Precision Reprogramming of Lung Cancer Biomarkers via CRISPR/Cas9: A Paradigm Shift in Personalized Immunotherapy

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Abstract: Lung cancer persists as the leading cause of cancer-related mortality globally, largely due to late detection, genomic complexity, and limited durability of existing therapeutic interventions. The integration of CRISPR/Cas9 gene-editing systems with biomarker-driven therapeutic strategies represents a transformative advance in precision oncology. Biomarkers—including genetic mutations, epigenetic alterations, protein signatures, and circulating analytes—enable early detection, patient stratification, and dynamic monitoring of therapeutic response. CRISPR/Cas9 offers a unique opportunity to directly reprogram these biomarkers or the pathways regulating them, enhancing tumor immunogenicity, reversing immune evasion mechanisms, and strengthening anti-tumor immune responses. Preclinical models demonstrate that CRISPR-mediated biomarker editing can restore antigen presentation, augment T-cell cytotoxicity, sensitize resistant tumors to immunotherapy, and improve tumor regression. Early clinical trials further validate the feasibility and safety of CRISPR-engineered immune cells in patients.

However, major challenges persist, including off-target editing, inefficient delivery to solid tumors, tumor microenvironment–mediated suppression, and ethical considerations linked to genome manipulation. Rapid advancements in editing fidelity, lipid nanoparticle systems, viral vectors, engineered vesicles, high-throughput biomarker discovery, and artificial intelligence–assisted CRISPR design are expected to accelerate clinical translation. This review synthesizes the current landscape, mechanistic underpinnings, emerging applications, and future directions of CRISPR/Cas9-enabled biomarker engineering for lung cancer immunotherapy, positioning this technology as a cornerstone of next-generation personalized oncology.

Keywords: CRISPR/Cas9, lung cancer, biomarkers, immunotherapy, precision oncology, PD-L1, ctDNA, antigen presentation, genome editing, T-cell engineering.

1. INTRODUCTION

Lung cancer is responsible for approximately 1.8 million deaths annually, cementing its position as the most lethal cancer worldwide [1,2]. The disproportionately high mortality rate results from late-stage diagnosis, rapid tumor progression, extensive heterogeneity, and resistance to current therapeutic strategies. The incorporation of molecular biomarkers into clinical workflows has significantly improved diagnosis, prognostication, therapeutic selection, and disease monitoring [3]. Advances in sequencing technologies and computational biology have led to the discovery of highly actionable genomic, proteomic, and epigenetic signatures that increasingly inform personalized treatment.

The development of CRISPR–Cas9 genome editing has transformed biomedical research through its ability to generate precise, programmable DNA modifications [4]. Within the context of oncology and lung cancer specifically, CRISPR/Cas9 offers a powerful platform to reprogram immunologically relevant biomarkers that govern tumor–immune dynamics. Although immune checkpoint inhibitors have improved survival in subsets of patients, resistance remains common. Tumors frequently evade the immune system by suppressing antigen presentation, altering regulatory pathways, or overexpressing inhibitory ligands such as PD-L1 [2]. CRISPR/Cas9 enables precise manipulation of these molecular determinants, providing a means to reverse immune escape and potentiate therapeutic responses.

Through direct editing of genes involved in antigen presentation, immune checkpoint regulation, tumor–immune interactions, and treatment resistance, CRISPR/Cas9 offers novel avenues to enhance the effectiveness of immunotherapies as shown in Figure

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1. This review examines current biomarker-directed strategies for lung cancer immunotherapy and evaluates the increasingly prominent role of CRISPR-enabled editing in shaping the future of personalized cancer treatment [3].

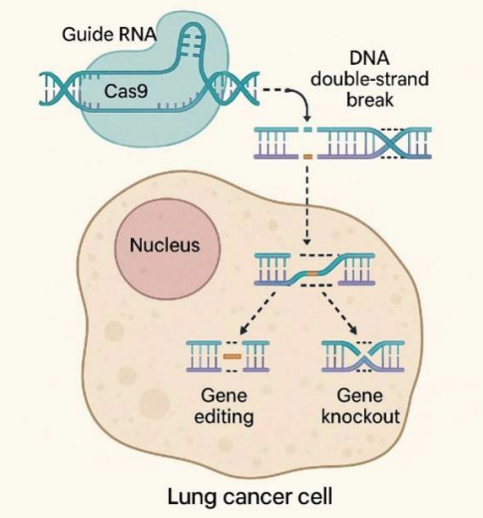

Figure 1: Malignant lung epithelial cell harboring genetic and epigenetic alterations.

