

Stem Cell-Based Therapies for Scarless Skin Repair

Elham Zendedel¹, *Atena Donya², Zahra Barabadi³, Amirhossein Zendeheel⁴

¹ Department of Tissue Engineering and Applied Cell Sciences, School of Advanced Technologies, Shahrekord University of Medical Sciences, Shahrekord, Iran.

² Department of Biology, Faculty of Sciences, Mashhad Branch, Islamic Azad University, Mashhad, Iran.

³ Sabzevar University of Medical Sciences, Sabzevar, Iran.

⁴ Department of Biomedical Engineering, Islamic Azad University of Mashhad, Mashhad, Iran.

Corresponding author: Elham Zendedel, top1360@yahoo.com

Received: 17 Jul. 2026, Revised: 10 Aug. 2026 Accepted: 16 Aug. 2026

Abstract

Scar formation after skin injury remains a significant clinical problem, affecting millions of people each year with important aesthetic, functional and economic sequelae. Mammalian fetal skin regenerates in a scarless manner until about 24 weeks gestation, unlike adult wound healing where it results in avascular, disorganized scar formation, thus providing a developmental model for adult repair. This review summarizes recent advances in stem cell-based therapy for scarless skin regeneration, including cutaneous stem cell niches (EpSCs, HFSCs, MSCs, ASCs), key signaling pathways (Wnt/ β -catenin, YAP, TGF- β /Smad), and novel strategies such as extracellular vesicles and biomaterial-assisted delivery. Preclinical studies have demonstrated the ability of stem cell transplantation to improve wound healing by accelerating wound closure, increasing vascularization and modulating inflammation. Nevertheless, issues of reproducibility, standardization, and inadequate preclinical model systems limit their use in the clinic. Future integration of genome editing, organoids, bioprinting and multi-omics will fill this gap. Overall, stem cell-based approaches represent a paradigm shift from passive anti-fibrosis to active regeneration, with transformative potential for functional skin restoration.

Keywords: Scarless wound healing; Stem cell therapy; Skin regeneration; Fibrosis; Tissue engineering.

1. Introduction

These cells are secreted at the phagocytogenic cytokines, phagocytogenic cytokines. 2. Phagocytosis, the generation of reactive oxygen species (ROS), and the development of neutrophil extracellular traps (NETs), which produce a healing-promoting milieu, are the methods used to destroy the pathogen (3). Each year millions of individuals develop scars following surgery, trauma or burns.(4) and the worldwide burden of scarring is considerable. Scars are classified into four types based on their morphology and pathophysiology: normal scars with collagen aligned in the same direction, atrophic scars with dermal thinning and sparse collagen, hypertrophic scars with excessive deposition of type I collagen and fibroblast proliferation, and keloids with aggressive

fibroproliferation extending beyond the margins of the wound. Keloids are the most severe kind with persistent inflammation, extensive angiogenesis and high recurrence rates. Each form of scar requires different clinical care strategies based on its histological and pathophysiological properties (5).

1 Scar tissue not only leads to cosmetic problems, but also degrades skin and joint function by creating neuropathic discomfort, surface abnormalities, itching, stiffness, and debilitating contracture dysfunction, resulting in a considerable economic burden on society (6). Shifting scar prevention and treatment strategies from passive anti-fibrosis to proactive promoting regeneration guiding wounds toward true structural and functional regeneration emerges as a promising

