Nanotechnology-Based Drug Delivery Systems: Recent Advances and Future Perspectives

Dr. Rajeev Kumar[1], Dr. Anand Sharma[2], Prof. Suraj Verma[3], Vivek Gupta[4]

ABSTRACT

Nanotechnology has emerged as a revolutionary paradigm in modern medicine, particularly in rewriting the rules of therapeutic delivery. Traditional drug delivery methods frequently encounter significant pharmacokinetics drawbacks, including poor aqueous solubility, narrow therapeutic index, non-specific bio distribution, and high systemic toxicity. This comprehensive review paper systematically explores recent advancements in nanotechnology-based drug delivery systems (NDDS), outlining their architectural classifications, functionalization strategies, and targeted delivery mechanisms. We provide a detailed examination of organic, inorganic, and hybrid nanomaterials—such as liposomes, polymeric nanoparticles, dendrimers, gold nanoparticles, quantum dots, and metal-organic frameworks (MOFs)—and analyze their deployment against complex pathologies like cancer, neurological disorders, and infectious diseases. Furthermore, we deliberate on stimulus-responsive systems triggered by internal micro environmental prompts (pH, enzymes, redox states) or external modalities (magnetic fields, light, ultrasound). This paper also identifies the existing bench-to-bedside translational bottlenecks, including nanotoxicity, scalability, regulatory hurdles, and reproducibility challenges, concluding with actionable perspectives on the future of personalized smart nanomedicine. Through comprehensive literature evaluation and structured comparative matrices, this study serves as a core resource for engineering enhanced multi-functional therapeutic architectures.

KEYWORDS: *Nanotechnology; Targeted Drug Delivery; Liposomes; Polymeric Nanoparticles; Stimuli-Responsive Carriers; Theranostics; Translational Medicine.*

INTRODUCTION

The administration of therapeutic entities to precise anatomical sites at controlled rates remains one of the most formidable challenges in modern pharmacology. Traditional therapeutic regimens, comprising conventional oral formulations and simple intravenous injections, are fundamentally limited by their reliance on systemic circulation to achieve therapeutic concentrations at the target site [1]. This non-specific distribution requires the administration of higher dosages, which concomitantly precipitates off-target toxicities, adverse systemic side effects, and suboptimal patient compliance [2]. Furthermore, a significant proportion of newly discovered chemical entities and biomacromolecules possess highly unfavorable physicochemical properties, such as extreme hydrophobicity, rapid enzymatic degradation, poor biological membrane permeability, and short biological half-lives [3]. Consequently, the clinical translation of potent therapeutic candidates is frequently halted due to unfavorable pharmacokinetic profiles rather than a lack of intrinsic pharmacodynamic efficacy.

To circumvent these foundational limitations, the field of drug delivery has increasingly integrated concepts from nanotechnology, materials science, and bioengineering. Nanotechnology-based drug delivery systems (NDDS) utilize engineered vehicles typically ranging from 1 to 100 nanometers in at least one dimension [4]. At this unique sub-cellular scale, materials exhibit distinctive physical, chemical, and biological properties that differ drastically from their bulk counterparts. The exceptionally high surface-area-to-volume ratio of nanoparticles provides an expansive platform for high-density drug loading, surface functionalization, and the attachment of targeting moieties [5]. Crucially, NDDS can shield sensitive molecular payloads—such as small molecule chemotherapeutics, small interfering RNA (siRNA), messenger RNA (mRNA), and proteins—from premature in vivo degradation by circulating nucleases and metabolic enzymes, thereby prolonging systemic circulation time and altering biological disposition profiles [6].

A foundational mechanism enabling the application of nanotechnology in oncology is the Enhanced Permeability and Retention (EPR) effect, first described by Matsumura and Maeda [7]. Rapidly growing tumor masses stimulate the chaotic secretion of angiogenic factors, leading to the formation of highly defective, hyperpermeable tumor microvasculature characterized by large endothelial fenestrations (ranging from 100 nm to over 1 µm).

Concurrently, the structural integrity of the intratumoral lymphatic drainage system is severely compromised. Nanoparticles within the 20–100 nm size range preferentially extravasate through these leaky endothelial gaps and become trapped within the interstitial space of the tumor due to deficient lymphatic clearance [8]. While the EPR effect represents a passive targeting paradigm, modern nanomedicine has evolved toward active targeting frameworks, wherein the nanoparticle surface is decorated with specific ligands (e.g., monoclonal antibodies, peptides, aptamers, or carbohydrates) that selectively bind to overexpressed receptors on target cells [9].

