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Exosome-Mimetic Nanoparticles for Intercellular Signal Interception in Metastatic Cancer

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ABSTRACT

Metastatic cancer progression is critically driven by intercellular communication networks that enable tumor cells to remodel distant microenvironments, evade immune surveillance, and acquire invasive phenotypes. Among the most influential mediators of this communication are exosomes nanoscale extracellular vesicles that transfer proteins, lipids, and nucleic acids between cells. Tumor-derived exosomes actively orchestrate pre-metastatic niche formation, promote epithelial–mesenchymal transition, and reprogram stromal and immune cells to support dissemination. However, directly targeting endogenous exosomes remains technically challenging due to their heterogeneity, abundance, and physiological roles. Exosome-mimetic nanoparticles have emerged as a powerful alternative, designed to structurally and functionally resemble natural exosomes while offering superior controllability, scalability, and engineering flexibility. These biomimetic systems can competitively intercept pathological signaling, sequester oncogenic cargo, or deliver therapeutic agents that disrupt metastatic communication pathways. This review provides a comprehensive analysis of exosome-mimetic nanoparticles as tools for intercellular signal interception in metastatic cancer. We examine their design principles, mechanisms of action, and applications in blocking tumor–stromal, tumor–immune, and tumor–tumor communication. Preclinical progress, translational challenges, and future directions toward clinical implementation are critically discussed, highlighting their promise in next-generation anti-metastatic therapies.
Keywords: exosome-mimetic nanoparticles; metastatic cancer; intercellular communication; signal interception; biomimetic nanomedicine

INTRODUCTION

Metastasis is responsible for the vast majority of cancer-related mortality and represents a complex, multistep process requiring precise coordination between tumor cells and their microenvironment[1, 2]. Central to this coordination is intercellular communication, through which cancer cells exchange molecular signals with neighboring tumor cells, stromal fibroblasts, endothelial cells, immune cells, and distant organ niches[3–6]. While soluble factors such as cytokines and growth factors play important roles, extracellular vesicles, particularly exosomes, have emerged as dominant mediators of long-range and highly specific signaling in metastatic cancer.

Exosomes are membrane-bound vesicles, typically 30–150 nm in diameter, generated within multivesicular bodies and released upon fusion with the plasma membrane. Their cargo includes proteins, lipids, messenger RNAs, microRNAs, long non-coding RNAs, and even DNA fragments[7–9]. Tumor-derived exosomes carry oncogenic signatures reflective of their cell of origin and are capable of reprogramming recipient cells at both local and systemic levels. In metastatic disease, exosomes contribute to nearly every stage of the metastatic cascade. They promote epithelial–mesenchymal transition, enhance migratory and invasive capacity, stimulate angiogenesis, suppress anti-tumor immunity, and condition distant organs to form permissive pre-metastatic niches[10–12].

A defining feature of exosome-mediated signaling is its efficiency and specificity. Surface proteins such as integrins, tetraspanins, and adhesion molecules dictate organotropism, allowing tumor exosomes to preferentially home to particular tissues such as the lung, liver, or brain[13–15]. Upon uptake, exosomal cargo

alters gene expression and metabolic states in recipient cells, effectively extending the influence of the primary tumor beyond its anatomical boundaries. This ability to remotely manipulate cellular behavior makes exosomes attractive targets for anti-metastatic intervention.

However, therapeutic targeting of endogenous exosomes is fraught with challenges. Exosomes are heterogeneous, abundantly produced, and essential for normal physiological processes, including immune regulation and tissue repair[8, 16, 17]. Systemic depletion or broad inhibition of exosome biogenesis risks unacceptable toxicity. Furthermore, the nanoscale size and dynamic composition of exosomes complicate their selective neutralization in vivo. These limitations have driven interest in alternative strategies that modulate pathological communication without disrupting normal vesicular trafficking.

Exosome-mimetic nanoparticles address this challenge by replicating key structural and functional attributes of natural exosomes while allowing precise control over composition, targeting, and function. Rather than attempting to eliminate endogenous exosomes, these engineered systems are designed to intercept, compete with, or reprogram intercellular signaling networks central to metastasis[18–20]. By acting as decoys, signal sinks, or targeted delivery vehicles, exosome-mimetic nanoparticles offer a sophisticated approach to disrupting metastatic communication with high specificity and reduced systemic impact.

2. Design Principles of Exosome-Mimetic Nanoparticles

Exosome-mimetic nanoparticles are engineered nanostructures designed to recapitulate the size, morphology, surface chemistry, and biological behavior of natural exosomes. Their design is guided by a balance between biomimicry and synthetic control, enabling functional replication of exosome-mediated interactions while overcoming the limitations of naturally derived vesicles[21, 22].

A central design consideration is size and membrane architecture. Most exosome-mimetic nanoparticles are fabricated within the 50–150 nm range to optimize cellular uptake and biodistribution. Lipid-based nanoparticles, polymeric vesicles, and hybrid systems incorporating cell membrane coatings are commonly employed. Membrane composition is critical, as lipid rafts, cholesterol content, and membrane fluidity influence vesicle stability and fusion with target cells[22–26].