2. CHALLENGES IN IDENTIFYING EARLY DETECTION BIOMARKERS

Early detection profoundly improves lung cancer outcomes. However, identifying reliable early-stage biomarkers is hindered by several biological and methodological constraints:

2.1. Minimal Tumor-Specific Alterations

Early-stage lung tumors are typically small and oftenlack distinct morphological or molecular features that differentiate them clearly from healthytissue. This makes early biomarker identification difficult and limits sensitivity.

2.2. Molecular Diversity in Lung Cancer

Lung cancer includes a range of subtypes—most notably non-small cell lung carcinoma (NSCLC) and small cell lung carcinoma (SCLC)—each characterised by unique molecular signatures. This diversity necessitates a broad and subtype-specific biomarker repertoire to achieve accurate and inclusive detection.

2.3. Non-Invasive Sample Limitations

For clinical utility, biomarkers should ideally bemeasurable in non-invasive or minimally invasive samples such as blood, sputum, or exhaledbreath. However, achieving both high sensitivity and specificity in these sample types istechnically demanding and often requires advanced detection platforms.

2.4. Validation and Standardization Barriers

Although many candidate biomarkers have shown promise in preclinical or small cohort studies, their translation to clinical practice is hindered by the need for validation across large, diverse patient populations. Robust standardisation is essential to confirm diagnostic accuracy and reproducibility. Addressing these challenges will require sustained research efforts to identify novel biomarkers, improve detection technologies, and rigorously validate biomarker performance in diverse clinical settings [5, 6].

3. PROMISING BIOMARKER CANDIDATES FOR EARLY LUNG CANCER DETECTION

The discovery and validation of biomarkers suitable for detecting lung cancer at an early, more treatable stage is critical to improving clinical outcomes. Several classes of biomarkers haves hown considerable promise in recent years, although each face unique technical and translational hurdle.

3.1. Circulating Tumor DNA (ctDNA)

Ct DNA comprises tumor-derived DNA fragments that circulate in the blood and can carry key genetic mutations found in lung cancer, including EGFR, KRAS, and TP53 alterations. While ct DNA concentrations are typically low in early-stage disease, high-sensitivity technologies such as next-generation sequencing (NGS) and droplet digital PCR (ddPCR) now allow for mutation detection with unprecedented precision. These methods support ctDNA 's utility in non-invasive screening and molecular profiling. [7,8].

3.2. CIRCULATING TUMOR CELLS (CTCS)

CTCs are shed from primary tumors into the bloodstream and represent a minimally invasive window into tumor biology. Although their numbers are extremely low in early-stage disease, advances in isolation techniques—such as microfluidic enrichment and immunomagnetic separation—have significantly improved the ability to detect and analyse these rare cells, positioning CTCs as strong candidates for early detection [9].

3.3. MicroRNAs (miRNAs)

miRNAs are small, non-coding RNAs that modulate gene expression and play critical roles in oncogenesis.

Certain miRNAs—including miR-21, miR-155, and miR-486—are consistently upregulated in lung cancer and are detectable in body fluids like plasma and sputum, offering a stable and accessible biomarker source. Despite encouraging data, further large-scale validationis required to confirm their diagnostic and prognostic utility [10,11].

3.4. Autoantibodies against Tumor Antigens

Tumor-associated autoantibodies emerge in response to antigens expressed by tumor cells and can be detected well before clinical symptoms appear. Panels targeting antigens such as p53, NY-ESO-1, and MAGE-A4 have demonstrated potential in detecting early-stage lung cancer with moderate accuracy. Nevertheless, improving assay sensitivity and refining biomarker panels remain essential for clinical application [12].

