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MINI REVIEW

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Advancements in cutaneous gene editing: The role of CRISPR technologies

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ABSTRACT

The discovery of Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) and associated Cas nucleases has revolutionized gene editing by enabling precise, efficient, and cost-effective modifications to the human genome. In dermatology, CRISPR has emerged as a transformative tool, offering potential cures for a range of inherited and acquired skin diseases. Genodermatoses such as Epidermolysis Bullosa (EB) and Ichthyosis have been among the first to benefit from ex vivo correction using CRISPR-Cas9, demonstrating restored gene function in keratinocytes and fibroblasts.

Beyond genetic disorders, CRISPR has been applied to target cutaneous pathogens. Studies have successfully disrupted viral DNA in models of human papillomavirus (HPV) and herpes simplex virus (HSV), suggesting a role in eradicating persistent skin infections. In melanoma research, CRISPR has facilitated gene knockout screens to identify novel therapeutic targets, including tumor suppressors and immune evasion pathways.

Despite its promise, CRISPR-based therapies face hurdles such as off-target effects, immune responses to Cas proteins, and challenges in delivering gene editors to skin cells in vivo. Advances in delivery vectors—like lipid nanoparticles and microneedle patches—alongside high-fidelity Cas variants, are helping overcome these barriers. Continued innovation may soon make CRISPR a clinical reality in dermatology.

KEYWORDS

CRISPR; Dermatology; Gene Editing; Genodermatoses; Viruses; Cutaneous Disease; Viral Skin Infections; High-Fidelity Cas Variants

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Introduction

Gene editing technologies have significantly advanced the field of biomedical research and offer promising opportunities for understanding and treating both inherited and acquired diseases. Among these, the CRISPR system has emerged as a powerful tool, utilizing programmable RNA-guided Cas (CRISPR-associated) nucleases to precisely alter, delete, or insert genetic material at specific genomic sites [1]. Compared to earlier gene-editing platforms such as zinc finger nucleases (ZFNs), meganucleases (MNs), and transcription activator-like effector nucleases (TALENs), the CRISPR-Cas system stands out for its simplicity, efficiency, and ease of design, owing primarily to its customizable guide RNA sequences [2].

Dermatologic diseases represent especially attractive targets for CRISPR-Cas–based therapies. Many well-characterized monogenic skin disorders, including epidermal blistering conditions, present ideal opportunities for gene correction strategies [3]. Moreover, the skin's accessibility allows for convenient harvesting and in vitro culture of target cells, as well as direct delivery of therapeutic agents via topical application, grafting, or injection. Additionally, because of its external visibility, the skin enables straightforward monitoring of treatment outcomes and detection of adverse effects.

Current research in this area is rapidly evolving, with both ex vivo and in vivo approaches under investigation. Dermatology, given its unique characteristics, is positioned at the forefront of translational applications for CRISPR therapies. Notably, one of the earliest human clinical trials involving CRISPR-Cas9 focuses on treating resistant forms of melanoma [4,5]. This review explores the ongoing developments and future prospects of CRISPR-Cas technology in the field of dermatology.

Mechanisms of Genome Engineering with CRISPR-Cas

Several types of CRISPR-Cas systems (Types I–III and their subtypes) have been discovered across bacterial and archaeal species, but the Type II CRISPR-Cas9 system is by far the most extensively studied, particularly in therapeutic research including dermatology. In its natural bacterial context, the Type II CRISPR-Cas system functions as an adaptive immune mechanism, enabling bacteria to defend against foreign DNA from viruses and plasmids [6]. When bacteria encounter foreign genetic material, they integrate short fragments of the invader's DNA into their own genome. These sequences are later transcribed into CRISPR RNAs (crRNAs), which pair with trans-activating crRNAs (tracrRNAs) to guide the Cas9 protein to a matching target in double-stranded DNA, resulting in a precise double-strand break (DSB).

