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Stealth missiles with precision guidance: A novel multifunctional nano-drug delivery system based on biomimetic cell membrane coating technology

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ABSTRACT

Nanodrug delivery systems (NDDSs) have demonstrated broad application prospects in disease treatment, prevention, and diagnosis due to their nanoscale size advantages and high drug-loading capacity. However, their clinical translation still faces multiple challenges, including rapid clearance by the reticuloendothelial system (RES), nonspecific targeting, and insufficient efficiency in crossing biological barriers. Cell membrane-coated biomimetic delivery systems (CMC-BDS), which integrates natural cell membranes onto nanoparticle (NPs) surfaces, provides nanodrugs with a versatile "biomimetic cloak," representing a highly promising surface engineering strategy. This approach enables nanocarriers to inherit the intrinsic biological properties of different cell sources, endowing them with immune evasion, prolonged circulation, dynamic targeting, biocompatibility, and biodegradability, while supporting the integration of diverse biomedical functions. Furthermore, surface functionalization modifications can enhance their programmability, multifunctionality, and biointerface adaptability, thereby optimizing targeted delivery efficiency and extending in vivo circulation time. This review first outlines the development and key preparation steps of cell membrane coating technology. It then discusses the selection strategies for various cell membrane types—including leukocyte, erythrocyte, platelet, dendritic cell, tumor cell, and bacterial membranes—while comparing their respective advantages and limitations. Finally, the review highlights recent advances in applying cell membrane-coated nanoparticles (CMC-NPs) for treating tumors, ischemic stroke, and inflammatory diseases.

1. Introduction

Conventional drugs are often limited by poor targeting specificity and significant systemic toxicity [1,2]. With the advancement of nanotechnology, engineered nanodrug delivery systems (NDDS) have demonstrated tremendous potential in biomedicine due to their small size, high drug-loading capacity, and functional programmability. Compared to traditional drugs, NDDS offer several distinct advantages [3–11]: **(1) High drug-loading capacity and functionalization potential:** the small size and large surface area-to-volume ratio of nanoparticles enable efficient drug encapsulation and surface modification; **(2) Drug protection:** nanocarriers can shield drugs from enzymatic degradation, improving stability and solubility; **(3) Enhanced permeability and retention (EPR) effect:** NPs (5–250 nm) can selectively accumulate in tumor tissues through the EPR effect. For example, given that tumor cells consume over 10 times more glucose than normal cells,

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precisely engineered nanocarriers can deliver glucose oxidase (GOD) to target tumor regions, where it catalyzes the oxidation of glucose into gluconic acid and H_2O_2, depleting glucose in the tumor microenvironment (TME) and ultimately "starving" the tumor cells to death [2].

However, current NDDS still face multiple challenges [12–15]: **(1) Rapid clearance by the reticuloendothelial system (RES):** conventional NPs are prone to plasma protein adsorption, leading to RES recognition and clearance, resulting in insufficient circulation time; **(2) Non-specific targeting:** traditional nanodrugs lack targeting specificity, unable to precisely deliver to disease sites while potentially damaging healthy tissues; **(3) Inefficient transendothelial transport:** conventional NPs demonstrate poor efficiency in crossing biological barriers like the blood-brain barrier.

To improve nanoparticle biocompatibility and functional integration, researchers often employ synthetic polymers (e.g., peptides, polysaccharides, polyethylene glycol (PEG)) for surface modification. For instance, polyethylene glycolylation can reduce protein adsorption through hydration layer effects to prolong circulation time [16]. Nevertheless, PEG modification has notable limitations: **(1) Antibody induction:** repeated injections may trigger anti-PEG antibody production, causing accelerated blood clearance; **(2) Limited biomimicry of complex biointerfaces:** PEG modification cannot replicate active targeting or dynamic environmental responsiveness; **(3) Insufficient targeting:** although target ligands (e.g., antibodies, peptides) can be conjugated, PEG's steric hindrance often interferes with ligand-receptor binding; **(4) Complex modification processes:** polyethylene glycolylation involves multiple steps including activation, conjugation, and purification, resulting in poor batch-to-batch consistency.

The cell membrane serves not only as a physical barrier but also participates in crucial biological processes such as signal transduction and immune regulation through membrane proteins and glycocalyx structures [17]. Inspired by this, scientists developed cell membrane-coated biomimetic delivery systems (CMC-BDS): wrapping natural cell membranes onto nanoparticle surfaces, enabling NPs to inherit surface antigens, receptors and signaling molecules from source cells, thereby acquiring both the physicochemical characteristics of natural cell membranes and the biological features of the source cells [18–29]. The advantages of cell membrane coating technology include [30–34]: **(1) Immune evasion and prolonged circulation:** The cell membrane coating allows NPs to "deceive" the immune system, avoiding rapid recognition and clearance by the RES. Rui et al. demonstrated that CMC-NPs showed significantly reduced macrophage uptake within 8 h [35]. **(2) Dynamic targeting:** CMC-NPs can actively migrate to specific tissues or organs according to physiological/pathological signals and cross impenetrable biological barriers to reach disease sites. **(3) Biocompatibility/biodegradability:** Natural membrane components ensure safety and avoid toxicity issues of synthetic materials. **(4) Flexible functional modification:** The fluidity of cell membranes facilitates functionalization, while combining with gene editing technology enables precise regulation of membrane proteins to enhance targeting or environmental responsiveness. **(5) Multifunctional integration potential:** Coating nanoparticles with fused membranes from different cell types can simultaneously endow them with diverse biomedical functions.

Therefore, this article provides a systematic overview of cell membrane coating technology. First, the review introduces the historical development and key procedures of cell membrane coating technology. Second, it elaborates on different strategies for cell membrane selection while comparing their respective advantages and limitations. Finally, the review discusses the diverse biomedical applications of cell membrane coating technology in fields including tumor diagnosis and treatment, ischemic stroke, and inflammatory diseases.

2. Historical development and key procedures of cell membrane coating technology

The evolution of cell membrane coating technology can be systematically examined along two key dimensions:**(1) Expansion of cell membrane sources:** The diversification of membrane sources has been a primary driver of technological advancement. In 2011, Zhang et al. pioneered the field by coating poly(lactic-co-glycolic acid) (PLGA) nanoparticles with natural red blood cell (RBC) membranes, demonstrating reduced macrophage uptake and systemic clearance—a breakthrough that ignited widespread interest in biomimetic membrane coating strategies [36]. Subsequent progress came in 2015 when the same team expanded membrane sources to include bacterial cells, significantly broadening the technology's scope [37].Research subsequently revealed a trend toward membrane source diversification, with scientists developing coating systems derived from various mammalian cells: leukocytes [38], erythrocytes [39,40], platelets [41], and dendritic cells [50]. Notably, the introduction of tumor cell membranes [42]and bacterial membranes [37] opened new avenues for targeted therapy and immunomodulation. Parallel advancements included genetically engineered biomembranes [43] and extracellular vesicles [44], marking the technology's transition into precision-controlled applications. Building upon these developments, hybrid membrane technology emerged as an innovative approach [24,45,46]. By fusing distinct cell membranes (e.g., dendritic-tumor cell [47], cancer-RBC [48,49], macrophage-cancer cell [50], and bacterial vesicle-cancer cell [51] hybrids), researchers achieved multifunctional synergies unattainable with single-membrane systems, significantly enhancing nanocarrier adaptability in complex physiological environments. **(2) Innovation in nanoparticle core design:** Concurrently with membrane source expansion, core material engineering underwent transformative changes. Early studies predominantly employed biodegradable polymers like PLGA [36], which, while reliable, offered limited functionality. To overcome this constraint, researchers developed diverse organic systems including liposomes [52] and peptide/protein-based carriers [53–56]. The incorporation of inorganic materials—such as black phosphorus nanoparticles [57], mesoporous silica nanoparticles [58], and gold nanoparticles [59]—not only expanded the technology's applicability but also endowed nanocarriers with unique photothermal and imaging capabilities. These advances have elevated CMC-NPs from simple delivery vehicles to multifunctional theranostic platforms integrating diagnosis and treatment.

The preparation process of cell membrane coating technology can be systematically divided into three critical steps that directly determine the performance and functionality of the final nanocarriers through optimization and innovation [32,60–63]: **(1) Cell membrane extraction:** For non-nucleated cells like erythrocytes and platelets, the membrane isolation involves isolating target cells from whole blood using density gradient centrifugation, disrupting cell membranes via hypotonic lysis or freeze-thaw cycles, removing soluble proteins by centrifugation, and processing the membrane components into nanoscale vesicles through extrusion or sonication. For nucleated cells including macrophages, tumor cells and stem cells, the extraction requires cell enrichment followed by membrane disruption through hypotonic lysis, mechanical homogenization or ultrasonication, removal of nuclei and cytoplasmic proteins via gradient centrifugation or differential centrifugation, and final preparation of nanoscale membrane vesicles through extrusion or sonication. **(2) Nanodrug core fabrication:** Based on application requirements, core materials can be selected from various candidates including lipid-based (liposomes), peptide/protein-based, polymeric, and inorganic materials [32,54–56], with emulsion-solvent evaporation and nanoprecipitation methods becoming predominant due to their operational simplicity, high encapsulation efficiency and excellent reproducibility. The establishment of these standardized protocols has significantly accelerated functionalized nanoparticle core development. **(3) Membrane-core fusion:** This final step determining biomimetic nanocarrier integrity and functionality currently employs

mainstream approaches including thin-film extrusion, ultrasonication-assisted fusion and electroporation-mediated integration, with each method offering distinct advantages for different applications.

The three membrane-core fusion methods are described as follows (Table 1): **(1) Extrusion method** [62]**:** This approach involves mixing cell membranes with nanoparticle cores and repeatedly passing the mixture through polycarbonate porous membranes with varying pore sizes using an extruder, resulting in uniform membrane coating on the nanoparticle surface. Its advantages include producing relatively uniform particle sizes, operational simplicity, preservation of membrane bioactivity, and wide applicability; disadvantages encompass tedious procedures, time-consuming process, difficulty in mass production, and potential membrane material loss. **(2) Sonication method** [60,64]**:** This technique employs ultrasound treatment to coat purified cell membranes onto nanoparticle cores, requiring optimization of sonication conditions to maximize encapsulation efficiency without causing drug denaturation or leakage. While being rapid and straightforward, this method cannot guarantee coating integrity or controlled size distribution, and the transient high temperature/pressure generated during sonication may damage membrane proteins and lipid structures, potentially compromising biological functions - necessitating careful parameter optimization **(3) Electroporation method** [65–67]**:** This process utilizes electrical pulses in microfluidic devices to facilitate nanoparticle entry into cell membranes for fusion. It offers high fusion efficiency, good reproducibility, and uniform particle sizes, but suffers from high costs and requires precise parameter adjustment. Regardless of the coating method employed, a common challenge lies in achieving uniform coating integrity, as most nanoparticles typically obtain only partial coverage, which may consequently reduce loading efficiency. All three methods require careful optimization of critical parameters (e.g., extrusion cycles/pressure, sonication power/duration, or electric field strength/pulse duration). to balance coating quality with preservation of both membrane biological properties and drug activity.

Table 1
Comparative advantages and disadvantages of membrane-core fusion methods.

