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Exploring the Synergy of Immunotherapy and Conventional Treatments in Cancer Therapy

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Abstract:

Immunotherapy (cancer immunotherapy) is a promising approach to cancer treatment that recognizes and destroys cancer cells by employing immune-related components or by directing the immune system. Between 2017 and 2020, the R&D pipeline for cancer immunotherapies increased by 233%. Immunotherapy can be broadly divided into five broad categories and is suitable for 20 different types of cancer. With the introduction of immune checkpoint inhibitors and CAR-T cell therapies, this approach has revolutionized the way of cancer treatment, even treating some patients with advanced cancers, while not all cancer types respond well to this approach. The effectiveness of immunotherapy as a stand-alone treatment is often constrained by tumor heterogeneity, immune evasion, and resistance mechanisms. To address these issues, immunotherapy is being investigated in combination with other traditional treatment modalities such as radiotherapy, chemotherapy and targeted drugs as a way to improve treatment outcomes. This review explores the rationale behind these combination therapies and discusses the potential of these combined therapies to improve patient survival and quality of life.

Keywords: Cancer; immunotherapy; combination therapy.

1. Introduction

In the past few years, terrific progression has been made in the treatment of cancer cells. A cutting-edge innovation that has completely changed the field of therapy is immunotherapy Compared with typical therapies that straight target cancer cells, immunotherapy uses the body's immune system to identify and destroy cancer cells. Using the principle of cancer immunology - a rapidly broadening field in oncology - this strategy aims to enhance the inherent capacity of the immune system to fight cancer. The working approach of cancer immunotherapy is to use chemicals specific to tumour antigens, which are shared on the surface of cancer cells and discovered by immune elements such as T cell receptors or antibodies. Normally, these tumor antigens are differentiable proteins or other macromolecules from cancer cells, such as carbohydrates. In immunotherapy, these tumor antigens are bound by modified antibodies, which effectively mark the cancer cells and trigger the immune system to kill them. Although this is an innovative technique, the efficacy of cancer immunotherapy differs greatly due to the kind of cancer. In addition, certain subtypes of stomach cancer react favorably to immunotherapy, but not all subtypes do. Immunologists James P. Allison and Tasuku Honjo won the 2018 Nobel Prize in Physiology or Medicine for their work inhibiting negative immune regulation as a means of treating cancer, highlighting the significance of immunotherapy in the fight against cancer. But using the immune system against cancer is not a novel idea. Historical sources place the development of cancer immunotherapy in the 17th and 18th centuries, when it was first applied as septic dressings and purposeful infections to cure cancer. In 1891, the renowned pioneer in this field, American surgeon William Coley, started inoculating patients with incurable tumors with germs like Streptococcus pyogenes. Modern immunotherapy was made possible by Coley's discovery that some bacterial infections may cause tumor regression. Since immunotherapy is a stand-alone treatment that only works for specific cancers and individuals, research has begun to focus on integrating immunotherapy with other treatment strategies to boost efficacy and widen its use. Though they are often associated with major side effects and drug resistance, among other problems, traditional cancer treatments such as radiation and chemotherapy have long been regarded as the gold standard. Scientists seek to leverage the unique benefits of each approach to optimize cancer treatment effectiveness by combining these tactics with immunotherapy. Examples of targeted medications include chemotherapy and radiation, which

kill immune cells and make cancer cells more vulnerable to immunological attack, these medications also interfere with specific biochemical processes that tumors use to evade the immune system.

This review explores the possible advantages of integrating immunotherapy with various other therapies, focusing on how these mixes conquer the restrictions of a single treatment. The objective is to evaluate the existing evidence supporting these extensive techniques, evaluate the system behind their synergy, and review their effect on future cancer therapy. The goal of this evaluation is to sustain further initiatives to establish much more reliable and customized therapy prepare for cancer cells people.

