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Engineering Precision "Molecular Scalpels": A Review of Evolving CRISPR Platforms and Their Applications in Oncology

Junzhi Gong*

Shenzhen Middle School, Shenzhen, China

**Corresponding author: Junzhi Gong.*

Abstract

CRISPR is often called a molecular scalpel, but oncology precision also requires the intended product in the right cells, reproducible manufacture, and meaningful benefit without unacceptable toxicity. This focused narrative review compares nuclease, base, and prime editing through those requirements and separates platform demonstrations from human cancer evidence. Cas9 nuclease editing has enabled early clinical studies of engineered T cells in solid and hematologic malignancies. Base-edited allogeneic CAR T cells have also reached patients with T-cell acute lymphoblastic leukemia. Prime editing offers a broader menu of small sequence changes but remains preclinical for cancer treatment. Across platforms, off-target activity, large on-target lesions, cell-product heterogeneity, and tumor escape demand different assays and cannot be summarized by one editing-efficiency value. Controlled ex vivo engineering of immune cells currently has the strongest translational footing. Direct editing of heterogeneous tumors remains a delivery and selectivity challenge. We propose a framework linking application, editor chemistry, edit product, delivery, and clinical validation.

Keywords

CRISPR, base editing, prime editing, cancer immunotherapy, genomic safety

1. Introduction

Programmable CRISPR-Cas systems made targeted changes to DNA broadly accessible. In oncology, they can be used to discover cancer dependencies, engineer immune cells, or attempt to change a tumor genome directly. These applications have different evidentiary burdens. A knockout in a cell line is a mechanistic result; an edited immune-cell product that can be manufactured consistently is a therapeutic candidate; and a patient response is clinical evidence that also depends on conditioning treatment, disease biology, and follow-up. Treating all three as equivalent obscures where progress has occurred.

The scalpel metaphor requires a clear definition of precision. Sequence precision concerns intended and unintended changes; cellular precision, which cells change; functional precision, their therapeutic phenotype; and clinical precision, benefit relative to risk. A highly accurate editor delivered to the wrong tissue is not a precise cancer treatment. Conversely, a useful engineered T-cell product may depend on a robust gene knockout rather than a single-base correction.

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This focused narrative review examines peer-reviewed primary studies available through October 2026. Foundational editor papers establish what each chemistry can do; assay studies define failure modes; and published cancer studies show which applications have entered patients. Studies were selected to compare these distinct evidence levels, not to estimate a pooled treatment effect. Small, single-arm, early-phase cohorts cannot establish superiority over standard care. The question throughout is therefore: which aspects of precision have been demonstrated for a given product, and which remain proposed?

2. Editing Mechanisms and the Meaning of Precision

Cas9 nucleases create a double-strand break beside a compatible protospacer-adjacent motif. End joining can disrupt a gene; template-guided repair can install a sequence. In immune-cell therapy, knocking out inhibitory receptors, endogenous T-cell receptors, or antigens that cause CAR T-cell fratricide serves distinct purposes. Targeted insertion can place a receptor at a defined locus and reduce variability from random integration. Repair still yields mixed alleles, so high knockout frequency cannot establish the genotype of every cell in an infusion product.

High-fidelity Cas9 variants reduce mismatch cleavage in tested human cells [1], but "no detectable off-target effects" depends on the guide, cell type, and assay sensitivity. GUIDE-seq reveals guide-dependent off-target activity [2]. Discovery assays, candidate-site sequencing, and final-product analysis answer different questions; one negative assay cannot exclude all alterations.

Cytosine base editors convert C·G to T·A, and adenine base editors convert A·T to G·C, usually without an intended double-strand break [3, 4]. They are attractive when a defined nucleotide substitution can create a stop codon, change an antigen epitope, or disable a gene during multiplexed immune-cell manufacture. Their chemical range is limited: only compatible substitutions are possible, the accessible editing window matters, and neighboring bases can become bystander products. Editor performance also varies across targets and cell types. Avoiding a programmed double-strand break reduces one source of damage but does not eliminate the need to measure unintended DNA and RNA changes; transcriptome-wide RNA edits were observed for some base-editor designs [5].

