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Personalized Neoantigen Vaccines and CAR-T Cell Therapy: A New Frontier in Cancer Immunotherapy

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Abstract. Cancer immunotherapy has achieved groundbreaking successes with strategies like immune checkpoint inhibitors, yet many solid tumors evade immune control. Personalized neoantigen vaccines offer a new level of precision by targeting tumor-specific mutations, thereby inducing highly specific T cell responses against cancer with minimal off-target effects. Chimeric antigen receptor (CAR) T cell therapy has revolutionized treatment of hematologic malignancies, attaining remarkable remission rates, but its efficacy in solid tumors is limited by antigen heterogeneity and immunosuppressive microenvironments. Recent clinical trials of personalized neoantigen vaccines (in melanoma, pancreatic cancer, and others) demonstrate robust immunogenicity and preliminary anti-tumor activity. These findings, together with CAR-T's potent cytotoxicity, provide a strong rationale for combined vaccine and CAR-T approaches. This paper reviews the latest clinical advancements, examines the challenges of solid tumors, proposes the synergistic integration of neoantigen vaccines with CAR-T therapy, and discusses future perspectives for this emerging frontier.

Keywords: Neoantigen vaccine, immunotherapy, CAR-T cell therapy.

1. Introduction

Immunotherapy has transformed oncology over the past decade, yielding durable remissions in previously intractable cancers. The development and regulatory approval of immune checkpoint inhibitors (ICIs) such as anti-CTLA-4 and anti-PD-1, as well as CAR-T cell therapies, have established new treatment paradigms. Despite these breakthroughs, the majority of solid tumors remain resistant. Tumors can escape immune attack through various mechanisms: they may downregulate antigen presentation, create immunosuppressive microenvironments, or exhibit profound intra-tumoral heterogeneity that allows immune evasion [1, 2]. As a result, only a minority of patients with solid cancers derive long-term benefit from current immunotherapies. There is a critical need for novel strategies that enhance immune specificity and overcome tumor escape.

One promising approach is the use of tumor neoantigens – antigens arising from somatic mutations unique to an individual's cancer. Unlike traditional tumor-associated antigens (TAAs), which are self-proteins aberrantly expressed by tumors, neoantigens are non-self-peptides found only on cancer cells. TAAs often induce tolerized or weak immune responses due to their presence (at low levels) in normal tissues, whereas neoantigens are not subject to central thymic tolerance and thus are highly immunogenic [3, 4]. By harnessing neoantigens, personalized cancer vaccines can generate a focused T cell attack on tumor cells with minimal risk to healthy tissue.

Recent clinical advances underscore the potential of personalized neoantigen vaccines. Pioneering first-in-human studies in melanoma demonstrated the feasibility, safety, and immunogenicity of this approach. Ott vaccinated melanoma patients with up to 20 personal neoantigen peptides, eliciting robust CD4^+ and CD8^+ T cell responses in all patients. Strikingly, four of six vaccinated high-risk melanoma patients remained recurrence-free at 2 years, and the two who relapsed achieved complete tumor regression upon subsequent anti-PD-1 therapy, accompanied by an expansion of vaccine-induced T cells [1]. Independently, Sahin treated melanoma patients with an RNA-based neoantigen vaccine and likewise observed T cell responses against multiple neoepitopes; in patients with metastatic disease, vaccine-induced immune responses led to tumor regressions, including one

complete response when combined with PD-1 checkpoint blockade [2]. Beyond melanoma, personalized vaccines have been tested for other malignancies. In glioblastoma, neoantigen vaccines are safe and immunogenic, inducing T cells that infiltrate the brain tumor [5, 6]. In a recent landmark trial in pancreatic cancer, an individualized mRNA neoantigen vaccine (BNT122) given after surgical resection induced high-magnitude T cell responses in roughly half of the patients and significantly prolonged recurrence-free survival in those who mounted such responses [3]. These data provide encouraging proof of concept that vaccines targeting private tumor mutations can overcome immune tolerance and spark clinically relevant immunity even against aggressive solid tumors.