In laboratory settings, this system is simplified through the use of a single-guide RNA (sgRNA), which mimics the crRNA-tracrRNA complex and directs Cas9 to the desired genomic site. The sgRNA can be easily customized, enabling researchers to target a wide range of genes with relative ease and scalability. Once a DSB is created in eukaryotic cells, the break can be repaired via two primary cellular mechanisms: non-homologous end joining (NHEJ) or homology-directed repair (HDR) [7,8]. NHEJ is error-prone and often leads to

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insertions or deletions that disrupt gene function, while HDR offers more precise editing using a DNA template to correct specific mutations. However, HDR is typically less efficient than NHEJ, and improving its reliability remains an active area of research.

In dermatology, most CRISPR gene-editing strategies to date have utilized ex vivo approaches, wherein patient-derived cells are genetically modified outside the body before being reintroduced through autologous transplantation. This method allows for the careful screening and expansion of successfully edited cells, improving safety and efficacy [9]. However, challenges such as cellular differentiation during expansion-particularly with induced pluripotent stem cells (iPSCs)-and the technical complexity of cell-based transplantation limit widespread application (Figure 1).

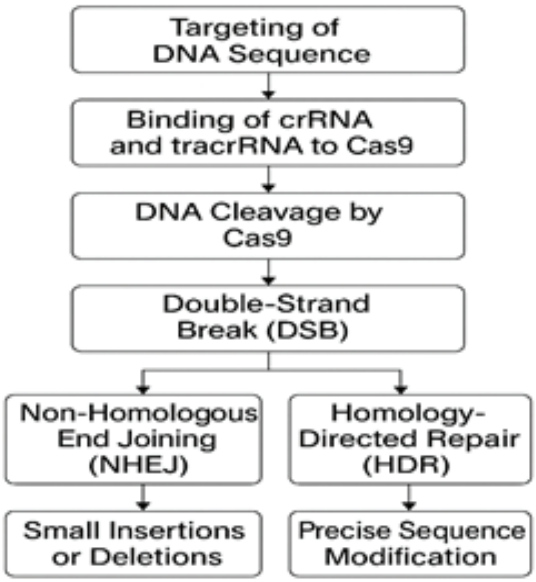

Figure 1. Mechanisms of genome engineering with CRISPR-Cas9

Alternatively, in vivo gene editing targets somatic cells directly within the body through systemic or localized delivery of CRISPR-Cas components (as DNA, RNA, or protein). While this approach holds promise for treating both localized and systemic skin conditions, it presents significant challenges in delivery specificity, safety, and the ability to monitor off-target effects. Continued development of precise and safe in vivo delivery systems is essential for translating CRISPR-based treatments into clinical practice.

Genodermatoses

Many genodermatoses are monogenic, making them prime candidates for gene therapy due to their single-gene defects and lack of effective treatment options. Current treatments mainly aim to alleviate symptoms. However, initial success with gene therapy in inherited epidermolysis bullosa (EB) has opened the door to the development of curative approaches. In 2006, a patient with nonlethal junctional EB (JEB) received successful long-term skin grafts using autologous keratinocytes corrected with a retroviral vector encoding the beta 3 subunit of laminin-332 [10].

This marked a transition from traditional gene therapy, which typically involves random integration of genes, to precise genome editing via nucleases like ZFNs, TALENs, and CRISPR-Cas systems. CRISPR-Cas allows for targeted gene additions, mutation corrections, and the removal of faulty sequences. Dominant disorders like EB Simplex (EBS) and dominant dystrophic EB (DDEB) particularly benefit from CRISPR's ability to disrupt disease-causing alleles [11]. For instance, CRISPR-Cas9-induced HDR was used to repair mutated KRT14 in EBS and modify the COL7A1 gene in DDEB to express a non-harmful version of collagen 7.

In contrast, autosomal recessive forms like JEB and recessive dystrophic EB (RDEB) require gene correction to restore protein function. This has been demonstrated using HDR or exon skipping techniques. Exon 80 of COL7A1 is a common mutation site in RDEB. Targeted excision using Cas9/sgRNA RNPs restored collagen 7 expression and adhesion properties in grafted keratinocytes and in vivo mouse models [12,13].

Despite promising outcomes, limitations such as low targeting efficiency, absence of long-term data, and potential off-target effects remain. Moreover, electroporation in human skin, especially in fragile EB patients, presents technical and safety challenges.

