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Synergistic Mechanisms and Clinical Research Progress of CAR-T Combination Therapies in Solid Tumours

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Abstract. Chimeric Antigen Receptor T-cell (CAR-T) Immunotherapy has achieved revolutionary breakthroughs in the treatment of hematological malignancies. However, its application in solid tumours has consistently been hampered by multiple challenges, including poor tumour infiltration capacity, an immunosuppressive tumour microenvironment, and tumour antigenic heterogeneity, resulting in extremely low clinical response rates of CAR-T monotherapy. To overcome this challenge, CAR-T combination therapy has emerged as a key area of research. By leveraging the complementary mechanisms and synergistic effects of different therapies, it is possible to simultaneously address the multiple obstacles encountered in the treatment of solid tumours. This article systematically reviews the classification and synergistic mechanisms of CAR-T combination therapy, summarises the latest clinical developments in this field, compares the efficacy, toxicity and suitable patient populations of different combination strategies, analyses the core issues currently faced in the field, and outlines future directions for development. Existing research indicates that CAR-T combination therapy can effectively overcome the therapeutic bottlenecks in solid tumour treatment, and different combination strategies are applicable to patients with different tumour characteristics.

Keywords: CAR-T combination therapy, solid tumours, synergistic mechanisms, immunotherapy, advances in clinical research

1. Introduction

Chimeric Antigen Receptor T-cell (CAR-T) Immunotherapy, as a leading form of adoptive cell immunotherapy, has achieved revolutionary breakthroughs in the treatment of hematological malignancies, bringing long-term survival hope to patients with advanced hematological malignancies. However, as this therapy has expanded into the field of solid tumours, it has faced multiple unbreakable therapeutic bottlenecks. The clinical response rate of CAR-T monotherapy for solid tumours has remained persistently low, making it difficult to replicate the success achieved in hematological malignancies [1]. Multiple factors, including the unique histopathological features of solid tumours, complex tumour immunosuppressive microenvironment and tumour antigen heterogeneity, jointly restrict the infiltration, activation and in-vivo persistence of CAR-T cells, making it difficult for monotherapy to achieve effective control of solid tumours [2].

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To overcome this challenge, researchers have begun exploring combination strategies involving CAR-T therapy and other treatment modalities, aiming to break through multiple therapeutic bottlenecks in solid tumour treatment via the complementary mechanisms and synergistic effects of different treatment modalities. In recent years, research and clinical exploration into CAR-T combination therapies have experienced explosive growth. Ranging from optimized combination of T-cell subsets, combination with immune checkpoint inhibitors (ICIs) and chemoradiotherapy, to the adjuvant application of novel biological agents, various combination regimens have demonstrated significantly superior efficacy compared to monotherapies in preclinical studies and early-phase clinical trials. These regimens provide new breakthrough opportunities for solid tumour immunotherapy. This paper focuses on the complementary and synergistic effects of different combination strategies, clarifies the clinical advantages of optimised regimens such as triple combinations, and provides a theoretical basis and treatment guidelines for personalised immunotherapy in solid tumours. It promotes the translational application of CAR-T combination therapy from basic research to clinical practice, paving the way for novel, more effective and safer treatment strategies for patients with advanced and refractory solid tumours, and possesses important scientific significance and clinical application value.

2. Classification and synergistic mechanisms of CAR-T combination therapies

2.1. Combined strategies and synergistic mechanisms of CAR-T cell subsets

The function of CAR-T cells is closely linked to the T-cell subsets to which they belong. CD8+ T cells are the primary effector cells, capable of directly killing tumour cells; CD4+ T cells, on the other hand, play a supporting role by promoting the activation, proliferation and infiltration of CD8+ T cells, whilst also secreting cytokines to exert a direct anti-tumour effect. Based on this mechanism, researchers have developed a dual combination strategy of CD4+ and CD8+ CAR-T cells to exert synergistic anti-tumour effects via their functional complementarity.

The most representative of these is the combination therapy involving CD4+ anti-TGF-β CAR-T cells and conventional CD8+ CAR-T cells. TGF-β is a key immunosuppressive factor in the solid tumour microenvironment. It suppresses T-cell function, induces the differentiation of Tregs, and exacerbates the immunosuppressive state of the microenvironment [3, 4]. In contrast, CD4+ anti-TGF-β CAR-T cells can block TGF-β signaling, reverse immune suppression and remodel the tumour immune microenvironment. They can also secrete effector molecules such as IFN-γ to directly kill tumour cells, whilst simultaneously upregulating the expression of activating receptors such as NKG2D, thereby enhancing antitumour activity [5, 6].

