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CRISPR-Cas9 PROTEIN: PHARMACEUTICAL FRONTIERS IN GENETIC RESTORATION

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Abstract: The advent of CRISPR-Cas9 has revolutionized gene editing, offering vast possibilities for disease treatment. This review dissects its mechanisms, structural aspects, and classification, underlining its superiority to preceding technologies such as ZFNs and TALENs. The evolution of CRISPR's applications is traced from bacterial immunity to the forefront of genome editing. Delivery methods—both viral and non-viral—are assessed, with detailed exploration of emerging solutions like lipid nanoparticles and DNA nanocarriers. CRISPR/Cas9 demonstrates significant therapeutic value in oncology by inactivating oncogenes, restoring tumor suppressors, and enhancing immunotherapeutic strategies. Furthermore, promising results have emerged in treating inherited disorders such as beta-thalassemia and sickle cell anaemia, as well as in clinical studies focused on T-cell therapies and viral diseases. Although these developments are encouraging, obstacles persist, including off-target genetic modifications and the intricacies of delivery. Innovations, notably base editing and prime editing, show promise to resolve these barriers. This review highlights the immense therapeutic prospects of CRISPR/Cas9 and stresses the importance of sustained research to fully unlock its clinical benefits.

Keywords: CRISPR-Cas9, Targeting oncogenes, Gene therapy, Cas9 variants, Gene editing, Beta-Hemoglobinopathies (HbF), Support vector machines (SVMs)

1. Introduction

Genome editing represents a transformative approach for addressing both hereditary and acquired diseases. Early genome modification efforts centred on Zinc Finger Nucleases (ZFNs) and Transcription Activator-Like Effector Nucleases (TALENs), which locate and alter specific DNA sequences by designing custom protein DNA-binding domains.¹ However, this process is costly, labour-intensive, and often suboptimal, restricting widespread genome editing adoption.²

The introduction of Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR)/Cas9 addressed these constraints.³ CRISPR/Cas9 comprises a Cas9 protein and a single guide RNA (sgRNA), which together enable sequence-specific cleavage at user-defined genomic sites through accurate base pairing. Unlike

previous methods, the design of sgRNAs is programmable, versatile, and less expensive, making CRISPR/Cas9 widely preferred in biotechnology.⁴

The naturally occurring CRISPR/Cas9 system, integral to bacterial immunity, uses Cas9 endonuclease programmed by sgRNAs to recognize and cut DNA containing a short protospacer adjacent motif (PAM). CRISPR locus transcripts produce small crRNAs, which, with Cas proteins, enable sequence-specific targeting and neutralization of foreign genetic material.⁵

2. Types of CRISPR-Cas Systems

The CRISPR-Cas systems are grouped into two main classes (I and II) and further divided into six types (I–VI).⁶

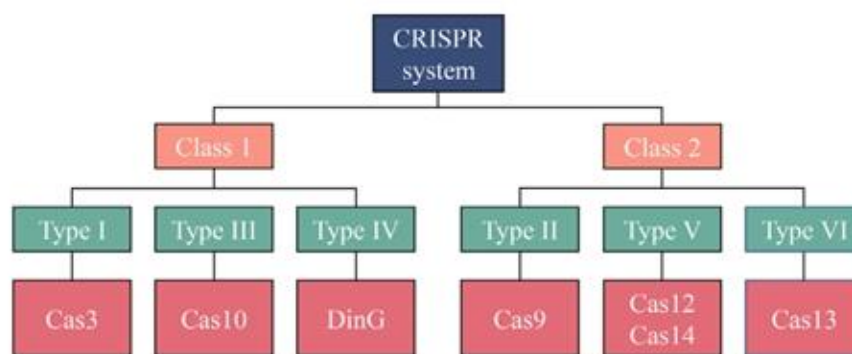


Figure 1: Classification of CRISPR Systems Based on Cas Protein Types and Functional Mechanisms

- **Class 1:** Types I, III, and IV operate through multi-protein complexes, responsible for functions via endonucleases such as Cas3, Cas10, and DinG.
- **Class 2:** Types II, V, and VI act with a single Cas protein (e.g., Cas9, Cas12–Cas14, Cas13), allowing targeted RNA-guided DNA or RNA cleavage.