Epidermolytic palmoplantar keratoderma (EPPK), a dominantly inherited keratin disorder characterized by thickened skin on palms and soles, is caused by mutations in KRT9. Luan et al. demonstrated phenotypic improvement in an EPPK mouse model using lentiviral vectors delivering CRISPR-Cas9 targeting the mutant KRT9 allele [14]. Treated mice showed normalized skin structure and reduced mutant protein levels. However, limited off-target analysis and unassessed long-term safety and immune response highlight the need for further research.

Cutaneous viruses

CRISPR-Cas systems, originally evolved in bacteria as a defense mechanism against invading bacteriophages, have been adapted to similarly combat viral infections in human cells. Infected human cells harboring latent viruses, which often evade immune detection and resist conventional antiviral treatments, may be targeted by CRISPR-Cas enzymes. These systems allow precise targeting of viral genomic sequences, enabling the disruption or complete removal of viral DNA segments [15]. Beyond therapy, certain Cas enzymes have also been utilized for highly sensitive viral detection in human tissue samples.

For example, Cas12 and Cas13 enzymes, known for their ability to indiscriminately cleave single-stranded DNA once activated by a specific target sequence, have enabled ultra-sensitive viral diagnostics platforms such as DETECTR, SHERLOCK, and HOLMES. While much antiviral CRISPR research has focused on systemic viruses without primary skin involvement, the skin's accessibility makes CRISPR-based therapeutics and diagnostics particularly promising for treating cutaneous viruses. Current studies indicate CRISPR-Cas technologies could effectively target human papillomavirus (HPV), herpes simplex virus (HSV), and Kaposi sarcoma-associated herpesvirus (KSHV).

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HPV is a double-stranded DNA virus that infects the basal layer of stratified epithelium, integrating its genome into host DNA. High-risk HPV strains (like 16, 18, 31, and 33) express E6 and E7 proteins that promote malignant transformation by disabling tumor suppressors p53 and Rb, leading to cancers such as anogenital squamous cell carcinoma [16]. Low-risk strains, including types 6 and 11, cause benign lesions like genital warts through E7-mediated uncontrolled epithelial cell growth. Researchers have successfully used CRISPR-Cas9 to disrupt E6 and E7 genes in cervical cancer cells both in vitro and in animal models. A pioneering clinical trial will soon test in vivo targeting of HPV E6/E7 in cervical neoplastic cells. While fewer studies have targeted dermatological HPV manifestations, there is emerging work on CRISPR constructs aimed at HPV-associated anal cancer and genital warts [17]. For instance, using adeno-associated viral vectors, researchers delivered Cas9 with guide RNAs targeting HPV-16 E6 and E7 to reduce tumor size in a mouse model of HPV-16 anal cancer. In vitro work also showed partial disruption of HPV-6 and -11 E7 genes in keratinocyte lines, though complete in vivo validation remains pending.

CRISPR-Cas systems hold promise not only for therapy but also for diagnostics. The DETECTR platform employs Cas12a with a fluorescent reporter to detect HPV DNA in patient samples with remarkable sensitivity and speed, distinguishing viral genotypes within an hour, making it a potential rapid and affordable point-of-care diagnostic tool [18].

Herpesviruses, including HSV-1, HSV-2, and KSHV, are large double-stranded DNA viruses that establish lifelong latent infections. HSV-1 and HSV-2 infect oral and genital mucosa causing ulcers, then enter latency in sensory ganglia as episomal DNA. KSHV infects endothelial cells and causes Kaposi's sarcoma. Latent herpesviruses evade immune clearance by minimizing viral gene expression, making them resistant to antiviral drugs [19]. CRISPR-Cas9 offers an alternative by directly targeting viral DNA. In vitro, CRISPR has been used to effectively halt HSV-1 replication by disrupting essential viral genes in fibroblasts and other cell types without significant off-target effects. Similarly, CRISPR has reduced KSHV levels in latently infected cells by targeting the viral latency-associated nuclear antigen (LANA).

However, challenges remain. While active HSV-1 replication can be inhibited, completely eradicating latent HSV-1 in neurons is more difficult, likely due to epigenetic modifications that hinder Cas9 access. Although other nucleases have shown success against latent HSV, CRISPR's efficacy here needs further validation. Additionally, in vivo demonstrations of CRISPR-based treatments for HSV and KSHV are still in early stages. Fortunately, HSV latency is confined to specific ganglia, potentially simplifying targeted delivery methods.