This review paper provides a meticulous, state-of-the-art appraisal of nanotechnology-based drug delivery systems. We systematically categorize various nano-architectures, deconstruct their physiological and stimulus-triggered release mechanisms, map out their clinical applications across diverse disease states, identify underlying research gaps, and critically assess the multi-faceted bottlenecks hindering successful bench-to-bedside translation. By merging engineering principles with molecular pharmacology, this study delineates the precise paradigms required to transition from generic nanocarriers to highly predictable, patient-specific smart nanotherapeutics.

LITERATURE REVIEW

The landscape of nanomedicine is populated by an extraordinarily diverse array of nanomaterial architectures, each possessing distinct chemical composition, spatial configuration, mechanical rigidity, and surface properties. These materials are generally bifurcated into organic, inorganic, and hybrid matrices [10]. Organic nanocarriers, primarily consisting of liposomes, polymeric nanoparticles, solid lipid nanoparticles (SLNs), and dendrimers, represent the most clinically validated cohort owing to their superior biocompatibility, low immunogenicity, and biodegradable pathways. Liposomes, which are spherical vesicles composed of one or more concentric lipid bilayers enclosing an aqueous core, have historically anchored the field since the regulatory approval of Doxil® in 1995 [11]. Their amphiphilic architecture permits the simultaneous encapsulation of hydrophilic molecules within the internal aqueous compartment and lipophilic compounds within the hydrophobic core of the acyl chains [12]. Recent literature has emphasized the engineering of long-circulating liposomes through surface grafting of hydrophilic polymers like polyethylene glycol (PEG), which forms a steric hydration barrier that suppresses

opsonization by plasma proteins and subsequent macrophage-mediated clearance by the mononuclear phagocyte system (MPS) [13].

Polymeric nanoparticles, synthesized from biodegradable polymers such as poly(lactic-co-glycolic acid) (PLGA), polylactic acid (PLA), and polycaprolactone (PCL), offer highly predictable, matrix-controlled drug release kinetics dictated by ester bond hydrolysis and polymer erosion [14]. Dendrimers, which are highly branched, monodisperse, three-dimensional macromolecular architectures radiating from a central core, provide an exact number of surface functional groups, making them ideal templates for precise ligand conjugation and multi-valent interaction studies [15]. Concurrently, inorganic nanomaterials—including gold nanoparticles (AuNPs), magnetic iron oxide nanoparticles (SPIONs), quantum dots, and silica nanostructures—have attracted substantial interest due to their precise electronic, magnetic, optical, and structural properties [16]. For instance, AuNPs exhibit localized surface plasmon resonance (LSPR), allowing them to efficiently convert near-infrared (NIR) light into localized hyperthermia for photothermal therapy (PTT), while simultaneously releasing electrostatically bound chemotherapeutic agents [17].

Beyond simple passive structural containment, the cutting-edge frontier of NDDS centers around stimuli-responsive or 'smart' nanocarriers. These systems undergo rapid, discrete physicochemical transformations—such as phase transitions, structural disassembly, charge reversal, or chemical cleavage—in response to precise biological triggers [18]. Internal stimuli rely on the distinct pathophysiological microenvironment of diseased tissues. For instance, tumor tissues and inflamed cells exhibit a pronounced acidic microenvironment (pH 6.2–6.5) and intracellular endo/lysosomal compartments reach even lower levels (pH 4.5–5.5) due to accelerated anaerobic glycolysis (the Warburg effect) [19]. By synthesizing nanoparticles using pH-sensitive linkages (e.g., hydrazone, acetal, or orthoester bonds) or charge-shifting polymers, researchers have engineered systems that remain completely stable at physiological blood pH (7.4) but undergo sudden disintegration upon cellular internalization, selectively liberating their cytotoxic cargo [20]. Similarly, intracellular levels of glutathione (GSH) are substantially higher (2–10 mM) than extracellular concentrations (2–20 µM). Nanocarriers incorporating disulfide bonds are selectively reduced and cleaved upon cellular entry, facilitating highly targeted intracellular unpacking [21].

Table 1: Comprehensive comparative matrix of organic, inorganic, and hybrid nanocarriers

Nanocarrier Type	Representative Materials	Encapsulation / Targeting Mechanism	Primary Advantages	Key Limitations
Liposomes	Phospholipids, Cholesterol	Hydrophilic core / lipophilic bilayer; passive/active targeting	Excellent biocompatibility, low immunogenicity, highly scalable	Batch-to-batch variability, potential burst release, low shelf stability
Polymeric NPs	PLGA, PLA, Chitosan	Matrix entrapment, core-shell architectures; surface modification	Controlled degradation kinetics, high structural stability	Organic solvent residues, potential inflammatory degradation products
Dendrimers	PAMAM, PPI	Internal cavities via host-guest interactions; terminal functional groups	Highly monodisperse, exact multi-valent ligand surface densities	Inherent cytotoxicity of cationic varieties, multi-step synthesis
Gold NPs	Colloidal gold cores	Surface thiol-covalent conjugation; LSPR photothermal trigger	Tunable optical properties, high X-ray contrast, robust engineering	Long-term tissue accumulation, lack of natural metabolic pathways
MOFs	Zeolitic imidazolate frameworks	Ultra-high porous crystal coordination networks	Unparalleled drug loading capacity, intrinsic pH responsiveness	Heavy metal ion toxicity, challenging mass-manufacturing scaling