Prime editing uses a Cas9 nickase fused to reverse transcriptase and a prime-editing guide RNA that encodes the desired sequence [6]. It can install a broader range of small substitutions, insertions, and deletions without a donor DNA template. This capability could create protective epitopes or correct specific variants, but editor size, guide design, incomplete products, and cell-specific repair responses complicate delivery and manufacture. In cancer, the distinction between generating a tumor model and treating a patient is especially important. A mouse engineered to carry a cancer-driving mutation demonstrates platform scope, not a therapeutic response.

Editor choice should follow the biological task. A characterized nuclease knockout may suffice for loss of function; a protective substitution may favor base editing if the target lies in a suitable window without harmful bystanders. Prime editing enables other small changes but does not solve delivery or product heterogeneity. Comparisons should use matched cells, loci, assay depth, and functional endpoints.

Precision cannot be ranked from editing efficiency alone. High intended-edit frequency does not reveal rare structural variants; a low measured off-target frequency does not describe bystander edits; and a clean early sample may not represent the expanded infusion product. Large deletions have been detected after Cas9 cuts and can escape short-amplicon assays [7]. In tested cell systems, base and prime editors produced such lesions less often than nucleases but did not eliminate them; the approximately twenty-fold difference reported by Hwang and colleagues is an experimental comparison, not a clinical safety ratio [8]. Product release criteria should therefore specify the exact allele distribution and the resolution of the assay used. Figure 1 summarizes the application routes, shared challenges, and evidence needed to assess precision.

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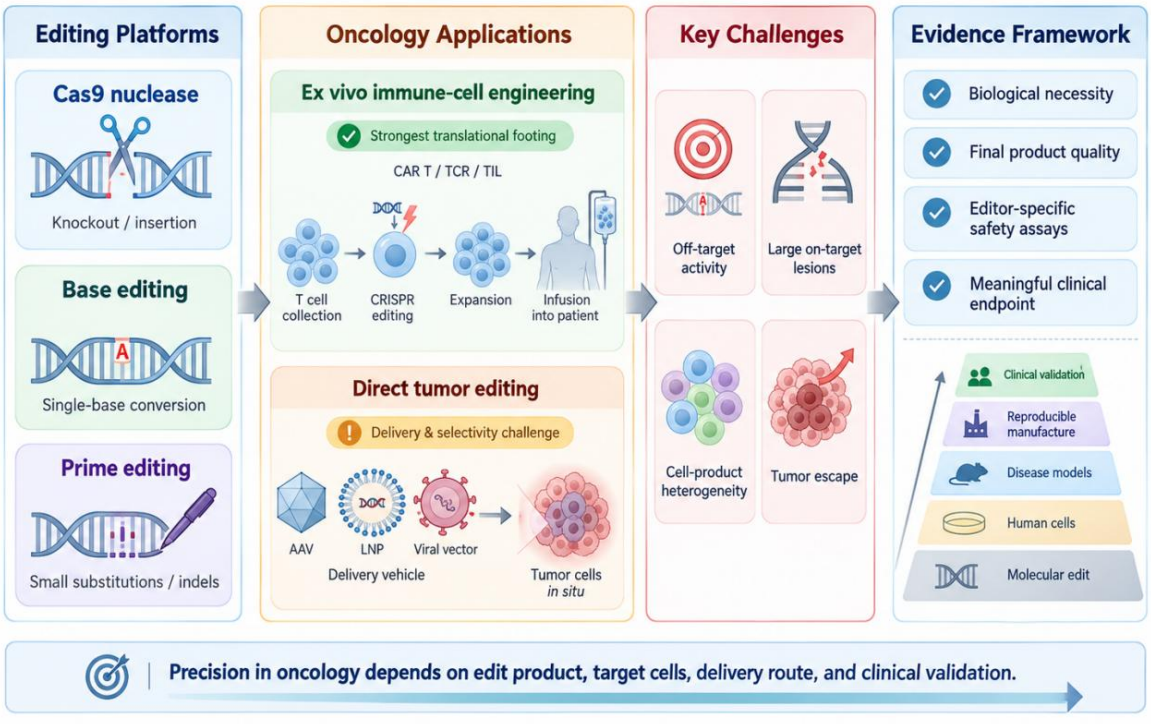
Figure 1: CRISPR platforms, oncology applications, risks, and evidence steps. Delivery examples overlap (AAV is a viral vector); direct tumor editing remains preclinical in the cited studies