3.5. Multi-Analyte Biomarker Panels

Integrating ctDNA, CTCs, miRNAs and autoantibodiesinto multi-analyte assays offers a path forward for improving diagnostic sensitivity and specificity. Coupling these platforms with machine learning and artificial intelligence is expected to enhance pattern recognition and risk stratification, enabling more personalised and effective early detection strategies as presented in Figure **2**.

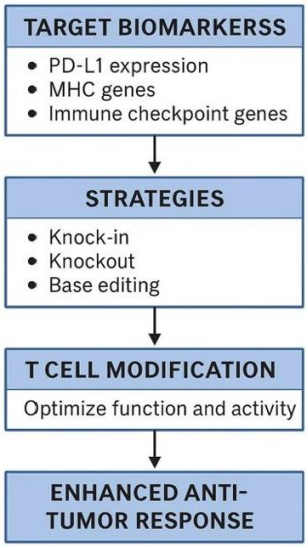

Figure 2: Genomic analysis identifies key mutations and immune-related biomarkers in lung cancer tissues.

Ultimately, the development of a robust, non-invasive, and highly sensitive biomarker panel capable of identifying lung cancer at its earliest stages could transform screening paradigms and significantly improve patient outcomes.

4. TYPES OF BIOMARKERS IN LUNG CANCER

Lung cancer biomarkers are broadly categorised based on their role in diagnosis, prognosis, and treatment guidance.

- Genetic mutations in driver genes such as EGFR, ALK, ROS1, KRAS, and BRAF play a criticalrole in lung cancer pathogenesis and are key determinants for targeted therapies, particularly tyrosine kinase inhibitors (TKIs) in patients with EGFR mutations [13].

- Protein biomarkers, especially programmed death-ligand 1 (PD-L1), are pivotal inimmunotherapy. PD-L1 expression levels predict response to PD-1/PD-L1 inhibitors, which have transformed the treatment landscape for lung cancer [14].

- Circulating analytes such as Circulating tumor cells (CTCs), detectable in peripheral blood, provide insights into tumor dissemination and are associated with disease progression and recurrence. Similarly, circulating tumor DNA (ctDNA) offers a minimally invasive method to assess mutational profiles in real-time, facilitating molecular diagnosis and monitoring [15].

- Non-coding RNAs, particularly microRNAs (miRNAs) such as miR-21 and miR-155, areemerging as valuable prognostic indicators. These molecules influence tumor biology and maypredict therapeutic response or disease aggressiveness.

- DNA methylationare gaining prominence in early detection strategies. For instance, hypermethylation of CDKN2A and SHOX2 has shown in Table-**1** to improve the sensitivity and specificity of non-invasive diagnostic methods [16-18]

These biomarkers enable diagnosis, guide targeted therapy, inform immunotherapy decisions, and support prognosis assessment.

5. TARGET BIOMARKERS FOR CRISPR/CAS9-BASED IMMUNOTHERAPY ENHANCEMENT

CRISPR/Cas9 gene-editing technology enables precise manipulation of specific biomarkers to enhance immune-mediated tumor clearance. Key targets include:

Table 1: Summary of Key Biomarkers in Lung Cancer and CRISPR/Cas9 Applications

Sr. No.	Biomarker	Role	Therapeutic Implication	CRISPR/Cas9 Application	References
1.	EGFR mutation	Oncogenic driver	Targeted by TKIs (gefitinib, erlotinib)	Gene correction/enhancement of drug sensitivity	Yuan *et al*., 2019
2.	ALK mutation	Oncogenic fusion	Targeted by ALK inhibitors	Potential gene editing to restore normal function	Yuan *et al*., 2019
3.	PD-L1 expression	Immune checkpoint ligand	Predicts response to PD-1/PD-L1 inhibitors	Modulation of expression to improve immunotherapy	Mino-Kenudson *et al*., 2022
4.	MHC molecules	Antigen presentation	Enhances tumour immunogenicity	Upregulation to boost immune recognition	Stefanoudakis *et al*., 2023; Cheung *et al*., 2018
5.	Circulating tumor DNA (ctDNA)	Disease monitoring	Assesses treatment response and resistance	Development of sensitive detection biomarkers	Das *et al*., 2023
6.	Prognostic signatures	Recurrence risk	Guides surveillance and adjuvant therapy	Elucidates relapse mechanisms	Zhou *et al*., 2023

