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2026 Volume 7, Issue 2
Cell Biology Research
ISSN: 2529-7627 (Online)

Research Progress on Nanomaterials in the Treatment of Ischemic Stroke

Jiangling Zhu[1], Bingcang Huang[2]*

[1]School of Gongli Hospital Medical Technology, University of Shanghai for Science and Technology, Shanghai 200093, China

[2]Department of Radiology Gongli Hospital of Shanghai Pudong New Area, Shanghai 200135, China.

***Corresponding author:** *Bingcang Huang, hbc01275@glhospital.com*

Abstract: The efficacy of ischemic stroke treatment is limited by a narrow therapeutic window, the blood-brain barrier, and secondary damage driven by oxidative stress and inflammatory cascades following reperfusion. Nanomaterials can enhance blood-brain barrier (BBB) penetration and increase accumulation in the ischemic penumbra through interface engineering and biomimetic modification. They can also integrate synergistic interventions such as antioxidant therapy, immune and inflammatory regulation, and vascular/neural repair, while incorporating imaging-guided delivery and microenvironment-responsive release to improve therapeutic precision. This review focuses on the preparation strategies and targeted design of nanomedicines for stroke. It summarizes the structural characteristics and mechanisms of action of liposomes, polymeric nanoparticles, inorganic/hybrid systems, and cell-mimetic delivery platforms. It discusses recent research progress regarding their roles in scavenging reactive oxygen species (ROS), regulating microglial polarization, inhibiting inflammatory amplification loops, and promoting neurovascular repair, and concludes by outlining key challenges in large-scale production, biosafety, and clinical translation.

Keywords: Ischemic stroke; Nanomedicine; Blood-brain barrier; Reactive oxygen species; Neurovascular unit

Online publication:

1. Introduction

Ischemic stroke begins with energy metabolism disorders and excitotoxicity triggered by acute ischemia and hypoxia, which further amplify oxidative stress and inflammatory cascades following reperfusion, ultimately leading to damage to the neurovascular unit (NVU)[1]. Current standard treatments remain largely constrained by safety considerations such as the therapeutic time window and the risk of hemorrhage. For example, the effective time window for intravenous thrombolysis is typically within approximately 4.5 hours of symptom onset. With the support of imaging screening, the applicable time window for endovascular therapy can be further expanded; however, the number of eligible patients remains limited, and risks such as hemorrhagic transformation still require rigorous assessment. Meanwhile, although the mechanisms of traditional neuroprotective strategies are well-defined, they often struggle to be consistently translated into standard clinical protocols due to blood-brain barrier (BBB) limitations and insufficient exposure to the target area[2].

Nano-delivery systems offer an engineerable strategy to partially overcome these limitations. Through surface ligand

modification or biomimetic coating, they can achieve receptor-mediated transport across the BBB[3], thereby enhancing drug exposure in the ischemic penumbra and improving pharmacokinetic behavior. In addition, nanoplatforms support the co-loading of multiple agents and controlled release, and can incorporate imaging modules such as magnetic resonance imaging (MRI) or fluorescence for treatment guidance and efficacy monitoring. Responsive designs triggered by reactive oxygen species (ROS), pH, or enzymes[4] further enable selective drug release within the lesion microenvironment, thereby reducing peripheral exposure and systemic toxicity and providing a materials basis for combining standard therapy with precision neuroprotection. Overall, research on nanotherapy for ischemic stroke has evolved beyond single delivery functions toward a broader framework that integrates engineering design, mechanistic regulation, and translational application. As shown in **Figure 1**, this field can be conceptualized as a systematic framework encompassing material development, lesion-targeted delivery, mechanism-based intervention, and translational evaluation. Despite these advances, several critical barriers still limit the further development of nanomedicine for ischemic stroke. First, the *in vivo* transport process of nanoplatforms across the BBB remains insufficiently characterized, and the efficiency and reproducibility of lesion-specific accumulation are still difficult to control precisely. Second, many studies focus predominantly on material performance itself, while the integrated relationship among biodistribution, mechanistic intervention, and functional outcome improvement has not yet been fully established. Third, substantial discrepancies persist between experimental models and real-world clinical scenarios, which weakens the translational value of many preclinical findings. Therefore, beyond demonstrating therapeutic potential, current research should move toward a more systematic framework integrating engineering design, mechanistic validation, and translational feasibility.