Types I, II, and V primarily edit DNA; Type III acts on both RNA and DNA, while Type VI exclusively modifies RNA. The mechanisms of Type IV are not yet fully understood.⁷

With a guide RNA, CRISPR-Cas systems facilitate insertion (“knock-in”), removal (“knockout”), and base editing (precise point mutation) in DNA or RNA. Base editing, in particular, combines CRISPR components with accessory enzymes to achieve direct sequence changes.⁸

3. Discovery and Development of CRISPR Technology

Currently, CRISPR-driven gene-editing tools are pivotal in modern biology. Initially discovered as a prokaryotic defence mechanism, the technology has advanced rapidly, evolving into tools for transcription regulation, single-nucleotide editing, RNA editing, and comprehensive genome modification.^{9,10}

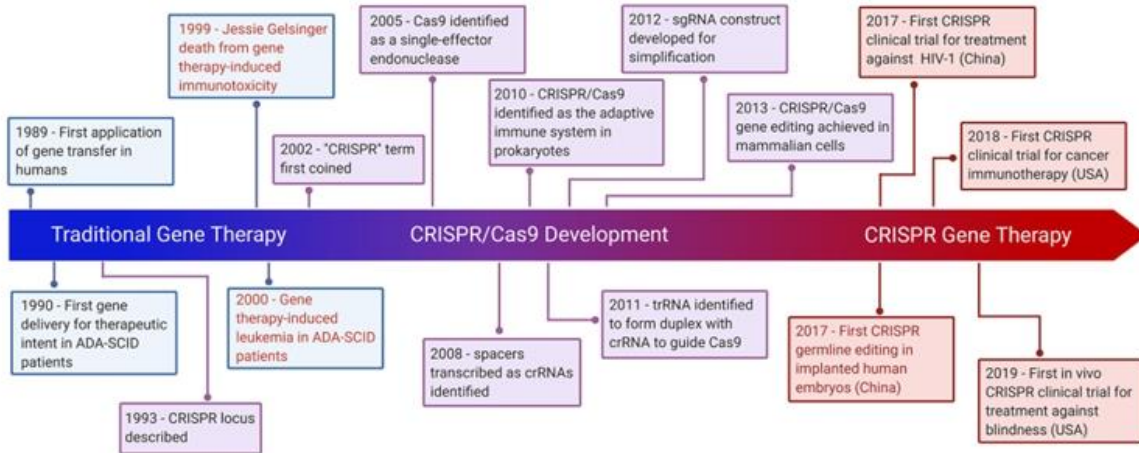


Figure 2: Timeline of Gene Therapy and CRISPR/Cas9 Development Milestones

4. Structural Features

CRISPR-Cas9 relies on two primary components: the guide RNA (gRNA) and the CRISPR-associated (Cas) nuclease. The gRNA contains a sequence that determines the target DNA region for editing, composed of a 17–20 nucleotide crRNA complementing the target site and a tracrRNA that anchors the complex.

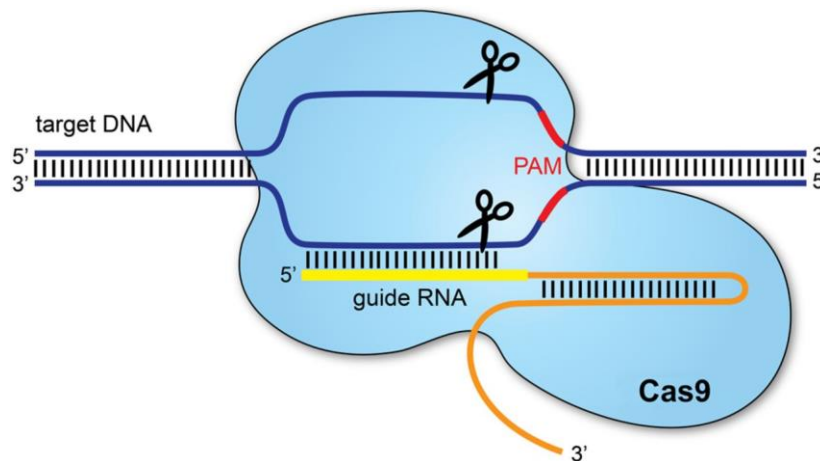


Figure 3: Mechanism of CRISPR-Cas9 Mediated DNA Cleavage

The Cas protein acts as a DNA endonuclease, making a double-strand break at the selected genome location. Among various Cas variants, the *Streptococcus pyogenes*-derived SpCas9 is the most extensively used.