5.1. PD-L1 Expression

Programmed death-ligand 1 (PD-L1) is one of the most clinically significant biomarkers in lung cancer immunotherapy. Tumors frequently upregulate PD-L1 to suppress T-cell activity. CRISPR/Cas9-mediated modulation of PD-L1 expression has been shown to alter tumor–immune interactions, sensitize tumors to PD-1/PD-L1 inhibitors, and reverse immunotherapy resistance [19].

5.2. MAJOR HISTOCOMPATIBILITY COMPLEX (MHC) GENES

Upregulationof MHC class I/II molecules is a common mechanism of immune escape. CRISPR-based upregulation of MHC genes significantly enhances antigen presentation and strengthens CD8+ T-cell recognition of lung cancer cells [20].

5.3. IMMUNE CHECKPOINT MOLECULES (CTLA-4, LAG-3)

CRISPR-mediated knockout of inhibitory checkpoint molecules including CTLA-4 and LAG-3 in T cells enhances cytotoxicity and promotes sustained immune engagement shown in Figure **3**. Preclinical studies show that eliminating these pathways markedly improves T-cell infiltration and tumor regression [21,22].

6. GENE EDITING STRATEGIES FOR BIOMARKER ENGINEERING

CRISPR/Cas9 offers multiple editing strategies suited for distinct biomarker targets and immune engineering goals:

6.1. Knock-In Approaches

Used to introduce beneficial regulatory genes, enhance antigen presentation, or engineer synthetic receptors that heighten immune sensitivity. For example, knock-in of PD-L1 into previously negative cell populations enables mechanistic interrogation of immune escape [24].

6.2. Knockout Approaches

Disrupting immunosuppressive genes, such as CTLA-4 and LAG-3, in T cells removes inhibitory signals, thereby boosting their anti-tumor function [24, 25]. Researchers have designed a gRNA targeting exon 3 of the CD274 gene (which encodes PD-L1). The successful knockout of PD-L1 reduced immune evasion in NSCLC cell lines, enhancing T- cell mediated cytotoxicity. This provided proof-of-concept that CRISPR can directly modulateimmune checkpoint biomarkers in lung cancer models. [26].

6.3. Base Editing

Base editingallows precise single-nucleotide conversions without generating double-strand breaks. This approach is particularly useful for:

- repairing point mutations,
- modulating SNPs affecting antigen presentation,
- or reducing off-target mutational risks [27].

6.4. Prime Editing

Prime editors expand precision editing by enabling targeted insertions, deletions, and substitutions without

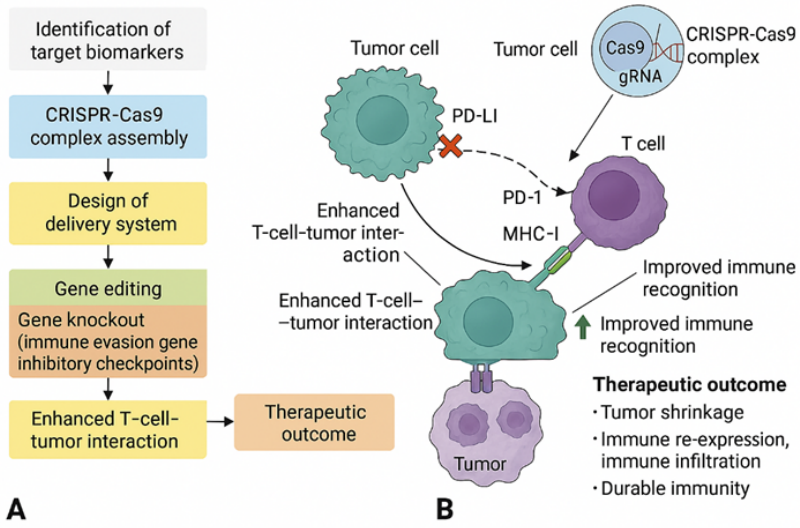

Figure 3: A Identification of genomic biomarkers (e.g., PD-L1, EGFR, ALK). **B** Down regulation of immune checkpoint expression or restoration of tumor suppressor function.

donor templates. Prime editing shows high promise for biomarker refinement and the correction of oncogenic mutations [28].