Surface functionalization represents another key element. By incorporating exosomal markers such as CD9, CD63, CD81, or tumor-specific integrins, exosome-mimetic nanoparticles can engage the same receptors and uptake pathways as tumor-derived exosomes. Cell membrane–camouflaged nanoparticles, derived from cancer cells, platelets, or immune cells, provide an especially high degree of biological fidelity and immune evasion[27, 28].

Cargo loading strategies distinguish exosome-mimetic nanoparticles from endogenous exosomes. Therapeutic payloads may include small-molecule inhibitors, siRNAs, antisense oligonucleotides, microRNA sponges, or proteins designed to neutralize oncogenic signals. Unlike natural exosomes, whose cargo is biologically constrained, exosome-mimetic systems allow precise tuning of cargo identity and dose[29].

Importantly, exosome-mimetic nanoparticles can be engineered for signal interception rather than delivery alone. Decoy nanoparticles displaying receptors for growth factors, chemokines, or exosomal ligands can sequester pro-metastatic signals before they reach recipient cells. This functional versatility makes exosome-mimetic nanoparticles uniquely suited for disrupting communication-driven metastatic progression.

3. Mechanisms of Intercellular Signal Interception

Exosome-mimetic nanoparticles intercept intercellular signaling through multiple, non-mutually exclusive mechanisms. One primary mechanism is competitive uptake. By mimicking the surface ligands of tumor-derived exosomes, these nanoparticles compete for binding to recipient cell receptors, reducing the internalization of oncogenic exosomes[21, 27]. This approach effectively dilutes the pathological signal without requiring direct elimination of endogenous vesicles. Another mechanism involves cargo sequestration. Exosome-mimetic nanoparticles can be engineered to bind and neutralize soluble mediators or exosomal cargo, such as pro-metastatic microRNAs or immunosuppressive proteins. Acting as molecular sponges, these systems reduce the bioavailability of signaling molecules critical for metastatic niche formation and immune evasion[30].

Signal redirection is an additional strategy. Some exosome-mimetic nanoparticles are designed to deliver antagonistic or corrective signals upon uptake, counteracting the effects of tumor-derived exosomes. For example, delivery of tumor-suppressive microRNAs to stromal or immune cells can reverse pro-metastatic reprogramming[31].

Finally, immune-mediated clearance represents an emerging mechanism. By decorating exosome-mimetic nanoparticles with immunogenic motifs, it is possible to promote recognition and clearance of both the nanoparticles and associated tumor exosomes by phagocytic cells [32, 33]. This indirect interception leverages innate immune pathways to disrupt metastatic communication networks.

4. Targeting Tumor–Stromal and Pre-Metastatic Niche Signaling

Tumor–stromal interactions play a decisive role in metastatic progression, particularly during the early stages of pre-metastatic niche formation. Prior to the arrival of circulating tumor cells, primary tumors actively condition distant organs through the release of soluble factors and extracellular vesicles, especially exosomes[34]. These tumor-derived exosomes transport proteins, lipids, and nucleic acids that reprogram

fibroblasts, endothelial cells, and resident immune populations, generating a permissive microenvironment for subsequent tumor cell colonization. Disrupting these preparatory signals represents a powerful strategy to prevent metastasis, and exosome-mimetic nanoparticles have emerged as a promising nanotechnological approach to achieve this goal[34].

Exosome-mimetic nanoparticles are engineered to replicate key structural and functional properties of natural exosomes, including size, membrane composition, and surface ligands, while offering greater control over cargo loading and targeting specificity[35]. By homing to stromal compartments in distant organs, these systems can act as decoys or delivery vehicles to interfere with tumor-derived signaling. For instance, exosome-mimetic nanoparticles presenting decoy receptors for vascular endothelial growth factor, transforming growth factor-β, or inflammatory cytokines can sequester pro-metastatic factors before they engage stromal cells[35]. This interception disrupts angiogenic activation, inflammatory priming, and fibroblast-mediated extracellular matrix remodeling hallmark features of pre-metastatic niche development[36, 37].

Preclinical studies have demonstrated that exosome-mimetic strategies effectively reduce vascular permeability, suppress fibroblast activation, and normalize extracellular matrix architecture in target organs such as the lung and liver[38]. By stabilizing the stromal microenvironment, these interventions limit tumor cell adhesion and extravasation, thereby reducing metastatic burden. Importantly, exosome-mimetic nanoparticles can be engineered to deliver inhibitory RNA molecules or small-molecule inhibitors directly to stromal cells, further reinforcing niche resistance to metastatic colonization[38].

