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Research Progress on Nanomedicine Delivery Systems in Tumor Immunotherapy

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Abstract. Tumor immunotherapy has altered the therapeutic landscape of melanoma, lung cancer, renal cancer, hematologic malignancies, and several mismatch repair deficient tumors, yet durable responses remain uneven across tumor types and patient populations. Major restrictions arise from inefficient antigen presentation, inadequate trafficking and persistence of effector lymphocytes, stromal exclusion, suppressive myeloid compartments, hypoxia, aberrant vasculature, and systemic immune toxicity after nonselective immune activation. Nanomedicine delivery systems provide a materials-based route to adjust where, when, and in which cellular compartment immunomodulators are released. Lipid nanoparticles, polymeric particles, protein and lipoprotein mimetics, inorganic nanomaterials, biomimetic vesicles, and responsive hybrid platforms can co-deliver antigens, adjuvants, checkpoint inhibitors, cytokine modulators, nucleic acids, photosensitizers, or metabolic regulators to tumors and lymphoid tissues. This review summarizes the immunological rationale for nanomedicine design in cancer immunotherapy, with emphasis on active targeting, stimulus-responsive release, multifunctional immune remodeling, checkpoint blockade delivery, nanovaccines, suppressive microenvironment modulation, and combined therapeutic strategies. Current evidence indicates that nanomedicine can improve pharmacokinetics, concentrate immune signals in relevant tissues, reduce systemic exposure, and synchronize innate and adaptive immune activation. Clinical translation still depends on reproducible manufacturing, validated biomarkers of delivery, immune safety, and trial designs that distinguish material effects from payload effects.

Keywords: Nanomedicine, cancer immunotherapy, nanoparticle delivery, tumor microenvironment, immune checkpoint blockade

1. Introduction

Cancer immunotherapy is grounded in the capacity of the host immune system to recognize malignant cells through antigenic differences and stress-associated signals. The cancer-immunity cycle describes a sequence in which tumor antigens are released, captured by antigen-presenting cells, presented to T cells, followed by T cell priming, trafficking, tumor infiltration, target recognition, and cytotoxic killing. Immune checkpoint inhibitors, adoptive cell therapy, tumor

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vaccines, cytokine therapy, and innate immune agonists intervene at different points in this sequence. Their clinical value is established, particularly through PD-1, PD-L1, and CTLA-4 blockade, but response rates vary widely. Primary resistance is frequent in tumors with low antigenicity, poor antigen presentation, T cell exclusion, suppressive myeloid infiltration, or defective interferon signaling. Adaptive resistance can emerge after an initial immune attack through induction of inhibitory ligands, recruitment of regulatory cells, or selection of antigen-loss variants [1].

The limitations of systemic immunotherapy reflect both tumor biology and drug distribution. Antibodies circulate for prolonged periods and engage targets in inflamed normal tissues as well as tumors. Cytokines and pattern-recognition receptor agonists can activate broad immune compartments before sufficient concentrations reach the tumor or draining lymph node. Nucleic acid therapeutics require protection from nucleases and access to cytosolic or endosomal compartments. Cell therapies face trafficking barriers, exhaustion, antigen heterogeneity, and local suppressive cues. These constraints have shaped the current interest in delivery systems able to concentrate immune modulation at lymphoid organs, tumor beds, or selected immune-cell subsets.

Nanomedicine delivery systems can change the biodistribution, persistence, cellular uptake, and intracellular routing of immunotherapeutic agents. Their dimensions overlap with biological transport pathways that control lymphatic drainage, phagocytic uptake, and tumor extravasation. Particles in the tens of nanometers can enter lymphatic vessels after local administration and reach draining lymph nodes, whereas larger or opsonized particles are more readily captured by mononuclear phagocytes. Surface charge, hydrophilicity, rigidity, ligand density, and protein corona formation further determine circulation time and cellular tropism. These properties allow engineered carriers to reduce premature degradation, buffer peak systemic exposure, and deliver combinations whose pharmacokinetics would otherwise be mismatched.