4.1 X-Ray Structure of CRISPR

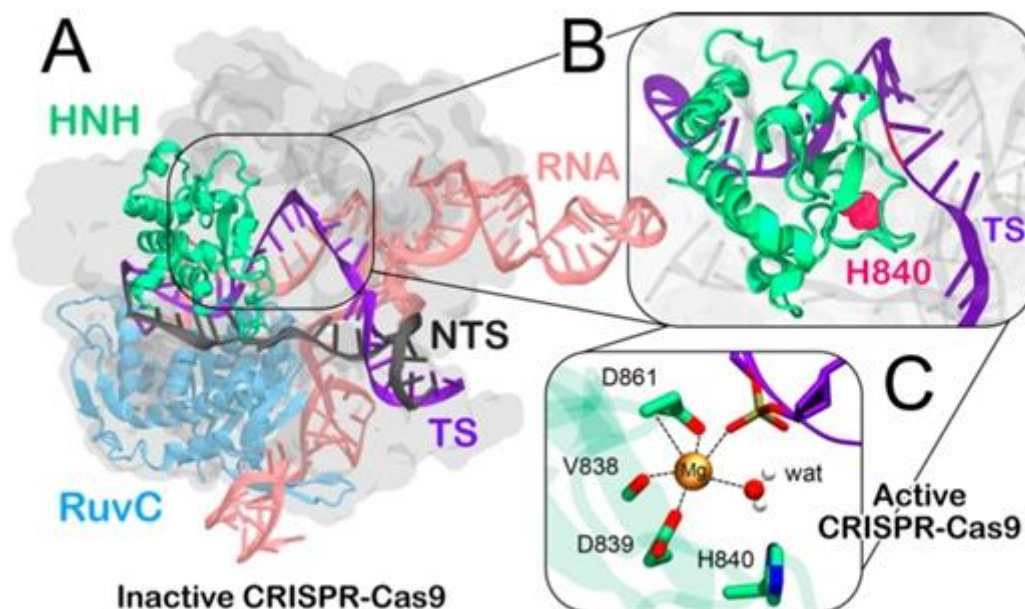


Figure 4: Structural Transition of CRISPR-Cas9 from Inactive to Active State

Description

- Depicts the crystal structure of the inactive CRISPR–Cas9 complex (PDB: 5F9R). The Cas9 protein is portrayed as a molecular surface, with the HNH and RuvC domains in green and blue, respectively. The RNA is coloured pink; the DNA strands (target and non-target) are violet and black, respectively. The catalytic HNH domain, shown, is positioned approximately 16–18 Å away from the cleavage site.^{3,11}
- A close-up of the activated state, derived from molecular simulations and FRET studies, showing HNH docked at the target cleavage site, away from RuvC.¹²
- Highlights Mg²⁺ ion coordination at the HNH catalytic site in the active state.¹³

4.2 CRISPR/Cas9 Composition

Prokaryotic cells are invaded by foreign DNA from phages or plasmids, segments are incorporated into the CRISPR array's spacer region. Upon subsequent infections, these spacers transcribe into pre-crRNA, some of which base-pair to tracrRNA. TracrRNA binds Cas9, forming a complex that, via RNase III processing and Cas9 action, generates a mature crRNA capable of guiding DNA targeting.

To streamline this, researchers created a crRNA-tracrRNA hybrid (the single guide RNA or sgRNA), substantially simplifying CRISPR/Cas9 use in eukaryotic cells. By designing a 20 bp sgRNA next to a PAM sequence, nearly any genome locus adjacent to PAM is accessible. Unlike ZFNs or TALENs, CRISPR specificity is determined by RNA-DNA pairing, making sgRNA design critical for target accuracy.

With SpCas9, target recognition depends on the presence of a PAM, triggering DNA strand separation. The ten crRNA bases directly adjacent to PAM—the “seed sequence”—initially bind, establishing an R-loop. REC2 and REC3 domains facilitate complete R-loop formation, activating the HNH and RuvC nucleases to cleave DNA.

However, editing via wild-type Cas9 variants (e.g., SpCas9, SaCas9) can result in off-target changes, chromosomal rearrangements, or large deletions, particularly constrained by PAM requirements. Cas9 modifications are therefore aimed at increasing specificity and expanding potential target sites.¹³