7. CURRENT STATUS OF CRISPR-MEDIATED BIOMARKER EDITING

The application of CRISPR/Cas9 for biomarker editing in lung cancer immunotherapy is advancing through multiple phases:

7.1. Preclinical Studies

CRISPR/Cas9 editing in lung cancer models has demonstrated enhanced immunotherapeutic responses and tumor suppression. *In vitro* assays with lung cancer cell lines and *in vivo* animal models reveal that biomarker modification improves immune recognition and tumor cell clearance. Knockout of immune checkpoint genes in T cells promotes sustained antitumor activity, while upregulation of MHC expression in tumor cells increases immune visibility[29,30].

7.2. Clinical Trials

CRISPR/Cas9 editing in lung cancer models has demonstrated enhanced immunotherapeutic responses and tumor suppression. *In vitro* assays with lung cancer cell lines and *in vivo* animal models reveal that biomarker modification improves immune recognition and tumor cell clearance. Knockout of immune checkpoint genes in T cells promotes sustained antitumor activity, while upregulation of MHC expression in tumor cells increases immunevisibility [29, 31].

7.3. Regulatory Approvals

Regulatory agencies rigorously assess gene-editing therapies to ensure safety and efficacy prior to clinical adoption [32].

7.4. Research and Development

Efforts are directed toward improving delivery vehicles, enhancing editing specificity, expanding target biomarkers, and integrating CRISPR/Cas9 with adjunct therapies such as chemotherapy and radiotherapy to achieve synergistic effects [33].

7.5. Challenges

Significant obstacles are as follows:

- Off-Target Effects: Unintended gene edits pose risks including oncogene activation ordisruption of essential genes. Strategies to mitigate these include highly specific guide RNAs,high-fidelity Cas9 variants, and comprehensive off-target screening [34]. CRISPRoff□target risks include oncogene activation (MYC) or essential gene disruption (TP53).Mitigation uses specific gRNAs (EGFR), high□fidelity Cas9 (KRAS), and GUIDE□seqscreening (PD□L1).

- Delivery: Efficient and targeted delivery of CRISPR components to tumor or immune cellsremains a major hurdle. Viral vectors (lentivirus, AAV), lipid nanoparticles, and novel non-viralsystems are under investigation to improve delivery efficiency and reduce immunogenicity (33, 35]

- Ethical and Safety Considerations: Permanent genomic alterations raise concerns about safety, consent, and germline editing. Robust ethical guidelines and transparent stakeholder engagement are imperative [36]. The crucial ethical distinction is that somatic editing impacts the individual level, but germline editing permanently alters the genetic makeup and passes it on to subsequent generations. Somatic editing affects cells in non-reproductive organs such the liver, lung, and blood, but no alterations are passed on to progeny [37]. There are currently many clinical therapies and approvals or studies based on clinical evidence for treating serious disorders, such as sickle cell disease, and there is a limited number of treated patients with risks and benefits. When the germline of reproductive cells—such as eggs, sperm, and embryos—is edited, the change is passed on to the following generations because these cells vary from somatic cells [38]. Furthermore, their procedures permanently alter the human gene pool, making consent impossible for future generations. The Cas9 approach is used to treat cancer DNA in cells by correcting their genes. Scientists are trying to improve these methods to lessen the frequency of side effects, and these treatments have been approved for possible safety hazards. Cas9 gene editing in cancer patients is safe and provides essential evidence of the concept's viability.

- Immune Response: Host immunity against CRISPR components may impair therapeutic efficacy. Approaches include use of humanized Cas9 variants and transient delivery to minimizeimmune activation [39].

- Tumor Microenvironment: Immunosuppressive tumor milieus can limit CRISPR efficacy.Combining editing with agents modulating the microenvironment, such as cytokines, mayenhance outcomes [40].

- Cost and Accessibility: High production costs and limited access hinder widespread clinicaluse. Advances in manufacturing and policy interventions are needed to improve affordability andequity [33].

