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Self-Assembling Nanotherapeutics for Precision Targeting of Cancer Metabolic Vulnerabilities

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ABSTRACT

Cancer cells reprogram their metabolism to sustain uncontrolled proliferation, survive hostile microenvironments, and evade therapeutic stress. These metabolic adaptations ranging from aerobic glycolysis and glutamine addiction to altered lipid and redox metabolism represent exploitable vulnerabilities for precision oncology. However, conventional metabolic inhibitors often suffer from poor bioavailability, systemic toxicity, and limited tumor selectivity. Self-assembling nanotherapeutics have emerged as a transformative strategy to overcome these limitations by integrating drug delivery, targeting, and therapeutic function within programmable nanoscale architectures. Through non-covalent interactions such as hydrophobic forces, hydrogen bonding, electrostatic attraction, and π–π stacking, small molecules, peptides, and polymers can spontaneously organize into functional nanostructures that respond to tumor-specific biochemical cues. This review critically examines the design principles, mechanisms, and therapeutic potential of self-assembling nanotherapeutics for targeting cancer metabolic vulnerabilities. We discuss how these systems enhance selective delivery of metabolic inhibitors, enable combinatorial and multi-pathway modulation, and exploit the unique metabolic and microenvironmental features of tumors. Current preclinical advances, emerging clinical prospects, and key translational challenges are also highlighted. Collectively, self-assembling nanotherapeutics represent a promising frontier for precision cancer metabolism–based therapies.

Keywords: cancer metabolism; self-assembly; nanotherapeutics; metabolic vulnerabilities; precision oncology

INTRODUCTION

Cancer metabolism has evolved from a descriptive hallmark of malignancy into a central pillar of targeted therapy development. Tumor cells undergo profound metabolic rewiring to support rapid proliferation, biosynthesis, and survival under conditions of hypoxia, nutrient deprivation, and immune pressure[1, 2]. The most classical example is the Warburg effect, characterized by elevated glucose uptake and preferential conversion of glucose to lactate even in the presence of oxygen. While energetically inefficient, aerobic glycolysis provides metabolic intermediates for nucleotide, amino acid, and lipid synthesis, thereby conferring a proliferative advantage. Beyond glycolysis, tumors exhibit dependency on glutamine for anaplerosis, altered lipid metabolism for membrane biogenesis and signaling, and reprogrammed mitochondrial function that supports redox balance rather than maximal ATP production[3].

These metabolic adaptations are not uniform across cancers but are shaped by oncogenic drivers, tissue of origin, and microenvironmental context. For example, MYC-driven tumors often exhibit glutamine addiction, while KRAS-mutant cancers display enhanced macropinocytosis and altered lipid utilization[4]. Hypoxic tumor regions rely heavily on glycolysis and reductive carboxylation, whereas nutrient-rich niches may support oxidative metabolism. This heterogeneity creates both opportunities and challenges for therapeutic targeting. Metabolic vulnerabilities arise when cancer cells become excessively dependent on specific pathways, enzymes, or substrates that are less critical in normal tissues[5, 6].

Despite the conceptual appeal of targeting cancer metabolism, clinical translation has been limited. Many metabolic enzymes are ubiquitously expressed, raising concerns about systemic toxicity. Additionally, metabolic plasticity allows tumors to bypass single-pathway inhibition by activating compensatory routes[7]. Poor pharmacokinetics, off-target effects, and limited tumor accumulation further constrain the efficacy of small-molecule metabolic inhibitors. These challenges underscore the need for delivery strategies that enhance tumor selectivity, enable spatial and temporal control of drug action, and support combination targeting of multiple metabolic nodes[7].

Nanotherapeutics offers a compelling solution by leveraging size, surface chemistry, and functional modularity to improve drug delivery and therapeutic index[8, 9]. In particular, self-assembling nanotherapeutics distinguish themselves by forming nanostructures directly from therapeutic components or simple building blocks, eliminating the need for complex carriers. These systems can be engineered to respond to metabolic hallmarks of tumors such as acidic pH, high glutathione levels, elevated lactate, or overexpressed enzymes thereby releasing or activating drugs selectively within cancer cells. By aligning nanotherapeutic design with cancer metabolic vulnerabilities, it becomes possible to transform metabolic liabilities into precision treatment opportunities[10, 11].

2. Principles of Self-Assembly in Nanotherapeutic Design

Self-assembly refers to the spontaneous organization of individual components into ordered structures driven by non-covalent interactions. In nanotherapeutics, self-assembly enables the formation of nanoparticles, nanofibers, micelles, or hydrogels from small molecules, peptides, lipids, or polymers without extensive chemical modification[12]. The resulting architectures are dynamic, reversible, and highly tunable, making them particularly attractive for biomedical applications.