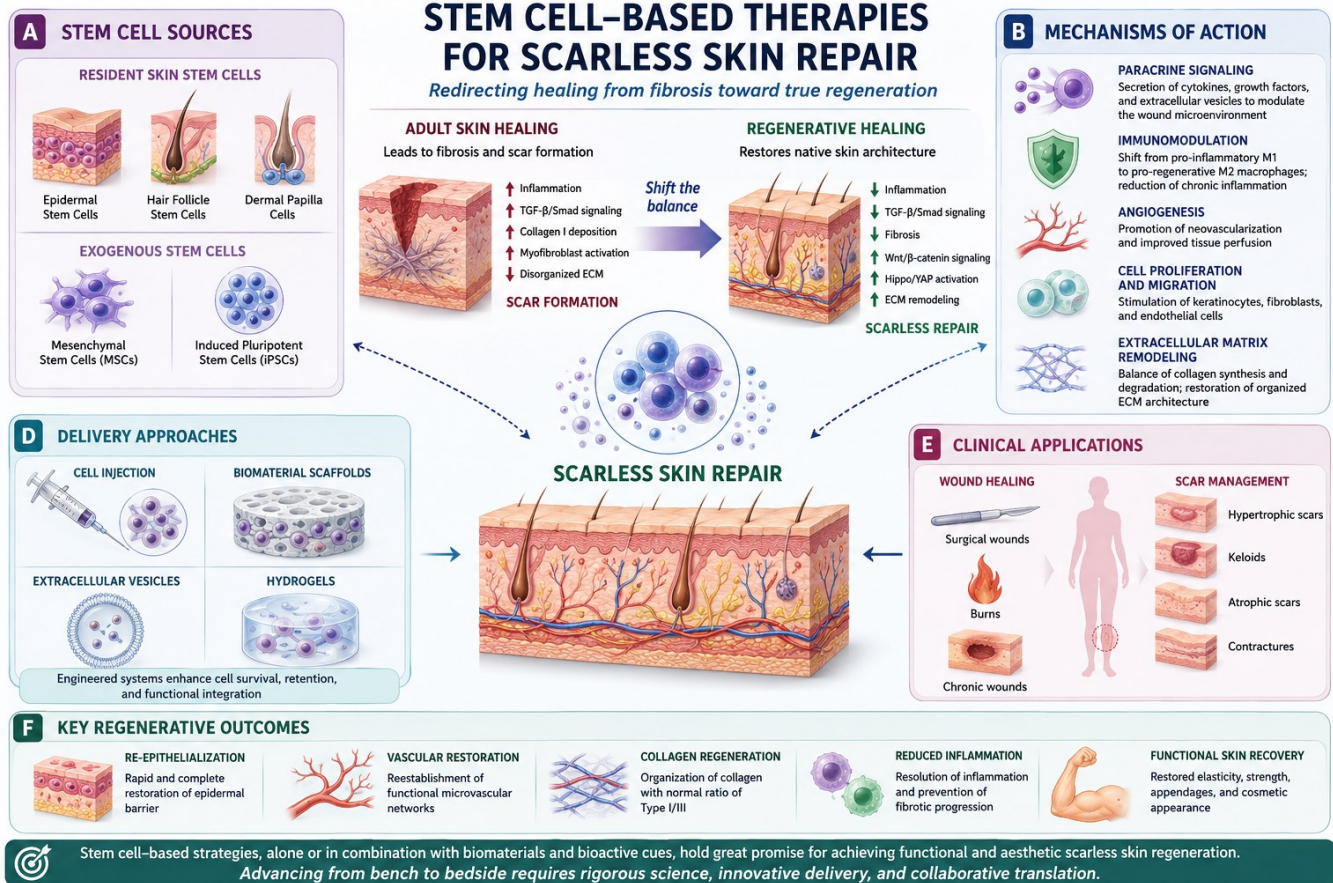


Figure 1. Overview of stem cell-based approaches for scarless skin repair. (A) Stem cell sources; (B) mechanisms of action; (C) regenerative healing process; (D) delivery approaches; (E) clinical applications; and (F) regenerative outcomes

Hair follicle stem cells (HFSCs) are adult stem cells characterized by modest periodicity, multipotent differentiation potential, and robust in vitro proliferation, expressing important markers such as CD34, K15, Gli1, and SOX9. These cells remain quiescent in vivo but exhibit remarkable clonal growth and multilineage differentiation capacity in vitro, enabling them to differentiate into neuronal, glial, melanocytic, and smooth muscle cells, as well as epidermal, hair follicle, and sebaceous gland lineages, thereby actively participating in skin wound healing. The function of the formation of the ar rectification of the formation of the HFSCs, the arrector.(3) During vertebrate terrestrial transition, numerous regeneration-associated genes, including the anamniote-specific ag1, were lost in mammals, contributing to their limited reparative capacity. some study demonstrates that transgenic reintroduction of *Xenopus ag1* into mice accelerates cutaneous wound clo-

sure by ~20% and upregulates scarless-healing markers such as Col3a1, Twist2, and Tnc, indicating functional integration into mammalian repair networks. These findings support the paradigm that resurrecting evolutionarily lost regeneration regulators may offer a viable therapeutic strategy to improve skin healing in mammals, including humans (9). Although there are various methods for scar treatment, due to the complexity and high recurrence rate of scars, scarless wound healing remains difficult to achieve. In addition to traditional treatment methods, emerging biomedical technologies, including stem cell technology and nanotechnology, offer effective alternatives for preventing scar formation .(6)

2. The Fetal Blueprint: Lessons from Scarless Wound Healing

mammalian fetal skin has an extraordinary capacity for scarless regeneration before a certain gestational age. In humans this occurs before about 24 weeks gestation (10).