The value of nanomedicine in immunotherapy is not limited to passive tumor accumulation. Early assumptions about enhanced permeability and retention have been revised by quantitative analyses showing that only a small fraction of injected nanoparticle dose reaches solid tumors in many models. For immune applications, this limitation reframes design priorities. A carrier can be useful if it directs an adjuvant to dendritic cells, protects mRNA during lymphatic transport, maintains local checkpoint blockade at a surgical margin, or repolarizes tumor-associated macrophages after intratumoral administration, even when bulk tumor accumulation is modest. The design priority shifts from maximizing total tumor deposition to matching material behavior with a defined immunological bottleneck.

2. Tumor immune mechanisms and delivery requirements

2.1. Antitumor immune cycle and rate-limiting steps

Effective antitumor immunity requires coordinated innate sensing and adaptive effector function. Dendritic cells capture tumor antigens, migrate to lymph nodes, and cross-present peptide fragments on MHC class I molecules to CD8-positive T cells. Costimulatory signals and inflammatory cytokines then determine the magnitude, differentiation state, and memory potential of the T cell response. Activated lymphocytes must leave lymphoid tissue, enter the tumor vasculature, cross endothelial and stromal barriers, and maintain cytotoxic activity during repeated antigen encounter. Any weak link can limit therapeutic response, especially in tumors with sparse dendritic cell infiltration, low type I interferon tone, abnormal vessels, or dense extracellular matrix.

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Delivery needs differ across these steps. Antigen and adjuvant formulations must reach antigen-presenting cells in a form that supports cross-presentation without causing systemic cytokine release. Checkpoint inhibitors must engage inhibitory pathways at the tumor-immune interface while avoiding broad autoimmunity. STING and TLR agonists require intracellular delivery to endosomal or cytosolic compartments and often have narrow windows between immune activation and inflammatory toxicity [2, 3]. Nucleic acids require endosomal escape, while protein antigens require preservation of conformational or peptide epitopes. As summarized in Fig. 1, nanomedicine platforms are most rational when they are designed around the cancer-immunity cycle and the specific immune or stromal bottleneck that limits antitumor response.

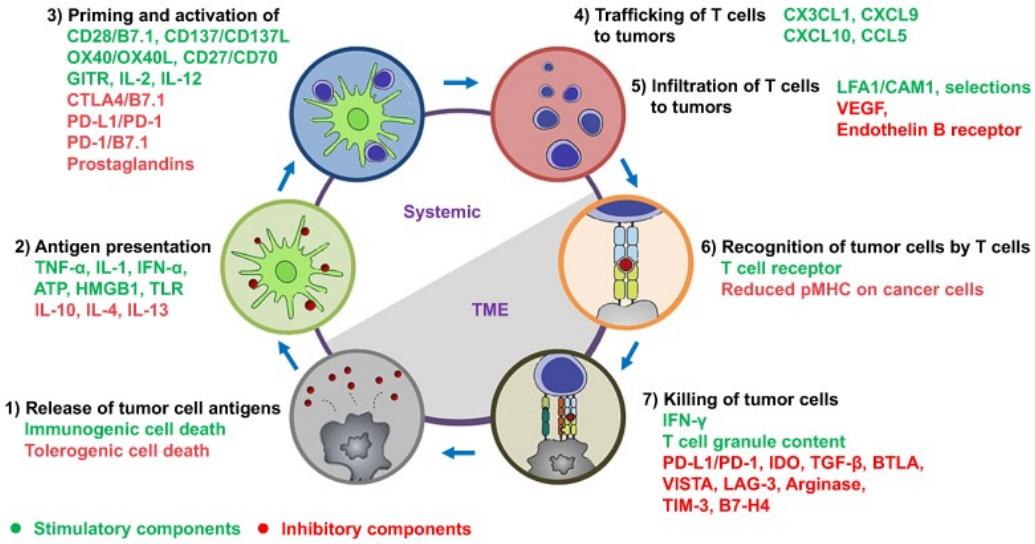

Figure 1. Cancer-immunity cycle and major stimulatory or inhibitory factors that define nanomedicine delivery targets in tumor immunotherapy

2.2. Immunosuppressive microenvironment

The tumor microenvironment contains malignant cells, fibroblasts, endothelial cells, extracellular matrix, resident and recruited immune cells, metabolites, and soluble mediators. Suppressive mechanisms include regulatory T cells, myeloid-derived suppressor cells, tumor-associated macrophages, indoleamine 2,3-dioxygenase activity, adenosine accumulation, prostaglandin signaling, lactate enrichment, hypoxia, TGF-beta, IL-10, and chronic antigen stimulation that favors T cell dysfunction [4]. Stromal architecture can physically separate cytotoxic T cells from malignant nests. In pancreatic, lung, breast, and colorectal tumors, fibroblast-rich compartments and abnormal vasculature often restrict lymphocyte penetration, reducing the effect of antibodies that require direct contact between T cells and tumor cells.