Key driving forces include hydrophobic interactions, electrostatic attraction, hydrogen bonding, and π–π stacking. By rationally balancing these interactions, researchers can control particle size, morphology, stability, and drug-loading capacity[13]. Importantly, self-assembling systems often exhibit stimulus-responsiveness, disassembling or transforming in response to environmental cues such as pH, redox state, enzyme activity, or metabolite concentration. This feature is especially relevant in cancer, where metabolic reprogramming creates distinct biochemical gradients between tumors and normal tissues[14].

From a therapeutic perspective, self-assembly allows drugs themselves to serve as structural components of the nanomaterial. For example, amphiphilic prodrugs can self-assemble into nanoparticles with high drug content and minimal excipients[15, 16]. Peptide-based systems can incorporate targeting ligands, enzyme-cleavable sequences, or mitochondrial localization motifs within the same molecular framework. Such integration simplifies formulation and enhances functional precision.

Crucially, self-assembled nanotherapeutics can be designed to remain stable in circulation while undergoing rapid disassembly within the tumor microenvironment or intracellular compartments[17]. This conditional stability reduces premature drug release and systemic exposure, addressing a major limitation of conventional chemotherapy and metabolic inhibitors.

3. Targeting Glycolysis and Lactate Metabolism with Self-Assembled Nanostructures

Aerobic glycolysis and enhanced lactate production, commonly referred to as the Warburg effect, are hallmark metabolic features of many solid tumors. This metabolic reprogramming enables rapid ATP generation, supports biosynthetic demands, and facilitates tumor progression even under hypoxic conditions[18]. Consequently, glycolytic enzymes such as hexokinase (HK), pyruvate kinase M2 (PKM2), and lactate dehydrogenase A (LDHA), as well as lactate transporters, have emerged as attractive therapeutic targets. However, the systemic inhibition of glycolysis is often limited by toxicity in normal tissues that also rely on glucose metabolism. Self-assembling nanotherapeutics offer a promising strategy to overcome these limitations by enabling spatially controlled delivery and activation of glycolytic inhibitors within tumors[18].

Self-assembled nanostructures including amphiphilic polymers, peptide assemblies, and supramolecular micelles can encapsulate or conjugate glycolytic inhibitors while maintaining stability during circulation[19]. These systems exploit tumor-associated physicochemical cues to achieve selective accumulation and activation. Acidic pH, a direct consequence of lactate export from highly glycolytic cancer cells, serves as a particularly effective trigger. pH-sensitive self-assembling nanoparticles undergo protonation-induced conformational changes in acidic environments, resulting in disassembly or membrane destabilization that facilitates drug release. This strategy ensures that glycolytic inhibitors are preferentially released within tumor regions exhibiting high glycolytic flux[19].

Redox-responsive self-assembled systems provide an additional layer of selectivity. Elevated intracellular glutathione levels, commonly observed in hypoxic and glycolytic tumor cells, can trigger the cleavage of disulfide bonds or other redox-sensitive linkages within nanostructures[20]. Upon internalization, these assemblies disintegrate in the reductive cytosolic environment, enabling intracellular delivery of glycolytic modulators. Such redox-responsive behavior enhances therapeutic precision while minimizing off-target effects[20].

Beyond direct metabolic inhibition, targeting glycolysis using self-assembling nanotherapeutics exerts profound effects on the tumor microenvironment (TME). Lactate accumulation contributes to extracellular acidification, angiogenesis, and immune suppression by inhibiting cytotoxic T-cell and natural killer cell function[21]. By

locally suppressing lactate production or export, nanotherapeutic intervention can partially normalize the TME, alleviate immune suppression, and improve responsiveness to immunotherapies. Thus, self-assembling nanostructures targeting glycolysis not only deprive tumor cells of energy and biosynthetic intermediates but also reprogram the metabolic and immunological landscape of the tumor, offering a multifaceted therapeutic benefit[21].

4. Exploiting Glutamine and Amino Acid Dependencies

In addition to glycolysis, many cancers exhibit a strong dependence on glutamine and other amino acids to sustain proliferation and survival. Glutamine serves as a critical carbon and nitrogen source, fueling the tricarboxylic acid cycle, nucleotide biosynthesis, amino acid synthesis, and redox balance through glutathione production[22, 23]. This phenomenon, often termed "glutamine addiction," creates a metabolic vulnerability that can be therapeutically exploited. However, systemic glutamine depletion or glutaminase inhibition can adversely affect normal proliferative tissues. Self-assembling nanotherapeutics offer a targeted approach to selectively disrupt glutamine metabolism in tumors[24].

Self-assembled nanostructures can be engineered to deliver glutaminase inhibitors, amino acid–depleting enzymes, or RNA-based therapeutics that suppress key regulators of amino acid metabolism[25]. Enzyme-responsive assemblies that disassemble in the presence of elevated glutaminase activity or tumor-associated proteases enable precise activation within glutamine-dependent cancer cells. Additionally, targeting moieties that recognize glutamine transporters, such as ASCT2, can enhance selective uptake by metabolically addicted tumors[25, 26].