5. Delivery Methods for CRISPR

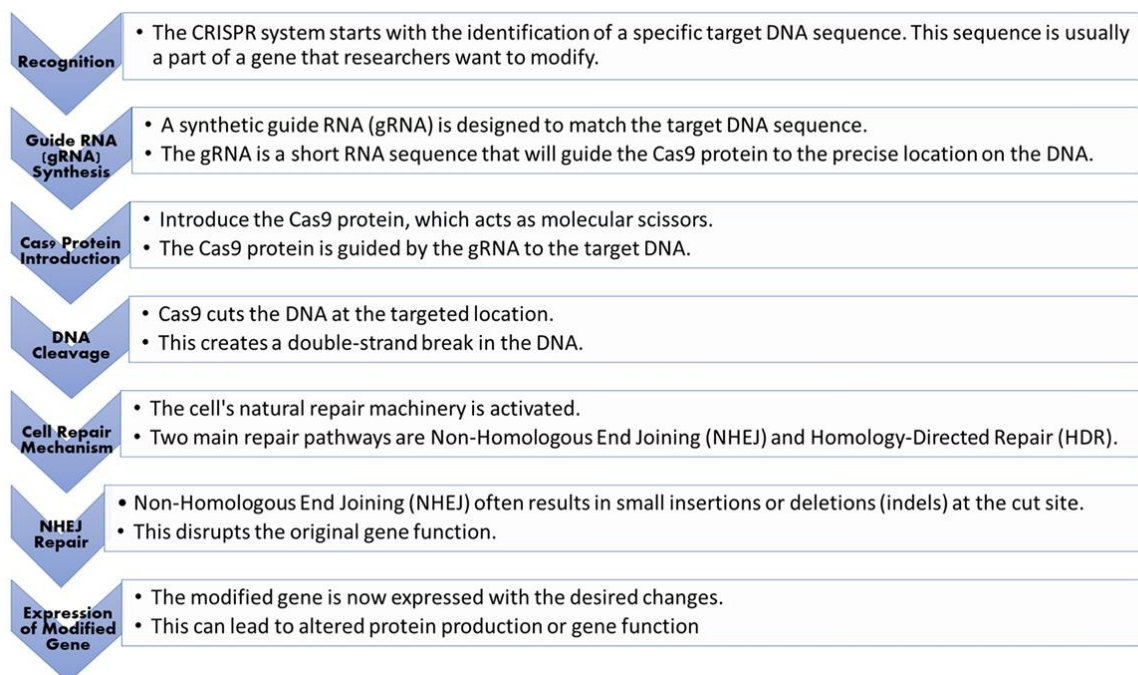
Plasmid DNA (pDNA) serves as a robust vehicle due to its durability and modifiability, enabling amplification and easy manipulation. Once inside cells, pDNA, via nuclear localization signals, is transcribed to produce Cas9 and sgRNA mRNA—although this method is cumbersome. Delivering Cas9/sgRNA as mRNA simplifies the process but faces degradation and stability issues. The Cas9-sgRNA ribonucleoprotein (RNP) complex, generated *ex vivo*, acts immediately but is challenging to deliver due to size and charge. Electroporation, cell-penetrating peptides, and, recently, exosome vectors are being explored to overcome these obstacles.

6. Structural Insights

Studies on *Streptococcus pyogenes* Cas9 (SpyCas9) and the smaller *Actinomyces naeslundii* Cas9 (AnaCas9) have clarified the essential domain organization of the Cas9 protein family.⁵

7. Guide Sequence Engineering

Guide RNAs are precisely tailored to match the desired DNA target, ensuring effective Cas9 recruitment and cleavage.^{14,15}



8. CRISPR-Cas9 Delivery Strategy

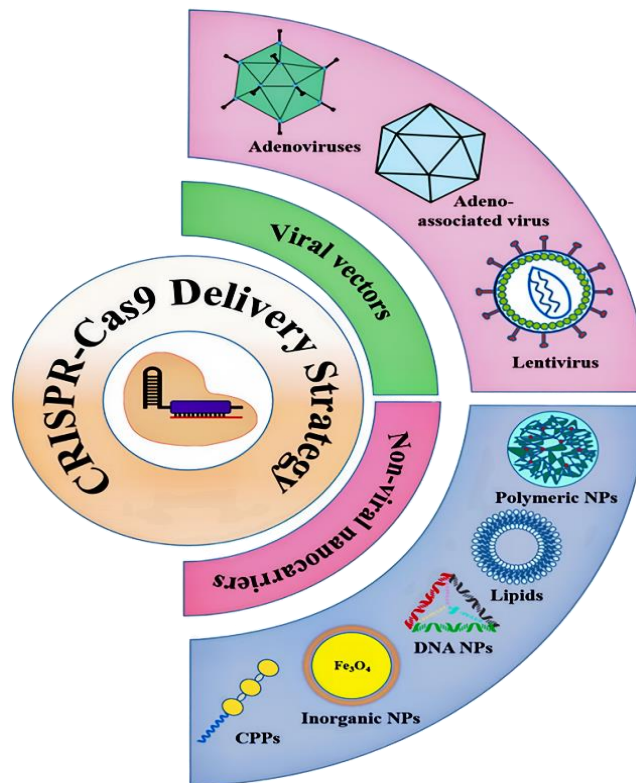


Figure 5: Delivery Strategies for CRISPR-Cas9 Gene Editing System

8.1 Viral Delivery Systems

Adeno-associated viruses (AAVs) are a leading vector for in vivo CRISPR-Cas9 delivery, providing excellent efficiency, low pathogenicity, and extended gene expression, though AAV immunogenicity and infection mechanisms remain research priorities.¹⁶ Lentiviral vectors, with greater payload capacities, integrate stably into genomes—ideal for larger or multiple gene insertions—but present risks, notably insertional mutagenesis. Each method's risk-benefit profile is important for therapeutic applications.¹⁷