Nanomedicine approaches can address these barriers through local immune remodeling. Carriers taken up by macrophages can deliver TLR7/8 agonists or other innate stimulators to shift macrophage phenotypes toward inflammatory states. Particles that alter hypoxia, degrade matrix components, normalize vessels, or deliver metabolic inhibitors may improve effector-cell access and function. The same design can also create harm if immune activation spreads beyond the intended tissue. Complement activation, cytokine release, hepatosplenic accumulation, and persistent inflammasome activation remain relevant safety concerns. Immune suppression is spatially

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heterogeneous, so a platform that works in a macrophage-rich tumor may have less value in a lymphocyte-excluded tumor dominated by fibroblasts or aberrant vasculature.

2.3. Functional requirements for nanomedicine delivery systems

A delivery system for tumor immunotherapy should satisfy several connected requirements. It should maintain payload integrity during preparation, storage, and administration. It should reach the anatomical compartment where the selected immune mechanism operates. It should favor uptake by relevant cells and release the payload in the compartment where the molecular target is accessible. It should avoid excessive accumulation in normal tissues that mediate immune toxicity. It should support scalable, reproducible manufacturing with measurable attributes such as size distribution, encapsulation efficiency, surface chemistry, endotoxin burden, and release kinetics [5, 6].

These requirements are more stringent when the platform carries more than one immune agent. Co-delivery can improve mechanistic coordination, but it also imposes constraints on loading chemistry, relative dose, temporal release, and analytical characterization. Antigen and adjuvant may need synchronized arrival in the same dendritic cell, whereas checkpoint blockade and photothermal therapy may require different spatial and temporal profiles. The most persuasive nanomedicine designs link each material attribute to a measurable immunological event, such as dendritic-cell maturation, antigen cross-presentation, macrophage repolarization, T cell infiltration, or reduced systemic cytokine exposure.

3. Design strategies for nanomedicine delivery systems

3.1. Active targeting and cell-specific delivery

Active targeting uses ligands, antibodies, peptides, aptamers, sugars, or biomimetic membranes to increase interactions with selected cells. For tumor immunotherapy, relevant targets include tumor antigens, endothelial markers, dendritic-cell receptors, macrophage receptors, lymph-node stromal components, and T cell surface molecules. Ligand density and orientation influence binding avidity, but targeting cannot overcome all transport barriers. A ligand has little effect if the particle never reaches the tissue or if a protein corona masks the binding interface. For this reason, active targeting is most effective when paired with a route of administration and particle architecture that already position the carrier near the target cell.

Cell-specific delivery can also exploit endogenous tropisms. Myeloid cells efficiently internalize many particles, which can be a liability for conventional chemotherapy but an advantage for immunomodulators aimed at dendritic cells or macrophages. Lymph-node trafficking after subcutaneous or intradermal administration can be tuned by size and surface chemistry, supporting vaccine applications [7]. As shown in Fig. 2, biomimetic nanocarriers can package antigens, adjuvants, or other immunotherapeutic payloads and enhance their interaction with antigen-presenting cells or tumor-associated immune compartments. Platelet-bound anti-PD-L1 delivery after tumor resection illustrates how a biological carrier can localize checkpoint blockade to a wound-like surgical niche and circulating tumor-cell interface.