When used in combination, the two exhibit significant synergistic effects: CD4+ CAR-T cells improve the immunosuppressive microenvironment, lift the suppression of CD8+ CAR-T cells and promote their infiltration [7]; CD8+ CAR-T cells perform the primary killing function, while alleviating CD8+ CAR-T cell exhaustion and extending their in-vivo survival time. Preclinical studies have verified that this combination regimen exhibits better anti-tumour efficacy than CAR-T monotherapy in solid tumour models including liver cancer and pancreatic cancer, and can even achieve complete tumour regression in partial models [7].

In addition to the combination therapies mentioned above, researchers are also exploring combinations of CAR-T cells with other genetically engineered immune cells, such as CAR-NK and CAR-M cells. CAR-NK cells exhibit strong tumour-infiltrating capacity and are less likely to trigger cytokine release syndrome (CRS). CAR-M cells can eliminate immunosuppressive cells and

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improve the stromal barrier. Such combination regimens can further enhance cytotoxic activity and reduce CAR-T cell exhaustion, and have demonstrated promising potential in preclinical studies [8].

2.2. Combined strategies and synergistic mechanisms of CAR-T therapy with immune checkpoint inhibitors

In the tumour immune microenvironment of solid tumours, persistent activation of inhibitory pathways including PD-1/PD-L1 and CTLA-4 induces CAR-T cell dysfunction, rendering them unable to exert cytotoxic effector functions. Accordingly, combining CAR-T therapy with immune checkpoint inhibitors (ICIs) reverses CAR-T cell exhaustion by blocking these inhibitory axes, emerging as a strategy with great clinical translational potential.

The core synergistic mechanism is that ICIs block inhibitory signals, restoring CAR-T cell activity, whilst simultaneously activating the patient's endogenous immune cells to form a synergistic response [9]. A Phase I clinical trial in malignant pleural mesothelioma demonstrated that CAR-T targeting mesothelin, in combination with the PD-1 inhibitor pembrolizumab, enabled CAR-T cells to persist in patients and maintained a robust IgG response [10]. The persistence of this regimen was significantly superior to that of CAR-T therapy alone [10]. Furthermore, these inhibitors can eliminate inhibitory M2 macrophages, thereby further improving the tumour immune microenvironment [11].

Building on this, researchers have further developed a 'sandwich' therapy comprising 'dual-target CAR-T + PD-1 inhibitor + radiotherapy', which simultaneously addresses multiple issues such as insufficient tumour infiltration, immunosuppression and antigenic heterogeneity. The core dual-targeting CD133/PD-L1 CAR-T cells can convert PD-L1 inhibitory signals into activating signals, thereby eliminating relevant tumour subclones. They can also exert potent cytotoxic effects through bystander activation [12].

Combining PD-1 inhibitors can further block residual inhibitory signals, while radiotherapy can shrink tumours, reduce the risk of CRS, induce immunogenic cell death, and promote the differentiation of CAR-T cells into tissue-resident memory T cells (Trm), thereby enabling long-term immune surveillance and preventing recurrence [12, 13]. This 'sandwich' approach provides a new paradigm for the combined treatment of solid tumours.

2.3. Combined strategies and synergistic mechanisms of CAR-T therapy with radiotherapy and chemotherapy

As conventional anti-cancer modalities, radiotherapy and chemotherapy can directly kill tumour cells and remodel the tumour immune microenvironment, thereby establishing a favourable niche for CAR-T cell function.` When combined with CAR-T therapy, these two approaches complement each other's strengths.

When CAR-T therapy is combined with radiotherapy, low-dose radiotherapy can enhance efficacy through multiple mechanisms. First, disrupt the physical tumour barrier and degrade the extracellular matrix. Second, normalise blood vessels and promote CAR-T cell infiltration. Third, induce immunogenic cell death in tumour cells and enhance the endogenous immune response [14]; Besides, modulate the composition of immune cells to improve the microenvironment [15]. More importantly, radiotherapy can induce CAR-T cells to differentiate into tissue-resident memory T cells, achieving long-term immune memory via the 'radiotherapy–E-cadherin–CD103–CAR-Trm' axis, thereby preventing tumour recurrence [12]. It should be noted that the dose and timing of radiotherapy have a critical influence. Low-dose radiotherapy (2–4 Gy) or a fractionated regimen of

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8 Gy × 3 can exert favourable immunomodulatory effects without impairing CAR-T cell viability and function. This makes them the preferred choice for combination therapy [16].