8. BIOMARKERS FOR PERSONALIZED TREATMENT

Biomarkers guide tailored therapeutic strategies, improving efficacy and minimizing toxicity:

Targeted Therapies: Mutations in EGFR and ALK predict responses to tyrosine kinas inhibitors. CRISPR/Cas9-mediated correction of such mutations may restore normal gene function or enhance drug efficacy, offering personalized treatment avenues [41].

Immunotherapy: PD-L1 expression predicts response to PD-1/PD-L1 checkpoint inhibitors. Editing PD-L1 levels in tumor cells can potentiate immunotherapy efficacy in patients with lowendogenous expression [42].

Biomarkers for Prognosis and Monitoring

Biomarkers also inform prognosis and therapeutic monitoring:

Recurrence Risk: Specific biomarker profiles predict recurrence risk, enabling individualized surveillance and adjuvant therapy planning. Editing prognostic biomarkers may elucidate mechanisms underlying relapse [43]**.**

Treatment Response: Biomarkers such as circulating tumor DNA (ctDNA) track therapeutic response and resistance emergence. CRISPR/Cas9 can facilitate development of precise markers to guide real-time treatment adjustments [6].

Advantages

CRISPR/Cas9-based biomarker editing offers distinct benefits:

Personalized Immunotherapy: Editing tailored to individual tumor profiles enhances treatment specificity and patient outcomes [2].

Overcoming Resistance: Biomarker editing can reverse immunotherapy resistance, restoring tumor sensitivity and prolonging efficacy[44].

Enhanced T Cell Activity: Gene editing in T cells augments anti-tumor potency and persistence, improving immune responses [45].

Precision and Safety: High specificity minimizes off-target effects, increasing therapeutic safety and efficiency [46].

9. APPLICATIONS OF BIOMARKERS

Biomarkers serve critical roles across lung cancer management, encompassing diagnosis, prognosis, and treatment stratification:

Diagnosis: Biomarkers aid differentiation between malignant and benign pulmonary nodules detected by imaging, reducing unnecessary invasive procedures and enabling timely intervention. CRISPR/Cas9-based diagnostics targeting specific genetic or epigenetic alterations promise enhanced diagnostic accuracy and rapidity [47].

Treatment Selection

Targeted Therapies: Mutations in genes such as EGFR, ALK, ROS1, KRAS, and BRAF predict responsiveness to targeted agents. CRISPR/Cas9-mediated correction or modulation of these mutations could restore gene function or sensitize tumors, refining personalized treatment strategies [41].

Immunotherapy: PD-L1 expression guides immune checkpoint blockade efficacy. CRISPR/Cas9-driven modulation of PD-L1 levels may broaden patient eligibility and enhance response rates to PD-1/PD-L1 inhibitors [42].

Prognosis and Surveillance: Biomarkers facilitate recurrence risk assessment and treatment monitoring. Editing prognostic markers can elucidate tumor biology and improve relapse prevention. Additionally, CRISPR-enhanced biomarker assays may improve real-time evaluation of therapeutic efficacy and early resistance detection [48].

11. FUTURE DIRECTIONS

Advancements in CRISPR/Cas9 biomarker editing for lung cancer immunotherapy will focus on:

- Optimizing delivery platforms—engineered viral vectors, lipid nanoparticles, and exosomes—to increase targeting precision while minimizing immunogenicity [49,50]. Developing combination regimens integrating CRISPR editing with chemotherapy, radiotherapy, or immune checkpoint blockade to potentiate synergistic effects and overcome resistance [51].
- Establishing rigorous ethical frameworks and fostering inclusive discourse to guide the responsible application of permanent gene editing [52].
- Employing multiplex biomarker panels to enhance diagnostic precision and inform complex therapeutic decisions [48].
- Advancing non-invasive biomarker detection through liquid biopsy techniques coupled with CRISPR-based diagnostics for improved patient monitoring [53].
- Discovering novel biomarkers via high-throughput proteomic and genomic screening to enable earlier detection and more effective disease management [54].
- Integrating artificial intelligence and machine learning with CRISPR-enabled biomarker data to refine predictive models and personalize therapy [55].
- Using CRISPR-Cas9 systems to influence AI and machine learning for genome editing: Computer tools, particularly Artificial Intelligence (AI) and AI machine learning sub-tools, are becoming essential. Large datasets produced by genetic sequencing and research investigations can be swiftly analyzed by these cutting-edge technologies. By using AI and ML to identify the most lucrative gene targets for a particular tumor profile, CRISPR medicines can be quickly and precisely customized [56].
- Future Research Hypotheses with Particular Testable: Enhancing efficacy and guaranteeing safety are the current research priorities, and there are two hypotheses for further study withparticular relevance