A particularly powerful feature of exosome-mimetic systems is their capacity for organ-specific targeting. Tumor-derived exosomes exploit integrin-mediated homing mechanisms to preferentially accumulate in specific organs, such as α6β4 and α6β1 integrins for lung metastasis or αvβ5 integrin for liver metastasis[39]. By incorporating similar integrin profiles or targeting ligands, exosome-mimetic nanoparticles can selectively intercept metastatic signaling in organs at highest risk[39]. This precision minimizes systemic interference with normal physiological signaling and enhances therapeutic safety. Collectively, targeting tumor–stromal communication at the pre-metastatic stage using exosome-mimetic nanotechnology represents a paradigm shift from reactive to preventative anti-metastatic therapy.

5. Modulating Tumor–Immune Communication in Metastasis

Effective immune surveillance is a major barrier to metastatic dissemination; however, tumor-derived exosomes play a pivotal role in undermining antitumor immunity. These vesicles transfer immunosuppressive proteins, cytokines, and regulatory nucleic acids that reprogram immune cells locally and systemically[40]. Exosomal PD-L1, transforming growth factor-β, and immunosuppressive microRNAs suppress cytotoxic T-cell function, promote regulatory T-cell expansion, and polarize macrophages toward tumor-supportive phenotypes. Consequently, intercepting exosome-mediated immune modulation has emerged as an attractive strategy to restore immune control over metastasis[41].

Exosome-mimetic nanoparticles offer a versatile platform to disrupt tumor–immune communication. One approach involves the use of decoy nanoparticles displaying immune checkpoint receptors or antibodies on their surface[42]. These systems can sequester exosomal PD-L1 in circulation or within the tumor microenvironment, preventing engagement with PD-1 on T cells and alleviating immune suppression. By acting upstream of immune checkpoint signaling, decoy nanoparticles address a fundamental mechanism of resistance to immune checkpoint inhibitors[42].

Alternatively, exosome-mimetic nanoparticles can be designed to deliver immune-stimulatory cargo directly to antigen-presenting cells such as dendritic cells and macrophages[43]. Delivery of toll-like receptor agonists, cytokines, or immunostimulatory RNA can reprogram these cells toward pro-inflammatory, tumoricidal phenotypes. Such reprogramming enhances antigen presentation, T-cell priming, and immune-mediated clearance of disseminated tumor cells[43].

The integration of exosome-mimetic nanotechnology with immunotherapy is particularly compelling. By neutralizing exosome-driven immunosuppression, these systems enhance the efficacy and durability of immune checkpoint blockade and adoptive cell therapies[44]. Importantly, because exosome-mediated immune modulation occurs systemically, targeting this communication axis may be especially effective in preventing metastatic outgrowth rather than solely shrinking established tumors. Overall, exosome-mimetic modulation of tumor–immune signaling represents a strategic intervention point for combating metastasis and improving immunotherapeutic outcomes.

6. Translational Challenges and Safety Considerations

Despite the strong therapeutic promise of exosome-mimetic nanomedicines, several translational challenges must be addressed before clinical application can be realized[45]. Manufacturing scalability is a primary concern, as the complex architecture and biomimetic nature of these systems complicate large-scale production. Achieving batch-to-batch consistency in size, surface composition, and cargo loading is essential for reproducibility and regulatory approval. Advances in microfluidic synthesis and standardized membrane engineering may help overcome these limitations[45].

Safety considerations are particularly critical for biomimetic nanoparticles derived from or coated with cellular membranes. Residual bioactive components, such as membrane-bound proteins or nucleic acids, may trigger unintended biological responses or immunogenicity[46, 47]. Rigorous purification and characterization protocols are therefore required to ensure biosafety. Additionally, long-term stability and storage conditions must be optimized to preserve functionality without degradation or aggregation.

Pharmacokinetics and biodistribution represent additional challenges. While biomimicry enhances targeting and circulation time, excessive resemblance to endogenous exosomes may interfere with normal intercellular communication[48]. Off-target accumulation in healthy tissues could disrupt physiological signaling pathways, necessitating careful dose optimization and biodistribution studies[49].

Finally, regulatory frameworks for complex biomimetic nanomedicines are still evolving. Clear guidelines for characterization, quality control, and preclinical evaluation are needed to facilitate clinical translation. Addressing these challenges through interdisciplinary collaboration will be essential to unlock the full potential of exosome-mimetic nanotechnology in metastatic cancer therapy.

7. Conclusion and Future Perspectives

Exosome-mimetic nanoparticles represent a compelling strategy for intercepting intercellular signaling pathways that drive metastatic cancer progression. By combining the biological sophistication of natural exosomes with the controllability of synthetic nanomaterials, these systems enable precise disruption of tumor–stromal, tumor–immune, and long-range metastatic communication. While significant translational challenges remain, advances in nanofabrication, molecular engineering, and systems biology are rapidly addressing these barriers. Future integration with personalized oncology, guided by exosomal biomarkers and metastatic risk profiling, may further enhance therapeutic precision. As understanding of exosome biology deepens, exosome-mimetic nanoparticles are poised to become a central component of next-generation anti-metastatic therapies.

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