3. Oncology Applications and Evidence Levels

The clearest path from editing to patients is ex vivo immune-cell engineering. Cells can be edited outside the body, expanded, sampled, and tested before infusion. In a first-in-human study, Stadtmauer and colleagues administered multiplex CRISPR-edited, NY-ESO-1-specific T cells to three patients with refractory cancer [9]. Edited cells persisted, while chromosomal translocations were detected. The result established the feasibility of manufacture and persistence; it did not establish broad antitumor efficacy. In advanced non-small-cell lung cancer, Lu and colleagues infused PD-1-edited T cells into twelve patients in a phase I study [10]. Treatment-related adverse events were grade 1 or 2, but clinical activity was limited. Candidate-site sequencing in that trial should not be read as a genome-wide guarantee of zero off-target risk.

Personalization adds another dimension. Foy and colleagues used non-viral CRISPR editing to replace endogenous T-cell receptors with patient-specific neoantigen receptors and treated sixteen patients with refractory solid tumors [11]. This was an important demonstration that individualized receptor selection and cell manufacture can be performed within a clinical workflow. It also showed the difficulty of translating a tailored molecular product into durable tumor control: the reported clinical outcomes did not support a general efficacy claim. Product heterogeneity, neoantigen quality, tumor infiltration, and an immunosuppressive microenvironment remain possible explanations for variable responses.

Base editing has entered oncology through allogeneic T-cell products. A 2026 report of universal base-edited anti-CD7 CAR T cells described nine children in a phase I study plus two adults treated under compassionate use for T-cell acute lymphoblastic leukemia [12]. Nine of eleven attained deep remission permitting transplantation, but subsequent allogeneic transplantation, treatment complications, and CD7-negative relapse complicate attribution of long-term benefit to the edited cells alone. The study supports clinical feasibility and substantial early activity in a defined setting; it does not show that base editing is clinically safer or more effective than nuclease editing.

Other edited immune-cell strategies continue to broaden the evidence base. In a first-in-human study of CISH-knockout tumor-infiltrating lymphocytes for metastatic colorectal cancer, Lou and colleagues treated twelve patients; one had a sustained complete response, while adverse events and the co-administered conditioning and interleukin-2 limit causal interpretation [13]. A larger single-arm phase I/II study of allogeneic CRISPR-edited CD19 CAR T cells, CTX110, treated sixty-three adults with relapsed or refractory

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B-cell non-Hodgkin lymphoma [14]. Among fifty-seven given a first infusion at dose level three or higher, objective and complete response rates were 65% and 39%; only five of twenty-two complete responses remained ongoing at data cutoff. The cohort offers stronger feasibility and activity evidence than a three-patient study, but it remains a dose-ranging trial rather than a randomized comparison with autologous CAR T-cell therapy.

These studies differ in disease, cell source, target, conditioning, dose, and endpoint, preventing an efficacy ranking. Together, they show that edited immune cells can be manufactured and infused, with meaningful responses in some patients. Nuclease editing currently has the most developed published oncology clinical record; base editing has early human experience; prime editing has no comparably mature patient-outcome evidence in oncology. Direct comparative evidence is needed to claim a clinical advantage for any editor.