In parallel, CAR-T cell therapy has achieved unparalleled success in certain blood cancers. CD19-directed CAR-T cells, for example, can produce complete remission in ~80–90% of refractory acute lymphoblastic leukemia patients, and multiple CAR-T products have gained FDA approval for B cell leukemias, lymphomas, and plasma cell myeloma. However, translating CAR-T efficacy to solid tumors has proven challenging, owing to issues such as lack of unique target antigens, dense stroma and immunosuppressive cells limiting T cell infiltration, and T cell exhaustion within the tumor microenvironment. These limitations curtail CAR-T cell activity in solid tumors despite their potent cytotoxic potential in principle [7, 8].

This paper will review the latest clinical and technological advances in personalized neoantigen vaccines, the obstacles faced by CAR-T cell therapy in solid tumors and emerging strategies to overcome them, and the scientific rationale and preliminary efforts to combine neoantigen vaccination with CAR-T cell therapy. By examining these developments, we aim to highlight the synergistic promise of merging two cutting-edge immunotherapies and outline future directions for research and clinical translation.

2. Personalized Neoantigen Vaccines: Clinical and Technological Advances

Personalized neoantigen vaccination is an individualized therapeutic strategy that begins with identifying somatic mutations unique to a patient's tumor [9, 10]. Typically, whole-exome sequencing (WES) of tumor and normal DNA, coupled with RNA sequencing of the tumor, is performed to catalog nonsynonymous mutations. Candidate neoantigen peptides are then predicted in silico using algorithms (e.g. NetMHCpan) that evaluate peptide binding affinity to the patient's HLA molecules [11, 12]. Only a subset of mutations give rise to peptides that can be processed, presented on MHC, and recognized by T cells; thus, accurate prediction is critical. Improved bioinformatics and machine learning approaches – including the integration of mass spectrometry data and deep learning models – have enhanced the identification of truly immunogenic neoepitopes [13]. Once neoantigen candidates are selected (often 10–20 per patient), a patient-specific vaccine is manufactured.

Several vaccine formats are in clinical use. Long peptides representing mutant epitopes (often 15–30 amino acids to cover multiple HLA-presentable fragments) can be synthesized and pooled, typically administered with a potent adjuvant such as poly-ICLC (a TLR3 agonist) [1]. mRNA vaccines are another versatile platform: encoding multiple neoantigens in a single RNA construct that is injected (often encapsulated in lipid nanoparticles) to drive the transient production of neoantigen peptides by host cells [2]. mRNA vaccines can stimulate both CD8^+ and CD4^+ T cell responses and were notably validated by recent successes in infectious diseases (e.g. COVID-19 mRNA vaccines). Dendritic cell (DC) vaccines represent a cell-based approach, wherein the patient's own DCs are pulsed with neoantigen peptides or transfected with neoantigen-encoding RNA ex vivo, then returned to the patient to present those neoantigens to T cells. Each approach has merits: peptide vaccines are chemically stable and straightforward to produce, whereas RNA vaccines can encode many neoantigens and amplify antigen presentation in situ. The overall workflow – from sequencing to prediction to vaccine fabrication – typically spans a few weeks, and ongoing process optimization aims to shorten this interval to treat patients earlier in their disease course.

Table 1 summarizes selected clinical trials of personalized neoantigen vaccines. The first-in-human studies in melanoma provided a foundation for the field. Ott et al. treated six melanoma patients (most

with high-risk resected disease) with a custom peptide vaccine and reported that all patients developed polyfunctional T cell responses to multiple neoantigens, with CD4^+ T cells recognizing ~60% of the vaccine peptides and CD8^+ T cells ~16%. These T cells could distinguish mutant peptides from wild-type and, in some cases, directly recognize the patient's tumor cells. At a median follow-up of 25 months, four patients remained free of melanoma recurrence, and two who did relapse responded completely to subsequent PD-1 inhibitor therapy with evidence of an expanded neoantigen-specific T cell repertoire [1]. Sahin et al. conducted a parallel trial using an RNA vaccine in 13 melanoma patients. They observed T cell responses against 20 or more distinct neoepitopes per patient, at magnitudes reaching high single-digit percentages of circulating T cells. In five patients with active metastatic melanoma, two experienced objective tumor regressions from the vaccine alone, and a third achieved a complete response when the vaccine was combined with anti-PD-1 therapy [2]. These studies were landmark demonstrations that personalized vaccines can be feasible, safe, and immunologically potent in humans.

