Review

The Efficacy of Nanoliposomes in Cancer Therapy: A Systematic Review of In Vitro Evidence

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ABSTRACT

Background: Cancer therapy is frequently limited by suboptimal drug internalization, chemical instability, off-target toxicity, and the rapid emergence of multidrug resistance. Nanoliposomes and related lipid-based nanocarriers offer a promising solution by enhancing the pharmacokinetic and cellular delivery profiles of anticancer agents. **Objective:** This systematic review critically appraises the available scientific evidence regarding the in vitro efficacy of nanoliposome formulations in cancer therapy. **Methods:** A systematic literature review (SLR) was conducted following the PRISMA framework across PubMed, Medline, and Google Scholar. From 988 initial records, 441 full-text articles were assessed against predefined eligibility criteria, ultimately yielding 15 original studies for qualitative synthesis. **Results:** The synthesized data consistently demonstrate that nanoliposome encapsulation significantly enhances the biological performance of anticancer agents compared to free drugs in cell culture models. This enhancement is characterized by markedly reduced IC_{50} values, decreased cancer cell viability, and increased apoptosis across diverse malignant cell lines. Mechanistically, these improvements are driven by liposome-facilitated active endocytosis, efficient endosomal escape via the Vesicle Budding-and-Collapse (VBC) pathway, and the ability to bypass P-glycoprotein-mediated efflux. Advanced formulations incorporating stimuli-responsive features, genetic payloads (siRNA, mRNA, CRISPR/Cas9), and tumor microenvironment-modifying enzymes demonstrated superior cytotoxic profiles. **Conclusion:** Nanoliposomes represent a highly functional nanotechnology platform for preclinical cancer intervention, effectively protecting active compounds and facilitating efficient intracellular delivery. However, the inherent limitations of two-dimensional cell cultures which lack the physiological complexity, dynamic tissue

barriers, perfusion, and immune interactions of intact organisms necessitate cautious interpretation and further validation in advanced 3D models and in vivo systems.

Keywords: Anticancer; cancer; drugs delivery; in vitro; liposome; nanoliposome.

Introduction

Cancer remains an enduring global health challenge, driving a paradigm shift toward more sophisticated and innovative therapeutic interventions.[1–3] Frontline chemotherapeutics, including cisplatin, doxorubicin, paclitaxel, and gemcitabine, are heavily relied upon but routinely fail to sustain clinical remission due to dose-limiting systemic toxicities and the rapid onset of multidrug resistance (MDR).[4] This chemoresistant phenotype stems from the chronic upregulation of intracellular pro-survival networks, particularly the PI3K/AKT/mTOR and ERK signaling pathways, alongside overexpression of transmembrane efflux pumps such as P-glycoprotein (P-gp).[2–5] Compounding these cellular defenses is the physical architecture of the tumor microenvironment (TME), where cancer-associated fibroblasts (CAFs) orchestrate a desmoplastic stroma packed with dense collagen and hyaluronic acid (HA), establishing an oppressive physical barrier that stifles interstitial perfusion and penetration of unencapsulated drugs.[6–8]

Overcoming these mass transport and cellular resistance barriers has driven the development of highly biocompatible, structurally tunable lipid vehicle architectures, including nanoliposomes, solid lipid nanoparticles (SLNs), and lipid nanoparticles (LNPs).[9] Encapsulating therapeutic payloads within these lipid matrices fundamentally alters their pharmacokinetics by insulating fragile compounds against extracellular degradation, extending biological half-lives, and shifting cellular entry from passive diffusion to active endocytosis.[4–9] Cellular internalization can be further directed via precise surface functionalization using aptamers, antibodies, or receptor-specific ligands.[10–12] Beyond active targeting, these matrices can be rendered stimuli-responsive to adapt dynamically to localized tumor signals, such as acidic pH, high intracellular glutathione (GSH), or overexpressed enzymes.[4–8]

The inherent modularity of nanolipid architectures also makes them exceptionally well-suited for transporting delicate macromolecular genetic cargo, including in vitro transcribed (IVT) mRNA, siRNA, microRNA (miRNA), and CRISPR/Cas9 machinery.[13–15] Intracellular delivery via these systems permits the direct rewiring of aberrantly expressed genetic networks to reverse chemoresistance.[13–16] Preclinical breakthroughs include reversing aggressive phenotypes in triple-negative breast cancer, knocking out oncogenic SOX2 loci in cancer stem cells, and reprogramming immunosuppressive tumor

microenvironments.[15–17]

