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Targeting Obesity-Induced Inflammation in Cancer Using Nanotherapeutics

Bizimana Rukundo T.

Faculty of Biological Sciences Kampala International University Uganda

ABSTRACT

Obesity drives a state of chronic, low-grade inflammation that reshapes tissue physiology and systemic immunity, accelerates carcinogenesis, and blunts responses to therapy. Adipocyte hypertrophy, hypoxia, and lipotoxicity trigger cytokine and chemokine cascades, recruit and polarize myeloid cells, and distort stromal architecture, yielding a tumor microenvironment that is immunosuppressed, fibrotic, and metabolically hostile. Nanotechnology offers a toolkit to precisely modulate this inflammatory circuitry while minimizing off-target toxicity. Liposomal and polymeric carriers, biomimetic vesicles, and inorganic or hybrid platforms can concentrate anti-inflammatory drugs, nucleic acids, and immune agonists in adipose depots and tumors, program release to match local pH/ROS/enzymatic cues, and bias delivery toward myeloid or stromal targets. This review synthesizes mechanistic links between obesity-induced inflammation and cancer, surveys nanotherapeutic strategies that dampen pathologic signaling or re-educate immune and stromal compartments, and outlines translational guidance for dosing, safety, and manufacturing in high-BMI populations. A unifying theme is context-aware engineering that aligns carrier physicochemistry and ligands with the altered vascular, immune, and metabolic landscapes of obesity to convert a systemic liability into a therapeutic entry point.

Keywords: nanotherapeutics; obesity-induced inflammation; tumor microenvironment; immunometabolism; cancer prevention and therapy

INTRODUCTION

The global rise of obesity has reframed cancer prevention and treatment. Excess adiposity is now firmly linked to higher incidence and poorer outcomes across multiple malignancies, including post-menopausal breast, endometrial, colorectal, pancreatic, hepatocellular, renal, and esophageal cancers[1–4]. Central to this association is a persistent, low-grade inflammatory state often termed metaflammation that originates in adipose tissue but radiates systemically. As adipocytes enlarge, local hypoxia develops, the extracellular matrix becomes denser, and endoplasmic reticulum stress escalates[5–8]. These cues prompt adipocytes and adipose-resident immune cells to secrete interleukins, chemokines, prostaglandins, and danger signals that recruit monocytes and neutrophils, polarize macrophages toward tumor-promoting states, and derail regulatory circuits. In parallel, insulin resistance and hyperinsulinemia elevate IGF-1 signaling, adipokine balance tilts toward leptin dominance and adiponectin deficiency, and lipid handling skews toward lipotoxic species such as ceramides[9, 10]. The net effect is an organism-wide rewiring of immunity and metabolism that primes tissues for malignant transformation, accelerates growth and invasion, and erects delivery barriers to therapy.

Within tumors, obesity-induced inflammation manifests as abundant tumor-associated macrophages with immunosuppressive programs, expanded myeloid-derived suppressor cell pools, exhausted cytotoxic T and NK cell phenotypes, and fibroblasts that deposit stiff collagen matrices[11–14]. Vasculature is leaky yet dysfunctional, raising interstitial fluid pressure and hindering convective transport. Metabolic gradients intensify; lactate accumulates; reactive oxygen species rise; and nutrient competition deprives antitumor lymphocytes of the substrates needed to sustain effector function[15, 16]. These same features that support tumor fitness also hinder small-molecule and biologic therapies by limiting penetration, amplifying off-target toxicity when the dose is escalated, and fostering adaptive resistance through paracrine buffering.

Nanotherapeutics aim to bend these liabilities into leverage. By packaging anti-inflammatory agents, pathway inhibitors, nucleic acids, or immune agonists in tailored carriers, it becomes possible to extend circulation, direct payloads toward inflamed adipose or tumor tissue, and program release upon encountering pH, redox, or This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited

enzymatic cues[17–19]. Liposomes and lipid nanoparticles can co-encapsulate hydrophobic drugs with siRNA or antisense oligonucleotides against inflammatory mediators[20–22]. Polymeric nanoparticles offer controlled and stimuli-responsive release and can be decorated with ligands that recognize receptors enriched on macrophages, endothelium, or fibroblasts in obese microenvironments. Biomimetic systems, including cell-membrane-coated particles and engineered extracellular vesicles, add immune stealth and homing motifs. Inorganic and hybrid materials contribute imaging contrast, photothermal or magnetothermal actuation, and catalytic scavenging of reactive oxygen species, enabling theranostic cycles in which dosing and distribution are verified in vivo[23–25].