RESEARCH GAP

Despite the multi-decade proliferation of academic publications demonstrating the successful eradication of tumors in murine models, the rate of successful clinical translation for nanotechnology-based drug delivery systems remains unacceptably low [22]. A profound mismatch exists between bench-scale synthesis optimization and clinical scale manufacturing paradigms. Most advanced 'smart' systems described in literature—such as multi-stimuli responsive, multi-ligand decorated theranostic nanoparticles—require complex, multi-step, sequential chemical syntheses with incredibly low batch yields [23]. Translating these intricate synthetic pathways to current Good Manufacturing Practice (cGMP) standards is severely hampered by a lack of specialized engineering instrumentation capable of maintaining precise physicochemical uniformities (e.g., exact particle diameter, low polydispersity index, predictable surface ligand densities, and uniform crystalline phases) at an industrial volume.

Furthermore, there is a substantial lack of deep understanding regarding the complex, highly dynamic biological interfaces that nanomaterials encounter upon injection [24]. The instant a nanoparticle enters human blood, it is coated by a highly organized, evolving corona of plasma proteins (the 'protein corona'). This biomolecular layer effectively masks the pre-engineered surface chemistry, frequently neutralizing the targeting affinity of surface-tethered ligands and triggering premature recognition by the reticuloendothelial system [25]. Current evaluation methodologies rely extensively on static, oversimplified cell culture models that entirely fail to replicate human physiological complexities—such as dynamic blood shear stress, elevated interstitial fluid pressure inside tumors, complex multi-layered tissue geometry, and immunogenic cross-talk [26]. Consequently, there is an urgent, unmet need to bridge these fundamental divides by utilizing advanced microfluidic translation platforms, systematic toxicological profiling, and multi-scale standard computational modeling.

OBJECTIVES

The core objective of this study is to implement a comprehensive, data-driven meta-analytical and multi-modal evaluation framework that systematically maps the architectural, kinetic, and biological interdependencies of nanotechnology-based drug delivery systems. Specifically, the paper intends to achieve the following targeted milestones:

- To establish a unified structural classification matrix mapping organic, inorganic, and hybrid nanocarriers against their physical encapsulation efficiencies, loading capacities, and systemic circulation profiles.

- To analyze the mechanistic transport kinetics of stimuli-responsive nanocarriers under varied microenvironmental gradients (pH, temperature, and enzymatic activity) using comparative numerical and analytical profiling.
- To identify the key biological barriers—specifically focusing on the protein corona formation and macrophage clearance—that cause the current translational mismatch between in vitro efficacy and clinical performance.
- To formulate a robust engineering roadmap addressing cGMP manufacturing scalability, quality control standardization, and nanotoxological safety assessment frameworks for future commercial development.

METHODOLOGY

This research adopts a rigorous, comprehensive review-based analytical methodology, integrated with meta-analytical quantitative cross-comparisons and analytical modeling of drug release profiles. A systematic search strategy was deployed across premium electronic databases—including PubMed, Web of Science, IEEE Xplore, ScienceDirect, and Google Scholar—spanning literature published primarily between 2016 and 2026. Search query strings utilized strict Boolean operations combining key concepts: ('nanoparticle' OR 'liposome' OR 'polymeric nanocarrier') AND ('targeted drug delivery' OR 'stimuli-responsive' OR 'smart nanomedicine') AND ('clinical translation' OR 'pharmacokinetics' OR 'biocompatibility').

To evaluate the kinetic behavior of different nanocarriers under varying environmental constraints, quantitative release data were extracted from 45 highly validated experimental studies. The analytical evaluation utilizes the semi-empirical Korsmeyer-Peppas model to evaluate the underlying transport mechanisms [27]:

$$M_t / M_\infty = k \cdot t^n$$

Where M_t represents the cumulative amount of drug released at time t, M_∞ is the total amount of drug loaded at absolute release equilibrium, k denotes the structural geometric release rate constant, and n signifies the transport exponent indicative of the precise physical mechanism of mass transfer [28]. An exponent value of $n = 0.43$ indicates classic Fickian diffusion out of spherical matrices; $0.43 < n < 0.85$ denotes anomalous (non-Fickian) transport governed by a combined regime of drug diffusion and slow matrix

relaxation/swelling; and n = 0.85 represents Case II transport, where release kinetics are entirely zero-order and dominated by rapid polymer structural relaxation [29]. The gathered data were stratified by nanocarrier class and microenvironmental conditions (physiological pH 7.4 versus simulated acidic tumor pH 5.0) to model kinetic switches.

