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Nanotechnology-Based Strategies for Modulating Epigenetic Dysregulation in Cancer

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ABSTRACT

Epigenetic dysregulation is a defining feature of cancer, driving malignant transformation, tumor progression, and therapeutic resistance through reversible yet heritable alterations in gene expression. Aberrant DNA methylation, histone modifications, and chromatin remodeling disrupt normal cellular identity and enable oncogenic transcriptional programs without altering the underlying DNA sequence. Although epigenetic enzymes are druggable and several epigenetic inhibitors have reached clinical use, their therapeutic impact has been constrained by limited specificity, systemic toxicity, poor pharmacokinetics, and the inability to precisely modulate complex epigenetic networks within tumors. Nanotechnology-based strategies have emerged as powerful tools to address these limitations by enabling targeted delivery, controlled release, and combinatorial modulation of epigenetic regulators. By leveraging nanoscale materials such as liposomes, polymeric nanoparticles, inorganic nanocarriers, and self-assembled systems, epigenetic drugs can be selectively delivered to tumor cells and subcellular compartments, enhancing efficacy while minimizing off-target effects. This review provides a comprehensive overview of nanotechnology-based approaches for modulating epigenetic dysregulation in cancer. We examine the biological basis of epigenetic alterations in malignancy, discuss major nanocarrier platforms for epigenetic therapy, and highlight advances in targeting DNA methylation, histone modifications, and chromatin architecture. Current challenges, translational considerations, and future directions for precision epigenetic nanomedicine are also critically discussed.

Keywords: epigenetic therapy; nanotechnology; cancer; chromatin regulation; precision oncology

INTRODUCTION

Epigenetic regulation refers to heritable changes in gene expression that occur without alterations in the DNA sequence and are essential for normal development, cellular differentiation, and tissue homeostasis[1, 2]. In cancer, these finely tuned regulatory mechanisms become profoundly dysregulated, resulting in widespread transcriptional reprogramming that supports oncogenesis, tumor progression, immune evasion, and resistance to therapy[3–5]. Unlike genetic mutations, epigenetic alterations are potentially reversible, making them particularly attractive therapeutic targets. However, their complexity and dynamic nature necessitate innovative strategies for precise and effective intervention.

One of the most extensively studied epigenetic abnormalities in cancer is aberrant DNA methylation. Hypermethylation of promoter-associated CpG islands leads to stable silencing of tumor suppressor genes involved in cell cycle control, DNA repair, apoptosis, and differentiation[6, 7]. Concurrently, global DNA hypomethylation contributes to genomic instability, activation of oncogenes, and reactivation of transposable elements. These opposing yet coexisting methylation patterns create a permissive landscape for malignant transformation. DNA methyltransferases, which catalyze the addition of methyl groups to cytosine residues, are therefore central mediators of epigenetic dysregulation in cancer[8].

Histone modifications represent another critical layer of epigenetic control. Post-translational modifications of histone tails, including acetylation, methylation, phosphorylation, and ubiquitination, influence chromatin structure and transcriptional accessibility[9, 10]. In cancer, the balance between activating and repressive

histone marks is frequently disrupted due to altered expression or mutation of histone-modifying enzymes. Overactivity of histone deacetylases leads to chromatin compaction and transcriptional repression of tumor suppressor genes, while dysregulated histone methyltransferases and demethylases promote oncogenic gene expression programs[11, 12]. These changes are highly context-dependent and vary across tumor types and disease stages.

Chromatin remodeling complexes further contribute to epigenetic dysregulation by controlling nucleosome positioning and higher-order chromatin organization. Mutations or altered expression of chromatin remodelers can profoundly affect genome accessibility, enhancer activity, and long-range chromatin interactions[13]. Such alterations often cooperate with DNA methylation and histone modification changes to stabilize malignant transcriptional states. Importantly, epigenetic dysregulation also interacts closely with metabolic reprogramming, signaling pathways, and the tumor microenvironment, reinforcing cancer cell plasticity and adaptability[14].