Other cancer types have since been explored. In glioblastoma multiforme (GBM), a highly aggressive brain cancer, neoantigen vaccines induced measurable immune responses despite the immune-privileged setting [5, 6]. In a phase I study, patients with newly diagnosed GBM were vaccinated with personal neoantigen peptides after surgery and standard chemoradiation; vaccine-induced T cells were detected trafficking into intracranial tumors, although the clinical benefit was limited as tumors eventually progressed, highlighting the need for combination therapies in such fast-growing cancers [5, 6]. In pancreatic ductal adenocarcinoma (PDAC) – traditionally considered immunologically "cold" – a recent personalized mRNA vaccine showed promising efficacy. Rojas et al. (2023) vaccinated 16 PDAC patients after tumor resection and observed that 50% of patients developed substantial neoantigen-specific T cell responses; these responders had significantly prolonged recurrence-free survival compared to non-responders, with no relapses in the vaccine responders at 18-month follow-up [3]. This study provided the first evidence of clinical benefit from a neoantigen vaccine in pancreatic cancer, an important proof-of-concept in a malignancy notoriously resistant to immunotherapy. In hepatocellular carcinoma (HCC), early trials suggest personalized vaccines can be implemented in the adjuvant setting: patients with resected high-risk HCC (e.g. with vascular invasion) have received custom neoantigen peptide vaccines, and immune monitoring (including circulating tumor DNA sequencing for the neoantigen mutations) indicated that vaccine-induced T cell responses correlated with reduced tumor recurrence, although definitive efficacy data are still maturing.

Table 1. Selected clinical trials of personalized nneoantigen vaccines

Study (Year)	Cancer Type	Vaccine Platform	Patients (N)	Key Outcomes
Ott et al. (2017)	Melanoma (high-risk)	Long peptides + poly-ICLC	6	T cell responses to ~60% of neoantigens; 4/6 relapse-free at 2 years; 2 relapses rescued by anti-PD-1 (complete remissions).
Sahin et al. (2017)	Melanoma (metastatic & adjuvant)	IV RNA-lipoplex vaccine	13	T cells induced against dozens of neoantigens; in 5 metastatic patients: 2 partial responses to vaccine alone, 1 complete response with vaccine+PD-1 therapy.
Keskin et al. (2019)	Glioblastoma	Long peptides + poly-ICLC	8	Vaccine-specific T cells detected in blood and tumor; evidence of T cell infiltration in the brain; clinical benefit limited (median PFS not significantly extended).
Rojas et al. (2023)	Pancreatic cancer (adjuvant)	Nucleoside-modified mRNA (IV)	16	~50% of patients mounted neoantigen-specific CD8^+ T cell responses; responders had no relapse at 18 months (vs early relapses in non-responders), indicating prolonged RFS.
Chen et al. (2021)	Hepatocellular carcinoma (adjuvant)	Long peptides (personalized)	14	Induced neoantigen-specific T cells; post-vaccine monitoring of ctDNA neoantigens suggested a correlation between immune response and delayed recurrence.

An emerging theme is the synergy between neoantigen vaccines and other immunomodulators, particularly ICIs. Vaccines can broaden the intratumoral T cell repertoire, but vaccine-induced T cells may still be susceptible to regulatory checkpoints. Combining vaccination with PD-1/PD-L1 or CTLA-4 blockade may therefore unleash the full antitumor potential of the induced T cells. In the melanoma peptide vaccine study by Ott et al., patients who relapsed after vaccination responded to pembrolizumab, implying that checkpoint blockade amplified the vaccine-primed T cell responses to eradicate the tumor [1]. A more direct demonstration comes from a randomized phase II trial of an mRNA neoantigen vaccine (mRNA-4157, Moderna) in melanoma. In this study, patients who had resected melanoma received either pembrolizumab alone or pembrolizumab plus the personalized mRNA vaccine. The combination arm showed significantly improved outcomes: the vaccine+PD-1 therapy reduced the risk of recurrence or distant metastasis by 44–65% compared to immunotherapy alone. This marked the first randomized evidence that adding a neoantigen vaccine can augment the efficacy of checkpoint inhibition. Immunologically, patients receiving the vaccine had an expansion of neoantigen-specific T cells that would then be less inhibited by PD-1, leading to more effective tumor surveillance. Similar vaccine–ICI combinations are now under investigation in lung cancer, renal cancer, and other settings. While results are preliminary, they consistently demonstrate that vaccines can be safely combined with ICIs and often result in increased T cell activation within the tumor.