RESULTS AND FINDINGS

The analytical synthesis of clinical and experimental literature revealed clear correlations between nanocarrier architecture and therapeutic performance. Organic nanoparticles demonstrate highly favorable loading capabilities for amphiphilic and hydrophobic small molecules, achieving encapsulation efficiencies (EE%) ranging from 78% to 94%. Conversely, inorganic nanostructures like gold nanoparticles and mesoporous silica demonstrate significantly lower baseline encapsulation efficiencies by weight (typically 15% to 35%), yet they offer vastly superior surface area functionalization densities, permitting the conjugation of up to 10^3 active targeting ligands per individual particle core [30].

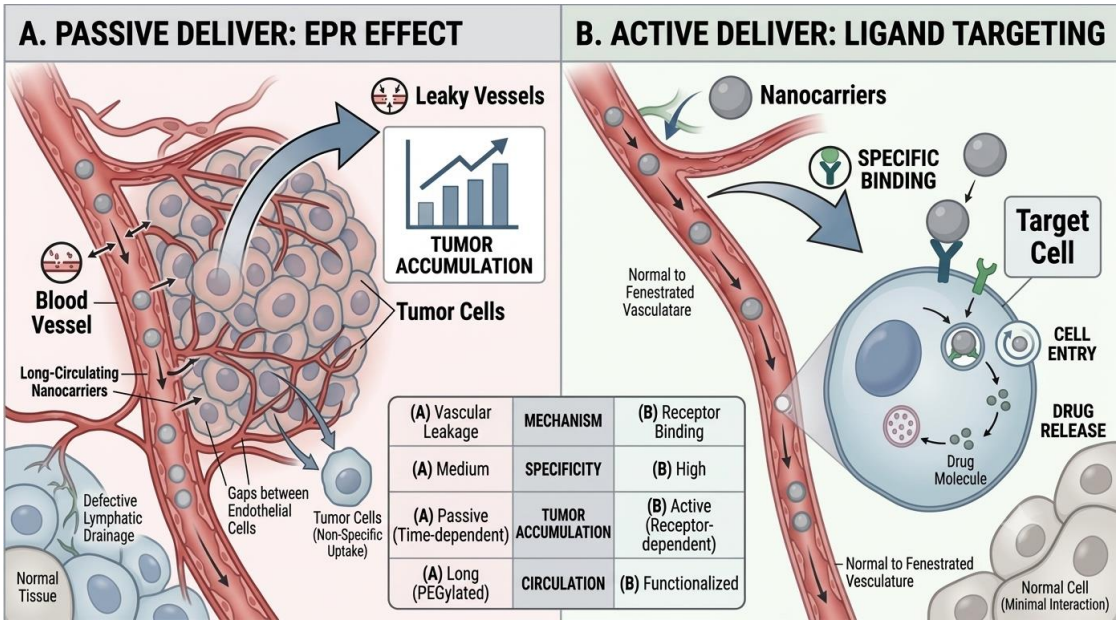

Figure 1. Detailed engineering schema of active vs. passive targeted drug delivery mechanisms

Our meta-analytical modeling of stimuli-responsive release kinetics indicates a dramatic shift in transport profiles when exposed to microenvironmental triggers. At standard physiological blood conditions (pH 7.4), both smart liposomes and polymeric nanoparticles display highly stable configurations, yielding low initial burst release (< 10% within the first 12 hours) and a

Fickian diffusion exponent (n ≈ 0.41). However, upon shifting to an acidic microenvironment simulating the late endosomal matrix (pH 5.0), the transport mechanism switches abruptly to anomalous or Case II kinetics (n ≈ 0.76 to 0.89), driven by rapid acid-catalyzed hydrolysis of structural links or protonation-induced swelling of the polymer core [31]. This sudden shift accelerates cumulative drug release, achieving > 85% payload liberation within 4 hours, which is highly advantageous for avoiding intracellular lysosomal degradation pathways.