Fusion Method	Advantages	Limitations	Reference
Extrusion	• Produces nanoparticles with uniform size distribution • Rapid and straightforward operation • Preserves membrane biological activity • Broad application spectrum	• Labor-intensive multi-step process • Time-consuming and difficult to scale up • Potential loss of membrane materials during processing	[18–20,31, 36,38,45, 68–71]
Sonication	• Simple and time-efficient procedure	• Inconsistent coating integrity and particle size distribution • Transient high temperature/ pressure may compromise membrane proteins and lipid structures	[18–20,31, 45,48, 72–75]
Electroporation	• High fusion efficiency • Excellent reproducibility with uniform particle sizes	• Requires significant equipment investment	[18–20,31, 45,67, 76–82]

3. Cell membrane selection strategies in membrane coating technology

Different cell membranes possess distinct surface molecules, advantages, limitations, and disease-specific applications (Table 2), enabling nanoparticles to acquire diverse biomedical functions. For example, RBC membranes leverage CD47-mediated "don't eat me" signals to effectively inhibit macrophage phagocytosis, making them ideal for prolonged systemic circulation [83]. Leukocyte membranes express monocyte chemoattractant protein-1 (MCP-1/CCL2) receptors, granting inherent inflammation tropism and tumor-targeting capabilities that enhance vascular barrier penetration and directional migration to lesions [84]. Platelet membranes, enriched with adhesion molecules like P-selectin, specifically recognize damaged vasculature and tumor microenvironments, offering unique advantages for treating vascular disorders and metastatic cancers [85,86]. Tumor cell membranes combine scalable production with homologous targeting via surface glycoproteins (e.g., CD44), significantly improving drug accumulation in tumor tissues [87, 88]. To date, functionalized membranes have been successfully extracted from multiple cell types for coating applications, including leukocytes, erythrocytes, platelets, dendritic cells, tumor cells, bacteria, and hybrid membrane systems [20] (Fig. 1). This diversity allows tailored solutions for varied therapeutic needs, with each membrane type offering unique biological properties that can be strategically exploited for targeted drug delivery, immune evasion, or tissue-specific accumulation. The selection of appropriate membrane sources depends on the intended application, desired circulation time, and specific biological barriers to overcome, with recent advances enabling even more precise engineering of membrane components for enhanced functionality.

3.1. Leukocyte membrane-coated nanoparticles (LM-CN)

Leukocytes, comprising approximately 1 % of peripheral blood cells, serve as the immune system's core effector cells and maintain host defense through diverse subsets and functions [116]. Notably, leukocytes' unique biological properties make them ideal biomimetic materials for nanodrug delivery systems. LM-CN have been widely explored as drug carriers [117,118], providing the following key benefits: **(1) Immune evasion:** By retaining the source cells' self-recognition markers and immune-modulating receptors, these membranes help nanoparticles avoid immune detection and clearance; **(2) Inflammation and tumor targeting:** Chemokine receptors and innate immune recognition molecules on the membrane surface facilitate active trafficking to inflammatory or tumor tissues, enhancing targeting precision. **(3) Transendothelial Migration:** Due to their high membrane fluidity and inherent endothelial penetration ability, monocytes/macrophages and neutrophils can transport nanoparticles across vascular barriers—overcoming a major hurdle for traditional delivery systems; **(4) Immune Modulation:** Membrane proteins derived from polarized monocytes/macrophages and neutrophils can engage with immune cell receptors, altering the local immune environment and enabling combined therapeutic effects.

3.2. Monocyte/macrophage membrane-coated nanoparticles (MM-CN)

Monocytes, as precursors of macrophages, possess the ability to migrate from blood circulation across endothelial barriers into various tissues where they differentiate into macrophages under local microenvironmental cues. The unique molecular composition of macrophage membranes endows them with distinctive inflammatory targeting capabilities and immunomodulatory functions, providing a natural biomimetic platform for nanodrug delivery. Specific receptors and adhesion molecules on macrophage membranes (e.g., CCR2, VCAM-1, and ICAM-1) mediate both targeting ability and immune evasion functions [119, 120]. For instance, monocytes utilize surface C-C chemokine receptor type 2 (CCR2) receptors to recognize chemokine (C-C motif) ligand 2

Table 2
Comparative analysis of cell membrane sources for biomimetic nanoplatforms.

Source	Key Markers	Advantages	Limitations	Applications	References
Leukocyte	CXCR4, CXCR2, CCR2, VCAM-1, etc.	Immune evasion; Inflammation/tumor targeting; Endothelial penetration	Limited extraction yield; Potential immune overresponse	Pneumonia, tumors, bone repair, Alzheimer's, atherosclerosis	[89–96]
Erythrocyte	CD47, C8bp, CR1, etc.	Immune evasion; Abundant source; Long circulation	Lack targeting specificity; Blood type compatibility	Tumors, diabetic wounds, pneumonia	[57,63, 97–100]
Platelet	CD47, P-selectin, etc.	Immune evasion; Inflammation/tumor targeting; Vascular injury homing	Thrombosis risk; In vivo aggregation	Tumors, inflammation, atherosclerosis, thrombosis	[62, 101–104]
Dendritic Cell	MHC complexes, CCR7, etc.	Antigen presentation; Immune activation	Short circulation; Limited source; Potential immune overresponse	Tumors	[105–108]
Tumor Cell	CD47, EpCAM, etc.	Immune evasion; Tumor homing; Scalable production	Safety concerns; Potential immune overresponse	Tumors	[109–111]
Bacterial	LPS, lipoproteins, etc.	Immune recognition; Deep tissue targeting; Scalable production	Safety concerns; Potential immune overresponse	Tumors, ischemic stroke	[52,61,112, 113]
Engineered	CD80/86, PD-1, etc.	Enhanced targeting; Multifunctional immune modulation; Deep penetration	Safety concerns; Potential immune overresponse	Tumors, inflammation	[43,59,114, 115]

Abbreviations: *CXCR4: C-X-C chemokine receptor type 4; CXCR2: C-X-C chemokine receptor type 2; CCR2: C-C chemokine receptor type 2; VCAM-1: Vascular cell adhesion protein 1; ICAM-1: Intercellular adhesion molecule 1; CD: Cluster of Differentiation; LPS: Lipopolysaccharide; MHC: Major histocompatibility complex; PD-1: Programmed cell death protein 1; BsAbs: Bispecific antibodies; SIRPα: Signal regulatory protein alpha.*

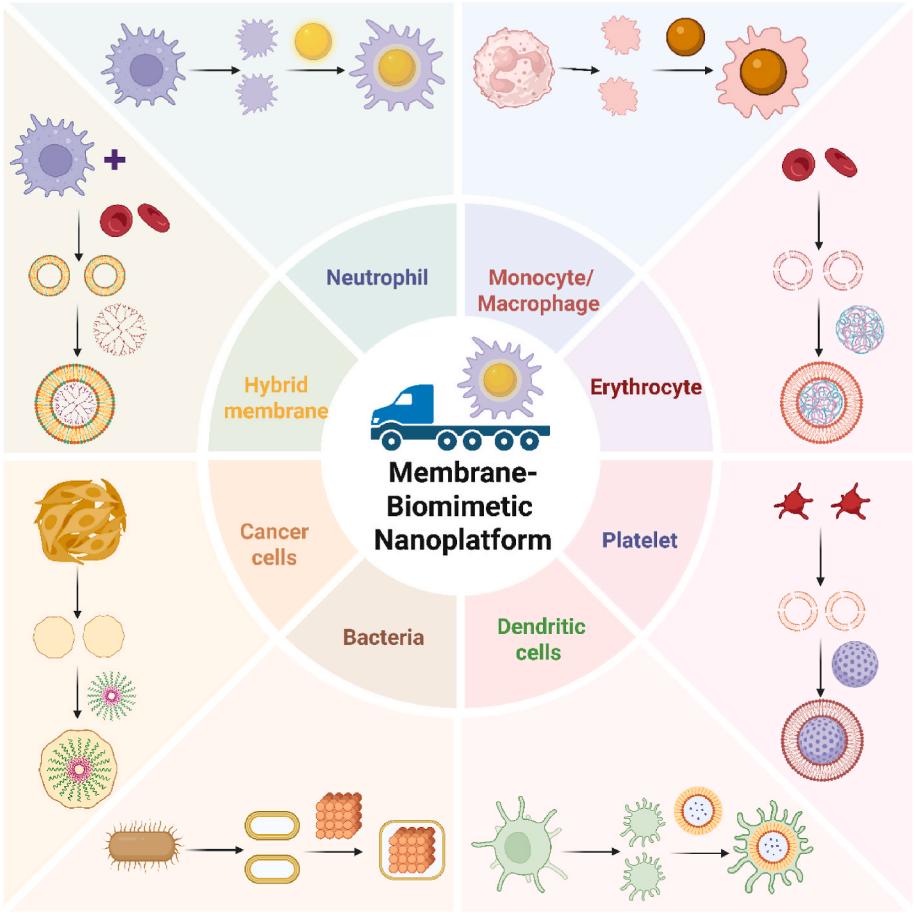

Fig. 1. Schematic of membrane sources for biomimetic nanoplatforms. This illustration demonstrates eight key membrane sources for biomimetic nanoparticles including neutrophil, monocyte/macrophage, hybrid, erythrocyte, cancer cell, platelet, bacterial, dendritic cell membranes and hybrid membrane. Created with Biorender **(Agreement number: MZ28A3FD68)**.

(CCL2) chemokines secreted by endothelial cells, enabling directed recruitment to inflammatory sites [84,121,122]; L-selectin facilitates macrophage adhesion and migration across endothelial cells [123]; while α4/β1 integrins enhance macrophage-tumor cell interactions through binding to tumor-overexpressed vascular cell adhesion protein 1 (VCAM-1) [124,125]. Membrane molecules such as CD45, CD11a, and glycans further reduce nanoparticle internalization by phagocytes or vascular endothelial cells, thereby improving tumor-targeting efficiency [126].

Building upon these inherent properties, MM-CN can effectively mimic the surface behavior of their source cells, with current biomimetic strategies falling into four main categories: **(1) Native macrophage membrane coating:** Wu et al. developed macrophage membrane-coated nanocomplexes that leverage natural inflammatory tropism to target fracture sites, co-delivering stromal cell-derived factor-1α peptide (sSDF-1α) and Ckip-1 small interfering RNA (siCkip-1) to significantly

enhance mesenchymal stem cell (MSC) recruitment and osteogenic differentiation, accelerating bone repair [89]. **(2) Macrophage membrane-liposome hybrids:** Fu et al. constructed macrophage membrane-hybridized lipid nanoparticles (Lipid NP@MM) through membrane fusion technology to protect and target-deliver THBS1-siRNA to vascular grafts, suppressing thrombospondin-1 (THBS1) expression and reducing intimal hyperplasia [90]. **(3) Polarization state-specific membrane applications:** Macrophage polarization primarily yields M1 (pro-inflammatory, anti-tumor) and M2 (anti-inflammatory, pro-tumor) phenotypes [119], enabling tailored therapeutic effects through selective membrane coating. For M1 applications, Liu et al. coated platinum nanoclusters with Porphyromonas gingivalis-stimulated M1 membranes (Pg-M-PtNCs) to target and eliminate brain bacteria while improving cognitive function in Alzheimer's disease model mice [91]. For M2 applications, Cao et al. designed M2 membrane-coated nanoparticles (M2FPPF@Cur) that minimize off-target effects while achieving efficient renal accumulation and prolonged retention in injured kidneys [92]. **(4) Hybrid membrane designs:** Xiong et al. created T cell-macrophage hybrid membrane nanoparticles (siIRF1@ZIF@HM NPs) that specifically target M1 macrophages to suppress interferon regulatory factor 1 (IRF1) and pyroptosis, effectively alleviating myocarditis [93].

3.3. Neutrophil membrane-coated nanoparticles (NM-CN)

Neutrophils, constituting 50 %–70 % of leukocytes, are the first effector cells to reach inflammatory sites and the most abundant immune population recruited during acute inflammation [127–129]. They eliminate pathogens through phagocytosis, neutrophil extracellular trap (NET) release, and reactive oxygen species (ROS) production [129–131], while their transendothelial migration capability enables penetration of physiological barriers like the blood-brain barrier (BBB). Neutrophil membranes express multiple receptors that recognize and respond to inflammatory signals. For instance, the abundant C-X-C chemokine receptor type 2 (CXCR2) on neutrophil membranes guides directional migration to inflammatory or tumor regions under C-X-C motif chemokine ligand 2 (CXCL2) chemokine guidance [132–135]. Additionally, L-selectin expressed on neutrophils mediates their recruitment to lesion sites [136–139], while β2 integrins interact with vascular endothelial adhesion molecules-1/2 (ICAM-1/2) ligands to facilitate endothelial barrier crossing [140–142]. These properties make neutrophil membranes ideal materials for biomimetic nanodrug design [143].

