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Liposomes in Breast Cancer Therapy: Applications and Recent Research Progress

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Abstract. Breast cancer presents marked molecular and phenotypic heterogeneity, often requires prolonged treatment, and remains prone to drug resistance and recurrence. Conventional chemotherapy is still constrained by systemic toxicity and injury to non-target tissues. With a phospholipid bilayer surrounding an aqueous core, liposomes can carry both hydrophobic and hydrophilic drugs. Control of particle size, membrane composition, surface charge, and ligand density can improve drug stability, pharmacokinetics, and tissue distribution. This review outlines liposome structure and preparation, then considers three functional groups used in breast cancer therapy: conventional liposomes, PEGylated long-circulating liposomes, and specialized systems such as thermosensitive, pH-sensitive, and ligand-targeted liposomes. Responsiveness to heat or acidity may release a payload within a tumor, whereas ligand modification can increase receptor-mediated cellular uptake. In practice, protein corona formation, receptor heterogeneity, PEG shielding, and the local tumor microenvironment can weaken these intended effects. Translation to the clinic is further limited by patient-to-patient variation in the enhanced permeability and retention (EPR) effect, storage and in vivo stability, photosensitizer-related phototoxicity, batch-to-batch consistency, and the demands of large-scale manufacturing. Future work should match tumor microenvironmental features with external stimuli, including temperature and light, to build delivery platforms with more predictable and stratified performance. Companion diagnostics, critical quality attribute control, and standardized clinical evaluation will also be needed to establish the safety, efficacy, and translational value of liposome-based therapy.

Keywords: breast cancer, liposomes, drug delivery, stimuli-responsive delivery, targeted therapy, clinical translation

1. Introduction

Breast cancer remains one of the major malignancies affecting women's health. International Agency for Research on Cancer (IARC) statistics show that newly diagnosed breast cancer in women represents about 11.6% of all new cancer cases worldwide, and breast cancer deaths account for about 6.9% of cancer mortality. It is currently the leading female malignancy in both incidence and mortality [1].

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An integrated treatment framework is now used for breast cancer, combining surgery, radiotherapy, chemotherapy, endocrine therapy, targeted therapy, and immunotherapy. As molecular biomarkers have been defined in greater detail, diagnosis and treatment have become more closely aligned with molecular subtypes. Considerable heterogeneity remains, however, both between subtypes and between individual patients. Treatment therefore has to be adjusted to disease stage, receptor status, and recurrence risk. The main unresolved problems are injury to normal tissues and systemic toxicity from chemotherapy, drug resistance after prolonged exposure, and the modest efficacy often seen in recurrent or metastatic disease [2].

Developments in molecular biology and drug-delivery technology have opened another route for addressing these limitations. Liposomes are particularly useful because their phospholipid bilayer and aqueous core can carry hydrophobic and hydrophilic drugs separately. This arrangement can improve dispersion, slow degradation, and alter pharmacokinetic behavior. PEGylation or the addition of targeting ligands may further extend circulation, improve interactions with breast cancer cells and the tumor microenvironment, and limit exposure of non-target tissues. These features explain why functionalized liposomes are being explored for stronger efficacy, fewer adverse effects, and combination treatment [3].

2. Basic characteristics of liposomes

2.1. Structure and composition

A liposome is a closed vesicle formed by one or more phospholipid bilayers around an aqueous core. The hydrophilic headgroups face the water on both sides of the vesicle, while the hydrophobic fatty acid chains point inward and create the nonpolar membrane region. Cell membranes have a similar phospholipid-and-cholesterol bilayer, which gives liposomes a degree of biomimicry and generally good biocompatibility. At the cell surface, they may be taken up through adsorption, membrane fusion, or endocytosis [4].

The lipid ingredients used in liposomes fall into four broad groups. Phospholipids provide the basic membrane framework; those with higher phase-transition temperatures often improve stability and can alter cellular uptake. Ionizable cationic lipids support nucleic acid encapsulation in lipid nanoparticles (LNPs) and may disrupt the endosomal membrane, promoting cytoplasmic release. PEGylated lipids create a hydrated steric layer that limits protein adsorption and macrophage uptake while helping to control particle size and colloidal stability. Cholesterol adjusts membrane rigidity and permeability and reinforces the integrity of the lipid bilayer [5].