Fetal skin in early gestational age is able to regenerate and heal rapidly after injury. However, the precise mechanisms and molecular pathways involved in this process are still largely unknown. Skin of early fetus shows many differences compared to skin in later developmental stages, which may provide clues for mechanisms of scarless healing (11). Mammalian skin wound healing is a dynamic, multi-phase process involving hemostasis, inflammation, proliferation, and remodeling. Each phase is driven by different cellular and molecular events including platelet-driven clotting, macrophage polarization from M1 to M2 phenotypes, and fibroblast-mediated ECM deposition. Macrophage phenotype switching and cytokine signaling are critical regulators of the transition from inflammation to proliferation. Re-epithelialization and angiogenesis restore barrier integrity and vascular networks. Ultimately, the outcomes of healing can range from scarless regeneration with reconstitution of functional tissue to fibrotic scarring or chronic wound formation. This is dependent on the severity of the injury and the regulatory dynamics of the healing response.(12) The fetal scarless healing is a developmental blueprint for improving adult wound repair (13). This time window indicates that the loss of regenerative potential is developmentally programmed and may be regulated by epigenetic, immunological, and mechanical switches. A major focus of contemporary research is to understand the switches regulating these processes, as their targeted modulation might allow the reactivation of latent regenerative programs in adult tissues.

3. The Cutaneous Stem Cell Niche: Architecture and Homeostatic Dynamics

The changing microenvironments known as stem cell niches control tissue homeostasis. The primary areas of the epidermal epidermis epidermal epidermal epidermal epidermal stem cells are the epidermal epidermal epidermal epidermal stem cells. Loss of chronological chronological chronological integrity. The juvenile rete epidermal epidermal epidermal epidermal epidermal atrophy. Differentiation Differentiation Differentiation Differentia-

tion .(14) We propose a “niche identity” concept where the follicular niche is determined by five associated structural-mechanical state variables: basement membrane integrity, adhesion apparatuses, fibrillar structure, mechanical set-points, and dynamic remodeling windows. We suggest that via interfacial continuity and the local mechanical state, the collagen network serves as an integrative center that regulates regeneration thresholds and lineage output stability. We postulate that under profibrotic remodeling, inflammation-driven matrix breakdown, and age-related stiffening, these niche elements are displaced coordinately, locking follicles into a low-output state. A niche-based translational framework is proposed to direct future research, and therapeutic relapse is described in terms of “signal–structure mismatch”.(15) Vascular endothelial cells (VECs) are emerging as potential components of the skin epithelial stem cell niche, although their defining signals and physiological contexts remain poorly understood. Skin Skin Skin Skin Skin Skin Skin Skin Skin Skin Skin stage. VEC-derived quiescence-inducing signals can influence hair follicle growth and alter homeostasis. Besides homeostasis, VEC–stem cell crosstalk is also indicated to be involved in the skin’s responses to pathological stressors, revealing an underexplored area that demands additional mechanistic exploration.(16)

4. Major Stem Cell Populations in Skin Regeneration

Stem cell loss of differentiated differentiated dedifferentiated differentiated differentiated differentiated cells. Nonetheless, the young, the reversion, the reversion, the reversal.(17) Adult stem cell therapeutics, regenerative stem cell therapies have evolved. Multiple populations of adult stem or progenitor cells, including mesenchymal stromal or stem cells (MSCs), epidermal stem cells (EpSCs), hair follicle stem cells (HFSCs), and adipose-derived stem cells (ASCs), exhibit properties highly relevant to wound healing, such as self-renewal, multilineage differentiation, immunomodulatory activity, and paracrine signaling. Preclinical investigations and early-phase clinical trials indicate that transplanted adult stem cells can expedite wound closure,

promote neovascularization, control inflammation, and improve tissue remodeling (18).