Given the accelerating expansion of these preclinical innovations, establishing a structured and consolidated consensus on their precise therapeutic boundaries is critical. This systematic literature review (SLR) provides a meticulous, transparent, and reproducible evaluation of current peer-reviewed evidence assessing the *in vitro* efficacy of engineered nanoliposomes in oncology. By mapping how these advanced lipid vesicles interact with cellular systems, alter cytotoxicity kinetics, and overcome microenvironmental barriers at the early preclinical phase, this study clarifies the true translational trajectory of liposomal platforms in modern cancer therapy.

Material and Methods

Study Design

This review employed a systematic literature review (SLR) design to identify, critically evaluate, and synthesize published evidence on the *in vitro* efficacy of nanoliposomes as drug delivery systems for cancer therapy. The SLR framework ensured that the identification and selection of studies followed a consistent, transparent, and reproducible procedure.

Search Strategy

A structured search was conducted across PubMed, Medline, and Google Scholar. Search strings comprised combinations of terms related to nanoliposomes, cancer, and *in vitro* experimental settings: *nanoliposome*, *liposome*, *lipid nanoparticles*, *cancer*, *tumor*, *in vitro*, *cell line*, *cytotoxicity*, *cell viability*, and *apoptosis*.

Eligibility Criteria

Inclusion criteria were: (1) original research evaluating nanoliposomes as anticancer delivery systems in *in vitro* models; (2) articles published within the specified time range; (3) full-text availability; and (4) studies reporting anticancer efficacy outcomes such as cytotoxicity, cell viability, apoptosis, or antiproliferative activity. Exclusion criteria comprised: (1) duplicate records; (2) articles not relevant to nanoliposomes or cancer; (3) non-original publications (reviews, editorials, conference abstracts); (4) purely *in vivo* studies without evaluable *in vitro* data; (5) studies not assessing cytotoxicity parameters; and (6) studies involving nano-formulations other than liposomes or lipid-based nanocarriers.

Study Selection and Data Extraction

Study selection followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) framework, comprising identification, screening, eligibility,

and inclusion stages. From 988 initial records, 162 duplicates were removed. After title/abstract screening, 525 reports were sought for full-text retrieval, of which 84 were unobtainable. A total of 441 full-text articles were assessed for eligibility, and 426 were excluded, ultimately yielding **15 studies** for final qualitative synthesis (Figure 1).

Data Synthesis

Data analysis employed a qualitative descriptive approach by grouping and comparing findings from selected studies. Synthesis focused on the efficacy of nanoliposomes in enhancing anticancer drug delivery and modulating key *in vitro* outcomes, including cytotoxicity, apoptosis, cell viability, and antiproliferative activity. Results were presented narratively to provide a comprehensive evidence-based understanding.

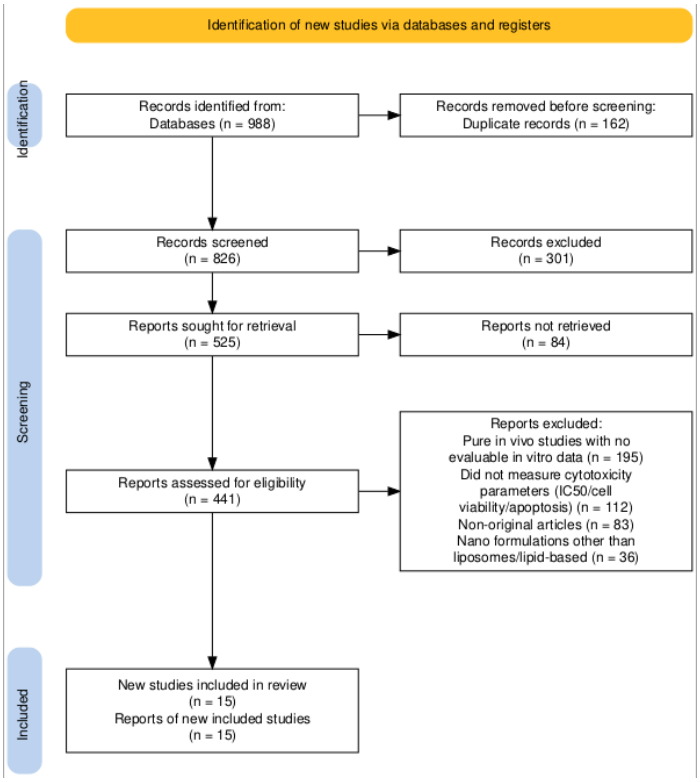

Figure 1. PRISMA flowchart of the systematic literature search and selection process

