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Biomimetic Nanoparticles for Precision Targeting of Cancer Stem Cells and Tumor Recurrence

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ABSTRACT

Tumor recurrence and therapeutic resistance remain major obstacles in effective cancer management, largely driven by a small but highly resilient subpopulation of cells known as cancer stem cells (CSCs). These cells possess self-renewal capacity, multipotency, and heightened resistance to chemotherapy, radiotherapy, and immunotherapy, enabling tumor regrowth and metastasis even after apparent clinical remission. Conventional anticancer strategies often fail to eradicate CSCs due to poor targeting specificity, systemic toxicity, and an inability to overcome the protective tumor microenvironment. Biomimetic nanoparticles have emerged as a promising precision nanomedicine strategy to address these limitations by emulating natural biological structures such as cell membranes, lipoproteins, exosomes, and extracellular matrix components. By inheriting biological functionalities including immune evasion, homotypic targeting, and receptor-specific interactions, biomimetic nanoparticles enable selective delivery of therapeutic payloads to CSC niches. This review comprehensively examines the biological basis of CSC-driven tumor recurrence, the design principles of biomimetic nanoparticles, and their application in selectively targeting CSCs. We discuss advances in membrane-coated, ligand-functionalized, and microenvironment-responsive biomimetic systems, highlight preclinical evidence of efficacy in preventing tumor relapse, and analyze translational challenges. Collectively, biomimetic nanoparticles represent a powerful platform for durable cancer control through precise CSC targeting.

Keywords: cancer stem cells; biomimetic nanoparticles; tumor recurrence; precision nanomedicine; targeted drug delivery

INTRODUCTION

Tumor recurrence remains a leading cause of cancer-related mortality despite significant advances in early detection and treatment[1, 2]. A growing body of evidence attributes recurrence, metastasis, and therapeutic resistance to a distinct subpopulation of tumor cells termed cancer stem cells (CSCs). CSCs are defined by their ability to self-renew, differentiate into heterogeneous tumor cell populations, and initiate tumor formation when transplanted into immunocompromised hosts[3-5]. These properties mirror those of normal stem cells but are deregulated by oncogenic signaling, epigenetic alterations, and microenvironmental cues.

CSCs have been identified across a wide range of malignancies, including breast, colorectal, lung, pancreatic, and hematological cancers. They are commonly characterized by specific surface markers such as CD44, CD133, ALDH1, EpCAM, and CXCR4, although marker expression is often context-dependent and dynamic[6-8]. Importantly, CSCs are not static entities; non-stem cancer cells can acquire stem-like properties through epithelial–mesenchymal transition, metabolic reprogramming, or therapy-induced stress. This phenotypic plasticity complicates eradication strategies and contributes to tumor relapse following treatment [8-11].

One of the defining features of CSCs is their intrinsic resistance to conventional therapies. CSCs often exhibit quiescence or slow-cycling behavior, rendering them less susceptible to cytotoxic agents that target rapidly dividing cells[9, 10, 12-15]. They also overexpress ATP-binding cassette transporters that actively efflux chemotherapeutic drugs, reducing intracellular drug accumulation. Enhanced DNA damage repair capacity,

elevated anti-apoptotic signaling, and robust antioxidant defenses further protect CSCs from radiotherapy- and chemotherapy-induced cytotoxicity[10,16-23].
The tumor microenvironment plays a critical role in maintaining CSC survival and function. CSCs preferentially reside in specialized niches characterized by hypoxia, extracellular matrix remodeling, immune suppression, and paracrine signaling from stromal cells. Hypoxic conditions stabilize hypoxia-inducible factors, which promote stemness, angiogenesis, and metabolic adaptation[11-13,24-28]. Interactions with cancer-associated fibroblasts, endothelial cells, and immune cells further reinforce CSC phenotypes and shield them from therapeutic insult.

Despite their central role in recurrence, CSCs remain difficult to target selectively. Many CSC-associated signaling pathways, such as Wnt/β-catenin, Notch, Hedgehog, and TGF-β, are also essential for normal stem cell maintenance, raising concerns about off-target toxicity[14, 15,29-33]. Additionally, CSCs often represent a minor fraction of the tumor mass, necessitating highly sensitive and specific delivery systems capable of reaching CSC niches without damaging normal tissues.
Traditional drug delivery approaches are poorly suited to meet these demands. Systemically administered drugs exhibit limited tumor penetration, rapid clearance, and nonspecific biodistribution[16,34-38]. Even advanced nanocarriers may be recognized by the immune system or fail to accumulate effectively in CSC-rich regions. These limitations have driven the development of biomimetic nanoparticles, which leverage natural biological features to improve targeting precision, circulation time, and therapeutic efficacy against CSCs[16,39-43]. By aligning nanotechnology with CSC biology, biomimetic strategies offer a rational path toward preventing tumor recurrence and achieving durable cancer remission.