5. Key Signaling Cascades Governing Scar-Free Versus Fibrotic Healing

Wnt/ β -catenin signaling induces profibrotic gene expression. Histone changes (H3K27 methylation and acetylation by Twist2) influence fibroblast maturation and myofibroblast differentiation. The YAP, EN-1 and Trps1 mechanosensitive pathways are implicated in fibrotic healing. SOX family transcription factors regulate embryonic epidermal differentiation. Fetal fibroblasts exhibit increased ROS detoxification and early apoptotic signaling, in contrast to adult fibroblasts, which overexpress proinflammatory and adhesion-related proteins, thus defining distinct transcriptional states that dictate scarless versus fibrotic outcomes (19). YAP is a mechanosensitive effector of the Hippo pathway that causes fibroblast activation and myofibroblast differentiation in response to matrix stiffening, and persistent YAP activity drives profibrotic gene expression and pathological scarring. While YAP inhibition upregulates Trps1 and activates Wnt signaling, which promotes hair follicle regeneration and reduces ECM deposition, mechanical intervention downregulates integrin-FAK and TGF- β /Smad signaling, lowering fibroblast activation and collagen overproduction. These therapies show a great regeneration potential (20).

6. The Paracrine Revolution: Extracellular Vesicles as Cell-Free Therapeutics

The paracrine revolution positions exosomes as attractive cell-free therapies, yet clinical success is significantly dependent on production platforms. Our data reveal that three-dimensional culture systems significantly outperform the classic 2D approaches in terms of exosome yield, protein cargo enrichment and stemness marker preservation, therefore boosting the pro-angiogenic and regenerative capabilities. Nonetheless, the translation is still requires, brief, delivery is still in vivo, and is still in vivo, linked, half-coupled (21). The maternal immune cargo is derived from

the maternal placenta, shuttle. In physiological pregnancy, this plays a role in immunological tolerance. A change in the exosomal composition toward proinflammatory and antiangiogenic molecules is linked to preeclampsia and FGR, indicating a possible function for these vesicles as mediators of maternal stress to systemic endothelial dysfunction. Preclinical findings reveal that extracellular vesicles from mesenchymal stromal cells can restore placental vascular remodeling, but more mechanistic studies are needed before translation to the clinic.(22)

7. Biomaterial-Based Strategies: Engineering the Niche for Directed Stem Cell Fate

A new strategy to overcome the limitations of single cell therapy is hybrid stem cell therapy which combines different cell types or cells with biomaterials. Co-transplantation of iPSCs and MSCs provides synergistic benefits via complementary mechanisms, while biomaterial scaffolds provide structural and biochemical cues that mimic native tissue. However, it is still a major challenge to ensure the stable construct and optimal properties of the biomaterials to support the behavior of stem cells in vivo .(23) Xue et al. designed a sprayable biomimetic double-layer polysaccharide dressing (MGF@BDD) with the immunomodulatory compound mangiferin to promote wound healing. Inspired by the architecture of human skin, the system self-assembles by sequential spraying into a dense Ca²⁺-Ba²⁺ reinforced top layer providing hemostasis and mechanical protection and a porous bottom layer acting as a pH responsive reservoir for mangiferin release. The dressing exhibited spatiotemporally coordinated bioactivity, including fast anti-inflammatory and anti-oxidative mangiferin release in the inflammatory phase and sustained co-release of mangiferin and Ca²⁺ in the proliferative phase to promote angiogenesis and tissue remodeling. MGF@BDD was demonstrated to speed up wound closure and decrease collagen disorganization in vitro and in vivo, representing an encouraging adaptive strategy for functional regeneration of complex wounds (24).

8. Preclinical Evidence: Successes and Limitations Across Animal Models

Fibromodulin (FMOD), a small leucine-rich proteoglycan, has been shown in studies in animal models (mice, rats, pigs) to be a major driver of regenerative wound healing, improving appearance, reducing scarring, speeding tensile strength recovery and clearing myofibroblasts. Clinical translation, however, is hampered by inefficiencies in production: bacterial/yeast systems are not effective and mammalian cell culture is expensive, variable batch-to-batch, and carries safety risks from adventitious agents. These manufacturing bottlenecks continue to be a major barrier for therapeutic use.(13) In mice, this regenerative capacity is observed before embryonic day 16 (E16) and in fetal lambs, wounds created prior to 120 days of gestation were demonstrated to heal without scarring as early as 1971 (10).