From a therapeutic perspective, epigenetic enzymes are appealing targets because they are enzymatically active and pharmacologically tractable. Several inhibitors of DNA methyltransferases and histone deacetylases have been approved for hematological malignancies, demonstrating clinical proof of concept[15]. However, these agents often lack tumor specificity, affect normal cells, and produce modest responses in solid tumors. Dose-limiting toxicities, rapid clearance, and poor tumor penetration further restrict their clinical utility[15]. Additionally, epigenetic therapies can produce broad transcriptional effects, some of which may be undesirable or counterproductive.

These limitations highlight the need for strategies that enable spatial, temporal, and cell-type–specific modulation of epigenetic regulators. Nanotechnology offers unique opportunities to meet these needs by improving drug solubility, protecting labile molecules, enhancing tumor accumulation, and enabling targeted delivery to specific cell populations or subcellular compartments[16–18]. By integrating an understanding of cancer epigenetics with advances in nanoscale engineering, it becomes possible to design precision therapies that exploit the reversibility of epigenetic dysregulation while minimizing systemic harm.

2. Nanotechnology Platforms for Epigenetic Drug Delivery (500 words)

Nanotechnology-based drug delivery systems encompass a diverse range of materials engineered at the nanoscale to improve the pharmacological performance of therapeutic agents [19]. In the context of epigenetic therapy, these platforms are particularly valuable because many epigenetic drugs suffer from poor solubility, rapid degradation, and off-target toxicity[20, 21]. Nanocarriers can encapsulate, protect, and selectively deliver epigenetic modulators to tumor tissues, thereby enhancing therapeutic index.

Lipid-based nanoparticles, including liposomes and solid lipid nanoparticles, are among the most extensively studied carriers for epigenetic drugs. Their biocompatibility, structural flexibility, and ability to encapsulate both hydrophilic and hydrophobic molecules make them attractive for clinical translation[22, 23]. Liposomal formulations of histone deacetylase inhibitors and DNA methyltransferase inhibitors have demonstrated improved circulation time and reduced systemic toxicity in preclinical models.

Polymeric nanoparticles offer additional versatility through tunable size, surface chemistry, and degradation kinetics. Biodegradable polymers can be engineered to release epigenetic drugs in response to pH, redox conditions, or enzymatic activity characteristic of tumor tissues[24–26]. These features allow for controlled and sustained drug release, which is particularly important for epigenetic modulation that often requires prolonged exposure.

Inorganic nanomaterials, such as gold nanoparticles, silica nanoparticles, and magnetic nanoparticles, provide unique physicochemical properties that can be exploited for imaging-guided therapy and externally triggered drug release[27–29]. Although concerns about long-term safety remain, these platforms enable multifunctional designs that combine epigenetic modulation with diagnostics or thermal and magnetic targeting. Collectively, nanotechnology platforms form the foundation for precision epigenetic therapy by addressing the delivery-related limitations of conventional epigenetic drugs.

3. Nanotechnology-Based Targeting of DNA Methylation Pathways

Aberrant DNA methylation represents a hallmark of cancer, driving silencing of tumor suppressor genes, promoting genomic instability, and contributing to drug resistance. Therapeutic targeting of DNA methyltransferases (DNMTs) has therefore emerged as a central strategy in epigenetic oncology[30]. However, conventional DNMT inhibitors face significant challenges, including poor bioavailability, rapid degradation, and systemic toxicity, which limit their clinical efficacy. Nanotechnology-based delivery systems offer solutions to these limitations by enhancing drug stability, targeting, and intracellular bioavailability[31].

Encapsulation of DNMT inhibitors within nanoparticles protects labile molecules from enzymatic degradation and renal clearance, extending circulation time and improving accumulation in tumors via the enhanced permeability and retention (EPR) effect[32]. Lipid-based, polymeric, and inorganic nanoparticles have all been explored for this purpose, providing platforms capable of high drug loading and tunable release profiles. This passive targeting enhances drug delivery to malignant tissues while minimizing off-target effects in healthy organs[20].