The coexistence of hydrophilic and hydrophobic domains allows liposomes to carry drugs with different physicochemical properties. Water-soluble compounds are mainly retained in the aqueous core, whereas lipophilic compounds partition into the phospholipid bilayer. Amphiphilic molecules tend to reside at the membrane-water interface. Altering phospholipid headgroups or hydrophobic tails, or adding functional molecules such as targeting ligands, can further support barrier crossing, targeted cellular uptake, and controlled drug release [6].

Liposomes are commonly described as multilamellar, small unilamellar, or large unilamellar vesicles according to lamellarity and size. From a functional perspective, they include conventional, long-circulating, actively targeted, stimuli-responsive, and biomimetic liposomes. Particle size, lamellarity, surface charge, membrane fluidity, and ligand density together shape circulation, tissue penetration, cellular uptake, and clearance. Formulation design should therefore start with the drug's physicochemical properties and the planned route of administration [7].

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2.2. Preparation methods

Thin-film dispersion, also called thin-film hydration, is among the most widely used bottom-up methods for preparing biomimetic liposomes. Lipids, cholesterol, and other membrane constituents first self-assemble into vesicles. Particle size, membrane architecture, and drug-loading capacity can be adjusted during the process, and targeting ligands or other functional molecules can be added later by surface functionalization. The method is useful at laboratory scale but involves several operations and relatively high production costs, which can make scale-up difficult [8].

Ultrasonic dispersion represents a typical top-down route. Probe or bath sonication applies cavitation and shear to break larger multilamellar vesicles into smaller ones. It is therefore often used to reduce particle size and refine formulations during small-scale preparation.

Reverse-phase evaporation is mainly used to produce large unilamellar liposomes with high encapsulation efficiency for water-soluble drugs. An organic lipid phase is emulsified with an aqueous drug phase to form a water-in-oil emulsion, and the solvent is removed under reduced pressure. Phase inversion then yields vesicles with a relatively large aqueous core. Residual solvent can remain, so an additional purification step is normally required.

3. Classification of liposomes for breast cancer therapy

For breast cancer therapy, liposomes can be grouped by surface modification and by their response to disease-associated signals. The three broad classes are conventional liposomes, long-circulating liposomes, and specialized functional liposomes. They differ in circulation, tumor accumulation, cellular uptake, and release behavior. Because breast cancer contains diverse subtypes and heterogeneous microenvironments, this classification has practical implications for choosing drug exposure, expected efficacy, and toxicity rather than serving as a naming scheme alone. [9, 10]

3.1. Conventional liposomes

Conventional liposomes generally contain phospholipids and cholesterol, without PEG, antibodies, or other targeting components at the surface. Hydrophilic drugs are held mainly in the aqueous core and hydrophobic drugs in the bilayer. Their relatively simple composition makes it straightforward to adjust the lipid type, cholesterol ratio, and particle size, and they can improve the dispersion of poorly water-soluble anticancer drugs [10].

The same unmodified surface also leads to rapid clearance. Plasma proteins adsorb readily to these liposomes, after which the mononuclear phagocyte system removes them through the reticuloendothelial system, particularly in the liver and spleen. Circulation is consequently short. In breast cancer treatment, conventional liposomes are reasonable starting or control formulations; when longer exposure or more efficient intracellular delivery is needed, their surface properties or membrane composition usually require further modification [11].

3.2. Long-circulating liposomes

Long-circulating, or stealth, liposomes usually carry PEGylated lipids in the membrane. The resulting hydrophilic steric layer reduces macrophage uptake and slows reticuloendothelial clearance, extending systemic circulation. A longer residence time may give the particles more opportunities to leave vessels with relatively high permeability and interact with breast cancer lesions [11].