9. Clinical Translation: Trials, Challenges, and Regulatory Landscapes

Despite significant advances, reproducibility, standardization and the limited capacity of animal models to reproduce the full range of human wound pathology are problematic. To close the translational gap and speed the development of effective wound healing therapies, novel biotechnologies need to be integrated with traditional models and ethical research practices must be embraced. This review provides an overview of the current status of preclinical wound healing research, with particular emphasis on traditional models, emerging technologies, assessment techniques, and endeavors to improve the translation of findings for clinical application .(25) Clinical trials play a special role in the process of translating laboratory discoveries into clinical practice. The high investments needed, as well as the strict regulatory restrictions to be complied with, usually lead to clinical trials only after a long period of pre-clinical research that has demonstrated great efficacy and fulfilled strict safety requirements (26). When treating burns, it is better to add fibroblasts to the keratinocytes. Early culture systems used irradiated mouse 3T3 feeder

layers because keratinocytes need the presence of fibroblasts to grow. Feeder-free media systems have been developed, but few have been accepted clinically; cell sprays and other alternatives have allowed earlier initiation of treatment. Defined clinical-grade media now make it possible to culture keratinocytes from patient biopsies efficiently in approximately 10 days, enabling earlier therapy and improved clinical translation (27).

10. Genome Editing, Organoids, and In Situ Reprogramming

Emerging Technologies and Their Intersection in In Vitro Wound Healing Research: A Revolution. Multi-omics, the integration of transcriptomic, proteomic, and metabolomic data, can provide a more systems-level understanding of tissue repair beyond static analyses of cellular behavior)

.(28)Nevertheless, new technologies are bringing experimental models closer to human physiology. Three-dimensional (3D) bioprinting enables the creation of complex tissue architectures with spatially organized cells and biomaterials (29). Microfluidic “wound-on-a-chip” platforms enable precise control of mechanical and biochemical stimuli with real-time monitoring (30). In parallel, iPSC-derived models provide opportunities for personalized wound healing and targeted therapy testing. Advances in computational tools and in silico modeling also support system design, outcome prediction and interpretation of high dimensional data (31).

11. Conclusion

Scarless wound healing is one of the most challenging and clinically important goals in regenerative medicine. This review summarizes the present status of stem cell-based therapies for skin repair, and is grounded in developmental insights into fetal scarless healing, cutaneous stem cell niche organization, and the distinct populations of adult stem cells (MSCs, EpSCs, HFSCs, and ASCs) that collectively provide multifaceted mechanisms of action by self-renewal, differentiation, immunomodulation, and paracrine signaling. Identification of critical signaling cascades such as Wnt/ β -catenin, YAP-mediated mechanotransduc-

tion and TGF- β /The Smad pathways have allowed for the molecular basis distinguishing scar-free regeneration and pathological fibrosis to be uncovered and have presented rational targets for therapeutic intervention. Furthermore, the emergence of extracellular vesicles as cell-free therapeutics and biomaterial-based strategies for engineering the stem cell niche has expanded the therapeutic arsenal beyond conventional cell transplantation, addressing critical limitations in stability, scalability, and delivery. Promising results have been demonstrated in preclinical studies using animal models, but there are still hurdles to overcome in clinical translation, such as reproducibility, standardization, and the inability of experimental systems to fully mimic human wound pathology. Looking forward, the integration of cutting-edge technologies genome editing, three-dimensional bioprinting, wound-on-a-chip platforms, organoid models, and multi-omics approaches holds transformative potential to bridge the translational gap. Taken together, these advances suggest a paradigm shift from passive anti-fibrotic management to proactive and programmable pro-regenerative strategies. While significant challenges remain, the convergence of stem cell biology, biomaterials science and bioengineering offers an unprecedented opportunity to realize the long-standing dream of scarless skin repair, restoring not only barrier integrity but the structural and functional complexity of native tissue.

Acknowledgments: The authors acknowledge the contributions of the scientific community whose foundational work has made this review possible. No specific funding was received for this manuscript.