NM-CN have demonstrated unique therapeutic advantages in treating various diseases. In cardiovascular diseases, Wang et al. utilized neutrophil membrane-coated polydopamine nanoparticles to deliver puerarin, which disrupts the detrimental crosstalk between macrophages and pyroptotic cardiomyocytes, thereby protecting myocardial cells and improving cardiac function [94]. For pneumonia treatment, Li et al. engineered biomimetic nanovesicles (DHA@ANeu-DDAB) by fusing neutrophil membranes containing activated β2 integrins with lung-targeting lipids, leveraging both pulmonary homing capability and β2 integrin-mediated adhesion to deliver anti-inflammatory drugs to inflamed pulmonary vascular endothelial cells [142]. Similarly, Yu et al. developed neutrophil membrane-coated PLGA nanoparticles (LVX@PLGA@Mem) that efficiently accumulate in chronic obstructive pulmonary disease (COPD) lesions upon inhalation, simultaneously alleviating infection and inflammation [95]. In atherosclerosis research, Zhang et al. designed a neutrophil membrane-based nanoplatform (NNP^{ST}) that evades immune clearance through its "stealth" surface properties, prolongs circulation time, and actively targets atherosclerotic plaques to release simvastatin, effectively inhibiting disease progression [96]. These applications showcase the versatility of neutrophil membrane coatings in enabling targeted drug delivery while overcoming biological barriers.

3.4. Erythrocyte membrane-coated nanoparticles (EM-CN)

Erythrocytes (red blood cells, RBCs) are the most abundant and long-lived cells in the body. Their key advantages include [118]: **(1) Immune evasion mechanism:** The CD47-SIRPα interaction ("don't eat me" signal) prevents macrophage-mediated clearance, enabling prolonged circulation of RBC-coated nanoparticles [144–149]. Membrane proteins such as CR1, C8bp, and CD59 further minimize immunogenicity by inhibiting complement activation [150]. **(2) High abundance and easy extraction:** As the most abundant blood cells with a simple, enucleated structure, RBCs allow efficient membrane extraction and purification, facilitating scalable production of biomimetic nanocarriers [151]. **(3) Extended circulation time:** RBCs naturally persist for 100–120 days in circulation [152], a trait inherited by RBC membrane-coated nanoparticles to enhance drug delivery kinetics. While RBC membranes lack intrinsic targeting ability and require blood-type compatibility, strategies such as peptide functionalization or hybrid membrane fusion (e.g., with tumor-homing cells) have been employed to impart tissue-specific targeting [153].

The biomimetic strategies employing erythrocyte membranes are primarily demonstrated in the following aspects: **(1) Erythrocyte membrane-coated core structures:** Srivastava et al. utilized erythrocyte membranes to coat near-infrared fluorescent semiconducting polymer nanoparticles (SPNs), achieving tumor contour visualization and biomarker detection [97]. Dai et al. designed erythrocyte membrane nanoparticles (M@AP) co-loaded with photosensitizer P2-PPh3 and immunostimulant Poly(I:C), significantly inhibiting tumor growth through combined photodynamic-immunotherapy [98]. **(2) Erythrocyte membrane-liposome hybrids:** Chiang et al. formed nanovesicles by hybridizing erythrocyte membranes with liposomes, enhancing tumor cell uptake of nanoparticles and subsequently releasing drugs to suppress triple-negative breast cancer progression [99]. Tang et al. constructed RC-Lip nanoparticles that adsorbed bacterial toxin Hlα and released curcumin, promoting diabetic wound healing by modulating M2 macrophage polarization [63]. **(3) Erythrocyte membrane vesicles (EMVs):** EMVs can encapsulate siRNA, preventing drug clearance by macrophages while endowing nanoparticles with excellent biocompatibility and pharmacokinetic properties [100]. **(4) Hybrid erythrocyte membranes:** Hybrid cell membranes combine the characteristics of two membrane types, enabling synergistic execution of complex biological activities in physiological environments. For instance, erythrocyte-melanoma cell hybrid membranes exhibit both long circulation and tumor-targeting properties [43]. Similarly, Liu et al. fused erythrocyte membranes with macrophage membranes to form hybrid membrane shells that further camouflaged drug-loaded liposomes, creating biomimetic liposomes (AB@LRM). Guided by macrophage membrane tropism and leveraging erythrocyte membrane-derived prolonged residency, these precisely targeted inflammatory sites to release black phosphorus quantum dots that significantly enhanced bacterial antibiotic sensitivity, while concurrently releasing the conventional antibiotic amikacin for effective antibacterial action [57].

3.5. Platelet membrane-coated nanoparticles (PM-CN)

Derived from megakaryocyte cytoplasm, platelets are anucleate discoid cell fragments with a diameter of 2–3 μm, and they play pivotal roles in hemostasis, inflammation, and immune regulation [154]. Owing to their unique biological properties, platelets have emerged as promising biomimetic nanocarrier materials [118]. Specifically, they possess the following characteristics:**(1) Immune evasion:** Platelets exhibit inherent immune evasion capabilities, enabling an extended circulatory lifespan (8–9 days) [36–40,155]; This property stems from: CD47-mediated "don't eat me" signals that suppress macrophage phagocytosis; Complement inhibitors (CD55, CD59) that mitigate complement system activation, collectively enhancing nanoparticle circulation time [120]. **(2) Inflammation and tumor targeting:** P-selectin

binds CD44 on leukocytes, endothelial cells, and tumor cells, enabling therapeutic applications in inflammation, atherosclerosis, and oncology [85,86,156,157]. Additionally, integrin αIIbβ3 (GPIIb/IIIa) facilitates platelet-tumor cell heteroaggregate formation, paradoxically supporting tumor immune escape [158]. **(3) Vascular injury homing:** Platelets actively target damaged vasculature via P-selectin-mediated adhesion to ischemic endothelium, promoting tissue regeneration and modulating localized inflammation [104]. **(4) Immunomodulatory activiy:** Platelet membranes detect pathogens through Toll-like receptors and release chemokines such as C-X-C motif chemokine ligand 4 (CXCL4) to attract leukocytes [159]. However, a major challenge in utilizing platelet membranes is their propensity to aggregate in vivo, which may lead to thrombotic complications.

Leveraging these characteristics, PM-CN have achieved breakthrough therapeutic advances across multiple diseases: For atherosclerosis, Zhang et al. designed platelet membrane-coated black phosphorus nanosheets (PBP@siR@PM) to deliver Ca^{2+}/calmodulin-dependent protein kinase γ (CaMKIIγ)-silencing siRNA into macrophages, significantly inhibiting atherosclerotic progression in high-fat diet-fed $ApoE^{-/-}$ mice [101]. In inflammatory diseases, Yan et al. developed PM@Pic/PtCD@NP nanoparticles that exploit neutrophil recognition of platelet membranes for active transport to colitis lesions, where they release resveratrol and platinum-carbon dot nanozymes to scavenge ROS, suppress neutrophil infiltration, and alleviate ulcerative colitis [102]. For cancer applications, Dai et al. constructed platelet membrane-coated AIE luminogen nanoparticles (Plt-M@P) enabling tumor-targeted photodynamic therapy through PLGA/PF3-PPh3 complexes [62]. Similarly, Yu et al. engineered platelet-mimicking PLGA nanoparticles replicating natural platelet size/surface properties for dual small-molecule/protein tumor targeting [103]. In thrombotic disorders, Sun et al.'s discoid platelet membrane nanostructures (PLT-NDs) neutralize pathological antibodies while preventing platelet depletion and maintaining hemostatic function [104].

3.6. Dendritic cell membrane-coated nanoparticles (DCM-CN)

Dendritic cell (DC), as the most potent professional antigen-presenting cells in the immune system, serves as crucial bridges between innate and adaptive immunity. Following antigen processing, DC membranes present these antigens to T cells through major histocompatibility complex (MHC)-peptide complexes, while simultaneously delivering essential co-stimulatory signals (such as CD80/CD86) and cytokines (including IL-12) to robustly activate both $CD4^+$ and $CD8^+$ T cell responses [160–162]. Mature DCs highly express MHC-II and C-C chemokine receptor type 7 (CCR7) receptors, enabling them to migrate to lymph nodes and initiate immune responses [163,164]. While tumor microenvironments often suppress DC function, ex vivo tumor antigen-loaded DC vaccines can restore anti-tumor immunity [160] However, traditional DC vaccines face limitations including low antigen delivery efficiency, weak lymph node homing, and risks associated with live-cell infusion. To overcome these challenges, researchers have developed DC membrane-based biomimetic strategies that preserve natural antigen-presenting molecules and co-stimulatory signals while avoiding the safety risks of live-cell therapies. The main limitations of DC membranes include short circulation time, limited availability and high cost, and potential risks of excessive immune activation.

The biomimetic strategies utilizing DC membranes are primarily manifested in the following aspects: **(1) Native DC membrane coating of core structures:** Xu et al. developed DC membrane-coated nanoparticles containing aggregation-induced emission (AIE) photosensitizers (DC@AIEdots), where the interaction between ligands on DC membranes and corresponding receptors on T cells enables T cell-mediated transport to breast cancer regions. The encapsulated AIE photosensitizers generate ROS under light irradiation to kill tumor cells while simultaneously activating immune responses [105]. Similarly, Nguyen Thi Nguyen et al. created DC membrane-coated nanovaccines (Si9GM) that upregulate cytotoxic T cells, reduce regulatory T cells (Tregs), and promote M1 macrophage polarization, synergistically enhancing anti-tumor effects when combined with PD-1 blockade therapy [106]. **(2) Genetically engineered DC membranes:** Wang et al.'s HybridDC nanovaccine incorporates multiple functional elements from engineered DC membranes, including CCR7 for enhanced lymph node targeting, tumor-associated antigen peptides/tumor-derived exosomes for specific antigen presentation, and co-stimulatory molecules for optimized T cell activation. This design significantly improves immune checkpoint blockade efficacy and induces long-term immune memory [107]. **(3) Hybrid DC membrane systems:** Xu et al. employed fusion membranes derived from tumor cells and DCs as a cancer vaccine shell, combined with a near-infrared-II (NIR-II) absorbing polymer core to construct hybrid membrane nanoconstructs (SPNE). The tumor cell membrane provides autologous antigens, while the DC membrane retains antigen-presenting functions, and the NIR-II photothermal core enables deep tumor ablation. This multicellular collaborative design achieves potent T cell immunity alongside photothermal therapy [108].

3.7. Tumor cell membrane-coated nanoparticles (TCM-CN)

Tumor cell membranes, as the outermost structural barrier of cancer cells, possess unique molecular compositions and biological properties that offer novel approaches for tumor-targeted therapy and immunomodulation. Compared to normal cells, tumor cell membranes exhibit four characteristic advantages: **(1) Immune evasion capability:** Specific molecules on tumor cell membranes enable cancer cells to evade immune recognition and clearance. For instance, CD47 protein ("don't eat me" signal) binds to SIRPα on macrophages to inhibit phagocytosis [165–167], while PD-L1 expression suppresses T cell activity [168–170]. These properties not only facilitate tumor immune escape but also provide biomimetic strategies for prolonging nanocarrier circulation. **(2) Enhanced tumor vaccine potential:** Tumor cell surfaces are rich in tumor-associated antigens (TAAs) like **epidermal growth factor receptor (**EGFR), **human epidermal growth factor receptor 2 (**HER2), and **mucin 1 (**MUC1), along with specialized glycolipids [171–174]. This multi-antigenic profile offers inherent advantages over single-antigen vaccines that are susceptible to tumor microenvironment suppression. **(3) Homologous tumor targeting:** Tumor cell membranes express abundant adhesion molecules (e.g., N-cadherin, galectin-3, EpCAM) that mediate unique homotypic/heterotypic adhesion, promoting tumor cell aggregation and microenvironment interactions [175–181]. Furthermore, selectin-ligand interactions between tumor membranes and platelets/leukocytes/endothelial cells in the tumor microenvironment (via P-selectin, L-selectin, E-selectin) enhance homologous targeting [182–185]. **(4) Unlimited proliferation and scalable production:** Tumor cells' infinite proliferative capacity and ease of in vitro culture enable rapid expansion and large-scale membrane preparation. Current limitations primarily involve incomplete biosafety understanding and potential risks of excessive immune responses.