2. Design Principles of Biomimetic Nanoparticles

Biomimetic nanoparticles are engineered nanoscale systems that imitate the structural, chemical, or functional properties of natural biological entities. Unlike conventional synthetic nanoparticles, biomimetic systems are designed to interact seamlessly with biological environments, thereby enhancing biocompatibility, immune evasion, and targeting specificity[17]. Their design typically integrates a synthetic core for drug loading with a biologically derived or bioinspired outer layer that confers functional advantages[17,44-48].
One of the most widely explored biomimetic strategies involves cell membrane coating. Nanoparticles cloaked with membranes derived from cancer cells, stem cells, immune cells, or platelets retain surface proteins, lipids, and glycans from the source cells[17–19, 49-51]. This molecular inheritance enables homotypic targeting, whereby cancer cell membrane–coated nanoparticles preferentially bind to and accumulate in tumors or CSC niches through self-recognition mechanisms. Additionally, membrane coatings can camouflage nanoparticles from immune surveillance, prolonging systemic circulation.
Ligand-based biomimicry represents another key design principle. By functionalizing nanoparticle surfaces with natural ligands such as hyaluronic acid, peptides, antibodies, or aptamers that recognize CSC-associated receptors, biomimetic nanoparticles achieve selective binding and internalization. Hyaluronic acid targeting of CD44-positive CSCs is a prominent example, combining receptor specificity with biodegradability and minimal immunogenicity[20, 21,52-54].
Biomimetic nanoparticles also exploit microenvironmental cues characteristic of CSC niches. pH-sensitive, redox-responsive, and enzyme-cleavable designs allow nanoparticles to remain stable during circulation but release their payload selectively within hypoxic, acidic, or protease-rich tumor regions. Such responsiveness enhances therapeutic precision while minimizing off-target effects[11–13,55-56].
Collectively, these design principles enable biomimetic nanoparticles to overcome biological barriers that limit conventional therapies, positioning them as highly adaptable platforms for CSC-targeted nanomedicine.

3. Biomimetic Targeting of Cancer Stem Cell Surface Markers

Cancer stem cells (CSCs) represent a small yet highly influential subpopulation within tumors, responsible for therapy resistance, metastatic dissemination, and disease recurrence. Selective recognition and targeting of CSC-specific surface markers is therefore central to precision oncology[22]. Biomimetic nanoparticles have emerged as a powerful strategy to exploit the overexpression of CSC-associated markers, including CD44, CD133, EpCAM, and CXCR4, enabling enhanced specificity and therapeutic efficacy while minimizing off-target effects[4, 22].
Biomimetic targeting leverages natural ligand–receptor interactions to achieve preferential CSC uptake. Hyaluronic acid (HA)–based nanoparticles exemplify this approach, as HA is a natural ligand for CD44, a marker highly enriched in CSCs across breast, ovarian, pancreatic, and colorectal cancers. HA-functionalized nanoparticles undergo CD44-mediated endocytosis, facilitating efficient intracellular delivery of chemotherapeutic agents, small interfering RNAs, microRNAs, or differentiation-inducing compounds[23]. These interventions suppress stemness-associated signaling pathways, reduce self-renewal capacity, and impair tumor-initiating potential.
Similarly, biomimetic nanoparticles targeting CD133 have demonstrated substantial efficacy in eliminating CSCs in glioblastoma, hepatocellular carcinoma, and colorectal cancer models. Functionalization with CD133-

specific peptides, antibodies, or antibody fragments enhances selective binding and internalization, overcoming the poor penetration and nonspecific distribution associated with conventional therapeutics[24, 25]. Beyond CD44 and CD133, targeting EpCAM and CXCR4 enables disruption of CSC adhesion, migration, and metastatic homing, addressing both local tumor persistence and distant dissemination[25].

A defining advantage of biomimetic nanoparticles is their capacity for multivalent interactions. By presenting multiple copies of targeting ligands on their surface, these systems achieve higher binding avidity than free ligands or monoclonal antibodies[26]. This multivalency is particularly important given the low frequency of CSCs within heterogeneous tumor populations. Enhanced internalization efficiency ensures that therapeutic payloads are preferentially concentrated within CSCs, amplifying antitumor effects while sparing normal stem and progenitor cells that express lower levels of these markers[26].

Collectively, biomimetic targeting of CSC surface markers represents a refined and adaptable strategy for selectively dismantling the cellular drivers of tumor recurrence and resistance, laying the foundation for durable cancer control.

4. Cell Membrane–Coated Nanoparticles for CSC Homotypic Targeting

Cell membrane–coated nanoparticles represent one of the most sophisticated biomimetic strategies for CSC targeting, offering unparalleled biological complexity and functional versatility[27]. By cloaking synthetic nanoparticle cores with natural cell membranes, these systems inherit membrane proteins, adhesion molecules, and antigens that mediate precise cellular interactions. This approach enables homotypic targeting, wherein nanoparticles preferentially bind to cells of the same origin, including CSC subpopulations[27].