Conflict of Interest Statement : The authors declare no competing financial interests.

References

- Huang Y, Wu X, Xu Y, Yang N, Xi P, Wang Y, et al. Organoids/organs-on-chips towards biomimetic human artificial skin. *Burns & Trauma*. 2025;13:tkaf029. doi:10.1093/burnst/tkaf029.
- Teertam SK, Setaluri V, Ayuso JM. Advances in micro-engineered platforms for skin research. *JID Innovations*. 2025;5(1):100315. doi:10.1016/j.xjidi.2024.100315.
- Yang Y, Cheng Q, Hu Y, Zhang Y, Ran N, Yang J, et al. Spatiotemporal immunomodulation by engineered hair follicle stem cell-derived nanovesicles enables scarless regeneration of infected wounds. *Chemical Engineering Journal*. 2026;172657. doi:10.1016/j.cej.2026.172657.
- Panda D, Nayak S. Stem cell-based tissue engineering approaches for diabetic foot ulcer: a review from mechanism to clinical trial. *Stem Cell Reviews and Reports*. 2024;20(1):88-123. doi:10.1007/s12015-023-10640-z.
- Chai H, Wang Z, Ju J, Sun J. Multifunctional hydrogel-based wound dressings for scar-free healing: Design principles, therapeutic mechanisms, and clinical translation challenges. *Advanced NanoBiomed Research*. 2026;6(1):e202500147. doi:10.1002/anbr.202500147.
- Wang X, Xu Z, Lv Y, Xiang D. Mesenchymal stem cells-derived exosomes: Next-generation nanomedicines toward scarless wound healing. *International Journal of Nanomedicine*. 2026;586955. doi:10.2147/IJN.S586955.
- Fu M, Huang Z, Li J, Li Y, Fang Z, Jiang Y, et al. Approaching scarless wound healing: From passive anti-fibrotic to proactive and programmable pro-regenerative strategies. *Advanced Science*. 2026;13(18):e21824. doi:10.1002/advs.202521824.
- Cao X, Wu X, Zhang Y, Qian X, Sun W, Zhao Y. Emerging biomedical technologies for scarless wound healing. *Bioactive Materials*. 2024;42:449-477. doi:10.1016/j.bioactmat.2024.09.001.
- Kosykh AV, Zhigmitova EB, Evtushenko NA, Gurskaia TH, Martynova AA, Silaeva YY, et al. Restoring Ag1, an ancient regeneration gene lost in amniotes, accelerates skin healing in mice. *Frontiers in Cell and Developmental Biology*. 2026;14:1706902. doi:10.3389/fcell.2026.1706902.
- Huang L, Ye X, Zhang Y, Ho C-K, Li Q. Impact of different mating and surgical protocols on the establishment of a mouse model for fetal scarless skin healing. *Biological Procedures Online*. 2025;27(1):38. doi:10.1186/s12575-025-00290-5.
- Gomes MLNP, Krijnen PA, Middelkoop E, Niessen HW, Boekema BK. Fetal skin wound healing: Key extracellular matrix components and regulators in scarless healing. *Journal of Investigative Dermatology*. 2025;145(2):280-302. doi:10.1016/j.jid.2024.05.027.