The applications of TCM-CN are demonstrated through several innovative approaches: Yin et al. developed 4T1 cancer cell membrane-coated Prussian blue nanoparticles (4T1-PB-CO NPs), where the membrane serves as exogenous TAAs to activate $CD4^+$ T cell responses and promote M1 polarization of tumor-associated macrophages. Concurrently, the released drug induces immunogenic cell death (ICD) in tumor cells, triggering damage-associated molecular patterns (DAMPs) release that enhances DC maturation and subsequent anti-tumor T cell responses [109]. Similarly, Li et al. created an arsenic-stimulated apoptotic H22 hepatoma cell-based biomimetic nanoparticle (arsenic/AB formulation). The tumor cell components facilitate macrophage-mediated delivery of arsenic to tumor sites, inducing hepatoma cell toxicity or apoptosis [110]. Furthermore, Huang et al. engineered cancer cell membrane-camouflaged poly(N-vinylcaprolactam)

(PVCL) nanogel vaccines. Leveraging membrane-mediated homologous targeting, these nanoparticles accumulate in tumor regions to release: (1) R837 for DC maturation and macrophage M1 polarization, (2) Mn^{2+} for enhanced MRI contrast, and (3) doxorubicin (DOX) for chemotherapy [111].

3.8. Bacterial membrane-coated nanoparticles (BM-CN)

Bacterial membranes possess distinctive structural and compositional features that confer unique biophysical properties, making them valuable platforms for nanomedicine applications. There are five key advantages for BM-CN: **(1) Immune recognition and targeted delivery:** Bacterial membranes are rich in pathogen-associated molecular patterns (PAMPs) like LPS and lipoproteins that are specifically recognized by immune cell PRRs [186,187]. This property enables dual applications: facilitating active uptake by immune cells for targeted delivery to disease sites, while their strong immunogenicity makes them ideal for developing bacterial vaccines or immune adjuvants capable of eliciting robust cellular and humoral immune responses. **(2) Deep tissue targeting capacity:** Certain bacterial strains express surface receptors that recognize tumor or lesion-specific antigens [188–191], allowing CMC-NPs to penetrate physiological barriers for precise deep-tissue delivery and enhanced drug accumulation. **(3) Unique biophysical properties:** The LPS layer of Gram-negative bacteria provides immune evasion capabilities, while the thick peptidoglycan layer of Gram-positive bacteria offers mechanical stability [192,193], making both membrane types attractive for engineering novel drug delivery systems. **(4) Natural vesicle systems:** Bacteria secrete extracellular vesicles (EVs, 20–250 nm) that naturally carry immunogenic antigens and PAMPs [194], serving as ideal drug carriers that can simultaneously modulate host immune responses. **(5) Scalable production:** Modern biotechnology enables large-scale production of engineered bacteria through genetic modifications to reduce toxicity, achieve surface uniformity, and ensure batch consistency [195–198], facilitating clinical translation. Current limitations primarily involve incomplete biosafety profiles and potential risks of excessive immune activation.

Capitalizing on the unique structural and functional properties of bacterial membranes, researchers have developed several groundbreaking delivery platforms with distinct therapeutic advantages: **(1) Bacterial membrane-coated core structures:** Addressing uneven nanoparticle distribution in tumors, Yang's team pioneered Fusobacterium nucleatum membrane-coated covalent organic frameworks (COFs). This design achieves dual benefits: the bacterial membrane significantly improves tumor-wide COF distribution and cancer cell uptake, while F. nucleatum components act as natural immunoadjuvants to activate $CD8^+$ T cells and B cells, converting immunologically "cold" tumors into "hot" microenvironments [61]. **(2) Bacterial membrane-liposome hybrids:** To overcome BBB limitations, a hybrid delivery system (B-Lipo) was created by fusing attenuated Salmonella VNP20009 membranes with liposomes. Co-loaded with 1-methyl-D-tryptophan (1-MT) and curcumin (Cur), the B-Lipo/1-MT&Cur nanoparticles exploit neutrophil-mediated transport: bacterial membrane components enable neutrophil recognition, leveraging these cells' chemotactic properties to cross the BBB and accumulate in brain tumors, triggering potent anti-tumor immunity [52]. **(3) Outer membrane vesicles (OMVs):** OMVs, as natural nanocarriers, have demonstrated exceptional therapeutic potential across multiple diseases. In ischemic stroke treatment, researchers encapsulated the neuroprotective agent pioglitazone (PGZ) into bacterial-derived OMVs to form OMV@PGZ nanoparticles, where OMVs mediate neutrophil phagocytosis, enabling PGZ to cross the blood-brain barrier via neutrophil transport and exert neuroprotective effects in cerebral ischemic regions [112]. For cancer therapy, OMVs can co-load cisplatin and photothermal conversion materials (PBIBDF-BT) - their surface PAMPs are recognized and engulfed by neutrophils, which then carry the nanoparticles to tumor sites for simultaneous chemotherapy and photodynamic therapy upon drug release [47]. Similarly, Sun et al. further showcased OMV versatility by chemically conjugating nanobodies targeting both CD47/SIRPα and PD-1/PD-L1 pathways onto OMV surfaces, enhancing tumor-specific delivery of these nanobodies for potent antitumor effects [113]. Chen et al. developed a novel biomimetic nanodelivery system by loading dihydroartemisinin (DHA) into iron-doped hollow mesoporous silica nanoparticles (Fe-HMSNs), followed by surface modification with OMVs to construct the D@FMN-M nanocomposite. This nanoparticle exhibits TME-responsive degradation, enabling synchronous release of Fe^{3+} and DHA to specifically induce ferroptosis in both tumor cells and pro-tumoral M2-type macrophages while remodeling the immunosuppressive tumor microenvironment [199]. These findings demonstrate that OMVs serve as an effective drug delivery platform with broad application prospects in the treatment of various diseases.

3.9. Genetically engineered cell membranes-coated nanoparticles (GECM-CN)

While natural cell membranes can confer favorable biocompatibility, immune evasion, and targeting capabilities to nanoparticles, their intrinsic properties often fall short of meeting the complex demands of disease treatment. To overcome these limitations, researchers have turned to genetic engineering techniques to precisely modify cell membranes, either by enhancing their native functions or introducing novel functional ligands. This approach significantly improves nanoparticle targeting, multi-effect immunomodulation, and deep tissue penetration capabilities [34,200–204]. However, current applications of genetically engineered cell membranes face limitations, primarily concerning incomplete biosafety profiles and potential risks of excessive immune responses.

In the application of GECM-CN, three major innovative strategies have emerged: **(1) Enhanced targeting:** Genetic engineering enables precise expression of specific targeting molecules (e.g., antibody fragments, ligands, or receptor-binding proteins) on cell membranes to improve nanoparticle delivery efficiency. For example, Ji et al. designed Prussian blue nanoparticles (SIRPα-M@nanoPB) coated with genetically modified macrophage membranes that stably overexpress SIRPα. By actively targeting and binding to CD47 on tumor cells, SIRPα effectively counteracts the immunosuppressive tumor microenvironment while restoring M1 macrophage polarization, thereby triggering a robust immune response [43]. Similarly, Nishta Krishnan et al. engineered cell membranes to express SpyCatcher membrane anchors, which readily form covalent bonds with SpyTag-modified ligands. When loaded with chemotherapeutic drugs, these modularly functionalized nanoparticles demonstrated strong targeting capabilities and significant tumor growth inhibition in ovarian cancer xenograft models [114]. **(2) Multifunctional immunomodulation:** Genetic engineering can introduce immune-regulatory molecules (e.g., co-stimulatory signals like CD80/CD86 or immune checkpoint inhibitors like PD-1 antibodies) to enhance anti-tumor immunity. Yusuke Ito et al. genetically modified leukemia K562 cells to express bispecific antibodies (BsAbs), co-stimulatory ligands, cytokines, and immune checkpoint-blocking antibodies. These engineered membrane components were then reassembled onto PLGA surfaces to create CMC-NPs capable of inducing potent anti-tumor immune responses [115]. **(3) Improved deep tissue penetration:** Huang et al. genetically engineered macrophage membranes to express the tumor-penetrating peptide tLyp-1, significantly enhancing nanoparticle chemotaxis and deep penetration into inflammatory tumor sites. These modified membranes were used to coat multifunctional biomimetic gold nanoparticles (GNR-SNO@MMT), which, under NIR-II laser irradiation, achieved deep tumor penetration and effective ablation [59]. In summary, genetically engineered cell membranes provide a highly customizable functionalization strategy for biomimetic nanoparticles, demonstrating immense potential in cancer therapy, gene editing, and immunomodulation. Current research continues to refine these techniques to optimize safety, efficacy, and clinical

translatability.

4. Therapeutic applications of CMC-BDS for different diseases

CMC-BDS demonstrates remarkable versatility across diverse biomedical applications. By harnessing natural cell membranes - including those from immune cells, red blood cells, or platelets - these innovative systems effectively encapsulate therapeutic payloads while achieving both targeted delivery and immune evasion. CMC-BDS technology shows particular promises for the therapy for the tumor, ischemic stroke and inflammatory conditions. This multifaceted applicability stems from the systems' unique ability to mimic biological cells, which significantly enhances drug stability, improves tissue-specific targeting, and minimizes off-target effects (Fig. 2).

4.1. Tumor therapy

The global cancer burden continues to escalate, with rapidly increasing incidence and mortality rates [205]. Current cancer treatment faces three major challenges: difficulties in early diagnosis, high risks of metastasis/recurrence, and severe toxic side effects from conventional radiotherapy/chemotherapy [206–210]. To address these challenges, NDDS leveraging the tumor-specific EPR effect have emerged as critical platforms for delivering chemotherapeutic agents, radiosensitizers, gene therapies, and immunomodulators [211,212]. However, mononuclear phagocyte system (MPS) clearance significantly limits therapeutic efficacy [213].

Against this backdrop, cell membrane-coated nanoparticle technology has emerged as an innovative solution, effectively addressing these critical challenges through the utilization of natural biological membranes or engineered hybrid membranes. The technology's core advantages lie in three key aspects: (1) The outer membrane coating provides a natural protective barrier for drug-loaded systems, enhancing nanostructural stability while minimizing drug leakage [214]; (2) Preservation of source cells' biological characteristics significantly improves tumor-targeting specificity; (3) Immune camouflage capability substantially prolongs blood circulation time. In recent years, CMC-BDS have achieved groundbreaking progress in cancer therapeutics, offering innovative delivery platforms for multiple treatment strategies.