8.2 Non-Viral Nanoparticle Systems

Lipid-based nanoparticles are advanced carriers for Cas9 and sgRNA, effectively protecting them from degradation and allowing for tailored size, charge, and target cell specificity. These nanocarriers enhance cellular uptake and gene-editing precision, but encounter challenges like immunogenicity and the need for optimal lipid-to-nucleic acid ratios.

Polymeric nanoparticles, constructed from biocompatible and biodegradable polymers, encapsulate Cas9 and sgRNA efficiently, enabling controlled release. Polymer type, size, surface properties, and degradation rate critically influence delivery success and gene-editing efficiency.

8.3 DNA Nanocarriers^{18,19}

Engineering of DNA-based nanostructures—like DNA nanoflowers, nanospheres, and nanotubes—has led to versatile platforms for drug and gene delivery due to their biocompatibility and capacity for stimuli-

triggered conformational changes. For example, Ding et al. introduced a non-cationic, DNA-crosslinked nanogel for CRISPR/Cas9 delivery; Shi et al. developed a miRNA-responsive DNA nanoflower system for targeted release in cancer therapy; Li et al. crafted a pH-activated nanocarrier co-delivering Cas9/sgRNA and DNAzyme, demonstrating potent anti-tumour effects after lysosomal activation; and Sun et al. designed DNA nano clews loaded with Cas9 RNP to achieve remarkably high delivery and gene knockout efficiency.

9. Clinical Applications and Trials

CRISPR-Cas technology's clinical use is divided into:

- **Ex vivo applications:** Patient cells are isolated, modified, and re-infused post-editing—applied in cancer immunotherapy, inherited disease treatment (e.g., sickle cell disease, beta-thalassemia), and prevention of viral infections.
- **In vivo applications:** Gene-editing agents are directly delivered to patient tissues.

9.1 Cancer

Despite advances curbing mortality, cancer remains a leading killer, owing largely to its molecular complexity and heterogeneity that complicate treatment. While therapies such as immunotherapy, targeted drugs, and surgery have improved outcomes, they bring challenges: high toxicity, cost, and incomplete remission.

CRISPR/Cas9 emerges as a gene-editing platform with accuracy and flexibility, opening doors for personalized medicine in oncology.^{20,21}

9.1.1 Structure and Mechanism of CRISPR/Cas9

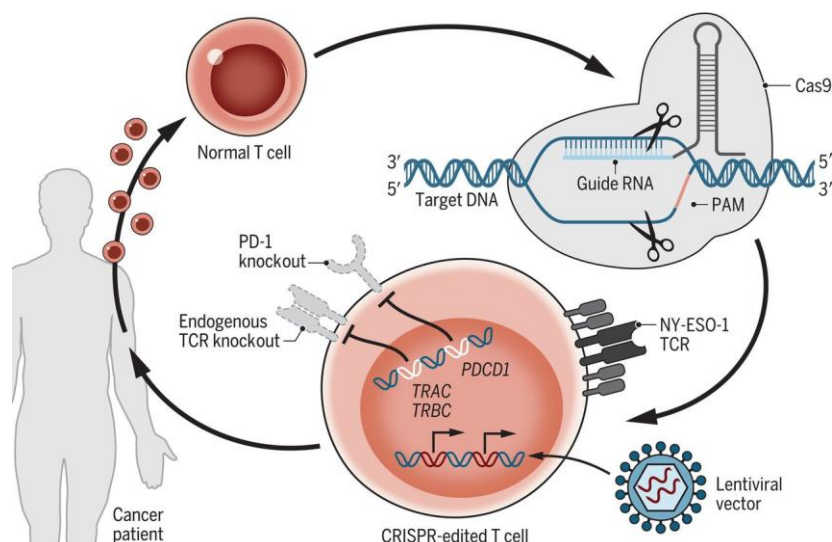


Figure 6: CRISPR-Cas9 Mediated T Cell Engineering for Cancer Immunotherapy

CRISPR/Cas9 consists chiefly of sgRNA and the Cas9 enzyme. The sgRNA contains a sequence that guides Cas9 to the gene of interest—typically the disease-affected region. Upon recognizing a PAM, Cas9, directed by the sgRNA, binds, unwinds the double-strand DNA at the appropriate locus, and introduces a cut. The cell's repair machinery resolves the resulting double-strand break (DSB) by either homologous recombination (HR)—enabling insertion of a correct DNA segment—or non-homologous end joining (NHEJ).