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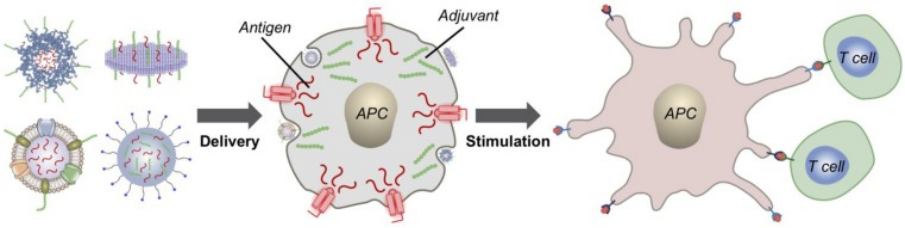

Figure 2. Biomimetic nanocarriers enhance the delivery of antigens and immunostimulatory agents to antigen-presenting cells for antitumor immune activation

Cellular specificity also depends on administration route. Intravenous particles encounter serum proteins, splenic filtration, hepatic sinusoidal endothelium, Kupffer cells, and circulating phagocytes before reaching tumors. Intratumoral administration bypasses part of this transport problem and can support potent local innate activation, although it is limited to accessible lesions and may not treat occult metastases unless systemic immunity develops. Intradermal and subcutaneous administration favor lymphatic access and are well suited to vaccines. Inhaled, intravesical, or intraperitoneal routes may be appropriate for selected anatomical sites. Route selection is a design variable rather than a procedural detail, because it changes the first immune cells encountered by the carrier.

3.2. Endogenous and exogenous stimulus-responsive release

Stimulus-responsive systems seek to reduce off-target exposure by releasing payloads in response to biochemical or physical cues. Endogenous triggers include acidic pH, high glutathione concentration, reactive oxygen species, enzymes, hypoxia, ATP, and tumor-associated metabolites. These cues are not unique to tumors, but their relative enrichment can be used to bias release in endosomes, lysosomes, or inflamed tumor tissue. Redox-sensitive antigen nanoparticles and pH-responsive STING nanovaccines illustrate how intracellular conditions can be used to increase antigen processing or cyclic dinucleotide release [8].

External triggers add spatial control. Light, ultrasound, magnetic fields, heat, and ionizing radiation can activate or enhance nanomedicine function at a defined site. Photothermal nanoparticles can induce local tumor destruction, release antigens, and create an inflammatory milieu that becomes more effective when paired with adjuvant delivery and checkpoint blockade. Radiation can increase antigen release, type I interferon signaling, and vascular changes, and nanomedicine can concentrate radiosensitizers or immunostimulants in the irradiated field. These strategies require careful calibration because tissue penetration, heat distribution, and dose heterogeneity can limit reproducibility across tumor locations.

3.3. Multifunctional synergistic immune regulation

Multifunctional nanomedicine platforms are designed to coordinate immune activation, tumor killing, and microenvironment remodeling. A single carrier may combine tumor antigen, TLR agonist, STING agonist, checkpoint inhibitor, chemotherapeutic, photosensitizer, or nucleic acid payloads. Co-localized adjuvant delivery can amplify immunogenic cell death by ensuring that antigens released from dying tumor cells are accompanied by maturation signals for antigen-presenting cells [9]. Nanodiscs and other vaccine platforms use lipid or polymer architectures to improve lymphoid delivery and sustain antigen display, thereby strengthening CD8-positive T cell priming.

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Synergy depends on sequence as much as composition. Innate activation before sufficient antigen availability may generate inflammation without durable specificity. Checkpoint blockade without T cell infiltration may have limited effect. Cytotoxic therapy can release antigens but can also deplete immune cells if delivered at the wrong dose or time. Nanomedicine can partially address this problem by aligning pharmacokinetics, but the platform must be tested against mechanistic endpoints rather than tumor volume alone. Designs that report antigen presentation, cytokine profiles, T cell receptor clonality, spatial immune infiltration, and memory responses provide stronger evidence than formulations evaluated only by survival curves.

4. Applications of nanomedicine delivery systems in immunotherapy

4.1. Delivery and potentiation of immune checkpoint inhibitors

Immune checkpoint blockade depends on sustained engagement of inhibitory receptors or ligands at sites of antitumor immune interaction. Systemic monoclonal antibodies have transformed cancer therapy, yet immune-related adverse events and limited tumor penetration support interest in localized or cell-associated delivery. Microneedle-assisted delivery of anti-PD-1 antibody was developed to retain antibody in the skin tumor region and improve local immune activation in melanoma models. Platelet-conjugated anti-PD-L1 has been used to release checkpoint inhibitor activity at surgical wounds and platelet-derived microparticle sites after platelet activation. As illustrated in Fig. 3, nanoparticle-mediated antibody delivery can reposition checkpoint blockade and costimulatory signaling to the tumor-immune interface rather than replacing the underlying antibody mechanism.