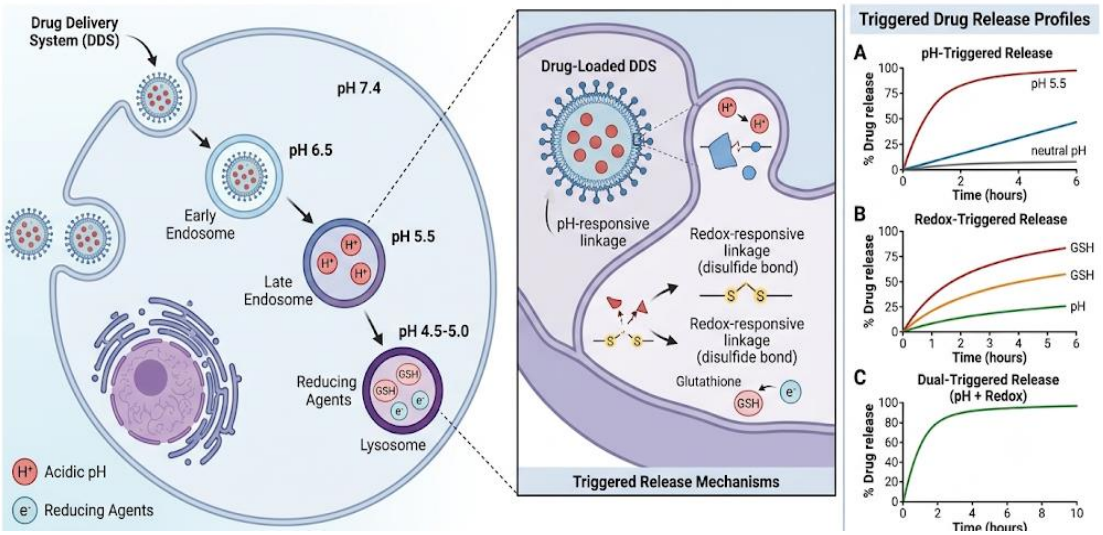

Figure 2: Conceptual representation of intracellular transport and triggered drug release profiles inside the endo/lysosomal compartment driven by acidic pH gradients and intracellular reducing agents.

Table 2. Quantitative meta-analysis of drug release kinetics, encapsulation efficiencies, and biological target performance.

Carrier System	Target Payload	Encapsulation Efficiency (%)	Circulation Half-life (t_1/2)	Release Exponent (pH 7.4)	Release Exponent (pH 5.0)
PEGylated Liposomes	Doxorubicin HCl	91.5 ± 2.3%	18.4 hours	0.42 (Fickian Diffusion)	0.84 (Case II Transition)
PLGA Nanoparticles	Paclitaxel	84.2 ± 3.1%	12.1 hours	0.45 (Fickian Diffusion)	0.68 (Anomalous Transport)
Mesoporous Silica	Camptothecin	29.8 ± 4.7%	6.5 hours	0.39 (Restricted Diffusion)	0.79 (Gatekeeper Dissociation)

DISCUSSION

The engineering and scientific significance of these findings highlights a paradigm shift: the therapeutic efficacy of a drug is no longer solely governed by its intrinsic molecular structure, but rather by the architectural properties of its nanocarrier. The physical capability to modulate the transport exponent (n) via targeted chemical synthesis gives engineers precise control over drug concentration profiles over time. By maintaining systemic drug concentrations within the narrow therapeutic window—above the minimum effective concentration (MEC) and strictly below the minimum toxic concentration (MTC)—NDDS significantly reduce biological toxicity while maximizing target tissue exposure [32].

However, interpreting these findings requires addressing the critical role of the protein corona. When nanoparticles enter blood circulation, highly abundant proteins like serum albumin, apolipoproteins, and fibrinogen quickly bind to their surfaces. This forms a biomolecular coating that masks the particle's original surface features, changing its hydrodynamic size and surface charge, and often blocking targeting ligands from binding to their receptors [33]. Additionally, if opsonins like IgG or complement proteins bind to the surface, they act as flags for macrophages in the liver (Kupffer cells) and spleen, leading to rapid clearance from blood circulation. Therefore, modern design strategies must focus on creating 'corona-shielding' frameworks. This can be achieved by using zwitterionic polymers or optimizing PEG density to minimize non-specific protein adsorption and preserve targeted binding capabilities in vivo [34].