12. Agramunt J, Kang Y, Rinkevich Y. Spatiotemporal dynamics of mammalian wound healing. *Cell Discovery*. 2026;12(1):4. doi:10.1038/s41421-025-00865-2.
13. Zheng Z, Ha P, Li C, Chang GX, Jiang W, Pang X, et al. Preclinical efficacy and safety study of a novel dermal fibroblast modulating drug, SLI-F06, in cutaneous wound healing. *MedComm*. 2026;7(6):e70761. doi:10.1002/mco2.70761.
14. Fang R, Hamaguchi R, Xu S, Lee W, Todorova K, Sol S, et al. Rete ridge topography as a determinant of epidermal stem cell identity: Implications for skin aging. *bioRxiv*. 2026:2026.04.07.716516. doi:10.64898/2026.04.07.716516.
Note: This is a preprint DOI, not a peer-reviewed journal DOI.
15. Jiang Z, Xu Y, Lou Y. From a stem-cell-centered to a niche-centered view: The core role of collagen networks in hair loss and hair follicle miniaturization. *Frontiers in Cell and Developmental Biology*. 2026;14:1824126. doi:10.3389/fcell.2026.1824126.
16. Tumber T, Ganguly T, McMahon CE, Tavallaei MA. Vascular endothelial cells as signaling niches for epithelial stem cells in the skin. *Frontiers in Cell and Developmental Biology*. 2026;14:1781544. doi:10.3389/fcell.2026.1781544.
17. Dimri-Wagh S, Bhattacharya S, Yassen G, Shorer O, Amitai-Lange A, Altshuler A, et al. Aged differentiated cells reverse into native stemness-like state by niche cytokines to sustain lifelong homeostasis and tissue repair. *Nature Communications*. 2026;17:5743. doi:10.1038/s41467-026-72331-w.
18. Lei R, Li G, Li N, Lin S, Chen Q, Yu X, et al. Adult stem cell therapy for skin wound healing: A translational perspective from bench to bedside. *Regeneration Repair Rehabilitation*. 2026. doi:10.1016/j.rerere.2026.06.003.
19. Niti A, Kouzi-Koliakou K, Michopoulou A. Fibroblast lineage switching as the developmental origin of scarring and target for regenerative healing. *Biology*. 2026;15(5):409. doi:10.3390/biology15050409.
20. Kang Y, Yang Y, Yao B, Zhang Z, Ji X, editors. Scar inhibition in wound healing: Mechanisms, design, and recent advances. *Exploration*. 2026;6(1):20240517. doi:10.1002/EXP.20240517.
21. Elakkawi MM, Zhu Y, Xiong Y, Li C, Li H, Chen J, et al. Study on the mechanism of hUCMSCs-derived 3D exosome combined with HAMA microneedles in the treatment of deep second-degree burns. *Bioactive Materials*. 2026;56:468-482. doi:10.1016/j.bioactmat.2025.09.031.
22. Miranda J, Maestre N, Devia M, Zapata R, Ochoa-Díaz MM, Annicchiarico W. Inflammation at the maternal–fetal interface: Mechanisms linking maternal–fetal immunity to preeclampsia and fetal growth restriction. *International Journal of Molecular Sciences*. 2026;27(9):3954. doi:10.3390/ijms27093954.
23. Choudhery MS, Arif T, Mahmood R. Aging puzzle: A closer look on the complex dilemma of autologous stem cell therapy. *World Journal of Stem Cells*. 2026;18(1):114119. doi:10.4252/wjsc.v18.i1.114119.
24. Xue Y, Lin Y, Lu Y, Zhang H, Chen B, Zhao Y, et al. Sprayable polysaccharide-based biomimetic double dressing with synergistic regulation for wound healing. *Biomaterials*. 2026;335:124328. doi:10.1016/j.biomaterials.2026.124328.
25. Jeyaraman N, Roy M, Muthu S, Khan S, Jeyaraman M. Preclinical insights into wound healing: Models, mechanisms, and translational strategies. In: *Management and Strategies for Wound Healing*. Springer; 2026. p. 287-309. doi:10.1007/978-3-032-12442-5_12.
26. Gultepe E, Valluru R, Brown NB, Sridhar S. The landscape of nanomedical clinical trials. *Nano Today*. 2026;66:102898. doi:10.1016/j.nantod.2025.102898.
27. Liao Z, Chen X, Hirt-Burri N, Porcello A, Marques C, Lourenço K, et al. Pharmacological and biological strategies as regulators of scarless wound healing and regeneration of skin. 2025. doi:10.5772/intechopen.1012960.
Note: This is a book-chapter DOI, not a journal-article DOI. The reference should therefore be reformatted as a book chapter.
28. Ceylan S, Demir D, Harris C, İpek SL, Vavourakis V, Manca M, et al. Skin in the game: A review of computational models of the skin. *BioData Mining*. 2025;18(1):55. doi:10.1186/s13040-025-00471-8.
29. Zhang M, Zhang C, Li Z, Fu X, Huang S. Advances in 3D skin bioprinting for wound healing and disease modeling. *Regenerative Biomaterials*. 2023;10:rbac105. doi:10.1093/rb/rbac105.
30. Biglari S, Le TY, Tan RP, Wise SG, Zambon A, Codolo G, et al. Simulating inflammation in a wound microenvi-