Nanomedicine can also potentiate checkpoint blockade indirectly. Platforms carrying TLR7/8 agonists, STING agonists, photothermal agents, or tumor antigens can convert poorly inflamed tumors into more permissive tissues for PD-1 or PD-L1 blockade. This rationale is strongest when treatment increases dendritic-cell activation, type I interferon signaling, intratumoral CD8-positive T cells, or the ratio of effector T cells to suppressive myeloid cells. Local or regional delivery may reduce systemic inflammatory exposure, but checkpoint-related toxicity can still occur if released agents enter circulation or induce widespread cytokine production. Translational studies need pharmacodynamic biomarkers that show where checkpoint engagement and immune activation occur.

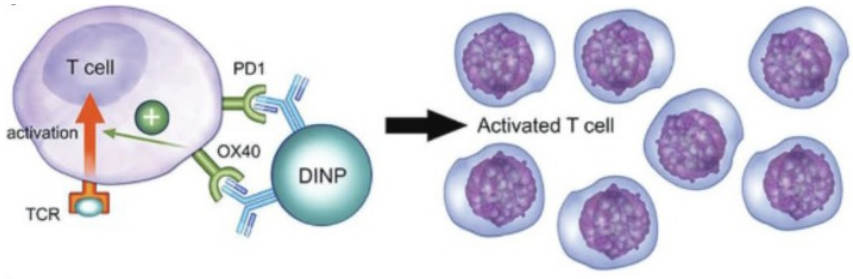

Figure 3. Nanoparticle-based antibody co-delivery can combine checkpoint blockade with costimulatory signaling to strengthen T cell-mediated tumor killing

4.2. Nanovaccines carrying tumor antigens and adjuvants

Cancer vaccines require antigen delivery, antigen-presenting cell activation, cross-presentation, and expansion of functional T cell clones. Soluble peptide or protein vaccines often distribute rapidly,

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lose co-localization with adjuvant, and produce weak CD8-positive T cell priming. Nanovaccines address these problems by packaging antigen and adjuvant in the same carrier, draining to lymph nodes, improving dendritic-cell uptake, and prolonging antigen presentation. Designer high-density lipoprotein-like nanodiscs carrying antigen peptides and adjuvants improved delivery to lymphoid organs and supported strong cytotoxic T lymphocyte responses in preclinical personalized vaccine models.

Adjuvant choice and intracellular routing are decisive. TLR7/8 agonist-loaded polymeric nanoparticles enhance dendritic-cell maturation and antigen presentation while reducing the uncontrolled diffusion associated with soluble agonists [10]. STING agonists require cytosolic access, so endosomolytic polymersomes and pH-responsive DNA nanovaccines were developed to improve cyclic dinucleotide activity after cellular uptake [11]. Neoantigen vaccines add another level of personalization, since antigen selection depends on sequencing, binding prediction, clonality, expression, and immune tolerance. Nanomedicine cannot compensate for poor antigen choice, but it can increase the probability that selected antigens and innate cues arrive in the same antigen-presenting cell at immunologically productive concentrations.

4.3. Regulation of the immunosuppressive microenvironment

Myeloid cells are major targets for nanomedicine because many nanoparticle systems accumulate in phagocytic compartments. Tumor-associated macrophages can suppress T cells, support angiogenesis, remodel extracellular matrix, and promote metastasis. TLR7/8 agonist-loaded beta-cyclodextrin nanoparticles have been shown to accumulate in tumor-associated macrophages and to suppressive myeloid cells with the aim of reducing arginase activity, IDO-associated tolerance, adenosine signaling, or anti-inflammatory cytokine production [12].

The extracellular and vascular compartments are additional targets. Dense matrix, high interstitial pressure, and abnormal vessels restrict drug distribution and lymphocyte access. STING-activating nanoparticles that affect the vascular-immune interface provide one example of using innate immune activation to remodel both immune and endothelial compartments. Hypoxia-modulating particles, matrix-responsive carriers, and vascular-normalizing strategies may improve infiltration by effector cells. These approaches need spatial analysis because average immune-cell abundance can obscure whether T cells contact malignant cells or remain trapped in stromal regions. For example, TLR7/8 agonist-loaded nanoparticles can promote inflammatory polarization of tumor-associated macrophages and thereby improve responses to checkpoint blockade in preclinical tumor models [13]. Other platforms that deliver small molecules, nucleic acids, or metabolic regulators may further suppress IDO activity, adenosine signaling, lactate-driven dysfunction, or anti-inflammatory cytokine production within the tumor microenvironment.