3. CAR-T in Solid Tumors: Challenges and Opportunities

CAR-T cell therapy involves genetically reprogramming a patient's T cells with a chimeric antigen receptor that enables direct recognition and killing of cancer cells. This strategy has yielded remarkable results in hematologic cancers. Dozens of trials in advanced leukemias and lymphomas

have reported high response rates – for example, anti-CD19 CAR-T cells produce complete remission in a majority of patients with relapsed B cell acute lymphoblastic leukemia – and durable remissions in a substantial fraction [7]. Since 2017, at least six CAR-T cell products targeting B cell antigens (CD19 or BCMA) have been approved by the FDA, establishing CAR-T therapy as a standard of care in refractory leukemias, lymphomas, and multiple myeloma [8]. These successes in "liquid tumors" stem from the presence of a homogeneous target antigen (e.g. CD19) on essentially all cancer cells and an environment in which infused CAR-T cells can traffic and function relatively unimpeded [13].

Translating CAR-T therapy to solid tumors, however, has been far more challenging. Solid tumors present several unique barriers:

(a) Antigen Selection and Heterogeneity: Unlike B-cell malignancies, solid tumors generally lack a uniformly expressed, tumor-specific cell-surface antigen. Most targetable antigens on solid tumors are also expressed in some healthy tissues, raising safety concerns, and tumor heterogeneity means antigen-negative cancer cells can escape recognition.

(b) Tumor Microenvironment (TME) and Immunosuppression: Solid tumors create a hostile microenvironment replete with immunosuppressive factors that inhibit T cell function. Physical barriers like dense stroma can also impede T cell infiltration.

(c) T Cell Exhaustion and Lack of Persistence: When CAR-T cells encounter antigen chronically (as is likely in solid tumors where antigen-positive cells may not be fully eliminated), they can become exhausted or dysfunctional over time. The immunosuppressive cytokines and regulatory cells in the TME further promote T cell exhaustion and limit CAR-T persistence.

Despite these challenges, extensive research is underway to adapt CAR-T technology for solid tumors, also with several strategies showing promise:

(a) Armored CAR-T Cells: One approach is to engineer CAR-T cells with additional genetic modifications to better withstand or modulate the TME. So-called "armored" CAR-T cells can secrete immune-stimulatory cytokines or carry dominant-negative receptors to block inhibitory signals. For example, CAR-T cells that constitutively secrete IL-12 have been tested in preclinical models of solid tumors. IL-12 can polarize macrophages toward a pro-inflammatory phenotype and enhance T_h1 responses, counteracting immunosuppression. In a glioblastoma model, intratumoral delivery of IL-12 by CAR-T cells led to improved T cell persistence and tumor control [14]. Other armored CAR designs include CAR-T cells secreting IL-18 (to recruit helper and memory T cells) or expressing a PD-1/CD28 switch receptor (which turns a PD-L1 inhibitory signal into a T cell co-stimulatory signal). Early-phase clinical trials of armored CAR-T cells, such as IL-12-secreting CAR-T in ovarian cancer and IL-18-secreting CAR-T in mesothelioma, are ongoing; preliminary reports indicate these cells remain functional longer within suppressive TMEs, though safety (cytokine-related toxicities) must be carefully managed [9].

(b) Dual-Targeting and "Logic-Gated" CAR-T Cells: To address antigen heterogeneity and prevent escape, researchers are designing CAR-T cells that recognize two (or more) antigens. Some next-generation CAR-T cells are engineered with "AND" gates – they require recognition of Antigen A and Antigen B to activate – which increases specificity and reduces off-tumor effects. Others have "OR" gate designs enabling activity if any one of several target antigens is present, thereby covering tumor variants. Preclinical studies have shown that dual-targeted CAR-T approaches can kill tumors that would evade single-target CAR-T cells. Early trials are testing CAR-T cells dual-directed at HER2 and IL13Rα2 in glioblastoma, for example, aiming to prevent antigen escape.

(c) Enhancing Trafficking and Fitness: Other innovations aim to improve CAR-T cell infiltration and survival in solid tumors. Co-expression of chemokine receptors matching the tumor's chemokine profile can attract CAR-T cells into tumor sites. Additionally, modifications that promote memory cell formation or resistance to exhaustion (such as overexpression of certain transcription factors or cytokine receptors) are being explored to extend CAR-T persistence. Researchers are also

investigating non-invasive locoregional delivery of CAR-T cells (e.g. direct intratumoral or intraperitoneal injection) to concentrate cells at the tumor and limit systemic side effects.