Results

The 15 included studies consistently demonstrate that lipid-based nanocarriers significantly enhance anticancer performance in preclinical settings compared to free compounds or non-encapsulated controls. The malignancies investigated spanned a broad spectrum, including breast, hepatocellular, pancreatic, lung, ovarian, colorectal, and head and neck squamous cell carcinomas, indicating wide applicability across diverse oncological profiles. The engineered formulations exhibited structural diversity, encompassing conventional nanoliposomes, solid lipid nanoparticles (SLNs), ionizable lipid nanoparticles (LNPs), exosome-liposome hybrids, and biomimetic membrane-coated configurations

tailored for specific chemotherapeutic, natural product, or genetic payloads.

Enhanced Cytotoxicity and Apoptosis Induction

Across all evaluated studies, lipid encapsulation was consistently correlated with enhanced cytotoxic profiles and robust apoptotic induction compared to unencapsulated control agents. Xu et al. (2025) demonstrated that aptamer-guided SLNs loaded with norcantharidin significantly enhanced cellular selectivity and cytotoxicity toward hepatocellular carcinoma cells (HepG2) while sparing normal cells.[10] Similarly, Li et al. (2022) reported that exosome-liposome hybrid nanoparticles co-delivering triptolide (TP) and miR497 effectively reversed cisplatin resistance in chemoresistant ovarian cancer cells by disrupting the upstream PI3K/AKT/mTOR survival pathway.[18]

Electroactive liposome-coated gold nanoparticles (Au-MIL), as reported by Chen et al. (2025), functioned as autonomous electron sinks to induce fatal mitochondrial and endoplasmic reticulum lipid oxidation, bypassing traditional reactive oxygen species (ROS) pathways.[2] Peng et al. (2025) further demonstrated that food-grade lipid nanoparticles (FLNs), when synchronized with autophagy inhibition, triggered destructive intracellular ROS surges via AMPK-mTOR-ULK1 pathway suppression, leading to severe mitochondrial dysfunction and massive tumor cell apoptosis.[3]

Targeting Precision and Microenvironmental Penetration

Surface functionalization using specific aptamers, lactobionic acid ligands, and nanobody co-assemblies significantly accelerated cellular accumulation and lowered IC_{50} values.[10–12] Masarwy et al. (2025) demonstrated that EGFR-targeted CRISPR-LNPs encapsulating Cas9 mRNA and sgRNA targeting SOX2 executed site-specific gene knockout, decreasing head and neck squamous cell carcinoma (HNSCC) viability by approximately 60% *in vitro*.[15]

Structural modifications addressing the tumor microenvironment proved highly effective. Sun et al. (2025) showed that quercetin liposomes conjugated with hyaluronidase enzyme successfully degraded the protective hyaluronic acid matrix in pancreatic tumors, radically boosting drug penetration and bioavailability.[7] Zhang et al. (2024) utilized CAF-homologous biomimetic liposomes to penetrate the dense pancreatic stromal barrier, precisely delivering the BET inhibitor JQ1 and pirfenidone to disrupt metabolic energy supply in pancreatic ductal adenocarcinoma (PDAC).[11]

Genetic and Immunomodulatory Payloads

Lipid nanoparticles proved exceptionally effective for genetic cargo delivery. Anthiya et al. (2023) demonstrated that functional LNPs encapsulating anti-mutant KRAS

siRNA achieved potent gene silencing, inhibiting tumor cell proliferation signaling pathways.[19] Xu et al. (2025) showed that LNPs co-delivering a STING agonist (cGAMP) and mutant KRAS mRNA successfully reprogrammed tolerogenic macrophages via Type I interferon signaling, reversing immunosuppression in pancreatic cancer liver metastases.[20] Fick et al. (2025) reported that LNP-delivered IFNα2 activated the Cxcl9 axis to drive cytotoxic T-cell recruitment and suppress lung metastasis.[17]