The need for CRISPR technology lies in its potential to enable highly personalized treatments, enhance patient survival and tolerability by reducing off-target effects and treatment-associated toxicities, and unlock novel therapeutic avenues for diseases that remain refractory to conventional interventions and it is useful to various cancers like breast, lung, colorectal, prostate and liver and its mainly worked by restoring the tumour suppressor genes (e.g. ETS1, CPEB2, BRCA1, etc.).^{20,22}

9.1.2 Current and Potential Clinical Applications in Cancer

- Gene Editing & Knockout: Targeting of oncogenes (RAS, EGFR, KRAS, MYC) and tumour suppressors (TP53, PTEN, BRCA1, APC) in cancer models suppresses tumour growth and clarifies mechanisms of chemoresistance (for example, PTEN knockout in lung cancer, AR deficiency in prostate tumours).
 - Restoring Tumour Suppressor Functions: CRISPR/Cas9 repairs or boosts tumour-suppressor activity, thereby repressing proliferation and progression.
 - Targeting Drug Resistance: The system is utilized to dissect molecular bases for therapeutic resistance, such as targeting ERK2 to heighten sorafenib sensitivity in liver cancers.
 - Cancer Modelling & Biomarker Discovery: Expedites identification of essential tumour genes and high-impact biomarkers through high-throughput screens.
- Augmenting Immunotherapy: Engineered T cells (e.g., PD-1/CTLA-4 knockouts; CAR-T cells) show amplified anti-tumour action and are under clinical evaluation for solid and haematological cancers, alongside editing T cell receptors for allogeneic use.^{20,21,23}

9.2 Gene Editing in Beta-Hemoglobinopathies

Beta-haemoglobinopathies, including sickle cell disease (SCD) and β -thalassemia (BT), are inherited disorders marked by aberrant beta-globin chain production. New approaches raise fetal haemoglobin (HbF) formation through gene editing rather than correcting adult haemoglobin defects. In practice, CD34+ hematopoietic stem cells are extracted, edited to promote HbF, and reinfused after chemotherapy—a process that narrows targeting and limits persistent CRISPR presence in the body.²⁴

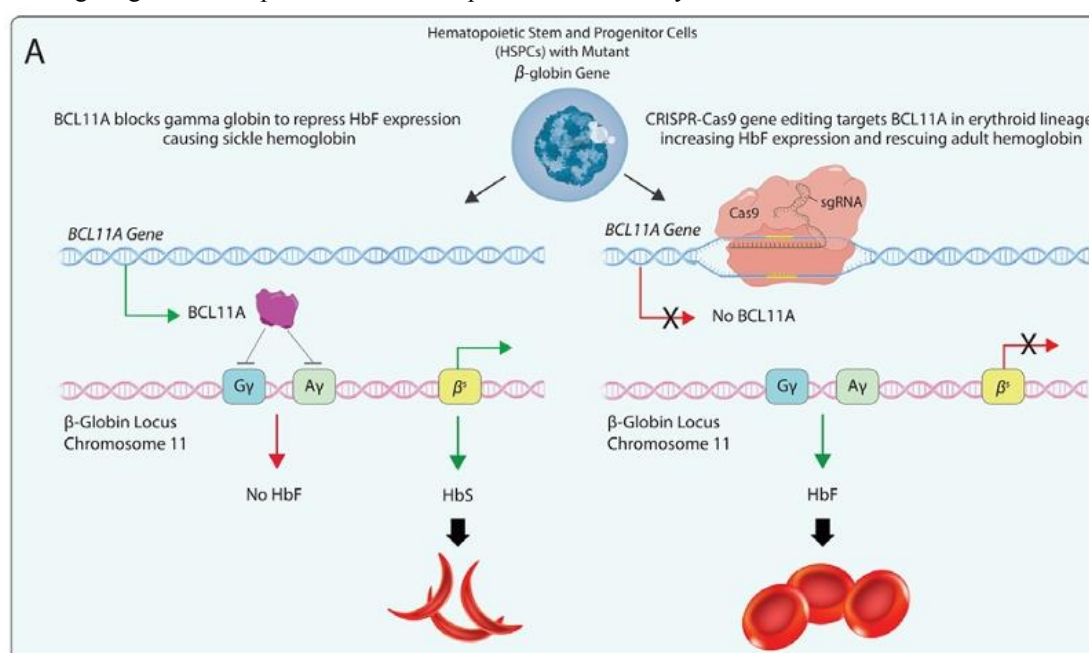


Figure 7: CRISPR-Cas9 Targeting of BCL11A to Reactivate Fetal Haemoglobin in Sickle Cell Disease