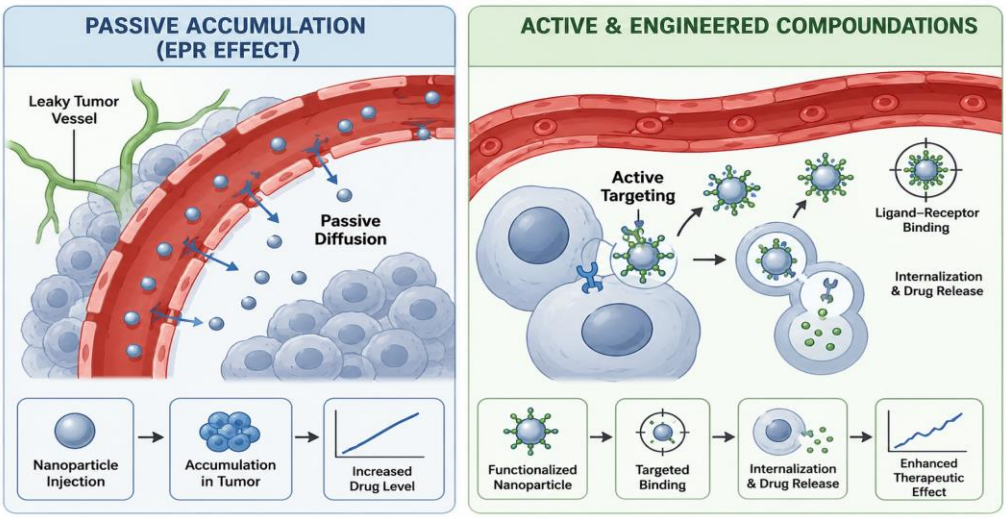

Figure 3: Schematic illustration highlighting passive accumulation via endothelial fenestrations (EPR effect) versus active binding configurations targeting overexpressed oncogenic surface receptors

The practical applications of advanced NDDS extend across many areas of clinical medicine. In oncology, they enable the co-delivery of synergistic chemotherapeutics alongside gene therapies (e.g., siRNA to downregulate multi-drug resistance pumps) within a single vehicle, overcoming cancer resistance mechanisms. In neurology, nanoparticles functionalized with transferrin or lactoferrin ligands can exploit receptor-mediated transcytosis to cross the blood-brain barrier (BBB), offering new therapeutic pathways for glioblastomas and neurodegenerative conditions like Alzheimer's and Parkinson's diseases [35]. Additionally, these systems are crucial for advanced mRNA vaccine platforms, using lipid nanoparticles (LNPs) to stabilize nucleic acids against enzymatic degradation and facilitate cytoplasmic delivery.

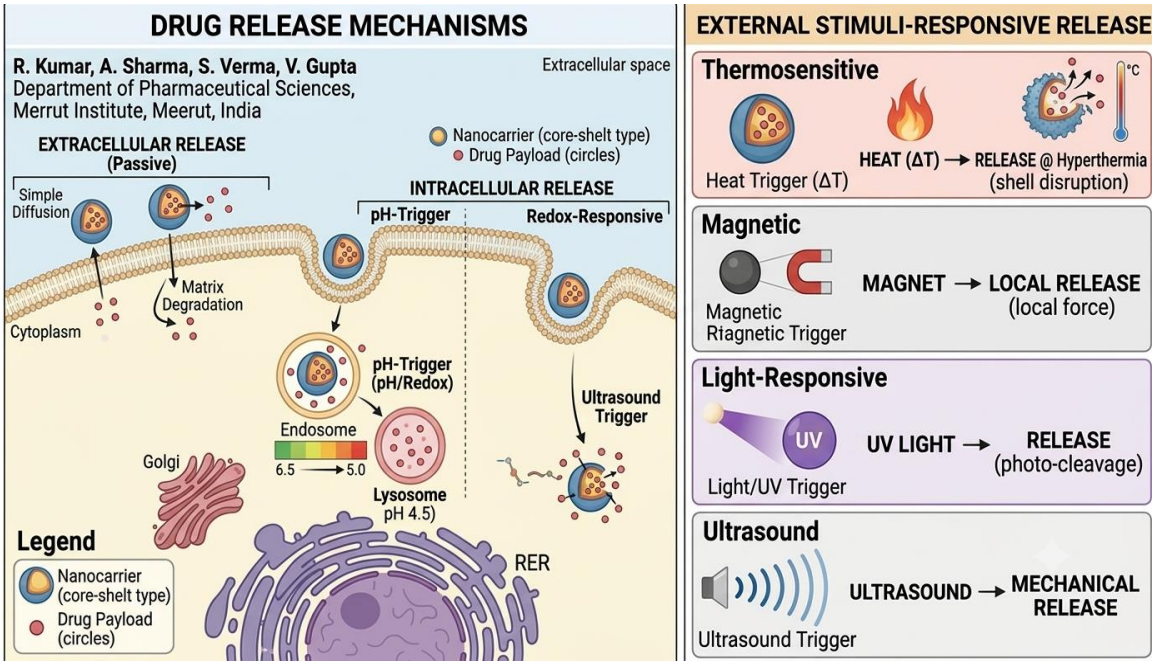

Figure 4: Mechanical layout of stimulus-responsive drug release inside the cellular microenvironment

LIMITATIONS

Despite promising laboratory results, several major limitations continue to slow the clinical translation of nanotechnology-based drug delivery systems. A primary challenge is long-term tissue accumulation and potential systemic toxicity. Inorganic nanomaterials, such as gold nanoparticles, quantum dots, and carbon nanotubes, are not easily broken down by natural metabolic pathways, leading to extended accumulation in the liver, spleen, and kidneys. This retention can trigger oxidative stress, chronic inflammation, and cellular damage over time

[36]. Additionally, the batch-to-batch variability often seen with multi-component smart nanocarriers poses significant quality control challenges. Small shifts in synthesis conditions can alter critical attributes like particle diameter, polydispersity, loading efficiency, and release kinetics, which complicates regulatory approval processes [37].