Peptide-based self-assembling nanostructures are particularly advantageous in targeting amino acid metabolism. Their modular design allows incorporation of amino acid–mimetic sequences that interact with transporters or metabolic enzymes. These peptides can self-organize into nanofibers or micelles that interfere with glutamine uptake or intracellular utilization[27]. Furthermore, co-assembly strategies allow simultaneous targeting of glutamine metabolism and compensatory pathways such as glycolysis or lipid oxidation, thereby reducing metabolic plasticity and therapeutic resistance[27].

The ability of self-assembling nanotherapeutics to integrate multiple metabolic inhibitors into a single platform is especially valuable in glutamine-targeted therapy, as tumors frequently activate alternative nutrient pathways in response to metabolic stress[28]. By synchronizing delivery and release, these systems provide sustained and coordinated metabolic disruption. Overall, self-assembling nanotherapeutics targeting glutamine and amino acid dependencies represent a powerful approach to exploit metabolic addictions while preserving normal tissue function.

5. Targeting Lipid Metabolism and Mitochondrial Function

Dysregulated lipid metabolism is a defining feature of aggressive and therapy-resistant cancers. Tumor cells exhibit increased fatty acid synthesis, uptake, and β-oxidation to support membrane biogenesis, energy production, and signaling[29]. Enzymes such as fatty acid synthase, stearoyl-CoA desaturase, and carnitine palmitoyltransferase 1 are frequently overexpressed, making them attractive therapeutic targets. Self-assembling nanotherapeutics provide a versatile platform to deliver lipid metabolism inhibitors with enhanced tumor selectivity[29].

Nanostructures incorporating lipophilic moieties exhibit preferential interactions with lipid-rich cellular compartments, facilitating intracellular trafficking and retention[30]. These assemblies can encapsulate inhibitors of lipid synthesis or oxidation while maintaining structural stability. Importantly, many lipid metabolic processes are closely linked to mitochondrial function, positioning mitochondria as a critical therapeutic target[30].

Mitochondria-targeted self-assembling nanotherapeutics often incorporate cationic or aromatic motifs that exploit the mitochondrial membrane potential to drive selective accumulation. Upon reaching mitochondria, these systems release metabolic inhibitors or disrupt oxidative phosphorylation, inducing energetic collapse and oxidative stress in cancer cells[31, 32]. Because normal cells typically exhibit lower mitochondrial membrane potential and metabolic flexibility, selective toxicity can be achieved.

By integrating mitochondrial targeting with lipid metabolic inhibition, self-assembling nanotherapeutics can induce profound metabolic stress that overwhelms tumor adaptive capacity. This dual targeting strategy exemplifies the modular and programmable nature of self-assembly and highlights its potential for precise metabolic intervention in cancer therapy.

6. Combinatorial and Microenvironment-Responsive Metabolic Nanotherapy

A key advantage of self-assembling nanotherapeutics lies in their capacity for combinatorial design and microenvironment responsiveness. Cancer metabolism is highly adaptable, with tumors frequently compensating for the inhibition of one pathway by upregulating alternative metabolic routes[33]. Co-assembling multiple metabolic inhibitors within a single nanostructure enables synchronized delivery and coordinated pathway suppression, reducing the likelihood of metabolic escape[33].

Self-assembled nanotherapeutics can integrate inhibitors of glycolysis, glutamine metabolism, and lipid oxidation, as well as signaling molecules that regulate metabolic adaptation. Defined stoichiometry ensures controlled ratios of each therapeutic component, enhancing reproducibility and therapeutic synergy[34]. This level of control is difficult to achieve with free drug combinations administered systemically.

Microenvironment-responsive assemblies further enhance precision by adapting to tumor heterogeneity. Hypoxia-, pH-, and enzyme-responsive systems activate selectively within specific tumor niches, enabling spatially resolved metabolic targeting. Such adaptability allows comprehensive coverage of heterogeneous tumor regions while minimizing systemic toxicity[34]. Collectively, combinatorial and microenvironment-responsive self-assembling nanotherapeutics represent a transformative approach to metabolic cancer therapy. By addressing both metabolic plasticity and spatial heterogeneity, these systems offer a rational pathway toward more effective and durable anticancer strategies.

7. Conclusion and Future Perspectives

Self-assembling nanotherapeutics offer a powerful and versatile strategy for precision targeting of cancer metabolic vulnerabilities. By integrating therapeutic function, targeting capability, and environmental responsiveness within minimalist nanoscale architectures, these systems overcome many limitations of conventional metabolic therapies. Advances in molecular design, understanding of tumor metabolism, and nanomaterial characterization have accelerated preclinical progress. However, challenges remain, including scalability, long-term safety, and translation across heterogeneous patient populations. Future efforts should focus on rational design guided by metabolic profiling, biomarker-driven patient stratification, and integration with immunotherapy and other modalities. As these challenges are addressed, self-assembling nanotherapeutics are poised to play a central role in the next generation of metabolism-based precision oncology.

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