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Longer circulation should not be equated with dependable tumor targeting. Liposomes acquire a protein corona soon after administration, and that corona can change both cellular interactions and tissue distribution. Cholesterol and other membrane components are metabolized as well, with possible effects on immune-cell responses. Dense PEG coverage may hinder internalization, and repeated dosing can produce accelerated blood clearance. The formulation must therefore balance circulation time with tissue penetration and cellular uptake [12, 13].

3.3. Specialized functional liposomes

Specialized functional liposomes add stimuli-responsive elements or targeting ligands so that release and uptake are linked to local pathological conditions. Their attraction is improved spatial and temporal selectivity together with better pharmacokinetic control. The additional structural complexity, however, makes reproducibility, quality control, and clinical translation more demanding [14].

3.3.1. Thermosensitive liposomes

Thermosensitive liposomes are designed to release their cargo when the surrounding temperature rises, commonly during local hyperthermia at temperatures above 40 °C. They are usually given intravenously and can extend the circulation half-life of the encapsulated drug. Heating increases lipid-bilayer permeability and triggers rapid release at the treated site. In vivo studies have reported substantially higher drug concentrations in the target region [9].

Wang et al. tested thermosensitive liposomal doxorubicin with mild hyperthermia and low-dose radiotherapy in an orthotopic mouse model of breast cancer. The sequential three-part regimen delayed primary tumor growth under low-dose chemotherapy, while treatment-related toxicity was similar to that seen with either thermosensitive liposomes or low-dose radiotherapy alone. The results support a role for thermosensitive liposomes as local sensitizing carriers in multimodal treatment [15].

Heating is not equally deep or uniform in every tumor, and the formulation must remain stable at physiological temperature while releasing the drug quickly once heated. Platforms that respond to both temperature and pH could consequently be useful for locally recurrent breast cancer [16].

3.3.2. PH-sensitive liposomes

The extracellular pH of a tumor may range from approximately 5.8 to 7.2, while lysosomes and endosomes are typically near pH 5.5. Both settings are more acidic than most normal tissues. pH-sensitive liposomes use protonatable lipids or acid-labile bonds to destabilize the membrane in an acidic environment. The resulting change can release the drug and, for some payloads, assist endosomal escape [9].

The approach may be useful for co-delivering chemotherapeutic agents and nucleic acids, including agents aimed at resistance pathways in triple-negative breast cancer. Pan et al. prepared the Dectin-1-targeted, pH-responsive formulation PLP-II/MGlu-Curd-Lips, which carried the acidic polysaccharide PLP-II from Plantago, and tested it in a 4T1 breast cancer mouse model. In the acidic tumor microenvironment, the formulation released its payload and promoted lysosomal escape while directing uptake to dendritic cells through Dectin-1. In vivo, it activated dendritic cells, shifted tumor-associated macrophage polarization, and increased cytotoxic T-lymphocyte infiltration, thereby remodeling the immunosuppressive microenvironment. These results suggest that pH

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responsiveness can work together with immune-cell targeting to improve breast cancer treatment [17].

The pH difference inside a tumor is nevertheless limited, and protein corona formation and phagocytic clearance may further blunt the response. Acid-triggered release measured in vitro should therefore not be treated as a direct predictor of therapeutic efficacy in vivo. [14, 16]

3.3.3. Ligand-targeted liposomes

Ligand-targeted liposomes are made by attaching antibodies, peptides, aptamers, or small-molecule ligands to the vesicle surface. The ligands bind receptors associated with breast cancer cells and can drive receptor-mediated endocytosis, increasing intracellular drug delivery. Ligands directed at HER2, folate receptors, transferrin receptors, or integrins may help recognize particular breast cancer subtypes [18].

Fonseca et al. developed F3 peptide-modified liposomes against nucleolin, a receptor overexpressed on both stem-like and non-stem-like triple-negative breast cancer cells, and used the particles to deliver a synergistic drug combination. Binding and treatment response tracked with nucleolin expression and cellular stemness. In vitro, both cell populations were completely eliminated. Targeting a receptor shared across these populations may therefore help address tumor heterogeneity and could lower the chance of recurrence [19].