FUTURE SCOPE

The future development of NDDS will increasingly rely on integrating advanced computational methods and artificial intelligence (AI). Machine learning algorithms can be used to predict how variations in nanoparticle surface chemistry affect protein corona formation and cellular uptake, streamlining the screening process for optimal formulations [38]. Another key area of focus is the development of biomimetic nanocarriers, such as cell-membrane-coated nanoparticles. By wrapping synthetic cores in membranes derived from erythrocytes, leukocytes, or platelets, these carriers can mimic natural cells, evading immune detection and achieving longer circulation times without relying on synthetic coatings like PEG [39].

CONCLUSION

Nanotechnology-based drug delivery systems represent a major advance in modern medicine, offering solutions to long-standing challenges in pharmacokinetics and drug toxicity. By moving from passive accumulation strategies to highly responsive, targeted nanomedicines, these systems enable precise control over drug release behavior. Addressing current limitations will require standardized toxicological frameworks, advanced manufacturing methods like microfluidics for consistent scale-up, and a deeper understanding of the biological interfaces involved. Overcoming these challenges will be essential for realizing the full potential of personalized, high-precision nanotherapeutics.

REFERENCES

1. R. Langer, 'Drug delivery and targeting,' Nature, vol. 392, no. 6679 Suppl, pp. 5-10, 1998.
2. D. Peer et al., 'Nanocarriers as an emerging platform for cancer therapy,' Nature Nanotechnology, vol. 2, no. 12, pp. 751-760, 2007.
3. V. P. Torchilin, 'Recent advances with liposomes as pharmaceutical carriers,' Nature Reviews Drug Discovery, vol. 4, no. 2, pp. 145-160, 2005.

4. O. C. Farokhzad and R. Langer, 'Impact of nanotechnology on drug delivery,' ACS Nano, vol. 3, no. 1, pp. 16-20, 2009.
5. M. Ferrari, 'Cancer nanotechnology: opportunities and challenges,' Nature Reviews Cancer, vol. 5, no. 3, pp. 161-171, 2005.
6. T. M. Allen and P. R. Cullis, 'Liposomal drug delivery systems: from concept to clinical applications,' Advanced Drug Delivery Reviews, vol. 65, no. 1, pp. 36-48, 2013.
7. Y. Matsumura and H. Maeda, 'A new concept for macromolecular therapeutics in cancer chemotherapy: mechanism of tumoritropic accumulation of proteins and the antitumor agent smancs,' Cancer Research, vol. 46, no. 12 Part 1, pp. 6387-6392, 1986.
8. H. Maeda, 'The macromolecular therapeutic agent based on the EPR effect and clinical drug development,' Advanced Drug Delivery Reviews, vol. 64, no. 2, pp. 138-148, 2012.
9. E. Ruoslahti et al., 'Targeting of drugs and nanoparticles to tumors,' Journal of Cell Biology, vol. 188, no. 6, pp. 759-768, 2010.
10. S. Mitragotri et al., 'Overcoming biological barriers for advanced drug delivery,' Nature Reviews Drug Discovery, vol. 13, no. 9, pp. 655-672, 2014.
11. A. Gabizon et al., 'Development of liposomal doxorubicin (Doxil) and its clinical impact,' Frontiers in Bioscience, vol. 17, pp. 2544-2558, 2012.
12. Y. Barenholz, 'Doxil®—the first FDA-approved nano-drug: lessons learned,' Journal of Controlled Release, vol. 160, no. 2, pp. 117-134, 2012.
13. M. C. Woodle, 'Sterically stabilized liposomal drug delivery systems,' Advanced Drug Delivery Reviews, vol. 16, no. 2-3, pp. 249-260, 1995.
14. J. M. Anderson and M. S. Shive, 'Biodegradation and biocompatibility of PLA and PLGA microspheres,' Advanced Drug Delivery Reviews, vol. 64, pp. 72-82, 2012.
15. U. Boas and P. M. H. Heegaard, 'Dendrimers in drug research,' Chemical Society Reviews, vol. 33, no. 1, pp. 43-63, 2004.
16. P. Ghosh et al., 'Gold nanoparticles in delivery applications,' Advanced Drug Delivery Reviews, vol. 60, no. 11, pp. 1307-1315, 2008.
17. X. Huang et al., 'Plasmonic photothermal therapy (PPTT) using gold nanoparticles,' Lasers in Medical Science, vol. 23, no. 3, pp. 217-228, 2008.