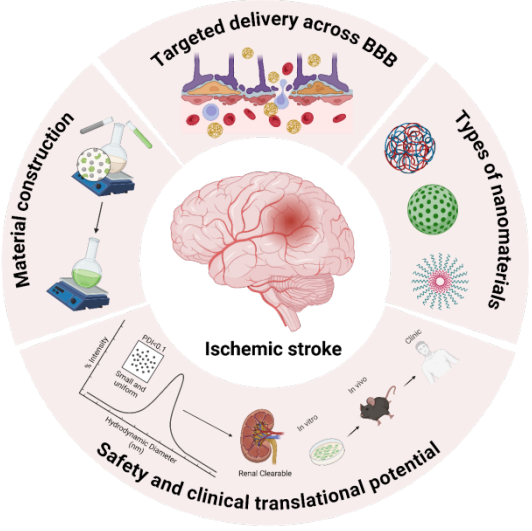

Figure 1. Schematic illustration of the engineering and translational framework of nanomedicine for ischemic stroke.

2. Preparation and Engineering Strategies for Nanomaterials

2.1. Preparation Routes: Organic Nanocarriers and Inorganic/Hybrid Systems

The translational potential of stroke nanomedicines primarily depends on the preparation routes and process scalability of the material systems. Organic nanocarriers (such as liposomes, polymeric micelles, and polymeric nanoparticles) are mainly prepared using bottom-up strategies such as self-assembly, emulsification-solvent evaporation, or nanoprecipitation[5], with well-established process windows and the potential for large-scale production and quality control. Their core-shell hydrophilic-hydrophobic structure facilitates the co-loading of small-molecule drugs, nucleic acids, and imaging probes[6]. In addition, the abundance of surface functional groups, such as hydroxyl, carboxyl, and amino groups, allows covalent coupling of polyethylene glycol (PEG) and targeting ligands, including transferrin, RGD peptides, and antibodies[7]. These features improve drug solubility and plasma half-life while reducing recognition and clearance by the

mononuclear phagocyte system (enhance blood-brain barrier (BBB) permeability and increase accumulation), thereby laying the foundation for BBB transport and targeted accumulation in the ischemic penumbra.

Inorganic and hybrid nanomaterials (such as metal oxides, sulfides, carbon-based nanostructures, and metal-organic framework derivatives) are typically synthesized via hydrothermal/solvothermal methods, sol-gel processes, or self-assembly assisted by hard/soft templates. These materials possess excellent physicochemical stability and are easily functionalized to integrate diagnostic and therapeutic capabilities. Taking cerium dioxide (CeO_2) nanoenzymes as an example, the Ce^{3+}/Ce^{4+} redox cycle on their surface can mimic the multi-enzyme activities of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), continuously scavenging ROS and effectively alleviating the oxidative stress cascade in ischemia-reperfusion injury[8]. Magnetic nanoparticles such as magnetite (Fe_3O_4) can serve as T_2-weighted MRI contrast agents, enabling real-time visual monitoring and navigation during treatment and providing technical support for precision medicine[9].

However, different nanomaterial systems also exhibit distinct limitations and engineering trade-offs. Organic nanocarriers generally possess favorable biocompatibility and formulation flexibility, but they may suffer from limited structural stability, premature drug leakage, and insufficient loading efficiency for certain cargos. In contrast, inorganic nanomaterials often show superior physicochemical stability, catalytic activity, and imaging capability, yet their long-term biodegradation, metabolic fate, and potential tissue accumulation remain important concerns. Hybrid systems can partially integrate the advantages of both categories, but their increased compositional and structural complexity may further complicate manufacturing reproducibility, quality control, and regulatory evaluation. Therefore, rational material selection for ischemic stroke therapy should balance therapeutic performance with manufacturability and translational safety.