i. Hypothesis (Efficacy Development): Compared to single-gene edited T-cells in comparable models of solid tumor organoids, specific complex CRISPR/Cas9 knockout of significant inhibitory checkpoint genes of immunity (such as LAG-3, PD-1, andTIGIT) will produce a measurable 30% increase in T-cell determination and tumorpenetration in patient-derived T-cells [57]. (This hypothesis evaluates the effectiveness ofsophisticated editing for humanizing toughness, a current immunotherapy limitation)

ii. Hypothesis (Precision and Safety): A model of machine learning educated on a dataset of obtainable off-target activity will be capable of calculating specific guide RNAsequences that decrease measurable off-target editing procedures by 50% in patient T-cells, thus considerably improving the security outline of precise CRISPR therapies [58].(This

hypothesis connects the necessity of computer tools to concentrate on thebasicethical distress regarding safety and unintended results.)

Moreover, artificial intelligence in combination with editing of genome and personalized medicine, enables precise treatments based on genetic profiles. It explored patients' genomic data, identifying disease-associated mutations, biomarkers, and variations, for example, that in cancer, Alzheimer's, diabetes, and more. It envisages personalized cures, considering efficacy, resistance, and toxicity to various drugs and therapies.

12. CONCLUSION

Biomarkers remain foundational to advancing personalized lung cancer management, enabling clinicians to stratify treatments, detect resistance earlier, and monitor disease progression with greater accuracy. The incorporation of CRISPR/Cas9 genome-editing technologies into biomarker engineering represents a transformational step forward, offering the ability to directly modify regulatory pathways that govern tumor progression, immune evasion, and therapeutic responsiveness.

Substantial evidence demonstrates that CRISPR-mediated editing of key biomarkers—such as PD-L1, MHC molecules, and inhibitory checkpoints—can enhance tumor immunogenicity, strengthen T-cell activation, and restore sensitivity to immune checkpoint inhibitors. These improvements directly address many of the limitations currently observed in immunotherapy for lung cancer, including adaptive resistance, weak antigen presentation, and suppressive tumor microenvironments.

Despite considerable promise, several challenges must be addressed before CRISPR-engineered biomarkers become standard clinical practice. Off-target mutations remain a persistent concern and necessitate improved guide RNA design, high-fidelity Cas variants, and extensive preclinical evaluation to ensure genomic safety. Delivery challenges—particularly in reaching dense and heterogeneous solid tumor sites—require further innovation in lipid nanoparticles, viral systems, and extracellular vesicle–based delivery strategies. Ethical considerations also remain central, particularly the distinction between somatic editing (clinically permissible) and germline editing (widely prohibited due to heritable risks). Future progress will rely heavily on interdisciplinary collaboration across immunology, genomics, computational biology, materials science, and regulatory policy. Rapid advancements in high-throughput biomarker discovery, CRISPR precision engineering, and multi-omic data integration are expected to support the development of more refined and clinically actionable biomarker panels. Moreover, the adoption of AI and machine learning will further enhance predictive modelling, risk stratification, and optimization of genome-editing tools, enabling safer and more personalized treatment decisions.

Ultimately, CRISPR/Cas9-driven biomarker reprogramming holds unprecedented potential to reshape the landscape of lung cancer therapy. By enabling therapies that are more targeted, more durable, and tailored to the unique molecular profile of each patient, this approach represents a major step toward the realization of truly personalized oncology and improved patient outcomes worldwide.

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CONFLICT OF INTEREST

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