Summary of Key Findings

In summary, all 15 studies reported that nanoliposome formulations consistently outperformed free drugs in:

1. Reducing IC_{50} values (indicating higher potency).
2. Decreasing cancer cell viability across multiple cell lines.
3. Increasing apoptotic markers (caspase activation, Bax/Bcl-2 ratio modulation).
4. Enhancing cellular internalization via endocytosis rather than passive diffusion.
5. Overcoming multidrug resistance by bypassing P-gp efflux pumps.

Discussion

Mechanisms of Enhanced Cytotoxicity and Apoptosis Induction

The synthesized evidence confirms that lipid-based nanocarriers elevate antitumor efficacy *in vitro* through deliberate modulation of cellular signaling and fitness cascades. Cytotoxicity assays, the primary evaluative benchmark, consistently report a profound reduction in IC_{50} values alongside a collapse in cancer cell viability. Rather than functioning as inert structural shields, these lipid-based platforms actively dictate the temporal and spatial exposure kinetics of the encapsulated agent.[16]

Programmed cell death, quantified via apoptotic markers, remains the definitive proof of nanocarrier performance. Surface-modified solid lipid configurations, such as those decorated with folate fragments, forcefully downregulate anti-apoptotic Bcl-2 while upregulating Bax, BAD, Caspase-9, and Caspase-3 expressions, causing extensive genomic fragmentation.[6] Simultaneously, formulating hydrophobic compounds into specialized lipid matrices successfully neutralizes P-glycoprotein-mediated MDR by depressing ROS levels and blocking pro-survival NF-κB pathways, doubling the intracellular accretion of cytotoxic agents.[9]

The chemo-genetic synergy observed in co-delivery systems where small-molecule therapeutics are combined with genetic materials like miR-145 using functional lipid nanoparticles facilitates dual-action suppression of tumor expansion while simultaneously providing diagnostic contrast enhancement.[18] Beyond classical apoptosis, engineered

vesicles can directly halt glycolytic flux, cutting off energy generation within desmoplastic matrices by targeting key metabolic networks such as the ALKBH5/JMJD8/PKM2 axis.[13]

Physicochemical Determinants: Size and Elasticity

The physical architecture of a lipid nanocarrier serves as a primary gatekeeper governing cellular internalization and transfection yields. The compiled literature suggests that restricting particle diameters to a strict nanoscale window predominantly between 80 and 150 nm is indispensable for maximizing drug bioavailability and preserving bilayer integrity.[3] Sizing within this window is biophysically ideal because configurations below 10 nm undergo rapid renal filtration, whereas particles exceeding 100–200 nm face immediate clearance via macrophage phagocytosis within the reticuloendothelial system (RES).[16] Nanoscale vehicles exploit energy-dependent endocytic entry mechanics, including macropinocytosis, clathrin-mediated endocytosis (CME), and caveolae-mediated endocytosis (CavME), allowing therapeutic payloads to circumvent extracellular enzymatic degradation.[9–16]

Beyond particle size, recent biophysical screenings reveal that nanoparticle elasticity is an essential structural parameter. By adjusting the cholesterol molar ratio or incorporating plant sterols such as β-sitosterol, the mechanical stiffness of lipid nanoparticles can be precisely engineered across a Young's modulus spectrum of 50 to 400 kPa.[16] Nanoparticles with intermediate mechanical properties (136–202 kPa) exhibit optimized membrane-wrapping energetics, yielding peak cellular uptake and potent mRNA translation in liver (HepG2) and ovarian (OVCAR8) cancer cells. Conversely, highly compliant, softer lipid matrices optimize mRNA delivery in non-small cell lung carcinoma lines (H1299), whose native membrane physics feature high baseline cholesterol concentrations and reduced surface fluidity.[16]