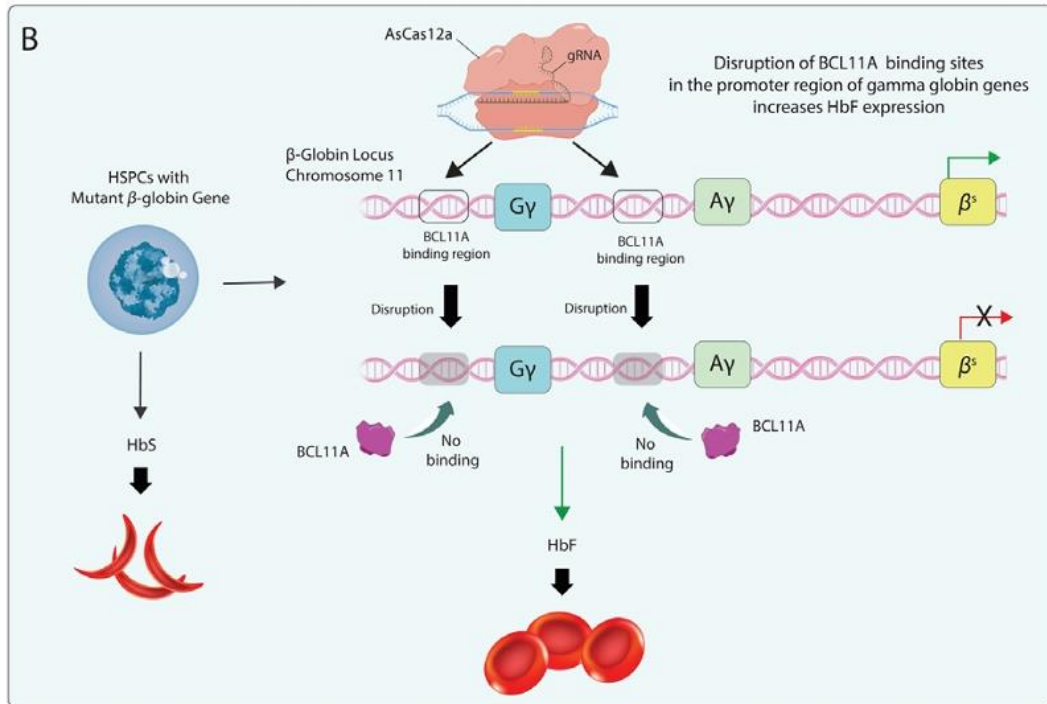


Figure 8: AsCas12a-Mediated Disruption of BCL11A Binding Sites to Induce Fetal Haemoglobin in Sickle Cell Disease