Ligands have also been used to deliver immunomodulatory payloads or to build biomimetic liposomes that reshape the immune microenvironment of triple-negative breast cancer. Their in vivo performance depends on receptor-expression heterogeneity, ligand density, linker length, and the extent of PEG shielding. Too many ligands can increase protein adsorption or non-specific binding in non-target tissues. The value of ligand targeting should thus be judged together with patient characteristics, in vivo biodistribution, and safety findings [20, 21, 18].

4. Clinical progress of liposome-based breast cancer therapy

Among liposomal products used clinically in breast cancer, pegylated liposomal doxorubicin (PLD) has the longest clinical track record. By changing where doxorubicin distributes and how long it circulates, PLD retains antitumor activity while markedly lowering myocardial exposure to free doxorubicin.

In a phase III randomized study of 509 patients with metastatic breast cancer, O'Brien et al. compared PLD with conventional doxorubicin as first-line therapy. Median progression-free survival was 6.9 months with PLD and 7.8 months with conventional doxorubicin; median overall survival was 21 and 22 months, respectively, indicating similar antitumor activity. Conventional doxorubicin produced 3.16 times more cardiac toxicity. Alopecia, nausea, vomiting, and neutropenia were less frequent with PLD, whereas hand-foot syndrome, stomatitis, and mucositis were more common [22].

Keller et al. compared PLD with vinorelbine or mitomycin C plus vinblastine in 301 patients with taxane-resistant advanced breast cancer. Median progression-free survival was 2.9 months for PLD and 2.5 months for the comparator, and median overall survival was 11.0 versus 9.0 months; neither difference was statistically significant. Patients without prior anthracycline exposure showed a trend toward longer progression-free survival with PLD, supporting its use as a salvage option after several previous lines of treatment [23].

Taken together, the clearest clinical contribution of liposomal formulations is redistribution of exposure and reduction of toxicity, rather than a consistent survival gain in every patient group.

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Outcomes vary with the EPR effect, tumor vascular permeability, cumulative anthracycline exposure, molecular subtype, and drug-resistance status. Hand-foot syndrome and other formulation-specific effects also require active management. Future trials should use molecular subtyping and pharmacokinetic biomarkers to select patients, while incorporating cardiac monitoring, dose adjustment, and preventive measures for treatment-related symptoms. Reproducibility will additionally depend on standardized manufacturing, long-term stability studies, and evidence from routine clinical practice.

5. Conclusions and future perspectives

Liposomes can carry both hydrophilic and hydrophobic drugs and, through PEGylation, ligand modification, and thermal-, pH-, or light-responsive designs, can alter pharmacokinetics, increase tumor accumulation, and control intracellular release. Their use has consequently moved beyond reducing anthracycline toxicity to include chemotherapy sensitization, nucleic acid delivery, immune modulation, and multimodal combination therapy.

The EPR effect is not uniform: tumor type, vascular architecture, interstitial pressure, and other local factors all matter. Nanoparticles may enter solid tumors through routes other than endothelial gaps, and active transendothelial transport can dominate in some settings. Liposomes also remain vulnerable to aggregation, drug leakage, protein corona formation, and batch-to-batch variation during storage and circulation. For photosensitizer-loaded liposomes, phototoxicity in non-target tissues and limited penetration depth add further constraints [24].

A useful next step is to combine tumor microenvironmental information with external stimuli and design liposomes that respond at defined thresholds of acidity, enzymatic activity, redox status, temperature, or light. Such systems could release a payload on demand within a tumor rather than simply responding to a broad environmental change. Tumor heterogeneity, the still incomplete understanding of the nano-bio interface, and the chemistry, manufacturing, and quality-control demands of scale-up remain major barriers to clinical translation [25].

Research should continue to refine lipid composition, particle-size control, sterilization, and continuous manufacturing, while defining critical quality attributes that correlate with in vivo behavior. Clinical trials need to assess efficacy, long-term safety, and quality of life together. Coordinated work on formulation design, companion diagnostics, and manufacturability could move liposomes from empirical carriers toward predictable, stratified, and personalized precision-therapy platforms.

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