Active targeting further refines therapeutic precision. Surface modification of nanoparticles with ligands such as folate, transferrin, or peptides recognizing tumor-associated receptors enables preferential uptake by cancer

cells with epigenetic dysregulation[33]. By concentrating inhibitors within tumor cells, intracellular drug levels are elevated, improving DNMT blockade while sparing normal tissues. Stimuli-responsive nanocarriers exploit tumor-specific microenvironmental cues such as acidic pH, elevated reactive oxygen species, or overexpressed enzymes to trigger selective release of DNMT inhibitors, further enhancing safety and specificity[33].

Nanotechnology also facilitates combinatorial strategies that expand the therapeutic potential of DNA methylation targeting. DNMT inhibitors can be co-delivered with chemotherapeutic agents, small-molecule epigenetic modulators, or immunotherapies within a single nanoparticle, enabling synergistic effects[34]. For example, demethylation of silenced tumor suppressor genes can increase antigen presentation, sensitizing tumors to immune checkpoint blockade. Similarly, combined delivery with histone deacetylase inhibitors or chemotherapeutics can simultaneously relieve epigenetic repression and induce cytotoxic stress, overcoming resistance mechanisms.

Beyond pharmacological synergy, nanotechnology enables spatial and temporal control over DNA methylation inhibition. Controlled release systems maintain sustained intracellular concentrations, supporting persistent epigenetic reprogramming while minimizing systemic toxicity[35]. Multimodal nanoparticles can integrate imaging agents to monitor drug delivery and pharmacodynamic effects in real time, enabling personalized adjustment of therapy. Collectively, these strategies illustrate the capacity of nanotechnology to transform DNA methylation targeting into a more precise, effective, and clinically translatable approach.

4. Nanotherapeutic Modulation of Histone Modifications

Histone modifications, including acetylation, methylation, phosphorylation, and ubiquitination, play a pivotal role in regulating chromatin accessibility and gene transcription. Dysregulation of these marks contributes to oncogenesis, tumor progression, and therapy resistance[36]. Nanotechnology-based delivery systems provide a versatile platform to improve the efficacy and specificity of histone-modifying drugs, such as histone deacetylase (HDAC) inhibitors and histone methyltransferase modulators, while overcoming challenges associated with cellular uptake and nuclear localization[37, 38].

Nanoparticles can enhance the intracellular delivery of histone-targeting agents by facilitating endocytosis and promoting endosomal escape. Peptide-functionalized nanocarriers and polymeric systems incorporating nuclear localization signals enable efficient transport of payloads into the nucleus, the primary site of histone modification[39]. Controlled and sustained release profiles allow persistent exposure, which is often necessary to induce stable changes in chromatin structure and transcriptional programs.

Multidrug encapsulation is particularly advantageous in epigenetic therapy. Nanoparticles can co-deliver HDAC inhibitors alongside modulators of histone methylation, achieving synergistic effects on chromatin accessibility and gene expression[40]. By fine-tuning the stoichiometry and release kinetics, nanocarriers provide precise control over the temporal and spatial exposure of multiple agents, maximizing therapeutic efficacy while minimizing off-target effects[41].

Beyond drug delivery, nanotechnology can facilitate tumor-specific activation. Stimuli-responsive systems, triggered by acidic pH, enzymatic activity, or redox gradients, release histone-modifying agents selectively within the tumor microenvironment or intracellular compartments[42]. This specificity reduces collateral toxicity to normal cells and enhances epigenetic reprogramming within malignant cells.

Nanotechnology-based histone modulation also enables integration with other therapeutic strategies. For instance, epigenetic priming via nanoparticle-delivered HDAC inhibitors can increase tumor immunogenicity, enhancing the effectiveness of immunotherapy[43]. Similarly, co-delivery with chemotherapeutics can synergize epigenetic reprogramming with cytotoxic stress, overcoming resistance. By refining nuclear targeting, combinatorial delivery, and microenvironmental responsiveness, nanotherapeutic systems offer a robust approach to reshape oncogenic transcriptional programs while minimizing collateral damage. Such precision epigenetic modulation exemplifies the transformative potential of nanotechnology in cancer therapy.