2. Combination of Chemotherapy and Immunotherapy

For a long time, chemotherapy has been the pillar of cancer cells treatment because it has the result of getting rid of quickly dividing cancer cells. Cytotoxic medications are utilized in chemotherapy to target and eliminate swiftly dividing cancer cells. These medications include topoisomerase preventions, alkylated compounds and antimetabolites, which contribute by interfering with important elements of the cell cycle. Alkylation substances, metabolites, topological isomerase inhibitors and antimicrobial drugs are some of their settings of action [1]. Chemotherapy is generally made use of to control or eliminate tumors by preventing the proliferation of tumour cells. Standard chemotherapy medicines interfere with the development and division of tumour cells via numerous systems, consisting of interference with DNA, RNA or healthy protein synthesis. In contrast, chemotherapeutic agents act on tumor cells by interfering with DNA replication and repair, RNA synthesis, and microtubule formation. Also, chemotherapy affects rapidly dividing cells, including those in the immune system, including myelosuppression, immunosuppression, lymphopenia, and other long-term effects. However, Chemoimmunotherapy is an emerging cancer treatment strategy that combines the cytotoxic effects of chemotherapy with the immune-boosting ability of immunotherapy to improve outcomes by targeting cancer cells with chemotherapy and boosting the immune system's ability to fight cancer with immunotherapy. More recent studies reveal a more complicated function, showing that in some circumstances, chemotherapy can greatly enhance antitumor immunity, especially when combined with immune checkpoint inhibitors (ICIs). This combined effect is the product of several interconnected mechanisms that improve ICI effectiveness and increase therapeutic potential against various cancer types.

Chemotherapy induces immunogenic cell death (ICD), which is one of the primary ways it increases antitumor immunity. Contrary to the non-immune cell death system, immune checkpoint disease (ICD) activates a series of immune actions and boosts anti-tumour resistance. Chemotherapy representatives, consisting of glycanoids, oxaliplatin and anthracycline, can generate ICD by putting pressure on tumor cells and triggering them to launch molecules referred to as risk-related molecular patterns (DAMP) [2]. High fluidness box 1 (HMGB1), calcin and adenosine triphosphate (ATP) are examples of DAMP, which as a signal to advise the existence of dead tumour cells in the immune system. Antigen-presenting cells (APCs), especially dendrital cells (DC), have pattern recognition receptors in charge of detecting them. The interaction between DAMPs and APCs promotes the absorption and treatment of tumour antigens, causing the activation and induction of cytotoxic T lymphocytes (CTL) in tumour cells. This sort of "chilly" tumor that infiltrates the least immune cells is transformed right into an "warm" tumour with a rise in immune cells, making it extra receptive to ICI. It also targets and eliminates immunosuppressive cells, such as bone marrow-derived inhibitory cells (MDSC) and regulative T cells (Tregs), which have an influence on the tumor microenvironment (TME). As we all recognize, effective anti-tumour responses are impeded by tregs and MDSCs with various mechanisms, such as the production of immunosuppressive cytokines and the motivation of immune resistance. Chemotherapy drugs particularly target these cells, thus lowering their amount and feature in TME, specifically when used at reduced or defeat dosages. For instance, modest doses of cyclophosphamide have been revealed to help in reducing Treg matter, which subsequently improves the general anti-tumour immune response. Likewise, medications such as amoxycillin and gicitabine can decrease the variety of MDSC and remove the main obstacles to the anti-tumour immune reaction.

In addition, chemotherapy has the capability to raise the task of all-natural killer (NK) cells, which is essential for the natural immune action against deadly tumors. Tumour cells that have experienced chemotherapy-induced DNA damages and stress reactions might show overexpression of NK cell receptor ligands, such as NKG2D, which boosts the sensitivity of tumour cells to NK cell-mediated fatality. Chemotherapy can also boost NK cell activity and reinforce the anti-tumour immunological atmosphere by increasing the manufacturing of cytokines such as interferon-γ (IFN-γ). With the decrease of MDSCs and Tregs, the boost in NK cell task boosts the efficiency of the immune action to the entire tumour.

The mix of ICI and chemotherapy needs to be utilized to supply better treatment outcomes. The functioning princi-

ple of ICI is to block repressive signals that restrict the activation and function of T cells, such as signals mediated by PD-1 and CTLA-4. Along with the straight effects of chemotherapy on tumors and immune cells, chemotherapy and ICIs jointly get rid of immune checkpoints that restrict the function of T cells to enhance the anti-tumour immune response. This combination is especially reliable in the treatment of initially drug-alone (ICI) immune tumors, consisting of specific kinds of triple-negative bust cancer cells (TNBC) and non-small cell lung cancer cells (NSCLC) [3]. Scientific trials prove that integrating chemotherapy with ICI can substantially boost the survival price and response rate of individuals. For example, when incorporated with PD-1 restraint, chemotherapy for non-small cell lung cancer dramatically enhances the total survival rate and progression-free survival price compared to chemotherapy used alone. Likewise, the combination of chemotherapy and PD-L1 preventions has actually proven an improvement in the success price of treatment in TNBC. The success of these mixes shows that chemotherapy can boost the immune atmosphere and minimize the tumor concern, both of which boost the effectiveness of ICI [4].