2.2. Surface Modification and Brain-Targeting Design

Nanocarriers are typically first modified through strategies such as PEGylation to prolong circulation time and reduce immune recognition[10]. Subsequently, targeting ligands are conjugated to their surface to enable receptor-mediated transport across the BBB[11]. Common targets include the transferrin receptor (TfR), low-density lipoprotein receptor-related protein-1 (LRP-1), and glucose transporter-1 (GLUT-1)[11], and corresponding modifications with transferrin, Angiopep-2 (an LRP-1 ligand), or glucose moieties can be employed to enhance brain delivery efficiency[10]. Furthermore, biomimetic cell membrane coatings (such as platelet membranes or stem cell membranes) can simultaneously enhance immunocamouflage and lesion adhesion/homing capabilities, thereby improving accumulation efficiency in the ischemic penumbra and treatment consistency[12, 13].

2.3. Stimulus Response and Controlled Release

By incorporating pH-sensitive, ROS-responsive, enzyme-triggered, or externally triggered (*e.g.*, by magnetic fields, ultrasound, or near-infrared irradiation) structural units, responsive delivery systems with controlled release capabilities can be constructed[14]. The microenvironment of stroke lesions typically exhibits mild acidification, elevated ROS levels, and upregulation of inflammation-related proteases, providing conditions for lesion-triggered selective drug release[4]. Under local stimulation, nanocarriers undergo disintegration, pore opening, or bond cleavage, thereby increasing effective drug concentrations within the lesion while reducing systemic exposure and toxic side effects.

This type of responsive design is particularly valuable in ischemic stroke because the pathological microenvironment evolves dynamically over time. In the hyperacute stage, ROS bursts, acidosis, and vascular dysfunction are more prominent, whereas in later stages inflammatory remodeling and tissue repair gradually become dominant. Accordingly, stimulus-responsive systems may provide not only spatial selectivity but also a certain degree of temporal adaptability, thereby improving the precision of drug release and enhancing the overall therapeutic index.

3. Summary of Research Progress by Material Type and Therapeutic Mechanism

3.1. Delivery-Oriented Nanoplatforms: Enhancing Brain Delivery and Lesion Accumulation

The core objective of delivery-oriented nanoplatforms is to improve BBB penetration efficiency and enhance drug exposure at the lesion site, thereby amplifying the efficacy and controllability of existing drugs. Existing studies predominantly utilize liposomes, polymeric carriers, exosomes, and inorganic nanosystems to deliver thrombolytic adjuvants, antioxidant/anti-inflammatory molecules, neurotrophic factors, and nucleic acid therapeutics, including small interfering RNA (siRNA), microRNA (miRNA), and antisense oligonucleotides (ASO), among others.[4,15-17] When evaluating delivery efficacy, in addition to assessing intracerebral distribution and retention time, it is necessary to quantify BBB penetration efficiency and specific accumulation in the ischemic penumbra, and to comprehensively evaluate the balance between enhanced therapeutic efficacy and the risk of hemorrhagic transformation.[4,15,16] The overall trend is to couple targeted accumulation with microenvironment-triggered release, thereby enhancing therapeutic efficacy while reducing peripheral exposure and systemic toxicity.[4,15]