Cutaneous bacterial infections

Beyond its antiviral potential, CRISPR-Cas technology is also emerging as a powerful tool to combat drug-resistant bacterial infections. The rise of antibiotic-resistant bacteria is a pressing public health issue, exacerbated by the continued overuse of antibiotics and a slowing pace in the development of new antimicrobial agents. Recently, CRISPR-Cas-based antimicrobials have been investigated as a next-generation solution for bacterial infections. Notably, CRISPR-Cas9 can be engineered to specifically target and eliminate genes that confer antibiotic resistance, effectively restoring bacterial susceptibility to existing treatments [20].

While systemic delivery of CRISPR antimicrobials remains complex, the skin offers a unique advantage due to its accessibility. This has made cutaneous bacterial infections a key area of focus in CRISPR-based antimicrobial research, especially through the use of topical formulations.

Staphylococcus aureus, a major skin pathogen, is well-known for its resistance to antibiotics. It is responsible for the majority (76%) of skin and soft tissue infections and contributes significantly to patient morbidity and mortality. Its resistance arises from its capacity to acquire plasmids and other mobile genetic elements that carry genes for antibiotic resistance and virulence [21]. Adding to the challenge, S. aureus commonly exists as a silent colonizer in 20–30% of healthy individuals, particularly in the nasal passages, enabling frequent outbreaks.

In a notable study, Bikard and colleagues developed a targeted CRISPR-Cas9 approach to eliminate resistant strains of S. aureus. They designed guide RNAs (gRNAs) to direct Cas9 to cut specific antibiotic resistance genes, such as mecA, which confers resistance to methicillin. Delivered via a phage-based system, these constructs selectively killed resistant bacteria and removed resistance-carrying plasmids in vitro. Moreover, topical application in a mouse model of skin colonization led to a marked reduction in resistant S. aureus populations, offering proof-of-concept for the use of CRISPR antimicrobials on the skin.

This research also opens up possibilities for multiplexed CRISPR antimicrobials, which could simultaneously target multiple resistance genes or different bacterial species. Importantly, these technologies may also influence the cutaneous microbiome, a growing area of interest in dermatological research [22]. Studies using metagenomic sequencing have linked imbalances in the skin microbiome, such as reduced microbial diversity and increased S. aureus presence, to conditions like atopic dermatitis. While CRISPR's use in treating atopic dermatitis has not yet been fully explored, targeting pathogenic S. aureus strains could potentially support treatments aimed at boosting beneficial microbial communities on the skin.

Melanoma

Some of the earliest clinical applications of CRISPR-Cas technology in humans have focused on cancer immunotherapy, particularly for conditions like melanoma and non-small-cell lung cancer (NSCLC). A key objective in these trials has been the gene editing of immune checkpoint regulators, such as programmed cell death protein-1 (PD-1) and cytotoxic T-lymphocyte-associated protein-4 (CTLA-4)-two proteins that naturally suppress T cell-mediated anti-tumor activity.

Melanoma, known for its strong immunogenicity due to a high mutation rate and the resulting production of neoantigens, is especially vulnerable to immune system attacks under ideal

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conditions [23]. However, in practice, the melanoma tumor microenvironment is highly immunosuppressive, and advanced-stage disease often responds poorly to standard treatments. This makes melanoma a prime candidate for innovative immunotherapies aimed at overcoming immune suppression.

One of the first CRISPR-Cas-based clinical trials for melanoma builds upon previous successes using PD-1 inhibitors and T cells engineered to express the NY-ESO-1 T-cell receptor (TCR). In this approach, patient-derived autologous T cells are genetically modified in two major ways. First, they are transduced with a lentiviral vector encoding the NY-ESO-1 TCR, enabling them to specifically recognize melanoma cells expressing the NY-ESO-1 antigen. Second, the same T cells are electroporated with CRISPR-Cas9 components designed to knock out genes encoding PD-1 as well as endogenous TCRα and TCRβ subunits.