4.1.1. Chemotherapy for tumors

Membrane coating technology has markedly enhanced conventional chemotherapy outcomes. The Lai team developed MM@DOX-Tet nanoparticles, where macrophage membrane-hybridized liposomes co-deliver DOX and the multidrug resistance reversal agent tetracycline (Tet), simultaneously improving tumor targeting, cellular uptake, and overcoming drug resistance [215]. Similarly, Sancho-Albero's group achieved efficient targeted doxorubicin delivery using

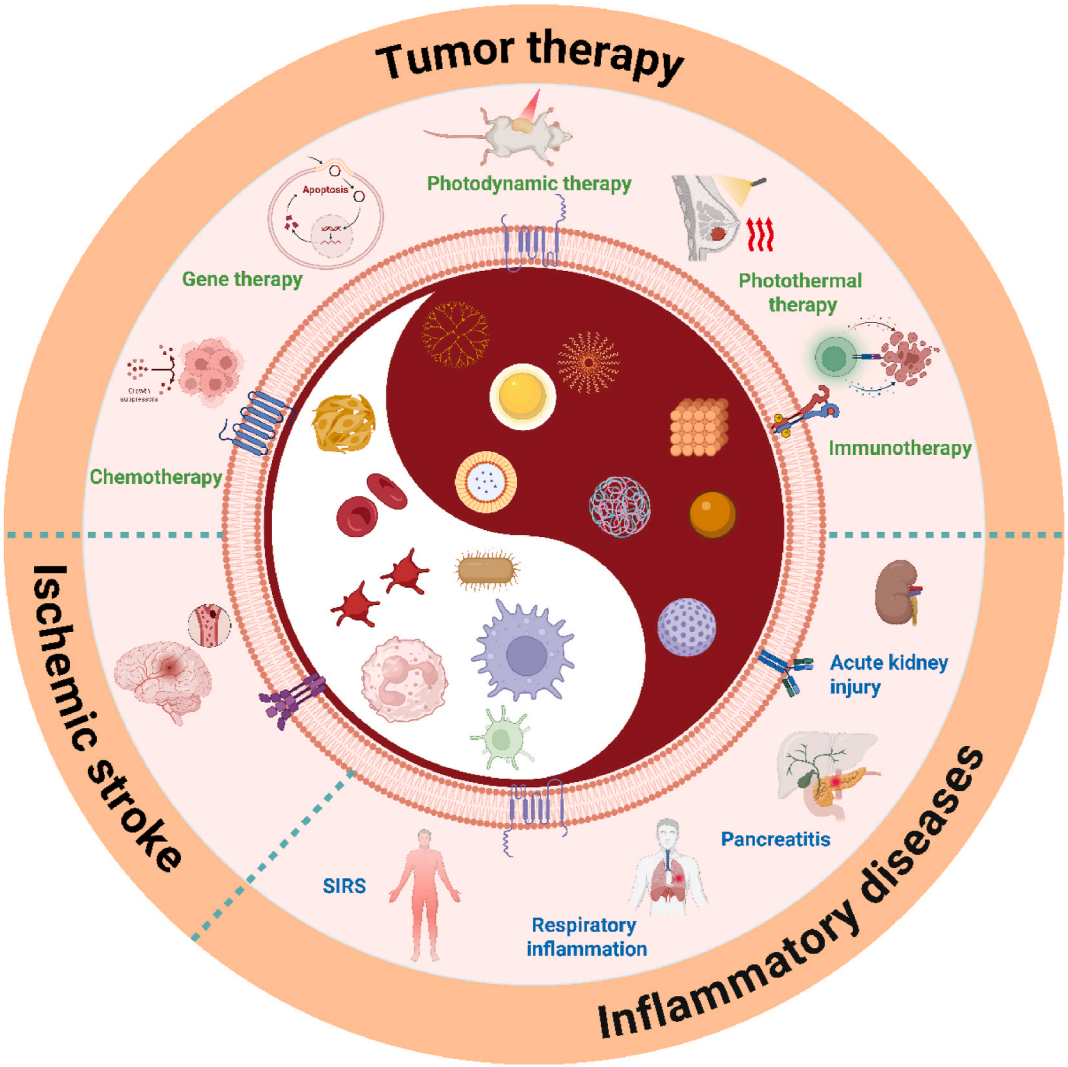

Fig. 2. Schematic for the applications of CMC-BDS in biomedicine. Created with Biorender (Agreement number: TZ28A3G7EU).

melanoma-derived extracellular vesicle membranes [216]. Furthermore, Zhou et al.'s mRDZ biomimetic nanosystem, camouflaged with macrophage membranes, accumulates in tumors to release DOX, effectively suppressing primary tumor growth, lung metastasis, and recurrence [217]. These advances provide critical insights for optimizing chemotherapeutic delivery.

4.1.2. Gene therapy for tumors

Membrane coating technology has pioneered novel delivery pathways for nucleic acid drugs in cancer gene therapy. Wu et al. developed an engineered macrophage membrane-hybridized liposome system (PMLip@siGFAT1) that prolongs nanoparticle circulation time while enhancing penetration into inflammatory vascular endothelium. Upon reaching tumor sites, these nanoparticles release specific small interfering RNA (siRNA) to suppress the hexosamine biosynthesis pathway in cancer cells, mitigating hyaluronic acid-mediated chemoresistance in pancreatic cancer and thereby potentiating chemotherapy [218] (Fig. 3). Similarly, Li et al.'s PH20/CCM@PMCS nanoparticles leverage hybrid membranes from cancer cells and fibroblasts to co-deliver carboplatin and p65-targeting siRNA (sip65), significantly improving

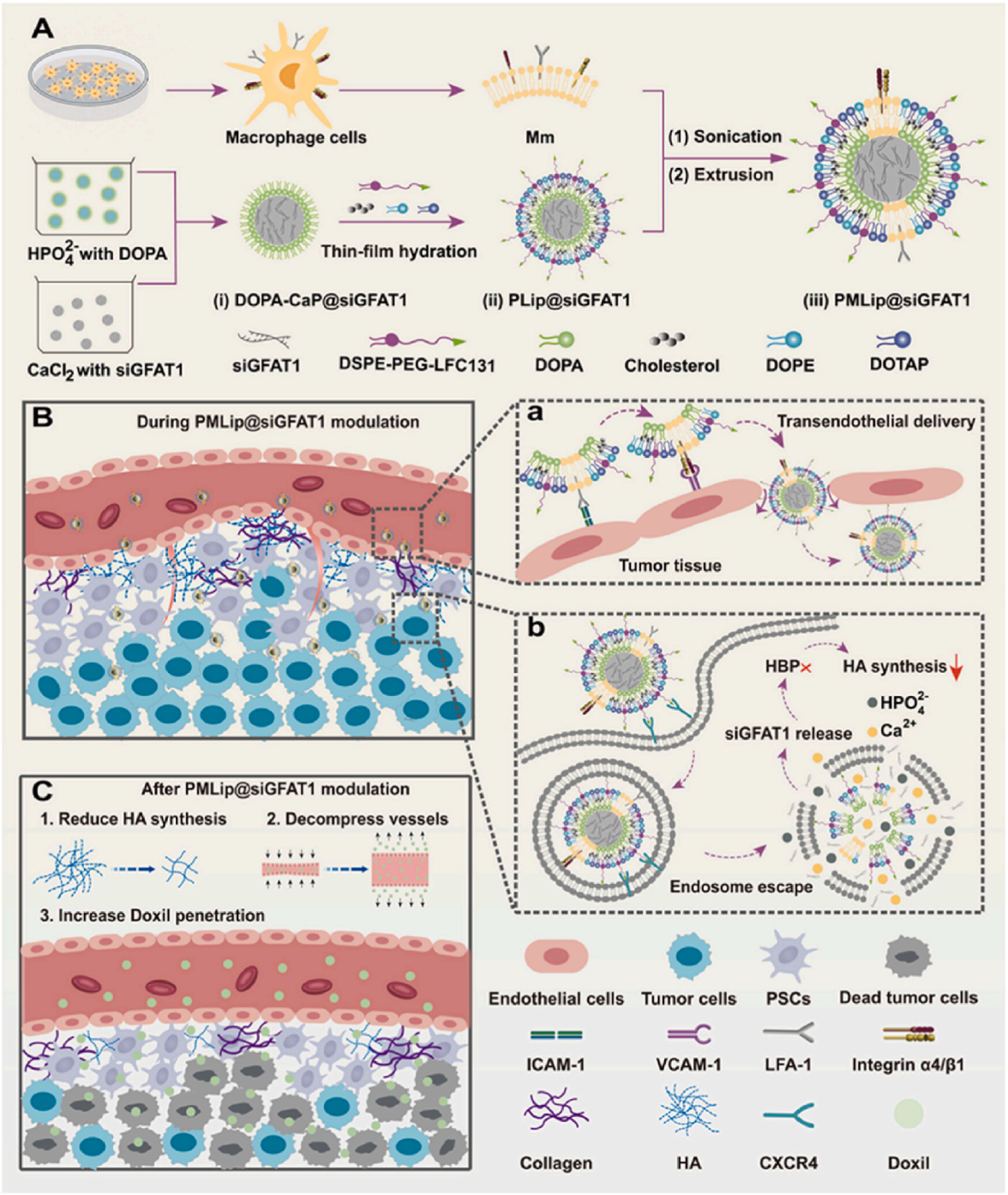

Fig. 3. (A) Schematic illustration of the PMLip@siGFAT1 assembly process. (B) Following intravenous injection, PMLip@siGFAT1 can evade immune clearance, traverse the tumor inflammatory endothelium, be preferentially taken up by tumor cells, and escape from endosomes, releasing siGFAT1 in the cytoplasm. (C) PMLip@siGFAT1 reduces HA turnover by silencing GFAT1, increasing the delivery and efficacy of Doxil. Mm: macrophage cell membrane. Reprinted from Ref. [218] with permission. Copyright © 2025 American Chemical Society.

ovarian cancer treatment outcomes [219]. These breakthroughs provide critical technical support for advancing gene therapy applications in oncology.

4.1.3. Photodynamic therapy for tumors

Photodynamic therapy (PDT), a non-invasive treatment modality, selectively destroys cancer cells through light-activated ROS generation by photosensitizers while exhibiting minimal toxicity to unirradiated normal tissues. Leveraging this principle, researchers have developed innovative membrane-coated nanodelivery systems: Zhang et al. engineered leukocyte/platelet hybrid membrane-coated dendritic large-pore mesoporous silica nanoparticles (DLMSNs) loaded with near-infrared (NIR) dye IR780 and DOX, creating LPHM@DDI nanoparticles. Under NIR laser irradiation, LPHM@DDI nanoparticles effectively suppressed triple-negative breast cancer (TNBC) growth and recurrence in mice through combined tumor ablation and anti-angiogenesis [58]. Yu et al.'s 4T1 cell membrane-coated TUC@ZMS nanoparticles generated singlet oxygen upon 980 nm NIR activation, inducing immunogenic cell death (ICD) while simultaneously treating primary breast tumors and inhibiting pulmonary metastases [220]. These advances significantly expand PDT's clinical potential.

4.1.4. Photothermal therapy for tumors

Photothermal therapy (PTT) utilizes light-absorbing agents to generate localized heat under high-intensity NIR laser irradiation, inducing thermal ablation of cancer cells. Yu et al. developed cancer cell membrane-coated F127/(R837 and IR1048) nanoparticles (CFRI), where the tumor cell membrane modification conferred superior tumor-targeting capability. These nanoparticles encapsulated NIR-II photothermal agent IR1048 and immunostimulant R837, demonstrating a high photothermal conversion efficiency of 49 % for effective ablation of primary tumors [221]. Zhang et al. constructed biomimetic nanoparticles (HM@I/NPs) by coating the photothermal agent indocyanine green (ICG) with H22 cancer cell membranes. Under NIR laser irradiation, ICG generated temperatures up to 56.7 °C for efficient tumor ablation, while pH-responsive release of DOX directly killed tumor cells. Concurrently, released sulindac suppressed cyclooxygenase-2 (COX-2) expression in the tumor microenvironment to enhance therapeutic efficacy [222] (Fig. 4). These advances provide novel strategies for combinatorial tumor therapy.

4.1.5. Immunotherapy for tumor

As one of the most promising frontiers in cancer therapeutics, tumor immunotherapy focuses on activating or restoring the body's anti-tumor immune response to control and eliminate malignancies. Nanomedicines are widely utilized in this field [223–225], with cell membrane coating technology emerging as an innovative strategy to overcome the limitations of conventional immunotherapy.