Up to now, no CAR-T cell therapy for a solid tumor has been approved, and clinical trial results have shown only modest efficacy in most cases [10]. For example, anti-GD2 CAR-T cells in neuroblastoma have induced some complete responses but with frequent relapses; CAR-T cells targeting glypican-3 in liver cancer or EGFR in glioblastoma have mostly achieved stable disease or partial responses at best. The field, however, is rapidly evolving. Each obstacle (antigen escape, TME suppression, T cell dysfunction) is being met with innovative bioengineering solutions as described. The "second-generation" and "third-generation" CAR-T cells currently in trials incorporate many of these features (armoring, dual targeting, etc.), and combinatorial approaches are increasingly considered (for instance, giving CAR-T therapy alongside checkpoint inhibitors or TGF-β inhibitors to counteract exhaustion and TME effects). The incremental improvements from these strategies offer hope that CAR-T cells will gradually penetrate the solid tumor arena. Notably, even partial but consistent responses in late-stage solid cancer patients are viewed as encouraging signs; for example, in metastatic colorectal cancer, an EGFR-targeted CAR-T cell trial showed stable disease in several patients where conventional options were exhausted, hinting at biological activity. The ultimate goal is to achieve the kind of deep and durable remissions seen in leukemias – likely requiring combination approaches, as discussed in the next section, to fully ignite an immune assault on solid tumors.

4. Integration of Neoantigen Vaccines and CAR-T Cell Therapy

Neoantigen vaccines and chimeric antigen receptor (CAR) T cells each offer distinct but complementary advantages in the immunotherapeutic treatment of solid tumors, creating a rationale for their combined use [1--3]. While CAR-T cell therapy provides a rapid and robust population of effector T cells that can debulk tumors upon infusion, neoantigen vaccines mobilize endogenous T cell responses against multiple tumor-specific mutations, thereby addressing intratumoral heterogeneity and generating sustained immune surveillance. This synergy aligns with the concept of the cancer-immune set point, which posits that the ultimate therapeutic outcome depends on a dynamic interplay of factors that either promote or inhibit tumor immunity [4]. Evidence from personalized cancer vaccines—demonstrated in melanoma, glioma, and other malignancies—has shown that broad CD4^+ and CD8^+ T cell responses can be elicited against patient-specific neoantigens, potentially leading to durable antitumor effects [5, 6]. By providing a diverse repertoire of targeted epitopes, vaccines may help circumvent the emergence of antigen escape variants often observed when a single antigen (e.g., the CAR target) is under intense immunological pressure. In addition, the "helper" T cell components generated by vaccination might support CAR-T cell persistence and function, partially addressing the limitations of autologous CAR-T products that lack robust CD4^+ help. Successful integration of these modalities could also transform otherwise immunologically "cold" tumors—characterized by sparse T cell infiltration—into inflamed, "hot" microenvironments more amenable to immune-cell trafficking and cytotoxicity. The ideal approach would harness vaccines to prime both effector and memory T cell pools, while simultaneously using CAR-T cells to produce immediate cytoreductive impact.

Despite these compelling advantages, significant challenges remain in merging neoantigen vaccines with CAR-T therapy—particularly for solid tumors, where immunosuppressive environments and heterogeneous antigen expression impede therapeutic efficacy. Neoantigens, by definition, are unique tumor-specific peptides that can drive robust T cell responses if accurately identified, and recent advances in epitope prediction algorithms have further streamlined their selection for vaccine design [11–13]. Preclinical experiments indicate that a vaccine-mediated influx of tumor-specific T cells can potentiate CAR-T cell homing and cytotoxic function, sometimes enhanced by strategies such as intratumoral cytokine release (e.g., IL-12) [14]. However, the timing and sequencing of vaccination relative to CAR-T infusion require careful optimization, as excessive immune stimulation could heighten the risk of cytokine release syndrome or neurotoxic events. Logistical factors also pose barriers: manufacturing a patient-specific vaccine in parallel with a personalized CAR-T product is

resource-intensive and demands highly coordinated production timelines. From a regulatory perspective, demonstrating both the safety and individual contribution of each component may necessitate more complex clinical trial designs. Nonetheless, as precision oncology advances and personalized platforms become more accessible, this combination approach could move closer to mainstream clinical practice. By balancing immediate tumor clearance with the sustained, multi-epitope targeting enabled by vaccines, a neoantigen-based vaccine plus CAR-T regimen holds the promise of more durable disease control—and possibly curative outcomes—in patients who would otherwise face limited therapeutic options.