This combined gene-editing approach boosts the T cells' ability to target and eliminate tumor cells. By eliminating PD-1 expression, the modified cells evade the tumor's immune suppression. Knocking out the native TCRs also reduces the risk of the engineered cells mistakenly targeting healthy tissues due to unintended antigen recognition. Once reintroduced into the patient, these tailored T cells are expected to mount a stronger, more focused immune response against melanoma cells expressing NY-ESO-1 [24].

Importantly, this method could also reduce off-target immune-related side effects. Unlike systemic PD-1 inhibitors that affect all T cells in the body, CRISPR-Cas9 editing confines PD-1 disruption to the engineered T cell population. This localized effect may offer a safer, more precise immunotherapy alternative with reduced systemic immune complications.

Perspectives and Future Directions

Collectively, a growing body of research highlights the potential of CRISPR-based therapies for treating genetic skin disorders, cutaneous infections, and melanoma. Ongoing and future investigations are expected to refine and broaden the clinical applicability of these therapies, expanding their reach to additional dermatologic conditions.

Many additional genetic skin disorders and cutaneous infections may be effectively addressed using CRISPR-Cas technology. Conditions like pachyonychia congenita and xeroderma pigmentosum, previously addressed with RNA interference and designer nucleases respectively, are strong candidates for CRISPR-mediated gene editing. Certain rare forms of EB, characterized by multiple simultaneous mutations, may be treatable using CRISPR systems designed to edit multiple genomic sites simultaneously.

The emergence of hypoimmunogenic universal donor induced pluripotent stem cells (iPSCs)—which are CRISPR-engineered to evade host immune detection—may enhance the accessibility of ex vivo gene editing strategies for genodermatoses. Although concerns persist regarding the tumorigenic risk of iPSCs, preclinical studies using CRISPR-modified iPSCs in a mouse model of recessive dystrophic EB (RDEB) showed no evidence of tumor formation. Improvements in differentiation protocols and the exclusion of potentially oncogenic cells could further mitigate these risks, supporting their future clinical use [25].

Gene editing holds significant promise for targeting cutaneous viruses, with Merkel-cell polyomavirus (MCPyV) and human T-cell leukemia virus type 1 (HTLV-1) standing out as key candidates. Merkel-cell polyomavirus (MCPyV), which accounts for most cases of Merkel cell carcinoma (MCC), integrates unpredictably into the genome of tumor cells. Removing MCPyV DNA using CRISPR-Cas9 could offer a new approach to treating this aggressive cancer, which is often resistant to conventional therapies. Early work has demonstrated that targeting MCPyV tumor antigens with CRISPR-Cas9 significantly reduces tumor cell proliferation in vitro.

HTLV-1, a retrovirus associated with adult T-cell leukemia/lymphoma and tropical spastic paraparesis, remains an unexplored target for CRISPR therapy [26]. However, its structural similarity to HIV—another retrovirus successfully targeted in CRISPR studies—suggests that HTLV-1 could also be eliminated from infected cells. Its relatively stable genome, compared to the highly variable HIV genome, makes it a particularly suitable candidate for CRISPR-based intervention.

As CRISPR-engineered T cell therapies continue to evolve, clinical trials are expected to assess new generations of more precise and effective treatments for melanoma. These include TCR-transduced and chimeric antigen receptor (CAR) T cells edited with CRISPR to eliminate endogenous T cell receptors and class I human leukocyte antigens (HLA-Is), creating universal donor T cells [27]. This innovation would enable the use of donor-derived T cells in patients regardless of their HLA type.

Safety measures are also being integrated into engineered T cells. For example, the inclusion of inducible safety switches like caspase-9 allows the cells to self-destruct upon administration of a specific molecule, such as AP1903. This controllable "kill switch" enhances patient safety by providing a mechanism to halt therapy in the event of adverse effects.

In addition to its therapeutic applications, CRISPR-Cas technology shows significant potential for advancing dermatologic diagnostics. Platforms utilizing Cas9, Cas12, Cas13, and Cas14 enzymes are paving the way for rapid, ultra-sensitive, and cost-effective detection of nucleic acids. These tools could revolutionize the diagnosis of cutaneous viruses and identify single-nucleotide mutations associated with skin cancers, making advanced diagnostics more accessible and portable.