(1) Tumor Antigens and Immunoadjuvants

The development of tumor vaccines is undergoing a transformative shift from traditional formats to nanotechnology-based approaches. Next-generation nanovaccines address critical challenges such as weak immunogenicity and inefficient delivery through the sophisticated integration of multiple tumor antigens and immune stimulants. These innovative vaccines offer dual advantages: enhancing antigen presentation by optimizing interactions with DCs while enabling sustained antigen stimulation through controlled release mechanisms [226]. The Krishnan team pioneered a pluripotent stem cell membrane-coated nanoparticle system loaded with adjuvants. Leveraging the immunostimulatory properties of membrane-bound carcinoembryonic antigens, this vaccine induced broad-spectrum anti-tumor immunity across multiple cancer models [227] (Fig. 5). The Lemjabbar-Alaoui group

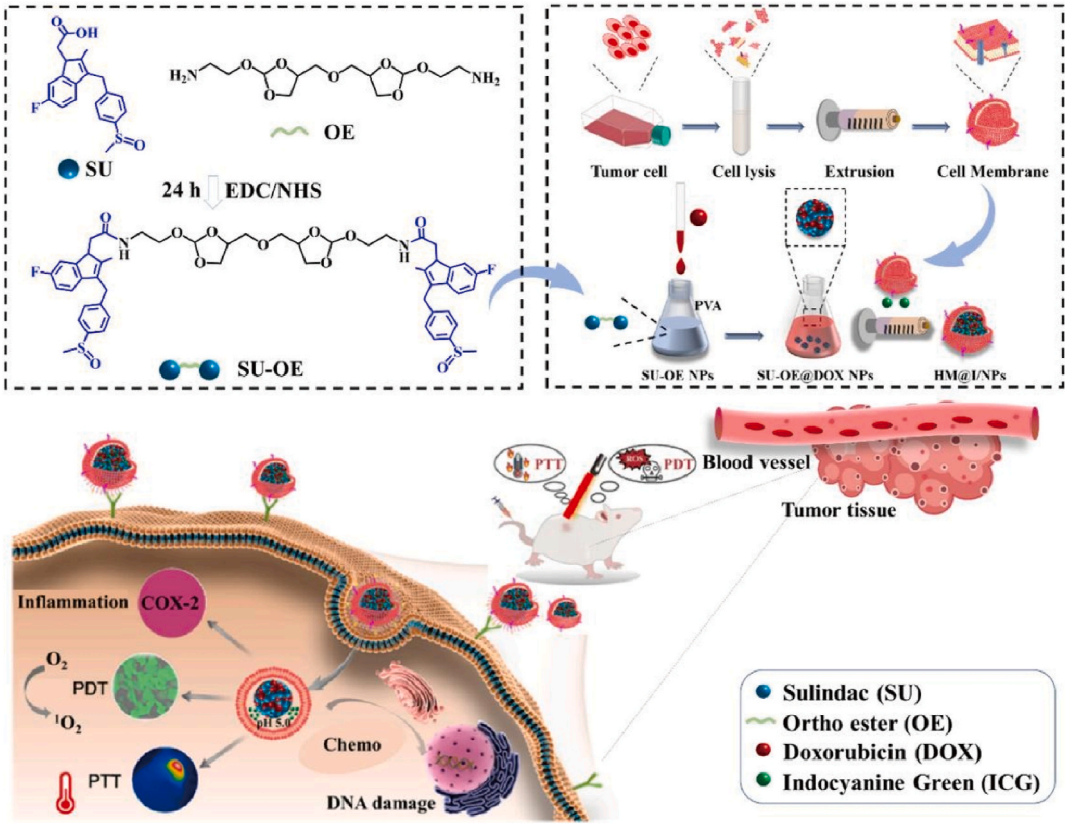

Fig. 4. Schematic diagram of construction of biomimetic nanodrugs for cancer cell membrane based on small molecule prodrug of sulindac-ortho ester and chemotherapy-phototherapy synergistic antitumor study in vitro and in vivo. Reprinted from Ref. [222] with permission. Copyright © 2025 Elsevier Ltd.

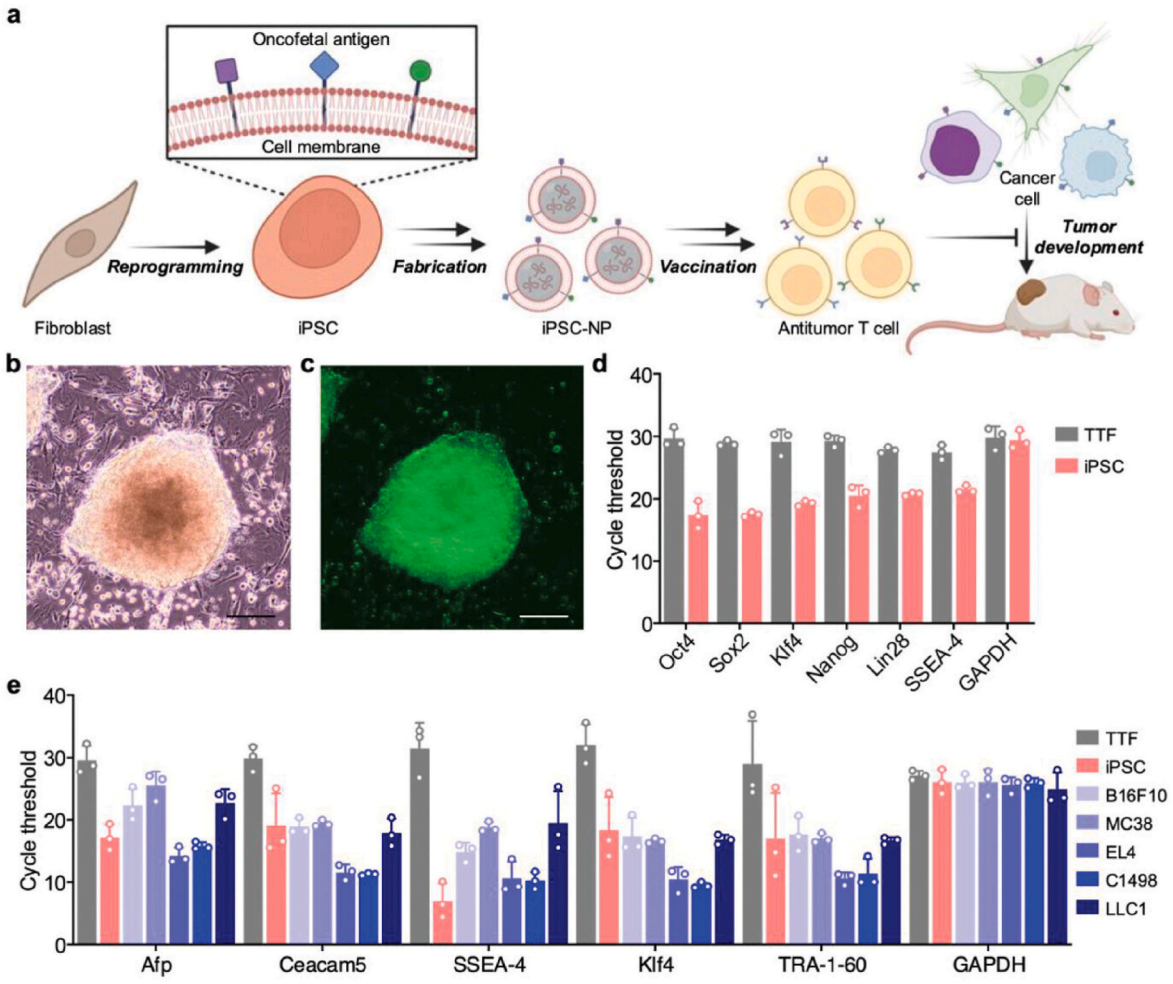

Fig. 5. Reprogramming of TTFs to iPSCs. a) TTFs are reprogrammed to iPSCs, which express oncofoetal antigens that are shared by various types of cancer. The membrane derived from these iPSCs is coated onto adjuvant-loaded polymeric cores, forming iPSC-NPs that can elicit antitumor T cell responses and be used as a broad-spectrum prophylactic cancer vaccine. Created with BioRender. b) Brightfield visualization of iPSC colony formation over a feeder cell layer. Scale bar: 100 μm. c) Alkaline phosphatase staining (green) of iPSCs indicating pluripotency. Scale bar: 100 μm. d) Cycle threshold for the RT-PCR analysis of pluripotency factor expression in TTFs and iPSCs (n = 3, mean +SD). e) Cycle threshold for the RT-PCR analysis of oncofoetal antigen expression in TTFs, iPSCs, and various cancer cell lines (n = 3, mean +SD). Reprinted from Ref. [227] with permission. Copyright © 2025 Wiley-VCH GmbH.

developed dendritic cell membrane-coated mRNA nanoparticles (DPNs), demonstrating the potential of ovalbumin (OVA) mRNA vaccines. Notably, ovalbumin-encoding DPN-OVA simultaneously activated robust cellular and humoral immunity, effectively suppressing tumor growth [228]. Zhang et al.'s M@BCG nanoparticles combined macrophage membrane targeting with the immunomodulatory effects of Bacillus Calmette-Guérin (BCG), offering a novel strategy for lung cancer immunotherapy [229].

(2) Immunomodulation of the Tumor Microenvironment

The immunosuppressive nature of the tumor microenvironment (TME) remains a major therapeutic barrier. To address this, membrane-coated nanoplatforms have been engineered to reprogram the TME through multiple mechanisms. While checkpoint molecules maintain immune homeostasis physiologically, tumors exploit their overexpression for immune evasion [230]. Xu et al.'s M1 macrophage membrane-coated PAP nanoparticles dualistically preserved M1 macrophage targeting capacity while delivering anti-PD-L1 siRNA to reactivate cytotoxic T lymphocyte (CTL)-mediated tumor killing [231] (Fig. 6).Cheng et al.'s PMNP-SAB@RTM hybrid system merged RBCs' prolonged circulation with fibroblast membrane's stromal targeting. By delivering salvianolic acid B (SAB) to degrade extracellular matrix (ECM), it enhanced T-cell penetration and synergized with checkpoint inhibitors, achieving successful "cold-to-hot" tumor conversion [232].

Although cell membrane-coated nanoparticle technology has demonstrated revolutionary potential in tumor therapy, several critical challenges must be addressed during its clinical translation: **(1) Deep tumor penetration barriers:** While current modifications with targeting peptides or specific ligands have significantly improved nanoparticle penetration, two characteristic barriers in the tumor microenvironment continue to limit therapeutic efficacy. On one hand, abnormally elevated interstitial fluid pressure creates a physical barrier; on the other, the dense ECM produces a molecular sieving effect. These factors collectively hinder complete nanoparticle penetration throughout tumor regions, allowing deep-seated tumor cells to evade treatment. Future research should focus on developing novel penetration-enhancing strategies, such as combining matrix-degrading enzymes with physical-assisted delivery technologies, to overcome these microenvironmental limitations. **(2) Personalized therapy challenges:** The high heterogeneity of tumors poses significant obstacles for treatment precision. Notable variations exist in microenvironment characteristics, membrane protein expression profiles, and drug sensitivity among different tumor types—and even among different patients with the same tumor type. Current standardized nanoformulations cannot meet individualized treatment requirements. There is an urgent need to establish precision

