



Biomimetic Nanotechnology Integrating Targeted Delivery and Immune Regulation in Stroke Therapy

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Ischemic stroke therapy has long been dominated by strategies aimed at restoring cerebral blood flow. Yet, accumulating evidence suggests that neuronal survival and functional recovery depend not only on reperfusion, but also on the resolution of post-ischemic immune dysregulation. This study (Chen *et al.*, *Prog Biochem Biophys*, 2026, **53**(3): 697-710. DOI:10.3724/j.pibb.2025.0541) advances this emerging paradigm by proposing a therapeutic strategy that integrates lesion-specific delivery with active modulation of the inflammatory microenvironment.

The authors employed a biomimetic nanotechnology approach, utilizing platelet membrane camouflage to endow lipid nanoparticles with intrinsic vascular injury-recognition properties. This design reflects a broader conceptual shift in nanomedicine—from passive accumulation to biologically instructed targeting.

By incorporating transforming growth factor- β 1, a cytokine with well-recognized but highly context-dependent immunoregulatory functions, the platform attempts to intervene in the complex cellular cross-talk that shapes the post-stroke neuroimmune landscape. Rather than functioning solely as a drug carrier, the system is positioned as a dynamic regulator of injury-associated immune responses. Emerging research increasingly recognizes that neurological recovery is tightly coupled to coordinated immune reprogramming involving microglia, infiltrating macrophages, and glial responses. By coupling biomimetic targeting with cytokine-mediated immunoregulation, this conceptual integration moves stroke nanotherapy beyond simple neuroprotection toward microenvironmental rebalancing. Despite its broad biological significance, the precise mechanisms through which exogenously delivered TGF- β influences immune responses within the ischemic neurovascular environment remain insufficiently understood. The interaction between nanoparticle-mediated cytokine delivery and endogenous signaling circuitry represents a critical unresolved question that will shape the future development of immunomodulatory stroke therapies.

This work illustrates an emerging shift in

nanotherapeutic design philosophy. Rather than viewing nanoparticles solely as passive drug carriers, there is increasing recognition that such platforms may function as biological modulators capable of influencing cellular communication networks. Stroke pathology, characterized by tightly coupled vascular, immune, and neural responses, represents a particularly relevant context for this integrative therapeutic concept. Nonetheless, achieving precise control over complex signaling systems poses substantial translational challenges, including pathway specificity, dosing stability, and long-term systemic safety.

Through its experimental findings, the study highlights a broader conceptual shift in stroke nanomedicine. Rather than designing nanoparticles solely to enhance drug accumulation, the authors position nanotherapeutics as tools capable of engaging in dynamic cellular communication networks. This perspective aligns with the emerging view that effective neuroprotective strategies must address the evolving interplay between vascular injury, immune activation, and neural tissue remodeling. Nonetheless, achieving precise control over complex signaling systems poses substantial translational challenges, including pathway specificity, dosing stability, and long-term systemic safety.

Taken together, the study highlights the promise of biologically inspired nanotechnology as a tool for addressing the multifactorial nature of ischemic brain injury. At the same time, it underscores the importance of mechanistic clarity in cytokine-based immune interventions. Future progress in this field will likely depend on integrating biomimetic delivery strategies with systems-level understanding of neuroimmune regulation, ultimately guiding the development of precision therapies for stroke and related neuroinflammatory disorders.

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