Hematologic malignancies and solid tumors pose different barriers to these products. A blood cancer can present an accessible lineage antigen, but effective targeting may also remove normal cells carrying that antigen. Solid tumors add trafficking, physical exclusion, antigen diversity, and suppressive local signals. The CISH-edited tumor-infiltrating lymphocyte and personalized neoantigen studies are consequently important tests of feasibility in solid disease, even though their early responses are insufficient for a broad efficacy conclusion. It would be misleading to transfer response rates from lymphoma to colorectal or lung cancer simply because the same editing enzyme appears in the manufacturing process.

4. Delivery and Tumor Biology

Editing malignant cells directly is harder than editing T cells outside the body. A solid tumor comprises genetically diverse malignant clones, stroma, vasculature, and infiltrating immune cells. A local dose may miss disseminated disease, whereas systemic delivery may expose healthy tissues. Incomplete editing leaves resistant clones; a successfully edited driver can be bypassed by another pathway. The genomic instability of many cancers can also make repair products difficult to predict. Thus, the ability to cut or rewrite a tumor allele in a laboratory model is only the first step toward clinical selectivity.

Delivery should be evaluated as a quantitative distribution rather than a yes-or-no event. Useful measurements include the fraction of malignant cells edited, differences between tumor sites, editor exposure in normal tissue, duration of editor expression, and the fraction of cells bearing unintended alleles. Sampling only the most accessible lesion can overstate coverage of metastatic disease. A tumor-shrinking effect in a small animal may also reflect a delivery pattern that does not scale to human anatomy. These measurements would help distinguish failure of the molecular edit from failure to reach the relevant cells.

Targeted lipid nanoparticles have delivered Cas9 components to tumors and produced antitumor effects in mouse models of glioblastoma and ovarian cancer [15]. That experiment supports a preclinical delivery strategy, not a claim of direct human tumor editing. Prime editing has likewise generated broad sets of somatic mutations in animal cancer models, but creating a disease model is not the same as reversing disease. For therapeutic use, an in vivo platform would need to demonstrate biodistribution, malignant-cell selectivity, a sufficient fraction of relevant clones edited, acceptable exposure in normal organs, and durable functional benefit.

Ex vivo manufacture offers controls that in vivo tumor editing presently lacks: a defined starting cell population, the option to discard substandard lots, and potency testing before exposure to a patient. Even this route faces trade-offs. Autologous manufacture is individualized and can be slow or fail in heavily pretreated patients. Allogeneic manufacture can make products available in advance, but additional edits may be needed to reduce graft-versus-host disease and immune rejection. Each added edit may improve a biological design while enlarging the space of possible genomic products and complicating release testing.

Another problem is target selectivity. A useful tumor antigen may also be expressed by healthy cells. Preclinical epitope prime editing has been used to shield hematopoietic cells from CD123-directed immunotherapy in models of acute myeloid leukemia [16]. This approach changes a normal-cell epitope while preserving a therapeutic target on malignant cells. It is a mechanistically informative way to address on-target, off-tumor toxicity, but patient benefit remains untested in the cited work. A clinical program would need to show sustained normal hematopoiesis, antitumor effect, and absence of new escape routes. A 2026 study

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corrected HAVCR2 in patient-derived lymphoma T cells and showed selective expansion of prime-edited cells in humanized mice [17].

5. Safety Assessment Beyond Off-Target Cuts

Genomic safety spans several scales. At the base-pair level, investigators need to quantify intended edits, indels, bystander substitutions, and unexpected sequence products. At the locus level, they must detect deletions, insertions, inversions, and rearrangements that short amplicons can miss. At the genome level, they need a guide- and cell-relevant off-target strategy. Multiplex nuclease editing introduces the additional possibility of translocations between intended cut sites. The relevance of a detected event depends on frequency, affected gene, whether the altered cell expands, and the lifetime of the product.

No single assay resolves all three scales. Short-read amplicon sequencing can measure common small variants efficiently but may miss alleles that fail to amplify after a large deletion. Long-range or long-read approaches can inspect local structure; genome-wide discovery methods can nominate unexpected sites for validation; and cytogenetic or copy-number analyses can detect some larger abnormalities. The testing plan should be matched to the proposed editing mechanism and should state lower detection bounds. It should also examine the final infusion lot, because selection during culture can change the abundance of rare genotypes between the editing step and administration.