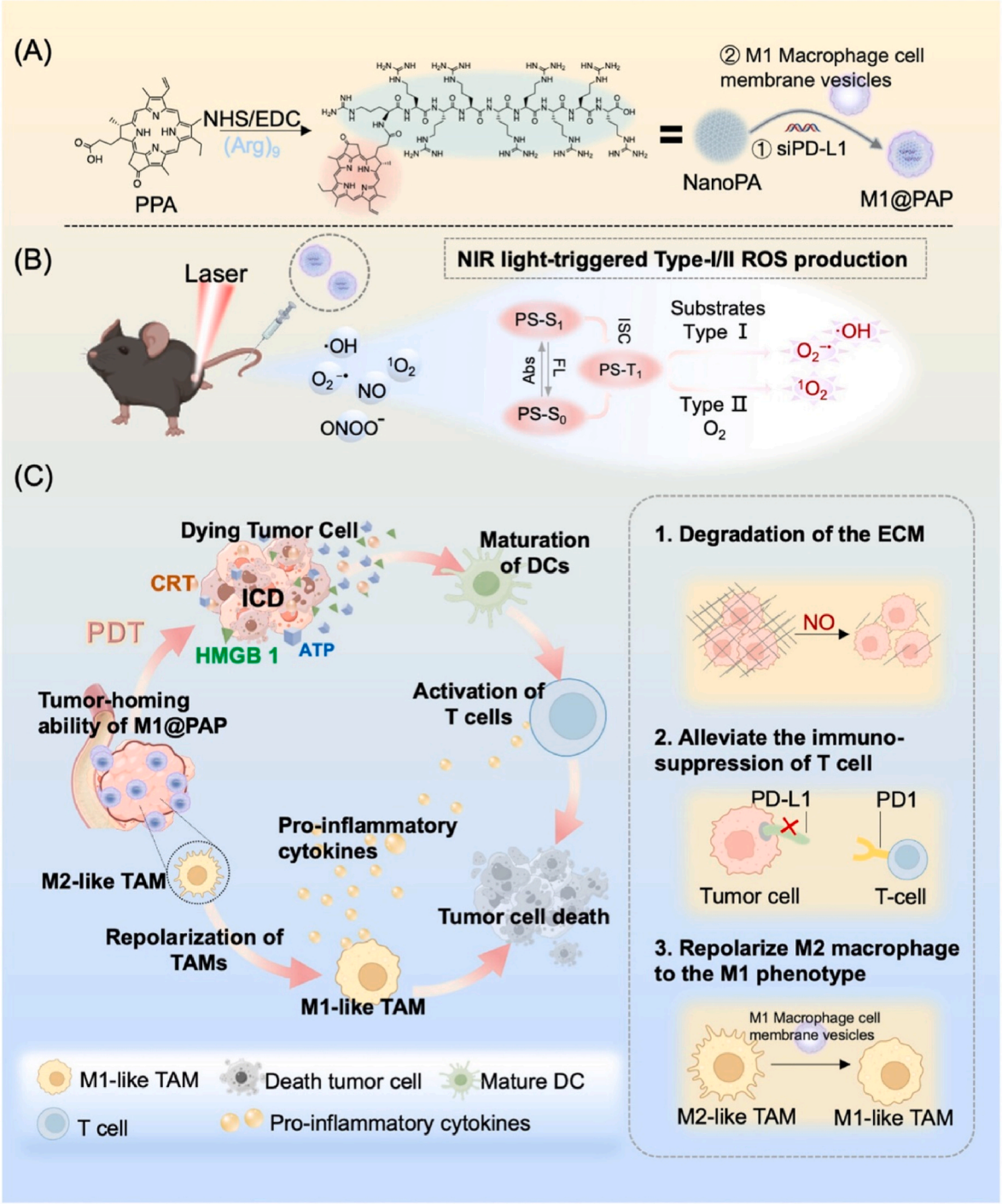

Fig. 6. (A) Preparation of M1@PAP. Arg_9 acted as a NO donor and was covalently linked to the photosensitizer PPA to prepare nanoparticles, then electrostatically adsorbed siPD-L1. The final preparation M1@PAP was formed by encapsulating with the prepared M1 macrophage membrane vesicles. (B) M1@PAP as a ROS supergenerator that integrated NIR light-triggered Type I/II ROS production, Arg_9 further generated NO and $ONOO^-$ under the stimulation of ROS. (C) M1@PAP modulated TME to enhance PDT combined with anti-PD-L1 immunotherapy. M1@PAP had excellent tumor targeting and breached the physical barrier. PDT can expose the CRT proteins on the surface of tumor cells, thus promoting the DCs maturation and antigen presentation. Also, M1@PAP can promote repolarization of TAMs, release pro-inflammatory factors, and further enhance $CD8^+$T activation. Reprinted from Ref. [231] with permission. Copyright © 2025 American Chemical Society.

modification platforms based on patient-specific tumor characteristics, integrating multi-omics analysis and rapid screening technologies to develop truly personalized nanomedicines. **(3) Biosafety concerns:** Although existing studies generally report good biocompatibility of CMC-NPs, long-term safety evaluations of special membrane sources (e.g., cancer cell membranes) remain inadequate. Particular attention should be paid to: potential risks from residual tumor-associated antigens on membranes, immunogenicity changes after repeated administration, and cumulative toxicity in vital organs. Therefore, more systematic and stringent safety evaluation systems must be established, including long-term tracking studies and large animal experiments, to provide comprehensive safety evidence for clinical applications.

4.2. Ischemic stroke

Ischemic stroke, one of the leading global causes of disability and mortality, presents significant therapeutic challenges. This acute condition, triggered by cerebrovascular occlusion, not only induces severe neurological deficits but may also lead to long-term disability [233, 234]. To address this clinical dilemma, CMC-BDS have emerged with unique therapeutic advantages, offering novel solutions for ischemic stroke treatment. For instance, Ma et al. designed a macrophage membrane-camouflaged nanocomplex (MM@AK137/FN) loaded with the antioxidant edaravone, achieving dual modulation of microglia and neurons, which significantly enhanced therapeutic efficacy in ischemic stroke [235]. The Pan research group developed an innovative OMV@PGZ system, utilizing extracellular vesicles to deliver PGZ. Through a neutrophil-mediated transport mechanism, this system not only suppressed NLRP3 inflammasome activation but also effectively inhibited ferroptosis, providing a new strategy to mitigate reperfusion injury [112]. Li et al. constructed a PUE@PDA@CMs nanoplatform by coating macrophage membranes and loading puerarin, which demonstrated excellent ROS-scavenging capability, significantly improving the oxidative stress microenvironment in ischemic brain tissue [236]. Wang et al. developed neutrophil membrane-coated porous metal-organic framework nanoparticles (M-MOF) loaded with NOX4-targeting siRNA (siNOX4). In the ischemic lesion area, siNOX4 specifically bound to activated endothelial cells, significantly suppressing NADPH oxidase 4 (NOX4) expression, reducing ROS production, and consequently alleviating cerebral damage while preserving neurological function [237] (Fig. 7).

These innovative studies have developed targeted therapeutic strategies against critical pathological processes in ischemic stroke (e.g., oxidative stress, inflammatory responses, and cell death) through diverse membrane engineering approaches. With deepening understanding of stroke's molecular mechanisms and continuous advancement of novel materials, cell membrane-coated nanomedicines are poised to bring transformative progress to ischemic stroke treatment. Future research should prioritize enhancing blood-brain barrier penetration efficiency and extending the therapeutic time window to further improve clinical translation potential.

4.3. Inflammatory diseases

Inflammatory diseases, as common clinical disorders, present significant therapeutic challenges including poor targeted delivery and low drug bioavailability. To address these issues, CMC-BDS have attracted considerable attention due to their superior inflammatory targeting capability and drug protection capacity. These systems demonstrate unique advantages for treating various inflammatory diseases.

In respiratory inflammation, Zhao's team innovatively developed a macrophage membrane-coated astaxanthin nanoparticle (Mϕ@Ast-NPs) intranasal delivery system. Leveraging macrophages' inherent inflammatory tropism, this system precisely delivers the potent antioxidant astaxanthin to inflammatory sites in acute lung injury/acute respiratory distress syndrome (ALI/ARDS), significantly enhancing anti-inflammatory efficacy [238]. **For pancreatitis,** Shi et al. developed macrophage membrane-coated selenized Poria cocos polysaccharide nanoparticles that combine targeted delivery through macrophage homing effects with autophagy restoration via protein kinase b/mechanistic target of rapamycin (AKT/mTOR) pathway modulation, effectively mitigating pancreatic inflammation [239] (Fig. 8). **In systemic inflammation,** Qu's group created an innovative macrophage membrane-coated nanosystem co-loaded with tannic acid and quercetin that synergistically combats SIRS (systemic inflammatory response syndrome) through ROS scavenging and mitochondrial repair [240]. **For acute kidney injury (AKI) therapy,** Deng et al. engineered a biomimetic MM-PtNCs platform utilizing macrophage membrane-encapsulated, ROS-responsive platinum nanozyme clusters that target renal inflammation through surface cytokine receptors while neutralizing pro-inflammatory factors [241]. In colitis treatment, Ma et al.'s Ber@MVs-CA nanoparticles effectively deliver berberine to inflammatory colon lesions by harnessing macrophage membrane

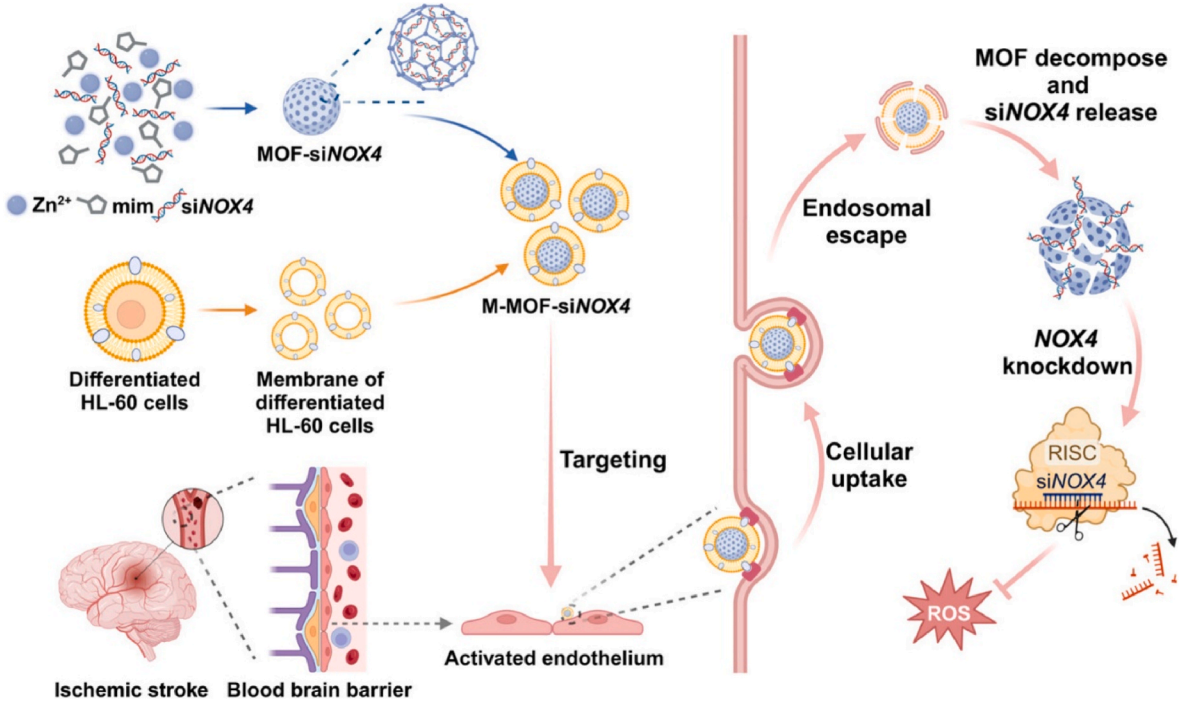

Fig. 7. The diagram illustrates a novel therapeutic strategy involving neutrophil-like cell membrane-coated porous metal-organic framework nanoparticles loaded with siNOX4 (M-MOF-siNOX4), which are designed to target damaged brain microvascular tissue. These M-MOF-siNOX4 nanoparticles specifically bind to activated endothelial cells, effectively reducing NOX4 expression, decreasing both reactive oxygen species (ROS) production and cell apoptosis, and restoring cell viability. Reprinted from Ref. [237] with permission. Copyright © 2025 Elsevier Ltd.