Nanoparticles coated with cancer cell membranes derived from CSC-enriched cultures have demonstrated enhanced accumulation within CSC niches in multiple preclinical models[15]. The preserved membrane proteins facilitate selective adhesion to parental tumor cells, enabling deep tumor penetration and preferential interaction with CSCs embedded within protective microenvironments. Importantly, this targeting strategy does not rely on synthetic ligands, reducing immunogenicity and simplifying design complexity.

Beyond cancer cell membranes, immune cell membrane coatings provide additional targeting and functional advantages. Macrophage- or T cell–membrane–coated nanoparticles exploit natural tumor-homing pathways and immune–tumor interactions, enabling access to CSCs residing in immunologically active niches[28]. These systems can simultaneously deliver cytotoxic agents and immune-modulating signals, enhancing CSC eradication through both direct killing and immune-mediated mechanisms.

A critical advantage of membrane-coated nanoparticles is immune camouflage. By presenting "self" markers such as CD47, these nanoparticles evade opsonization and clearance by the mononuclear phagocyte system, resulting in prolonged circulation times[29]. Extended systemic persistence increases the probability of nanoparticle accumulation in CSC-rich regions and supports sustained therapeutic exposure, which is essential for targeting slow-cycling or dormant CSCs.

Together, cell membrane–coated nanoparticles represent a biologically intuitive and highly effective approach for CSC targeting, combining precision, immune evasion, and multifunctionality within a single biomimetic platform.

5. Biomimetic Nanoparticles for Modulating CSC Niches and Plasticity

Targeting CSCs in isolation is insufficient without addressing the tumor microenvironment that sustains their survival and plasticity. CSC niches are characterized by hypoxia, altered extracellular matrix (ECM) composition, metabolic reprogramming, and paracrine signaling that collectively preserve stemness and promote therapy resistance. Biomimetic nanoparticles have been engineered to disrupt these supportive niches, thereby sensitizing CSCs to treatment and preventing their regeneration[30].

Nanoparticles mimicking ECM components, such as collagen- or fibronectin-inspired structures, enhance penetration through dense stromal barriers and facilitate access to CSC-enriched regions[31, 32]. Hypoxia-responsive biomimetic systems selectively activate within oxygen-deprived niches, releasing therapeutic payloads precisely where CSCs are most protected. This spatial selectivity minimizes systemic toxicity while maximizing niche-specific disruption[31].

In addition to niche targeting, biomimetic nanoparticles can modulate CSC plasticity—the dynamic interconversion between CSCs and non-stem cancer cells. Delivery of agents targeting epithelial–mesenchymal transition, epigenetic regulators, or metabolic pathways interferes with the molecular programs that enable CSC regeneration[33]. By suppressing plasticity, these systems reduce the replenishment of CSC pools following therapy, addressing a major driver of tumor relapse.

By simultaneously dismantling physical niches and molecular adaptability, biomimetic nanoparticles offer a comprehensive strategy to destabilize CSC ecosystems and restore therapeutic vulnerability[33].

6. Preventing Tumor Recurrence through Combinatorial Biomimetic Nanotherapy

Tumor recurrence is driven by interconnected intrinsic and extrinsic mechanisms, including CSC survival, immune evasion, and microenvironmental protection. Consequently, effective prevention requires combinatorial therapeutic strategies capable of addressing multiple pathways simultaneously[34]. Biomimetic nanoparticles

are uniquely suited for this purpose, enabling the co-delivery of diverse therapeutic agents within a single, coordinated platform.

These systems can encapsulate cytotoxic drugs alongside CSC pathway inhibitors, immune modulators, or anti-stromal agents, ensuring synchronized spatial and temporal action. Such coordination is difficult to achieve with separate drug administrations but is critical for overcoming compensatory survival mechanisms[35]. Biomimetic coatings further enhance targeting precision, ensuring that combination therapies are delivered directly to recurrence-driving cellular populations.

Integration of CSC-targeted therapy with immune activation has proven particularly effective. Biomimetic nanoparticles that combine CSC eradication with immune stimulation promote long-term tumor surveillance, reducing the likelihood of disease re-emergence[35–37]. Preclinical studies consistently demonstrate that combinatorial biomimetic nanotherapies outperform monotherapies in suppressing tumor regrowth, metastasis, and therapeutic resistance. In sum, biomimetic nanotechnology offers a versatile and powerful framework for preventing tumor recurrence by harmonizing precision targeting, combination therapy, and immune engagement into a single, clinically translatable strategy.

7. Conclusion and Future Perspectives

Biomimetic nanoparticles represent a powerful and versatile strategy for precision targeting of cancer stem cells and prevention of tumor recurrence. By emulating natural biological structures and interactions, these systems overcome key barriers in drug delivery, achieve selective CSC targeting, and modulate supportive tumor niches. Continued advances in biomaterials engineering, CSC biology, and translational nanomedicine are expected to accelerate clinical adoption. Future success will depend on scalable manufacturing, robust safety evaluation, and patient stratification based on CSC biomarkers. As these challenges are addressed, biomimetic nanoparticles hold strong promise for achieving durable cancer remission and improving long-term patient outcomes.

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