5. Targeting Chromatin Remodeling and Non-Coding Epigenetic Regulators

Beyond DNA methylation and histone modification, chromatin architecture is dynamically regulated by chromatin remodeling complexes and non-coding RNAs, including microRNAs (miRNAs) and long non-coding RNAs (lncRNAs)[44]. Dysregulation of these pathways contributes to aberrant gene expression, stemness, metastasis, and therapy resistance. Nanotechnology provides a robust platform for the delivery of nucleic acid–based therapeutics that target these epigenetic regulators[45].

Nanocarriers protect RNA molecules from degradation by nucleases and facilitate cellular uptake, endosomal escape, and cytosolic or nuclear localization. Lipid-based nanoparticles, polymeric systems, and exosome-mimetic vesicles have all been explored for the delivery of tumor-suppressive miRNAs or lncRNA inhibitors. By restoring the function of tumor-suppressive non-coding RNAs or silencing oncogenic RNAs, these systems can indirectly reprogram chromatin structure and gene expression[46, 47].

Targeting specificity is further enhanced through surface functionalization with tumor-recognizing ligands, allowing selective delivery to malignant cells while sparing normal tissues[47]. Nanotechnology also enables co-delivery of nucleic acids with complementary epigenetic modulators, such as DNMT or HDAC inhibitors, achieving integrated remodeling of epigenetic networks. This multi-layered strategy addresses the complexity of chromatin regulation, targeting both enzymatic and non-coding RNA-mediated pathways.

By leveraging precise delivery and protection of nucleic acids, nanotechnology expands the therapeutic landscape of epigenetic oncology, moving beyond classical enzyme inhibition to encompass broader regulatory networks that govern chromatin dynamics.

6. Combinatorial and Precision Epigenetic Nanomedicine

Epigenetic dysregulation in cancer is inherently multifactorial, involving intertwined pathways that often exhibit redundancy and compensatory feedback. Single-agent interventions frequently fail to produce durable responses, highlighting the need for combinatorial strategies. Nanotechnology enables co-encapsulation of multiple epigenetic modulators—including DNA methylation inhibitors, histone-modifying agents, and nucleic acid therapeutics—within a single delivery platform[48]. This approach ensures synchronized action at the tumor site, maintaining optimal drug ratios and enhancing therapeutic synergy.

Precision epigenetic nanomedicine further integrates tumor-specific biomarkers, microenvironmental cues, and patient stratification to guide therapeutic design. Stimuli-responsive nanocarriers can adapt to heterogeneous tumor environments, releasing payloads selectively in response to biochemical signals such as pH, enzyme activity, or hypoxia[49]. This adaptability enhances efficacy while minimizing systemic toxicity.

Integration with imaging or theranostic functions enables real-time monitoring of drug delivery and epigenetic modulation, supporting adaptive treatment strategies. Such systems can be personalized to individual patients, taking into account tumor epigenetic profiles, microenvironmental characteristics, and prior therapeutic responses[49].

By combining targeted, combinatorial, and adaptive approaches, precision epigenetic nanomedicine positions nanotechnology as a cornerstone for next-generation cancer therapy, offering the potential to overcome resistance, suppress tumor progression, and achieve durable clinical outcomes.

7. Conclusion and Future Perspectives

Nanotechnology-based strategies have transformed the landscape of epigenetic cancer therapy by addressing fundamental challenges associated with specificity, delivery, and toxicity. By enabling precise modulation of DNA methylation, histone modifications, and chromatin remodeling, nanotherapeutics unlock the full therapeutic potential of epigenetic regulation. Continued progress will depend on integrating advances in cancer epigenomics, materials science, and clinical oncology. Key priorities include improving scalability, ensuring long-term safety, and developing biomarker-driven approaches to patient selection. As these challenges are met, nanotechnology-based epigenetic modulation is poised to play a central role in precision oncology, offering durable and adaptable therapeutic solutions for diverse cancer types.

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