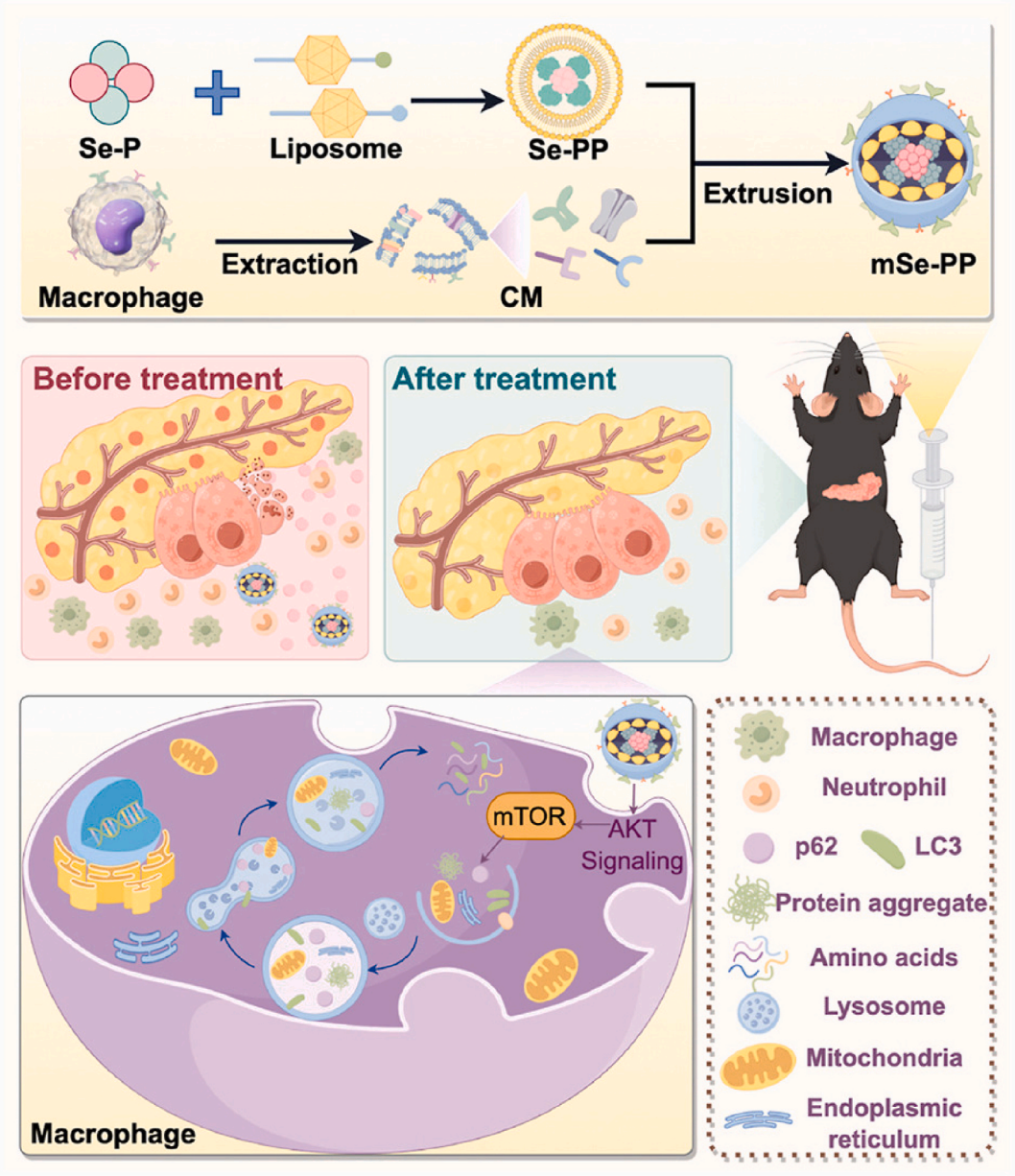

Fig. 8. The diagram illustrates a surface masking strategy that involves coating the surface of selenylated Poria cocos polysaccharide nanoparticles with a layer of macrophage plasma membrane to circumvent MPS sequestration. Mechanistic studies elucidate that macrophage membrane-biomimetic selenylated Poria cocos polysaccharide nanoparticles primarily mitigate pancreatic inflammation by inhibiting the AKT/mTOR pathway to reverse autophagic flux impairment. Reprinted from Ref. [239] with permission. Copyright © 2025 Elsevier Ltd.

tropism, showing outstanding therapeutic performance against ulcerative colitis [242].

To address this challenge, we herein demonstrate a surface masking strategy that involves coating the surface of selenylated Poria cocos polysaccharide nanoparticles with a layer of macrophage plasma membrane to circumvent MPS sequestration, thereby enhancing the therapeutic efficacy of selenylated Poria cocos polysaccharide nanoparticles. Nanoparticles encapsulated with macrophage membranes can simulate the active homing efficacy of macrophages to inflamed lesions during AP, resulting in excessive infiltration of macrophages in pancreatic inflammation sites and prolonged tissue retention time. Mechanistic studies elucidate that macrophage membrane-biomimetic selenylated Poria cocos polysaccharide nanoparticles primarily mitigate pancreatic inflammation by inhibiting the AKT/mTOR pathway to reverse autophagic flux impairment.

These studies conclusively demonstrate that CMC-BDS can achieve targeted drug delivery and effective therapy by precisely addressing the pathological characteristics of various inflammatory diseases. With deepening understanding of inflammatory mechanisms and advancements in nanotechnology, such biomimetic systems are expected to bring revolutionary breakthroughs in the treatment of inflammatory disorders. Future research should prioritize addressing critical challenges including scalable production and clinical translation to accelerate their progression toward clinical applications.

5. Discussion

NDDS shows promise for treating critical diseases, but conventional nanodrugs have issues like short circulation, poor targeting, and off-target effects. Researchers have developed biomimetic CMC-NPs to address these challenges. CMC-NPs combine natural cell membranes with synthetic nanomaterials, offering advantages such as: **(1) Immune**

evasion and prolonged circulation; **(2) Precise targeting through natural or engineered mechanisms**; **(3) Reduced toxicity and clearance risks**; **(4) Flexible surface modifications to meet complex needs**. Currently, leukocyte, erythrocyte, platelet, dendritic cell, tumor, and bacterial membranes are used in oncology and inflammation therapies, with hybrid membranes further enhancing functionality.

However, the clinical translation of CMC-NPs still faces multiple bottlenecks: **(1) Technical challenges:** Current membrane isolation methods, such as differential centrifugation and ultrasonic disruption, yield low purity and variable results. The physicochemical compatibility between nanocarriers and biological membranes also needs optimization. **(2) Quality control:** Ensuring clinical safety and efficacy requires rigorous evaluation, including assessments of immunogenicity of heterologous membrane proteins, long-term toxicity of the nanocarrier core, and potential pathogen contamination risks. **(3) Translational barriers:** Research relies heavily on murine models, which cannot fully replicate human disease complexity. Standardized protocols for scaling up to good manufacturing practice (GMP)-compliant production are still lacking.

Future research breakthroughs should concentrate on four critical dimensions: (1) Technological innovation: Develop high-precision membrane isolation techniques (e.g., microfluidic sorting, affinity chromatography) with AI-driven proteomics analysis for customized screening of membrane components. Optimize membrane protein expression via CRISPR-Cas9 or synthetic biology to reduce immunogenicity risks. **(2) Enhanced evaluation systems:** Implement comprehensive in vitro and in vivo evaluations prioritizing human clinical trials to assess immunotoxicity and hematotoxicity of NDDS. Establish a robust safety assessment framework covering acute/chronic toxicity, carcinogenicity, and immunostimulatory effects. **(3) Interdisciplinary integration:** Use advanced machine learning to construct 3D interaction networks (nanocarrier-biomembrane-disease target) for precision drug formulation design. **(4) Standardized manufacturing:** Standardize production processes for biomimetic nanomedicines using advanced analytics to minimize batch variability and ensure consistent quality and performance for clinical translation.

CMC-BDS has emerged as a revolutionary platform in biomedicine due to its biomimetic properties, functional programmability, and disease adaptability. While challenges remain in preparation techniques, safety profiles, and scalable manufacturing, advancements in hybrid membrane technology, gene editing, and nanomaterials science position CMC-NPs to play a pivotal role in personalized medicine and combination therapies.

6. Abbreviations

1-MT	1-methyl-D-tryptophan
AKI	Acute kidney injury
ALI/ARDS	Acute lung injury/Acute respiratory distress syndrome
AIE	Aggregation-induced emission
BM-CN	Bacterial Membrane-Coated Nanoparticles
BsAbs	Bispecific antibodies
BBB	Blood-brain barrier
CaMKIIγ	Ca^{2+}/calmodulin-dependent protein kinase γ
CCR2	C-C chemokine receptor type 2
CCR7	C-C chemokine receptor type 7
CMC-BDS	Cell membrane-coated biomimetic delivery systems
CMC-NPs	Cell membrane-coated nanoparticles
COPD	Chronic obstructive pulmonary disease
siCkip-1	Ckip-1 small interfering RNA
CD	Cluster of differentiation
DPNs	Coated mRNA nanoparticles
COFs	Covalent organic frameworks
Cur	Curcumin
CXCR	C-X-C chemokine receptor type
CXCL	C-X-C motif chemokine ligand
COX-2	Cyclooxygenase-2
CTL	Cytotoxic T lymphocyte
DAMPs	Damage-associated molecular patterns
DCM-CN	Dendritic Cell Membrane-Coated Nanoparticles
DCs	Dendritic cells
DHA	Dihydroartemisinin
DOX	Doxorubicin
EPR	Enhanced permeability and retention
EGFR	Epidermal growth factor receptor
EMVs	Erythrocyte membrane vesicles
EM-CN	Erythrocyte Membrane-Coated Nanoparticles
ECM	Extracellular matrix
EVs	Extracellular vesicles
GECM-CN	Genetically Engineered Cell Membranes-Coated Nanoparticles
GOD	Glucose oxidase
GMP	Good manufacturing practice
HER2	Human epidermal growth factor receptor 2
ICD	Immunogenic cell death
ICG	Indocyanine green
ICAM-1	Intercellular adhesion molecule 1
IRF1	Interferon regulatory factor 1
LM-CN	Leukocyte Membrane-Coated Nanoparticles
LPS	Lipopolysaccharides
MHC	Major histocompatibility complex
MSC	Mesenchymal stem cell
M-MOF	Metal-organic framework nanoparticles
MCP-1/CCL2	Monocyte chemoattractant protein-1/chemokine (c-c motif) ligand 2
MM-CN	Monocyte/Macrophage Membrane-Coated Nanoparticles
MPS	Mononuclear phagocyte system
MUC1	Mucin 1
NOX4	NADPH oxidase 4
NDDSs	Nanodrug delivery systems
NPs	Nanoparticles
NIR	Near-infrared
NIR-II	Near-infrared-II
NET	Neutrophil extracellular trap
NM-CN	Neutrophil Membrane-Coated Nanoparticles
NLRP3	NOD-like receptor family, pyrin domain containing 3
siNOX4	NOX4-targeting siRNA
OVA	Ovalbumin
sip65	p65-targeting siRNA
PAMPs	Pathogen-associated molecular patterns
PRRs	Pattern recognition receptors
PDT	Photodynamic therapy
PTT	Photothermal therapy
PGZ	Pioglitazone
PM-CN	Platelet Membrane-Coated Nanoparticles
PLGA	Poly(lactic-co-glycolic acid)
PVCL	Poly(N-vinylcaprolactam)
PEG	Polyethylene glycol
PD-1	Programmed cell death protein 1
AKT/mTOR	Protein kinase B/Mechanistic target of rapamycin
ROS	Reactive oxygen species
RBC	Red blood cell
Tregs	Regulatory T cells
RES	Reticuloendothelial system
SAB	Salvianolic acid B
SIRPα	Signal regulatory protein alpha
siRNA	Small interfering RNA
sSDF-1α	Stromal cell-derived factor-1α peptide
SIRS	Systemic inflammatory response syndrome
Tet	Tetracycline
THBS1	Thrombospondin-1
TLR4	Toll-like receptors
TNBC	Triple-negative breast cancer
TCM-CN	Tumor Cell Membrane-Coated Nanoparticles
TME	Tumor microenvironment
TAAs	Tumor-associated antigens
VCAM-1	Vascular cell adhesion protein 1

CRediT authorship contribution statement

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Methodology, Investigation. **Wei Du:** Validation, Supervision, Software. **Zhenghui Yang:** Writing – review & editing, Methodology, Investigation. **Jiayu Li:** Software, Investigation, Data curation. **Wanshun Li:** Visualization, Validation, Methodology, Investigation. **Jiaheng Xu:** Software, Resources, Methodology. **Ying Duanmu:** Software, Methodology, Investigation, Data curation. **Ting Yu:** Validation, Methodology, Investigation. **Fengping Cai:** Software, Methodology, Investigation. **Wenhao Li:** Writing – review & editing, Supervision, Project administration, Formal analysis. **Zhendong Jin:** Writing – review & editing, Resources, Funding acquisition. **Wencheng Wu:** Writing – review & editing, Resources, Funding acquisition. **Haojie Huang:** Writing – review & editing, Resources, Project administration.

Ethics approval and consent to participate

Not applicable.

Consent for publication

All authors in the paper agree to be published.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Reports a relationship with that includes:. Has patent pending to. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

No data was used for the research described in the article.

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