3.2. Antioxidant and Nanozyme Strategies: Inhibiting ROS-Driven Cascading Damage

The surge in ROS during the reperfusion phase is often considered a key driver of secondary injury, capable of inducing mitochondrial dysfunction, lipid peroxidation, and amplifying cell death processes[18-20]. Nanozyme systems can mimic the activity of antioxidant enzymes such as SOD, CAT, and GPx, achieving sustained scavenging of multiple ROS through catalytic cycles, thereby overcoming the limitations of short half-lives and insufficient local exposure associated with small-molecule antioxidants[19]. Their advantages primarily lie in their broad-spectrum scavenging capacity for multiple ROS, sustained effects at low doses, and the ability of certain materials to maintain activity through redox cycling[19]. Importantly, ROS overproduction in ischemia-reperfusion injury is not merely a direct source of oxidative damage, but also an upstream driver of multiple downstream pathological events, including mitochondrial dysfunction, blood-brain barrier disruption, inflammatory amplification, and various forms of regulated cell death. Therefore, nanozyme-mediated ROS modulation may exert protective effects beyond simple antioxidant scavenging by reshaping the local redox microenvironment and interrupting multiple self-amplifying injury cascades. This broader mechanistic significance further highlights the therapeutic value of catalytic nanomaterials in stroke intervention. Existing studies suggest that nanozyme intervention can reduce infarct size, improve indicators of mitochondrial damage, and promote neurological recovery[18,19].

3.3. Immune-Reprogramming and Repair-Oriented Strategies for Neurovascular Unit Reconstruction

Post-stroke neuroinflammation is typically characterized by pro-inflammatory polarization of microglia and peripheral macrophages, activation of the NOD-like receptor family pyrin domain containing 3 (NLRP3) inflammasome, and sustained release of inflammatory mediators, thereby forming a secondary injury amplification loop[18]. Nanotechnology-based strategies can reprogram the inflammatory microenvironment through multi-pathway regulation, including promotion of anti-inflammatory phenotypic conversion, inhibition of key inflammasome signaling nodes, and coupling of ROS scavenging with inflammation suppression to disrupt positive feedback loops[18,21,22]. In addition, nucleic acid delivery strategies, such as those based on specific miRNAs or siRNAs, provide an expandable approach for fine regulation of inflammatory signaling. However, their delivery consistency, off-target effects, and long-term safety still require systematic evaluation[22,23].

In the subacute phase, angiogenesis and neuroplasticity become increasingly important structural foundations for functional recovery. Nanomaterials can promote endothelial cell migration and microvascular reconstruction by delivering pro-angiogenic and chemotactic signals in combination with local scaffolding systems such as scaffolds or hydrogels[24], thereby improving microcirculatory perfusion in the ischemic penumbra[22,23]. Holistic protective strategies targeting the NVU emphasize the synergistic maintenance of the homeostasis of neurons, glial cells, pericytes, and the basement membrane; sequential or combined delivery of neurotrophic and vascular repair signals is a common design

approach[23]. Further integration of MRI or near-infrared probes into a diagnostic-therapeutic integrated platform facilitates dynamic monitoring of perfusion and tissue remodeling processes, providing imaging evidence for personalized treatment adjustments[22]. Notably, the therapeutic priorities of ischemic stroke are stage-dependent. In the acute phase, suppressing oxidative stress, limiting inflammatory expansion, and preserving BBB integrity are central goals for salvaging the ischemic penumbra. In the subacute and chronic phases, however, promoting angiogenesis, synaptic remodeling, axonal regeneration, and neurovascular unit reconstruction becomes increasingly important for long-term functional recovery. Therefore, future nanoplatform design should move toward stage-specific and mechanism-coordinated intervention strategies rather than relying on a single static treatment paradigm.

4. Common Challenges and Evaluation Frameworks

4.1. Animal Models and Endpoint Assessment

Preclinical research on nanomedicines for cerebral ischemia often employs models such as middle cerebral artery occlusion (MCAO)[16,25,26], and generally requires separate endpoint assessments for the acute and recovery phases. The acute phase (approximately 24-72 h) primarily focuses on safety outcomes such as neurological deficits, infarct burden, and hemorrhagic transformation[16,25]; whereas the recovery phase, often extending over several weeks, places greater emphasis on long-term behavioral performance and neuroplasticity-related outcomes[25,26]. At the mechanistic level, it is recommended to concurrently evaluate key pathway indicators such as inflammatory responses, oxidative stress, and BBB integrity[16,26-28], thereby establishing a chain of evidence where distribution, mechanisms, and outcomes mutually corroborate one another. Nevertheless, the absence of unified evaluation criteria remains a major obstacle in this field. Differences in animal species, modeling methods, administration timing, dosing regimens, and endpoint selection make it difficult to directly compare outcomes across studies. In addition, many reports emphasize short-term infarct reduction while providing insufficient data on long-term behavioral recovery, biosafety, and reproducibility. Establishing more standardized and clinically relevant evaluation systems will therefore be essential for improving the reliability and translational value of preclinical stroke nanomedicine research.