Nevertheless, some variables, such as the selection of chemotherapy drugs, just how much to take and when to take them belong to immune checkpoint inhibitors, will certainly affect the impact of the chemotherapy-immunotherapy combination. The best strategy must endanger in between security and effectiveness to reduce adverse effects and optimise treatment benefits. The objective of the present research is to identify the best timetable, series and combination that is most valuable to individuals [5].

3. Combination of Radiotherapy and Immunotherapy

By using subatomic particles or targeted X-rays, radiation treatment harms the DNA of cancer cells through ionizing radiation. This radiation can be applied internally by brachytherapy, in which radioactive sources are positioned within or next to the tumor, or externally through the use of a device similar to a linear accelerator, which concentrates high-energy photons or electrons on the tumor. The intention is to cause deadly damage to DNA in cancer cells, which are more vulnerable to radiation than normal cells because they have compromised repair systems. Usually, this damage causes a mitotic catastrophe, which is the interruption of cell division that culminates in cell death. While providing cumulative harm to cancer cells, fractionating the radiation dose—delivering it in several tiny doses—allows normal cells to heal in between treatments, thereby enhancing the therapeutic effect while minimizing damage to surrounding healthy tissues [6]. However, Acute, consequential, and late impacts are the several types of side effects that might result after radiation therapy. Within weeks after starting therapy, acute effects often show up as erythema, mucositis, and diarrhea in quickly reproducing tissues such the skin, mucosa, and gastrointestinal tract. If left unchecked, they can develop into long-lasting side effects that come beyond the first course of therapy. The sluggish division of tissues, such as the heart, lungs, and gastrointestinal organs, can have late consequences that manifest months or years later. These might result in problems such fibrosis, necrosis, and vascular damage. Skin irritation, xerostomia, neurocognitive deficits, radiation pneumonitis, heart dysfunction, gastrointestinal problems, and reproductive system harm are among the specific adverse effects [7].

When RT is utilized with other immunotherapy, it offers an natural strategy to get over drug resistance, boost the immune system targeting cancer cells, and make use of the complementary process of these therapies to enhance the diagnosis of patients. The primary feature of radiotherapy (RT) is to directly damage the DNA of cancer cells, bring about cell death and the development of an immunogenic setting. Tumour antigens and damage-related molecular patterns (DAMP) were launched, setting off the procedure. Then, antigen existing cells (APC) procedure these antigens to turn on T lymphocytes. RT is a vital mechanism that upregulates the capacity of the main histocompatibility complicated Class I (MHC-I) particles of tumor cells to make them extra apparent to cytotoxic T lymphocytes [8]. In addition, RT advertises the phagocytosis of APC on tumor cells by causing calkin to be displaced to the cell surface area and downregulate CD47, which can typically prevent phagocysis. The activation of T cells and the onsum of a wider immune response to tumours rely on this device [9]. On top of that, brand-new antigens produced by RT-induced DNA damages improve the immune reaction by increasing the worry of mutations and supplying new targets for immune acknowledgment. In order to make complete use of this immunogenic change, RT must be utilized with immunotherapy, especially immune checkpoint inhibitors (ICI), which can interfere with the tumor avoidance system to set off an immune reaction. Immunotherapy inhibitors (ICI) targeting CTLA-4 or PD-1/ PD-L1 will certainly damage signals that protect against T cell activation and boost the ability of the immune system to identify and destroy cancer cells. When made use of with RT, these inhibitors benefit from radiation-induced tumor microenvironmental modification, consisting of enhanced antigen presentation and raised antigen release. In addition, via the launch of cytoplasmic DNA through damaged tumor cells, RT stimulates the STING (interfer-

on genetics stimulant) path, which subsequently triggers the type I interferon reaction and advertises the systemic anti-tumour immune feedback, which is boosted by ICI therapy.