The synergism between CAR-T therapy and chemotherapy mainly depends on chemotherapy-induced lymphodepletion and tumour microenvironment remodelling. Cyclophosphamide and fludarabine are commonly used in clinical practice as pre-treatment prior to CAR-T infusion to eliminate immunosuppressive cells such as Tregs and MDSCs from the patient's body, thereby reducing competition from endogenous lymphocytes, promoting CAR-T cell proliferation, and enhancing therapeutic efficacy [17, 18]. Furthermore, chemotherapeutic agents can upregulate the expression of tumour target antigens and stress-related molecules. For example, carboplatin can induce tumour cells to express DAMPs, thereby enhancing their sensitivity to CAR-T cells [19]. However, the chemotherapy dose must also be optimised, with low-dose pre-treatment being the optimal choice, as this avoids toxicities such as myelosuppression and damage to CAR-T cells.

In addition to the mainstream combination strategies mentioned above, researchers have also explored the combination of CAR-T therapy with other novel therapies, thereby further expanding the scope of combination therapy. For instance, combining CAR-T with anti-angiogenic agents such as VEGF/VEGFR neutralizing antibodies can remodel tumour vascular architecture and facilitate CAR-T cell tumour infiltration [20]. In neuroblastoma models, GD2-targeted CAR-T combined with bevacizumab can enhance CAR-T cell tumour infiltration and amplify anti-tumour cytotoxicity [21]. Furthermore, the combination of CAR-T therapy and tumour vaccines has also demonstrated significant potential. For instance, the combination of CLDN6-targeting CAR-T cells with mRNA vaccines can significantly promote T-cell proliferation and has shown promising results in clinical trials [22].

3. A clinical comparison of CAR-T combination therapies in solid tumours

In recent years, clinical research into CAR-T combination therapies has entered a phase of rapid development, yielding a series of encouraging research findings.

Different combination regimens target different obstacles, and there are significant differences in their efficacy, toxicity and suitable patient populations. To provide a clearer comparison of the characteristics of different regimens, we have summarised the current mainstream combination strategies, as shown in the table below:

Table 1. The current mainstream combination strategies

Combined Strategy Type	Objective Response Rate (ORR)	Median Progression-Free Survival (mPFS)	Major Toxicological Effects	Types of Cancer / Patient Groups
Combination of CD4+/CD8+ Cell Subsets	►The cyclophosphamide + fludarabine (Cy/Flu) group -- the ORR reached 82% (with a complete remission rate [CRR] of 64%). ►The cyclophosphamide-only group --the ORR was 50%.	►The Cy/Flu group -- mPFS has not yet been reached. ►The cyclophosphamide-only group -- mPFS was 1.5 months. (Subgroup analysis showed that patients with a CD4/CD8 ratio < 1.12 in the infusion product had a 6-month PFS rate of 76%, which was significantly higher than the 31% observed in the high-ratio group.)	►Manageable immune-related toxicity: A. the incidence of sCRS was 12.5%. B. the incidence of Grade ≥3 neurotoxicity was 28%. ►the high-dose group (2×10^7 cells/kg) – a risk of toxicity; the standard dose (2×10^6 cells/kg) – good safety	Relapsed/refractory B-cell non-Hodgkin's lymphoma (NHL) and B-cell acute lymphoblastic leukaemia (B-ALL). Currently used primarily for CD19-positive hematological malignancies.

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Table 1. (continued)

CAR-T + ICIs	▶Taking the ZUMA-6 trial as an example, the ORR was 75% (with a CRR of 54%).	▶The mPFS was 9.0 months, and the 48-month PFS rate was 31.5%.	All toxicities were manageable and reversible: the incidence of Grade ≥3 CRS was 9%, and that of Grade ≥3 neurotoxicity was 32%.	Relapsed/refractory diffuse large B-cell lymphoma (DLBCL).
The Triple-combination Regimen of Radiotherapy + CAR-T + ICIs	▶Achieve an ORR of 80–85% (with a CRR of approximately 70%), which was significantly superior to monotherapy or dual-agent combination therapy.	▶A mPFS of approximately 12–15 months, with a 2-year PFS rate of up to 57%	Toxicity is manageable: CAR-T-associated CRS (incidence approximately 10%) and neurotoxicity, local tissue reactions.	Relapsed/refractory CD30-positive lymphoma and certain solid tumours (such as liver cancer and glioblastoma).
OMV + CAR-T	▶The complete tumour regression rate for the combination therapy reached 100%, ▶The regression rate for CAR-T therapy alone was only 20%.	▶The mPFS in the combination therapy group has not yet been reached (at 60 days of follow-up, none of the mice had experienced tumour recurrence). ▶The mPFS in the CAR-T monotherapy group was only 20 days.	The preclinical models demonstrated good safety; no significant systemic toxicity was observed.	Solid tumours, particularly those with strong immunosuppression and high antigenic heterogeneity, such as triple-negative breast cancer.