Endosomal Escape: The Vesicle Budding-and-Collapse (VBC) Mechanism

To optimize cytoplasmic delivery, modern nanolipid platforms exploit the recently discovered Vesicle Budding-and-Collapse (VBC) mechanism. As the internalized vehicle moves along the endocytic pathway, the acidic environment of the maturing endosome (pH 6.0–6.5) induces protonation of tertiary amines on cationic ionizable lipids (CILs).[16] This pH-dependent charging destabilizes the core LNP structure, triggering particle disintegration and the insertion of free CILs into the inner leaflet of the endosomal membrane. Driven by hydrophobicity, these inverse cone-shaped lipids self-assemble into enriched cationic domains that electrostatically capture anionic nucleic acid segments. The intense negative intrinsic curvature generated causes the endosomal membrane to invaginate, forming a

nascent vesicle that buds outward into the cytosol and spontaneously collapses, releasing its molecular components.[16]

However, the VBC model reveals an additional post-escape challenge: the slow dissolution of cytoplasmic lipid/nucleic acid aggregates represents a critical kinetic bottleneck, particularly for large macromolecular cargos like mRNA. To accelerate this dissolution, structural modifications such as incorporating bulkier plant sterols or utilizing terminally branched CIL tail structures (e.g., SM-102 or ALC-0315) prevent tight crystalline lipid packing, facilitating rapid and complete cargo release into the cytosol.[16]

"Smart " and Stimuli-Responsive Platforms

The clinical potential of lipid architectures is expanded by transforming them into stimuli-responsive or "smart " platforms designed to exploit microenvironmental variations native to malignant tissues. Quercetin liposomes surface-modified with hyaluronidase enzymes actively degrade the dense hyaluronic acid barrier surrounding pancreatic tumors, significantly improving drug penetration and cellular bioavailability.[7] Camouflaging liposomes with homologous membrane fragments derived from CAFs facilitates effortless migration through dense stromal matrices to ensure site-specific delivery of encapsulated inhibitors.[11] These configurations can be biophysically tailored to undergo rapid structural degradation when exposed to localized tumor triggers, such as redox-sensitive disulfide bonds cleaved by high intracellular glutathione (GSH) or enzyme-sensitive cleavable peptide brushes.[9] Furthermore, these chemical architectures can be combined with exogenous triggers ultrasound-driven acoustic cavitation, superparamagnetic iron oxide core (SPION) heating, or near-infrared light-driven photothermal/photodynamic therapies (PTT/PDT) to enforce fatal oxidative shocks.[2–4]

Translational Challenges and Limitations

Despite these robust *in vitro* findings, several critical limitations must be acknowledged. Standard two-dimensional cell lines fail to replicate the complex tissue barriers, dynamic vascular perfusion, three-dimensional extracellular matrices, and intricate immunoregulatory landscapes of a living organism. Conventional cell cultures lack the physiological complexity, shear stress, and cellular heterogeneity of intact tumors.

Additionally, the spontaneous formation of a biomolecular protein corona upon intravenous administration can compromise targeting fidelity and trigger rapid immune clearance.[16–18] Industrial scale-up of complex lipid formulations presents further challenges, including batch-to-batch consistency, long-term stability, and sterilization without compromising integrity. Therefore, future research must prioritize validating these stimuli-

responsive platforms in complex 3D tumor organoids, patient-derived xenografts (PDX), and comprehensive *in vivo* models to drive successful progression toward clinical oncology.

Conclusion

This systematic review confirms that engineered nanoliposomes and related lipid nanocarriers significantly enhance *in vitro* cancer interventions by lowering IC_{50} values, reducing cell viability, and accelerating programmed cell death across diverse malignant lineages. These therapeutic outcomes are structurally governed by restricting particle diameters to an 80–150 nm window, tuning biophysical elasticity to optimize cell membrane wrapping, and exploiting the VBC endosomal escape pathway for cytosolic payload delivery. By bypassing P-glycoprotein efflux, degrading stromal barriers via enzyme conjugation, and enabling co-delivery of chemotherapeutics with genetic cargo (siRNA, mRNA, CRISPR/Cas9), these platforms provide a formidable alternative to conventional chemotherapy. However, the inherent limitations of two-dimensional cell cultures which lack the complex tissue barriers, dynamic perfusion, and immunoregulatory landscapes of living organisms necessitate cautious interpretation. Future research must prioritize validation in advanced 3D tumor organoids, patient-derived xenografts, and comprehensive *in vivo* models to overcome translational hurdles and accelerate the progression of lipid nanotechnology toward clinical oncology.

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