Conclusion

The increasing integration of CRISPR-Cas technology into dermatologic research and clinical care highlights its vast and transformative potential. This gene-editing system has shown remarkable promise across a broad spectrum of skin-related conditions, from repairing the genetic mutations that drive inherited disorders like epidermolysis bullosa to eliminating antibiotic-resistant bacteria responsible for chronic skin infections. CRISPR is also redefining cancer immunotherapy,

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particularly in melanoma, by enabling the engineering of T cells with enhanced tumor-targeting capabilities and reduced immunogenic risks. Beyond therapeutic applications, CRISPR-based diagnostic platforms are paving the way for a new era of molecular detection, offering rapid, ultra-sensitive, and cost-effective tools to identify viral infections and pinpoint genetic mutations associated with dermatologic diseases. These technologies hold the potential to make precision diagnostics widely accessible, even in resource-limited settings. As this field advances, the success of CRISPR-based interventions will depend heavily on the continued optimization of delivery methods, minimization of off-target effects, and implementation of built-in safety features such as inducible kill switches. With ongoing innovation, CRISPR-Cas systems are poised to revolutionize both the treatment and diagnosis of dermatologic conditions, bringing personalized and highly targeted medicine closer to routine clinical practice.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

1. Li G, Li X, Zhuang S, Wang L, Zhu Y, Chen Y, et al. Gene editing and its applications in biomedicine. Sci China Life Sci. 2022;65(4):660-700. https://doi.org/10.1007/s11427-021-2057-0
2. Cai L, Fisher AL, Huang H, Xie Z. CRISPR-mediated genome editing and human diseases. Genes Dis. 2016 ;3(4):244-251. https://doi.org/10.1016/j.gendis.2016.07.003
3. Baker C, Hayden MS. Gene editing in dermatology: Harnessing CRISPR for the treatment of cutaneous disease. F1000Res. 2020; 9:281. https://doi.org/10.12688/f1000research.23185.2
4. Bhat P, Garibyan L. The potential of CRISPR-guided therapies in the dermatology clinic. J Invest Dermatol Inno. 2022 ;2(2):100103. https://doi.org/10.1016/j.xjidi.2022.100103
5. Jacinto FV, Link W, Ferreira BI. CRISPR/Cas9-mediated genome editing: From basic research to translational medicine. J Cell Mol Med. 2020;24(7):3766-3778. https://doi.org/10.1111/jcmm.14916
6. Tetsch L. The adaptive bacterial immune system CRISPR-Cas and its therapeutic potential. Med Monatsschr Pharm. 2017;40(1):17-23. Available at: https://pubmed.ncbi.nlm.nih.gov/29952526/
7. Liu M, Rehman S, Tang X, Gu K, Fan Q, Chen D, et al. Methodologies for improving HDR efficiency. Front Genet. 2019; 9:691. https://doi.org/10.3389/fgene.2018.00691
8. Pawelczak KS, Gavande NS, VanderVere-Carozza PS, Turchi JJ. Modulating DNA repair pathways to improve precision genome engineering. ACS Chem Biol. 2018;13(2):389-396. https://doi.org/10.1021/acschembio.7b00777
9. Hirakawa MP, Krishnakumar R, Timlin JA, Carney JP, Butler KS. Gene editing and CRISPR in the clinic: current and future perspectives. Biosci Rep. 2020;40(4):BSR20200127. https://doi.org/10.1042/BSR20200127
10. Murauer EM, Koller U, Pellegrini G, Bauer JW. Advances in gene/cell therapy in epidermolysis bullosa. Keio J Med. 2015;64(2):21-25. https://doi.org/10.2302/kjm.2014-0013-RE
11. Maeder ML, Gersbach CA. Genome-editing technologies for gene and cell therapy. Mol Ther. 2016 ;24(3):430-446. https://doi.org/10.1038/mt.2016.10