In addition to immune checkpoint restraint, the mix of RT and immunotherapy gives additional capacity. For instance, RT can increase the expression of MHC particles and tumour-related antigens (TAA) on tumour cells. Tumour vaccines are made to cause a solid immune reaction to particular cancer cells antigens [10]. Higher antigen exposure makes vaccination much more efficient by enhancing its capability to trigger anti-tumour immune response. Preclinical researches reveal that when RT is incorporated with peptide or DC-based inoculation, it can significantly reduce the tumor growth of mouse designs and enhance survival. Scientific trials show that incorporating RT with injections such as Sipuleucel-T can boost the survival rate of prostate cancer cells patients. In a similar way, the potential of RT to alter the tumour microenvironment is beneficial to adoption cell therapy, such as chimeric antigen receptor (CAR) T cell treatment. Through the destruction of tumor vasculature and the reduction of immunosuppressive factors, radiation treatment (RT) improves the effectiveness of CAR-T cell therapy by increasing the infiltration of CAR-T cells into tumors. Preclinical research has shown that the anti-tumor efficaciousness of RT can be enhanced when combined with CAR-T cells. Furthermore, combining RT with cytokine treatments like GM-CSF or interleukin-2 (IL-2) may improve the immune response. RT increases these cytokines' capacity to activate immune cells and promotes the effectiveness of treatment [11].

4. Combination of Targeted Therapy and Immunotherapy

Unlike typical chemotherapy, which affects all rapidly proliferating cells, targeted therapy precisely targets genetic abnormalities and aberrant proteins that fuel tumor development. Mechanistically, depending on the precise molecular targets involved, targeted treatments may inhibit cell surface receptors, impede signal transduction pathways, or cause cell death. For example, monoclonal antibodies can be coupled to cytotoxic medications or radionuclides for direct targeting of tumor cells. These antibodies frequently target certain proteins on the surface of cancer cells or in the tumor environment, such as HER2 in breast cancer. Conversely, small-molecule inhibitors enter cells to block intracellular targets such as kinases that are important in the growth of cancer cells. Targeted medicines can still have serious adverse effects even with their accuracy. Common side effects include dry skin and acneiform rashes, especially when using EGFR inhibitors. Severe side effects include congestive heart failure when using medications that target HER2, hypertension when using VEGF inhibitors, and bleeding concerns when using EGFR inhibitors. It is frequently necessary to address the adverse effects with supportive care, modify the dosage, or stop the medication altogether in order to manage them, which emphasizes the importance of close observation [12].

However, targeted treatment with immunotherapy combines the best features of both modalities to increase overall anti-tumor effectiveness. Targeted treatments, such as monoclonal antibodies and small-molecule inhibitors, target certain molecular abnormalities, such as genetic mutations or overexpressed proteins, within cancer cells. These treatments work by obstructing pathways vital to the development and survival of cancer cells, therefore they can directly prevent tumor growth. For example, MEK inhibitors such as trametinib disrupt downstream signaling in the MAPK pathway, whereas BRAF inhibitors such as vemurafenib target the BRAF V600E mutation in melanoma [13]. Targeted therapy can lessen tumor burden and perhaps change the tumor microenvironment (TME) to make it more vulnerable to immune system attack by focusing on these particular oncogenic drivers.

Immunotherapy, which includes cancer vaccines and immune checkpoint inhibitors (ICIs), functions by boosting the immune system's defenses against cancerous cells. Immunoglobulin-producing antibodies (ICIs) like anti-PD-1/PD-L1 and anti-CTLA-4 block the proteins that prevent T cells from activating and functioning, which improves the immune system's capacity to identify and eliminate tumor cells. The shortcomings of each strategy when applied alone can be addressed by combining these with tailored medicines. BRAF inhibitors, for instance, not only shrink tumors but also increase the expression of melanoma differentiation antigens, which might increase the efficacy of ICIs by increasing T cell recognition of tumor cells [14]. In similar blood vessels, MEK inhibitors can change TME and advertise T cell seepage in the tumor to advertise T cell activity [15]. Additionally, the system of drug resistance can be solved by the mix of immunotherapy and targeted drugs. For example, compared to making use of BRAF inhibitors alone, the mix of BRAF and MEK inhibitors in melanoma has actually been verified to enhance clinical outcomes and minimise drug resistance. This mix can prevent or postpone the growth of drug resistance mechanisms that decrease the efficiency of single-dose targeted treatment [16]. For certain carcinogenic pathways, it can also change the immune setting of the tumor, thus decreasing the variety of immunosuppressive cells and improving the effectiveness of immunotherapy.