Base editors bring different liabilities. A bystander edit can alter an adjacent codon even when the intended base is correctly changed. Some deaminase designs can edit RNA outside the targeted locus [5]. Prime editors avoid a programmed double-strand break but can yield incomplete products or indels, and guide design affects purity. The right comparison is therefore a distribution of molecules and cell states, not a binary “edited versus unedited” count. Sequencing of the final lot after expansion is more directly relevant than sequencing immediately after transfection.

Cancer-treatment safety is broader still. Engineered T-cell therapies can cause cytokine-release syndrome, neurotoxicity, cytopenias, infection, and prolonged immune suppression. These outcomes arise from the whole treatment pathway, including conditioning and receptor biology, and cannot be assigned automatically to the editing enzyme. In the base-edited leukemia experience [12], early remission must be interpreted alongside infection and transplantation. In the allogeneic lymphoma experience [14], response and serious adverse events both matter. Functional assays should test tumor killing, persistence, exhaustion, and recognition of healthy tissue alongside genomic integrity.

Long-term surveillance is especially relevant because edited immune cells can persist or expand. Clonal tracking and repeated assessment for abnormal growth are more informative than one pre-infusion assay. A meaningful protocol should state when a genomic signal triggers investigation, what detection limits apply, and whether a rare abnormal clone is stable, declining, or expanding. Those questions cannot be resolved by a generic assertion that a platform is precise; they require product-specific follow-up.

6. An Evidence Framework for the Next Generation

Four questions can organize future studies. First, is an edit biologically necessary for the anticancer mechanism, or only technically feasible? Matched unedited or differently edited comparators should isolate its contribution in models. Second, what proportion of the final product has the intended genotype and phenotype? Allele-resolved sequencing and functional testing should be performed after expansion. Third, which rare harms are plausible for the chemistry used? Structural variants after double-strand breaks, bystanders after base editing, and complex or incomplete products after prime editing call for different assays. Fourth, which patient endpoint would demonstrate benefit? Response, duration, survival, and quality of life must be interpreted in light of disease stage and prior therapy.

The evidence ladder moves from molecular edits through human cells, models, manufacture, and clinical evaluation. Accuracy in a cell line or release test does not ensure performance in patient cells or tumors. Studies should report failed manufacture as well as successful infusion and should identify denominators for enrolled, manufactured, and treated patients.

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Early trials can improve interpretability before a randomized comparison becomes feasible. A protocol can specify the edit-dependent mechanism, the minimum acceptable product quality, dose cohorts, and the time points for genomic and functional monitoring. It can distinguish a response from stable disease, and it can report duration rather than only the best observed scan. Recording why a participant does not receive the manufactured product is essential for judging feasibility. In later comparative work, a matched comparator should be selected to test the incremental value of the edit rather than merely comparing an edited product with no cellular therapy.

The strongest near-term research opportunity is to connect an editor-specific genomic quality profile with a cell product's mechanism and longitudinal patient outcomes. Claims about prime editing in oncology should emphasize what it can uniquely install and how product purity is measured, while acknowledging the absence of comparable patient-outcome evidence. Claims about allogeneic edited CAR T cells should examine persistence and immune compatibility as well as initial response. This product-and-indication framing is more informative than declaring one generation of editors universally safer or more effective.

7. Conclusion

CRISPR precision in oncology is a property of an entire therapeutic system, not of a guide RNA alone. Nuclease editing has the most developed published cancer clinical experience; base editing has entered early human cell-therapy studies; prime editing offers broader sequence design but remains preclinical for cancer treatment. Progress requires explicit mechanistic rationale, allele-resolved product characterization, genomic and functional safety assays, and clinical endpoints that can separate an edited product's value from the rest of treatment. This framework makes the molecular-scalpel metaphor useful without overstating what present evidence can prove.

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Conflicts of Interest

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