12. Izmiryan A, Ganier C, Bovolenta M, Schmitt A, Mavilio F, Hovnanian A. Ex vivo COL7A1 correction for recessive dystrophic epidermolysis bullosa using CRISPR/Cas9 and homology-directed repair. Mol Ther Nucleic Acids. 2018; 12:554-567. https://doi.org/10.1016/j.omtn.2018.06.008
13. Wu W, Lu Z, Li F, Wang W, Qian N, Duan J, et al. Efficient in vivo gene editing using ribonucleoproteins in skin stem cells of recessive dystrophic epidermolysis bullosa mouse model. Proc Natl Acad Sci. 2017;114(7):1660-1665. https://doi.org/10.1073/pnas.1614775114
14. Zieman AG, Poll BG, Ma J, Coulombe PA. Altered keratinocyte differentiation is an early driver of keratin mutation-based palmoplantar keratoderma. Hum Mol Genet. 2019 ;28(13): 2255-2270. https://doi.org/10.1093/HMG/DDZ050
15. Wang J, Quake SR. RNA-guided endonuclease provides a therapeutic strategy to cure latent herpesviridae infection. Proc Natl Acad Sci. 2017;111(36):13157-13162. https://doi.org/10.1073/pnas.1410785111
16. Hsu DS, Kornepati AV, Glover W, Kennedy EM, Cullen BR. Targeting HPV16 DNA using CRISPR/Cas inhibits anal cancer growth in vivo. Future Virol. 2018;13(7):475-482. https://doi.org/10.2217/fvl-2018-0010
17. Zhen S, Li X. Oncogenic human papillomavirus: application of CRISPR/Cas9 therapeutic strategies for cervical cancer. Cell Physiol Biochem. 2018;44(6):2455-2466. https://doi.org/10.1159/000486168
18. Song R, Chen Z, Xiao H, Wang H. The CRISPR-Cas system in molecular diagnostics. Clin. Chim Acta 2024:119820. https://doi.org/10.1016/j.cca.2024.119820
19. van Diemen FR, Lebbink RJ. CRISPR/Cas9, a powerful tool to target human herpesviruses. Cell Microbiol. 2017;19(2): e12694. https://doi.org/10.1111/cmi.12694
20. Tao S, Chen H, Li N, Liang W. The application of the CRISPR-Cas system in antibiotic resistance. Infect Drug Resist. 2022:4155-4168. https://doi.org/10.2147/IDR.S370869
21. Lee GC, Dallas SD, Wang Y, Olsen RJ, Lawson KA, Wilson J, et al. Emerging multidrug resistance in community-associated Staphylococcus aureus involved in skin and soft tissue infections and nasal colonization. J Antimicrob Chemother. 2017;72(9): 2461-2468. https://doi.org/10.1093/jac/dkx200
22. Olesen CM, Clausen ML, Andersen PS, Agner T. The skin microbiome in atopic dermatitis-a potential treatment target?. Curr Dermatol Rep. 2018;7:199-208. https://doi.org/10.1007/s13671-018-0245-6
23. Bai X, Quek C. Unravelling tumour microenvironment in melanoma at single-cell level and challenges to checkpoint immunotherapy. Genes. 2022;13(10):1757. https://doi.org/10.3390/genes13101757
24. Mastaglio S, Genovese P, Magnani Z, Ruggiero E, Landoni E, Camisa B, et al. NY-ESO-1 TCR single edited stem and central memory T cells to treat multiple myeloma without graft-versus-host disease. Blood, J Am Soc Hemato. 2017 ;130(5):606-618. https://doi.org/10.1182/blood-2016-08-732636
25. Zhang LL, Wei JY, Wang L, Huang SL, Chen JL. Human T-cell lymphotropic virus type 1 and its oncogenesis. Acta Pharmacol Sin. 2017 ;38(8):1093-1103. https://doi.org/10.1038/aps.2017.17
26. Zhao W, Lei A, Tian L, Wang X, Correia C, Weiskittel T, et al. Strategies for genetically engineering hypoimmunogenic universal pluripotent stem cells. IScience. 2020;23(6). https://doi.org/10.1016/j.isci.2020.101162
27. Wellhausen N, Agarwal S, Rommel PC, Gill SI, June CH. Better living through chemistry: CRISPR/Cas engineered T cells for cancer immunotherapy. Curr Opin Immunol. 2022;74:76-84. https://doi.org/10.1016/j.coi.2021.10.008

https://doi.org/10.61577/amsd.2025.100001