Medical trials have actually shown the synergy between

targeted treatment and immunotherapy, which supplies proof of the effectiveness of these combinations. The mix of MEK inhibitor Kobemetinib and BRAF prevention Vimulafenib showed a far better impact in metastatic melanoma than BRAF alone. In addition, it has actually been proved that combining these targeted treatments with anti-PD-1 medicines can boost the diagnosis of individuals and boost the anti-tumour feedback. On the other hand, the trial of combining EGFR inhibitors with ICI reveals prospects in the therapy of non-small cell lung cancer cells (NSCLC), with greater survival prices and better illness control [17].

Targeted therapy targets details molecular abnormalities in cancer cells to ruin the essential way required for tumor development and survival. Examples of these treatments consist of BRAF inhibitors (Vimulafenni and Dabrafeni), MEK inhibitors (trimetinib), CDK4/6 inhibitors (Parbosilib and Abetilib) and PARP inhibitors (Nirapalib). In addition to decreasing the tumour problem, these drugs additionally have an effect on the tumor microenvironment (TME), which may boost the effectiveness of succeeding immunotherapy.

BRAF V600E mutation, which is frequently seen in melanoma, is the target of BRAF inhibitors, namely vemurafenib and dabrafenib. It has been demonstrated that these inhibitors promote the production of melanoma differentiation antigens (MDAs) and improve immune cell penetration into the tumor, especially CD8+ T lymphocytes. The tumor's enhanced antigen expression aids in the immune system's recognition of it. The anti-tumor effects are enhanced when paired with MEK inhibitors such as trametinib, which obstruct downstream signaling in the MAPK pathway. This combination not only increases responsiveness to immune checkpoint blockage (ICB) using PD-1 inhibitors, but also improves anti-tumor activity. Clinical studies have shown that the triplet combination of trametinib, dabrafenib, and pembrolizumab, a PD-1 inhibitor, improves overall response rates and progression-free survival when compared to BRAF and MEK inhibitors alone [18].

Immunostimulatory effects of CDK4/6 inhibitors, such palbociclib and abemaciclib, are mediated through many pathways. These inhibitors improve antigen presentation and CD8+ T-cell activity by downregulating the DNA methyltransferase DNMT1, which increases the production of endogenous retrovirus (ERV) elements and type III interferon (IFN). Furthermore, CDK4/6 inhibitors spare other T cell populations while specifically reducing CD4+ CD25+ regulatory T cells (TRegs). The immune landscape is changing to become more anti-tumor, which increases PD-1 blockade's efficacy. Preclinical models of colorectal cancer, such as MC38 and CT-26 models, have demonstrated encouraging outcomes when combined with PD-1 inhibition and CDK4/6 inhibitors. Combining abemaciclib with pembrolizumab has shown early promise in treating ER-positive/HER2-negative metastatic breast cancer, according to well-tolerated early-phase clinical studies [19].

PARP inhibitors, like niraparib, mainly cause cytotoxicity in cancers that are unable to repair homologous recombination by building up DNA damage. In addition to their direct lethal effects, PARP inhibitors cause a type I interferon response that is dependent on STING. This response improves anti-tumor immunity by decreasing myeloid-derived suppressor cells (MDSCs), enhancing the infiltration of effector CD4+ and CD8+ T cells, and improving antigen presentation. On the other hand, PARP inhibitors have the ability to increase PD-L1 expression in tumor cells, which may result in immune escape. Combining PARP inhibition with PD-1/PD-L1 blocking, which has been demonstrated to result in more robust tumor suppression and extended survival, can help to lessen this difficulty. Notably, the combination of niraparib with pembrolizumab has shown enhanced effectiveness in patients with platinum-resistant ovarian cancer in the current TOPACIO/KEYNOTE-162 study, accomplishing an aim to response rate of 19%, compared to lower ORRs with single-agent therapies [20].

5. Emerging Strategies in Immunotherapy Combinations

Exploring combinations of several immunotherapy methods is one of the most intriguing areas of study in cancer treatment. By focusing on several immune system components, these strategies seek to strengthen the anti-tumor immune response in concert with one another.