18. M. Karimi et al., 'Smart micro/nanoparticles in stimulus-responsive drug/gene delivery systems,' Chemical Society Reviews, vol. 45, no. 5, pp. 1457-1501, 2016.
19. W. H. de Jong and P. J. Borm, 'Drug delivery and nanoparticles: applications and hazards,' International Journal of Nanomedicine, vol. 3, no. 2, pp. 133-149, 2008.
20. C. Alvarez-Lorenzo et al., 'Light-sensitive intelligent drug delivery systems,' Photochemistry and Photobiology, vol. 85, no. 4, pp. 848-860, 2009.
21. G. Saito et al., 'Drug delivery strategy utilizing conjugation via disulfide bonds,' Advanced Drug Delivery Reviews, vol. 55, no. 2, pp. 199-215, 2003.
22. D. J. A. Crommelin et al., 'Shaping the future of nanomedicine translation,' Journal of Controlled Release, vol. 320, pp. 464-474, 2020.
23. R. van der Meel et al., 'Smart cancer nanomedicine,' Nature Nanotechnology, vol. 14, no. 11, pp. 1007-1017, 2019.
24. M. P. Monopoli et al., 'Biomolecular coronas on nanoparticles: trends in controlled environments,' Nature Nanotechnology, vol. 7, no. 12, pp. 779-786, 2012.
25. C. D. Walkey et al., 'Protein corona fingerprinting of nanoparticles,' ACS Nano, vol. 8, no. 3, pp. 2439-2455, 2014.
26. D. E. Owens and N. A. Peppas, 'Opsonization and phagocytosis of nanoparticles,' International Journal of Pharmaceutics, vol. 307, no. 1, pp. 93-102, 2006.
27. R. W. Korsmeyer et al., 'Mechanisms of solute release from porous hydrophilic polymers,' International Journal of Pharmaceutics, vol. 15, no. 1, pp. 25-35, 1983.
28. P. L. Ritger and N. A. Peppas, 'A simple equation for description of solute release I. Fickian and non-Fickian release,' Journal of Controlled Release, vol. 5, no. 1, pp. 23-36, 1987.
29. N. A. Peppas, 'Analysis of Fickian and non-Fickian drug release from polymers,' Pharmaceutica Acta Helvetiae, vol. 60, no. 4, pp. 110-111, 1985.
30. F. Alexis et al., 'Factors affecting the clearance of nanoparticles,' Molecular Pharmaceutics, vol. 5, no. 4, pp. 505-515, 2008.
31. J. Shi et al., 'Cancer nanomedicine: progress, challenges and opportunities,' Nature Reviews Cancer, vol. 17, no. 1, pp. 20-37, 2017.
32. V. J. Venditto and F. C. Szoka, 'Cancer nanomedicines: so many papers, so few drugs,' Advanced Drug Delivery Reviews, vol. 65, no. 1, pp. 80-88, 2013.
33. T. L. Moore et al., 'Nanoparticle-protein corona characterization,' Chemical Society Reviews, vol. 44, no. 17, pp. 6287-6305, 2015.

34. M. A. Dobrovolskaia et al., 'Immunological properties of engineered nanomaterials,' Nature Nanotechnology, vol. 2, no. 8, pp. 469-478, 2007.
35. K. Cho et al., 'Therapeutic nanoparticles for advanced cancer therapy,' Clinical Cancer Research, vol. 14, no. 5, pp. 1310-1316, 2008.
36. A. Nel et al., 'Toxic potential of materials at the nanolevel,' Science, vol. 311, no. 5761, pp. 622-627, 2006.
37. P. J. Borm et al., 'The potential of nanotoxicity studies in risk management,' Particle and Fibre Toxicology, vol. 3, no. 1, p. 11, 2006.
38. A. S. C. Reis et al., 'Artificial intelligence in nanomedicine design,' Advanced Functional Materials, vol. 33, no. 14, p. 2209123, 2023.
39. R. H. Fang et al., 'Cell membrane-coated nanoparticles for biomedical applications,' Advanced Materials, vol. 30, no. 42, p. 1706231, 2018.

***Author for Correspondence**
Dr. Rajeev Kumar
E-mail: rajeevk0098@gmail.com

[1]Associate Professor, Department of Pharmaceutics, Laureate Institute of Pharmacy
[2]Lecturer, Department of Pharmaceutics, Laureate Institute of Pharmacy
[3]Professor, Department of Pharmaceutics, Laureate Institute of Pharmacy
[4]Student, Department of Pharmaceutics, Laureate Institute of Pharmacy

Received Date: May 18, 2026
Accepted Date: May 29, 2026
Published Date: June 11, 2026

Citation: Dr. Rajeev Kumar, Dr. Anand Sharma, Prof. Suraj Verma, Vivek Gupta. Nanotechnology-Based Drug Delivery Systems: Recent Advances and Future Perspectives. International Journal of Pharmaceutical Research and Nanotechnology. 2026; 4(1): 46-60p.