentiation and sebaceous gland activity. These microbial cues can reprogram exosome biogenesis at the level of multivesicular body formation, selectively loading microRNAs and proteins that enhance tissue repair and immune tolerance. The result is a cascade in which microbial balance promotes anti-inflammatory exosome profiles that stabilize the follicular microenvironment and prolong the anagen phase of the hair cycle [54].

Conversely, dysbiosis disrupts this harmony. Overgrowth of lipase-producing organisms leads to excessive release of free fatty acids from sebum triglycerides, which irritate the follicular epithelium and trigger innate immune receptors. Keratinocytes exposed to such stress respond by releasing exosomes enriched in pro-inflammatory cytokines, matrix-degrading enzymes, and danger-associated molecular patterns. These exosomes

recruit neutrophils and macrophages to the perifollicular region, amplifying inflammation in a feed-forward manner. Chronic exposure to such inflammatory exosomes can damage dermal papilla cells, induce perifollicular fibrosis, and progressively shorten anagen duration, culminating in visible thinning and hair loss. This mechanism provides a molecular explanation for the microinflammation observed in conditions like androgenetic alopecia and chronic scalp folliculitis [55].

Sebaceous glands represent a critical intersection point for microbiome–exosome signaling. Sebum provides nutrients for commensal bacteria and fungi, and its composition shapes microbial diversity. When sebum production increases, lipophilic species such as Malassezia flourish, altering the local metabolome. Sebocytes sense these changes and respond by secreting exosomes containing antimicrobial peptides, lipases, and regulators of lipid metabolism. These vesicles can reduce microbial overgrowth, restore lipid balance, and dampen inflammation. In a healthy scalp, this feedback system keeps microbial proliferation in check and prevents excessive immune activation. However, if sebocyte function is impaired or microbial signals overwhelm homeostatic responses, exosomes with inflammatory cargo dominate, leading to erythema, scaling, and pruritus characteristic of seborrheic dermatitis [56].

The immune system adds another layer of complexity to this crosstalk. Commensal microbes normally help maintain hair follicle immune privilege by promoting regulatory T-cell differentiation and secretion of anti-inflammatory cytokines. In turn, immune cells produce exosomes carrying immunosuppressive microRNAs that further protect the follicle from immune attack. Disruption of this balance—whether through infection, stress, or systemic inflammation—can result in a switch toward pro-inflammatory exosomal signaling. Such a shift is implicated in autoimmune hair disorders such as alopecia areata, where cytotoxic T cells target the anagen bulb. Restoring microbial diversity or supplying tolerogenic exosomes could, in theory, re-establish immune privilege and facilitate regrowth [57].

Stem cell niches within the follicle also appear responsive to microbiome-derived cues. Certain bacterial metabolites serve as mitogenic signals that enhance bulge stem cell proliferation. When these metabolites are scarce, follicles may remain in telogen for prolonged periods. Exosomes released by activated stem cells can synchronize neighboring follicles by transferring growth-promoting microRNAs and signaling proteins. This interfollicular communication could explain patterns of synchronized shedding or regrowth observed clinically, suggesting that microbiome–exosome crosstalk operates not only locally but also in a coordinated fashion across the scalp surface [58].

Therapeutically, this axis represents a promising target for next-generation interventions. Probiotics, prebiotics, and postbiotics could be designed to optimize the microbial community toward producing metabolites that favor regenerative exosome profiles. Meanwhile, exosome-based therapies can be engineered to deliver anti-inflammatory or growth-promoting cargo, complementing microbiome modulation. Combination approaches might offer synergistic benefits rebuilding a healthy microbial ecosystem while simultaneously restoring the

vesicular signaling needed to trigger anagen entry. Innovative delivery systems, such as microneedling-assisted penetration or hydrogel-based topical formulations, are being developed to co-deliver microbial metabolites and exosomes directly into the follicular infundibulum, maximizing therapeutic impact.

Future research must integrate multi-omics strategies metagenomics, lipidomics, transcriptomics, and exosome proteomics to decode this complex network in detail. Mapping which microbial signatures correspond to specific exosomal cargo patterns will allow classification of scalp conditions into mechanistic subtypes, enabling precision therapy. Such an approach could transform management of alopecia, seborrheic dermatitis, and chronic folliculitis from empiric trial-and-error treatments to tailored regimens guided by an individual's microbial and exosomal profile [59].

5. Therapeutic Implications and Emerging Treatments

Therapeutic approaches targeting both the skin microbiome and exosome signaling represent a rapidly evolving field that holds promise for more precise and durable management of hair and scalp disorders. Microbiome modulation has shifted from broad-spectrum antimicrobials to more selective strategies that preserve beneficial organisms while suppressing pathogenic overgrowth. Topical probiotics, prebiotics, and postbiotics are being formulated to restore ecological balance and reduce inflammation without inducing resistance. Phage therapy and bacteriotherapy offer species-specific modulation of bacterial populations, and early data suggest they can significantly improve scalp health when combined with gentle keratolytics and sebum regulators [60].

Exosome-based interventions are progressing from experimental models to early clinical use. Mesenchymal stem cell–derived exosomes and dermal papilla cell exosomes are being developed as topical or injectable formulations aimed at stimulating hair follicle stem cells, enhancing angiogenesis, and prolonging the anagen phase. Bioengineered exosomes carrying customized microRNAs or growth factors are also under investigation for reversing follicular miniaturization and modulating immune responses in autoimmune alopecias. Integration with microneedling, laser stimulation, or platelet-rich plasma therapies appears to amplify their regenerative potential and accelerate visible results [61].

Combination therapies represent the most promising frontier. Simultaneous modulation of the microbiome and delivery of regenerative exosomes could address both upstream triggers and downstream follicular damage, offering a more comprehensive solution. Personalized treatment regimens based on scalp microbial sequencing and exosomal cargo profiling are expected to become feasible with advances in rapid diagnostic platforms [62].

Challenges remain in standardizing dosing, ensuring vesicle stability, and meeting regulatory requirements for live biotherapeutics and biologics. Long-term safety data, reproducibility of manufacturing, and accessibility must be established before widespread adoption. Despite these barriers, the convergence of microbiome science and exosome technology is poised to redefine therapeutic strategies for alopecia, acne, seborrheic dermatitis, and related disorders [63,64].

6. Conclusion

The interplay between the skin microbiome and exosome-mediated communication provides a powerful framework for understanding hair biology and associated disorders. Dysbiosis can disrupt immune tolerance and trigger inflammatory cascades, while exosomes act as both mediators of damage and drivers of regeneration depending on their cargo. Together, these systems influence follicular cycling, sebaceous activity, and scalp homeostasis. Therapeutic strategies aimed at restoring microbial balance and harnessing regenerative exosomes hold great promise for treating alopecia, acne, seborrheic dermatitis, and related conditions. Future research integrating multi-omics profiling, standardized exosome production, and microbiome-targeted interventions will be critical for developing precision scalp medicine and advancing safe, effective, and long-lasting solutions for hair health and regeneration.

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