For instance, adoptive cell therapy like CAR-T cells in conjunction with immune checkpoint inhibitors is being studied for both hematologic and solid cancers. Genetically customized T cells or CAR-T cells aim to precisely target tumour antigens and hence damage cancer cells. Nonetheless, the visibility of immunosuppressive tumour microenvironment and immune checkpoint molecules (such as PD-L1) hinders the efficiency of CAR-T cells in strong tumors. The mix of immune checkpoint inhibitors and CAR-T cell therapy can enhance the longevity and function of CAR-T cells in the tumor microenvironment, which can result in better medical outcomes.

One more example is to enhance the restorative impact. The new technique of cancer immunotherapy for bone marrow cells concentrates on reprogramming and modifying these cells in the tumour microenvironment (TME). These approaches consist of transforming the composition of bone marrow cells, preventing immunosuppressive tasks, reprogramming bone marrow cells in various

means, making use of cytokines, utilizing bone marrow cell therapy, and concentrating on brand-new indicators. Changing the composition of bone marrow cells in the tumour microenvironment (TME) is a key method to minimize the variety of tumor cells and boost the anti-tumour action [21].

The employment of bone marrow cells, such as bone marrow-derived inhibitory cells (MDSCs) and tumour-related macrophages (TAMs), depends on the CCL2-CCR2 axis. In metastatic anti-scastration prostate cancer, professional tests of human monoclonal antibody Kaluman (a CCL2 prevention) reveal that the success of monotherapy is restricted, and the objective action rate (ORR) is 0%; however, when made use of in mix with standard chemotherapy, its ORR is 37.5%, and when the response duration of response (DOR) is 6.3 months.

When used in conjunction with chemotherapy for pancreatic cancer, the oral CCR2 inhibitor PF-04136309 has shown response rates ranging from 23.8% to 48.5%; however, in 24% of instances, it was linked to pulmonary damage [22]. There has also been investigation into the blocking of CSF-1R, a crucial TAM regulator. AMG 820 and pexidartinib, for example, have demonstrated promise as inhibitors, especially when it comes to treating tenosynovial giant cell tumors (TGCTs), with pexidartinib demonstrating a 38% ORR in a Phase III study . CSF-1R inhibitors, however, have not always been successful in treating different forms of cancer; in fact, several trials have found that their use alone only partially reduces objective responses [23]. Reprogramming myeloid cells to improve their anti-tumor capabilities entails polarization shifts. When used in conjunction with stereotactic body radiation treatment, FLT3 ligands like CDX-301 have been shown to increase dendritic cell maturation and promote therapeutic responses, with a 31% partial response rate .

Reparixin, a CXCR1/2 inhibitor, demonstrated an ORR of 30% when combined with paclitaxel in metastatic breast cancer; however, subsequent Phase II trials did not show improved progression-free survival compared to paclitaxel alone [24]. Targeting the CXCR1/2 axis, which is involved in myeloid cell recruitment and angiogenesis, has produced mixed results. Targeting STAT3 is one of the novel cytokine-based strategies that aims to interfere with its function in immune suppression and tumor progression. In order to prevent the tumor-promoting effects of active STAT3, which is implicated in immune evasion and the development of myeloid cell tolerance, STAT3 inhibitors such as napabucasin and TTI-101 are being studied [25]. Beside these strategies, to further control myeloid cell connections and functions inside the TME, new targets including Siglec-15, TREM2, MARCO, LILRB2, and CLEVER-1 are being researched.

6. Conclusion

The chemoimmunotherapy combination exploits the ability of chemotherapy to induce immunogenic cell death (ICD) to enhance anti-tumor immunity through the release of danger-associated molecular patterns (DAMPs) and subsequent activation of antigen-presenting cells (APCs). But the broad effects of chemotherapy can also damage healthy cells that divide quickly, which can result in major side effects including immunosuppression and myelosuppression. Similarly, the combination of radiotherapy (RT) with immunotherapy has shown promising results because of its ability to induce DNA damage and generate an immunogenic environment conducive to antitumor responses. Targeted therapies, which use precise methods to disrupt specific molecular pathways of tumor growth, have also shown good potential when used in combination with immunotherapy. In conclusion, as the field of cancer therapy develops further, combining standard therapies with innovative techniques has become known as a major development in raising patient prognoses and efficacy. Chemotherapy has been the preferred option for treating cancer for a long time. Ongoing research and clinical trials are essential to refine the combination of these therapies and discover new strategies to fight cancer.

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