As shown in Table 1, the triple combination regimen of radiotherapy plus CAR-T plus ICIs yields the highest ORR and longest PFS, yet it is mainly applicable to patients with large-volume tumour burdens. In contrast, the cell-subset combination regimens are more suitable for patients with solid tumours exhibiting high levels of TGF-β-mediated immune suppression, as well as those with CD19-positive hematological malignancies. The immune checkpoint inhibitor combination regimen demonstrates superior efficacy in PD-L1-positive tumours. By contrast, the OMV combination regimen shows unique superiority in tumours with high antigen heterogeneity. These findings indicate that clinically, the optimal combination regimen should be selected according to individual tumour characteristics to realise personalised precision therapy [23-29].

4. Current issues and future prospects

4.1. Current issues

Although CAR-T combination therapies have achieved remarkable progress in solid tumour treatment, multiple unresolved challenges still restrict their clinical translation and broad clinical application.

Firstly, the research on the synergistic mechanisms of some combination regimens remains insufficiently thorough. At present, most combination regimens are based on preclinical observations, and their specific molecular mechanisms have not yet been fully elucidated. Such insufficient mechanistic understanding also leads to the absence of a clear theoretical basis for regimen optimisation.

Secondly, the issue of cumulative toxicity remains a significant concern. As each treatment modality carries its own toxicity profile, their combined use may result in cumulative toxicity. For example, the combination of CRS induced by CAR-T therapy and the immune-related toxicity of ICIs may increase the risk of serious adverse events. Striking a balance between efficacy and safety remains a key challenge to be addressed in clinical practice.

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Thirdly, there is a lack of uniform standards regarding the timing and dosage of administration. Currently, across different studies, there are no standardised guidelines for radiotherapy doses, the timing of CAR-T infusions, or the administration schedule for immune checkpoint inhibitors. There are significant variations in the treatment regimens used by different centres, which results in poor comparability of study results and makes it difficult to establish standardised treatment regimens.

Fourthly, combination therapies are costly to produce, making it difficult to promote on a large scale. CAR-T therapy is already expensive to produce, and combination therapies require the simultaneous use of multiple treatment modalities, which further increases the cost of treatment. This makes the treatment unaffordable for the ordinary patient, thereby limiting its widespread clinical adoption.

4.2. Future optimisations

In light of the above issues, future research needs to focus on optimisation in several areas:

Firstly, future research should delve deeper into the synergistic molecular mechanisms of combination therapies. By applying advanced technologies including single-cell sequencing and spatial transcriptomics, researchers can analyse dynamic alterations in the tumour immune microenvironment after combined treatment and clarify the molecular basis of therapeutic synergy. This will lay a solid theoretical foundation for subsequent regimen optimisation.

Secondly, future research should focus on optimising the timing and dosage of administration. Based on preclinical models and early clinical trial data, researchers can explore the optimal dosage and administration timing for various combination regimens and formulate precise administration strategies. This will minimise the risk of cumulative toxicity whilst ensuring therapeutic efficacy.

Thirdly, there is a need to conduct more multicentre, large-scale clinical trials in the future and to gather comprehensive long-term data on safety and efficacy, thereby providing sufficient evidence to support the approval and clinical application of combination therapies.

Fourthly, low-cost, high-efficacy combination strategies should be developed in the future, such as the development of universal CAR-T therapies. This would reduce the cost of cell products whilst optimising combination regimens, minimising unnecessary treatments, lowering overall treatment costs, and facilitating large-scale clinical application.

Finally, future research should combine AI technology with patients' individual characteristics to develop personalised combination regimens. AI-based analysis of patient tumour genomes and microenvironmental features enables individualised matching of optimal combination strategies, thus achieving precise personalised therapy.

5. Conclusion

The clinical application of CAR-T therapy in solid tumours has long been a core challenge in cancer immunotherapy, and the rise of combination strategies has offered a critical approach to breaking through this therapeutic bottleneck. At present, CAR-T combination therapies have developed into multiple distinct strategic modalities, covering cell subset optimized combination, as well as combined regimens with immune checkpoint inhibitors, radiotherapy and chemotherapy. Various regimens have demonstrated promising potential in preclinical and early-phase clinical studies. Among them, innovative strategies such as the triple regimen of CAR-T, radiotherapy and ICIs exhibit particularly prominent efficacy, providing novel therapeutic options for patients with various solid tumours.

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However, the field currently faces challenges such as unclear synergistic mechanisms, toxicities, and a lack of standardised protocols, all of which require further research and optimisation. In the future, with a deeper understanding of synergistic mechanisms and the development of personalised, precision treatment strategies, CAR-T combination therapy is expected to further break through the treatment bottlenecks in solid tumours, offering hope for long-term survival to more patients with advanced solid tumours, and becoming a core approach in the immunotherapy of solid tumours.

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