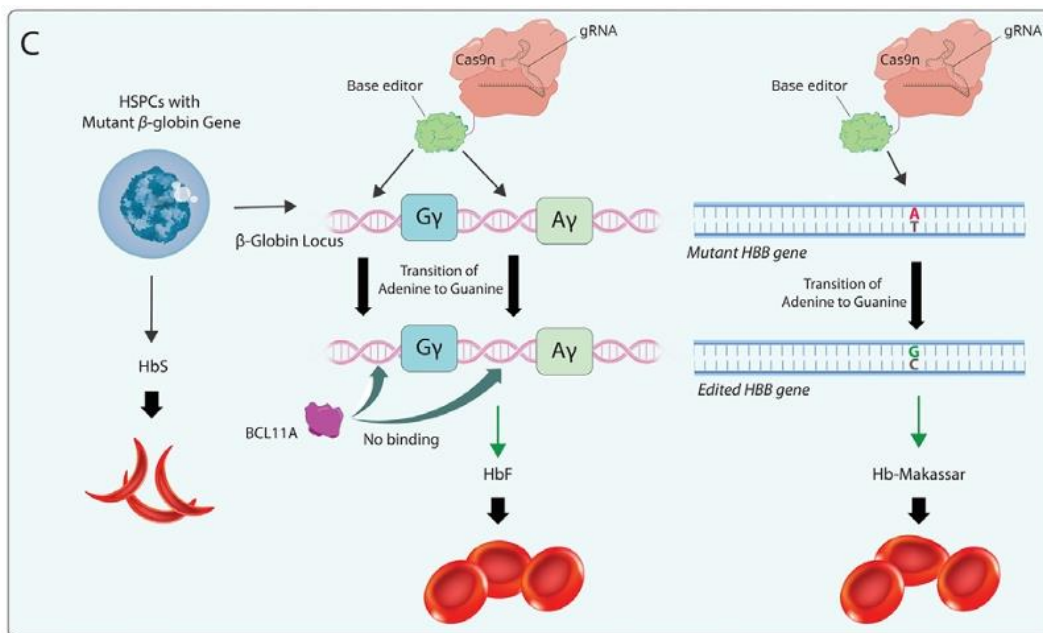


Figure 9: Base Editing Strategy Using Cas9n to Correct β -Globin Gene Mutations and Reactivate Fetal Haemoglobin



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- CASGEVY (Vertex/CRISPR Therapeutics) modifies the BCL11A erythroid enhancer to disrupt GATA1 binding, derepressing fetal haemoglobin synthesis.
- EDIT-301 harnesses AsCas12a to modify γ -globin promoters and thus diminishes BCL11A binding for increased HbF production.
- BEAM Therapeutics' base editors:
- BEAM-101 replaces A with G in the BCL11A interaction region of gamma-globin promoters;
- BEAM-102 directly corrects the sickle cell mutation in beta-globin.²⁵

9.3 Genetic Engineering of Pancreatic Cells for Diabetes

Type 1 diabetes (T1D) is driven by autoimmune-mediated destruction of pancreatic beta cells. CRISPR enables modification of pluripotent stem cell-derived pancreatic cells for immune evasion and subsequent allogeneic transplantation, potentially eliminating the need for chronic immunosuppression. Such strategies promise to generate "off-the-shelf," hypoimmunogenic iPSC lines, though multiple engineering steps heighten the risk of off-target genetic changes.^{26,27}

10. Limitations

Notwithstanding high specificity, CRISPR–Cas9 systems can yield off-target edits, including structural variations such as deletions and translocations, particularly when up to three mismatches between sgRNA and target DNA are tolerated. DNA repair of Cas9-induced breaks can cause complex genomic alterations, contributing to instability. Advances in predictive algorithms and novel Cas variants seek to mitigate these issues.

Delivering CRISPR components presents major challenges: viral vectors (e.g., AAVs) are efficient but can provoke strong immune responses (as seen in recent trials), especially at high doses; non-viral carriers (lipid nanoparticles, electroporation) have lower immunogenicity but limited specificity and durability.

11. CRISPR-Cas9 Protein Combination with Artificial intelligence

The combination of artificial intelligence (AI) with CRISPR-Cas9 and its derivatives is poised to transform precision genome editing by overcoming the limitations of predictability and efficiency in the present.²⁸ Machine learning algorithms, as cited in recent developments in drug discovery and genetic engineering (e.g., via Internet of Bio-Nano Things and neural network-based biomarker discovery tools), can refine the guide RNA design, prepredict off-target effects, and model repair outcomes with great accuracy.²⁹ For example, AI-based platforms such as those using convolutional neural networks (CNNs) or support vector machines (SVMs) may scan enormous genomic datasets of pandemics or long-term diseases to allow in-real-time customization of CRISPR therapies.³⁰ Coupled with Internet of Nanothings (IoNT) for nanoscale monitoring and delivery, as examined in healthcare applications for viral diagnosis and customized medicine, AI may enable closed-loop, automated systems for in vivo editing, which may expedite clinical trials in cases such as COVID-19-linked genetic susceptibility or inherited diseases and reduce risks and ethical issues.³¹

12. Future Perspectives

Cas9-induced double-strand breaks risk unpredictable repair outcomes; alternative tools are rising in prominence. Base editors—Cas proteins coupled with DNA-modifying enzymes—enable single-base conversions. Prime editing employs a Cas9 nickase–reverse transcriptase fusion and a prime editing guide RNA for precise sequence alteration—both now entering initial clinical evaluation. As of late 2024, a prime editing-based trial for chronic granulomatous disease (restoring NCF1 gene function in CD34+ cells) is underway and



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its concluded that CRISPR-Cas9 Protein as suitable promising candidate for Genomic restoration. These advancements leverage our understanding of genetics, genomics, and molecular biology to develop therapies that target the root cause of diseases, potentially leading to long-term cures.

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