4.4. Combined therapeutic strategies based on nanoplatforms

Combination therapy is central to nanomedicine-based immunotherapy because tumors often contain several resistance mechanisms at once. Photothermal therapy with immune-adjuvant nanoparticles and checkpoint blockade can combine local tumor injury, antigen release, dendritic-cell activation, and relief of inhibitory signaling. Co-localized adjuvant delivery after immunogenic cell death can reinforce antigen-specific immunity by coupling danger signals to tumor-derived antigens. Radiotherapy and nanomedicine combinations similarly use tumor damage as a source of antigen and inflammatory signaling, while nanoparticles shape the location and persistence of immunostimulatory payloads.

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Nucleic acid delivery adds programmable immune modulation. Biomimetic nanoparticles delivering mRNAs that encode costimulatory receptors have been reported to enhance T cell mediated antitumor immunity, illustrating how transient genetic programming may complement antibody or small-molecule approaches [14]. Nanomaterials for T cell immunotherapy also include particles for ex vivo cell engineering, in vivo T cell targeting, cytokine support, and modulation of chimeric antigen receptor T cell function [15]. For combined platforms, translation requires evidence that each component contributes to efficacy and that the combined safety profile is acceptable. A formulation with many payloads can be mechanistically attractive but difficult to manufacture, characterize, dose, and regulate.

5. Conclusions and outlook

Nanomedicine delivery systems have moved tumor immunotherapy from systemic immune stimulation toward spatially and temporally controlled immune intervention. The strongest evidence comes from platforms that solve defined delivery problems, including lymph-node co-delivery of antigen and adjuvant, intracellular delivery of STING or TLR agonists, local checkpoint blockade at tumor or surgical sites, macrophage-directed reprogramming, and synchronization of immunogenic tumor injury with innate activation. These systems do not remove the biological complexity of cancer immunity. They provide tools for testing whether a specific immune barrier can be altered without exposing the whole organism to the same intensity of stimulation.

Several issues remain before broad clinical use. Tumor delivery is variable across species, tumor sites, vascular phenotypes, and prior treatments. Rodent models often overestimate homogeneous accumulation and immune responsiveness. Human tumors contain larger diffusion distances, more heterogeneous stroma, and treatment histories that reshape immune compartments. Manufacturing adds another constraint. Small changes in lipid composition, polymer molecular weight, ligand density, residual solvent, or sterilization procedure can alter protein corona formation, complement activation, and payload release. For complex multifunctional particles, analytical methods must confirm not only average composition but also batch-to-batch distributions of payload ratio, surface presentation, and release behavior.

Clinical development will benefit from trials that pair therapeutic endpoints with delivery and immune pharmacodynamic measurements. Imaging, blood cytokine kinetics, tumor biopsies, spatial transcriptomics, single-cell profiling, and antigen-specific T cell assays can clarify whether a platform reaches the intended tissue and changes the intended immune step. Biomarker selection should follow the mechanism of the carrier. A lymph-node vaccine requires evidence of antigen-specific priming; a macrophage-targeted particle requires myeloid-state analysis; a checkpoint-delivery platform requires local receptor engagement and immune toxicity monitoring. Without these measurements, negative trials may not distinguish failed delivery from a wrong immune hypothesis.

Future nanomedicine immunotherapy is likely to favor fewer but better justified components, adaptable manufacturing, and patient stratification based on immune architecture. Tumors with absent priming may require vaccines or innate agonist delivery. Tumors with T cell exclusion may require stromal or vascular remodeling before checkpoint blockade. Tumors with inflamed but exhausted lymphocytes may need localized checkpoint or cytokine support. The field is moving toward mechanism-matched delivery rather than universal nanoparticle formulations. When material design, immune biology, and clinical biomarkers are aligned, nanomedicine can become a practical means of converting potent immune agents into treatments with more controlled distribution, stronger local activity, and narrower systemic toxicity.

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