4.2. Safety and Translational Feasibility

Different material systems vary in particle size distribution, surface charge, degradation products, and batch-to-batch consistency, and these differences may substantially influence *in vivo* behavior, immunocompatibility, and large-scale manufacturability. Long-circulating nanomaterials may accumulate in the MPS, such as the liver and spleen, *in vivo*; their absorption, distribution, metabolism, and potential chronic toxicity still require systematic evaluation. Although nanodelivery systems can enhance intracerebral distribution, their *in vivo* behavior, metabolic fate, and long-term safety remain issues that must be resolved before clinical translation.[16,25,26] Beyond biological safety, translational feasibility also depends heavily on formulation reproducibility, storage stability, sterilization compatibility, and the robustness of large-scale production processes. For stroke, where treatment decisions are highly time-sensitive, nanomedicine products must not only be effective but also operationally compatible with emergency clinical workflows. As a result, future development should place greater emphasis on simplifying formulation design, improving batch consistency, and aligning product characteristics with realistic clinical implementation requirements.

5. Future Prospects

To advance the clinical translation of nanomaterials for stroke treatment, material design should shift from a focus on functional stacking to an engineering-driven approach centered on manufacturability, biodegradability, and verifiability. Priority should be given to biodegradable or clearable systems to reduce formulation complexity and mitigate immune-

related risks. In terms of clinical strategy, a more feasible approach is to complement standard treatments such as thrombolysis and thrombectomy by enhancing neuroprotective effects, increasing intra-lesional exposure, and reducing systemic risks. Future efforts should establish a framework for staged dosing and stratified patient populations: during the acute phase, the focus should be on antioxidant, anti-inflammatory, and vascular protection to salvage the penumbra; during the recovery phase, the emphasis should shift to regenerative repair and plasticity reconstruction to support long-term functional recovery. In addition, the integration of advanced imaging, artificial intelligence, and precision medicine concepts may further promote the optimization of stroke nanotherapy. Imaging-guided systems can help monitor drug distribution and lesion evolution in real time, while data-driven approaches may improve patient stratification and support the rational matching of therapeutic strategies to disease stage and lesion characteristics. Importantly, future nanomedicine platforms should be designed as complements to existing reperfusion therapies, such as thrombolysis and thrombectomy, rather than as isolated interventions. Only by embedding nanotherapeutic strategies into real clinical pathways can their translational value be maximized.

6. Conclusion

In summary, nanomaterials have expanded the therapeutic framework of ischemic stroke from conventional drug delivery toward integrated intervention involving BBB targeting, redox regulation, inflammatory modulation, and neurovascular repair. Material preparation and engineering strategies play central roles in determining *in vivo* distribution, pharmacokinetic behavior, and therapeutic benefit within the limited treatment window. However, the major barriers to translation lie not merely in adding functional modules, but in achieving biosafety, manufacturing scalability, reproducibility, and compatibility with real clinical workflows. Guided by clearly defined clinical scenarios, future material systems should be optimized around deliverability, controlled release, evaluability, and manufacturability to facilitate the translation of mechanistic advances into clinically meaningful benefit.

Funding

This work was supported by the National Natural Science Foundation of China (Project No.: 82372029, 22205133), Discipline Construction of Pudong New Area Health Commission (Project No.: PWZxk2022-03), Shanghai Pudong New District Health Committee Health Industry Special Project (Project No.: PW2024E-02) and The Investigator-initiated Trial Program of Shanghai Pudong New Area Health Commission (the Cohort Study Program) (Project No.: 2025-PWDL-24).

Disclosure statement

The author declares no conflict of interest.

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