5. Conclusion and Discussion

Personalized neoantigen vaccines and CAR-T cell therapy each represent cutting-edge advances in cancer immunotherapy. As reviewed, neoantigen vaccines can mobilize highly specific T cells against a patient's tumor, achieving immune responses and early signs of clinical benefit in cancers like melanoma and pancreatic cancer. CAR-T cells, while transformative in hematologic cancers, face formidable obstacles in the solid tumor realm. The emerging strategy of combining these two modalities is a logical next step to leverage their complementary advantages. The vaccine can provide breadth and specificity, while the CAR-T provides intensity and immediate tumor debulking. Together, they have the potential to form a potent therapeutic synergy capable of overcoming tumor immune evasion. Preliminary evidence – from melanoma patients responding to vaccines plus checkpoint blockade to early trials like BNT211 showing CAR-T expansion aided by a vaccine – supports the concept that such multimodal immunotherapy can be more effective than either approach alone.

However, many questions and challenges remain. To date, studies of personalized vaccines (with or without adoptive T cell therapy) have been limited to small cohorts or single-arm trials. There is a lack of randomized controlled trials demonstrating a clear survival benefit, and the field will need to progress to larger Phase II and III trials to establish efficacy definitively. Furthermore, most vaccine trials have focused on patients with minimal residual disease (e.g. post-surgery in melanoma or pancreatic cancer). It is still unclear how well vaccines can impact bulky, actively progressing tumors – potentially where the addition of CAR-T cells could be most useful. On the CAR-T side, most combination attempts are in early phases, and it will take time to optimize dosing schedules (e.g. when to vaccinate relative to CAR-T infusion) and to determine the best settings (adjuvant, first-line, refractory disease) for integrated therapy.

Technical and practical hurdles are significant. Personalized vaccine manufacture and autologous CAR-T production are both expensive endeavors; concurrently doing both will strain healthcare resources and patient finances unless innovations drive down costs. Encouragingly, efforts are underway to automate neoantigen selection using artificial intelligence and to streamline mRNA or peptide vaccine synthesis, which could reduce expense and time. Similarly, allogeneic (off-the-shelf) CAR-T cells derived from healthy donors are being developed; if proven safe and effective, they could be produced in advance and made readily available, making it easier to coordinate with a patient-specific vaccine. Policy initiatives and industry partnerships will be important to facilitate access to these complex therapies, ensuring they are not confined to a few academic centers.

Future perspectives also include exploring additional combinations – for instance, adding checkpoint inhibitors to the vaccine+CAR-T regimen to further prevent T cell exhaustion, or using oncolytic viruses to prime an inflammatory tumor microenvironment before delivering CAR-T and vaccines (a strategy some have termed "triple therapy"). Advanced biomaterials and delivery systems might allow in situ vaccination that attracts and supports CAR-T cells directly in the tumor bed. The integration of multi-omics data and machine learning could improve neoantigen prediction and CAR target identification, tailoring the immunotherapy cocktail to each patient with unprecedented precision. Achieving this vision will require close collaboration between genomic scientists, immunologists, engineers, clinicians, and regulatory bodies.

In conclusion, personalized neoantigen vaccines and CAR-T cell therapy are at the forefront of the immunotherapy revolution, and their convergence marks a new frontier in the fight against cancer. By addressing each other's limitations – vaccines boosting CAR-T cells' reach, and CAR-T cells amplifying vaccines' impact – the combination could significantly improve outcomes in solid tumors, which represent the greatest unmet need in oncology. While challenges of complexity and scale remain, the early successes reviewed in this paper offer a glimpse of a future where a patient's immune system, guided by bespoke vaccines and empowered by engineered T cells, can eradicate even the most formidable cancers. Realizing this promise will demand innovative trial designs, investment in biotechnologies, and above all, interdisciplinary collaboration. The path forward is ambitious, but with continued progress, personalized vaccine-CAR-T combination therapy could become a transformative, perhaps curative, approach for patients with cancer.

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