The therapeutic logic is twofold. First, dampen initiating signals that couple obesity to carcinogenesis by neutralizing cytokines, chemokines, and prostaglandins or by correcting maladaptive lipid signaling. Second, re-educate the tumor microenvironment by repolarizing macrophages, reducing myeloid suppression, normalizing vasculature and matrix, and restoring metabolic niches that permit durable antitumor immunity[26, 27]. Success depends on aligning nanocarrier properties with obese pharmacokinetics and tissue permeability, integrating short courses of vascular or matrix normalization to improve access, and adopting imaging-guided dosing to cope with inter-patient heterogeneity. This review maps the pathways most amenable to nanoscale intervention, examines platform choices and design rules for inflammatory targeting, considers safety and manufacturing in dyslipidemic contexts, and sketches clinical translation paths that reflect the reality that many patients with cancer live with obesity[28].

2 Pathways and Microenvironmental Features that Link Obesity to Cancer and Invite Nanoscale Intervention

Obesity reshapes inflammatory signaling through interlocking cytokine and lipid networks. Adipocytes and infiltrating macrophages secrete interleukin-6, tumor necrosis factor, interleukin-1 family members, CCL2 and related chemokines, and prostaglandin E2 via cyclooxygenase-2 induction[29]. These mediators converge on NF- κ B, STAT3, and AP-1 transcriptional programs in epithelial and stromal cells, upregulating survival genes, anti-apoptotic effectors, and matrix remodeling enzymes. Lipotoxic ceramides and saturated fatty acids activate TLR4 and inflammasomes, adding feed-forward loops that maintain cytokine production[30]. In circulation, hyperinsulinemia and elevated IGF-1 spur the PI3K–AKT–mTOR axis, favoring anabolic metabolism in premalignant and malignant cells. Leptin surges promote angiogenesis and migration, whereas adiponectin deficiency removes a brake on inflammation and proliferation[30].

The tumor microenvironment formed in this setting is structurally and functionally distinct. Macrophages adopt immunosuppressive, pro-angiogenic phenotypes and express receptors such as CD206, scavenger receptors, and folate receptor- β . Myeloid-derived suppressor cells accumulate and impair T-cell priming[15, 31–33]. Cancer-associated fibroblasts deposit collagen I and hyaluronan, stiffening tissues and elevating interstitial pressure. Endothelial cells respond to VEGF and inflammatory cues with disorganized sprouting and increased permeability but inadequate perfusion[30]. Metabolically, hypoxia and excess lactate suppress T cells while sustaining macrophage and fibroblast programs; reactive oxygen species rise and oxidize lipids, generating immunomodulatory aldehydes[34].

These biological signatures create address codes and triggers for nanotherapy. Receptors on macrophages and endothelium can be targeted with mannose, galactose, RGD and related integrin ligands, or folate analogs. Enzyme-rich milieus provide protease-cleavable linkers, while acidic pH and elevated reactive oxygen species offer release switches for acid-labile or ROS-responsive chemistries[34]. Hyaluronidase-sensitive matrices can be locally softened to relieve pressure and improve convection[35, 36]. Importantly, obesity modifies protein corona formation on nanoparticles through dyslipidemia and glycation, altering opsonization and mononuclear phagocyte system uptake; therefore, stealth coatings and corona management become integral to design. By co-opting these features, nanotherapeutics can localize to inflamed adipose depots and tumors, re-time drug exposure to the microenvironmental clock, and change the cellular recipients of therapy from indiscriminate to intentioned.

3 Lipid-Based Nanotherapeutics to Reprogram Inflammation at the Adipose–Tumor Axis

Liposomal and lipid nanoparticle platforms are well-suited to ferry anti-inflammatory and immunomodulatory agents because their membranes mirror biological bilayers and can integrate signals for endosomal escape and controlled release[37–40]. Encapsulation of small-molecule inhibitors of cyclooxygenase-2 or microsomal prostaglandin E synthase can suppress prostaglandin-driven angiogenesis and immune suppression with reduced gastrointestinal and cardiovascular toxicity relative to free drugs. Lipid nanoparticles carrying siRNA or antisense oligonucleotides against interleukin-6, CCL2, or STAT3 effectors can silence upstream drivers of the inflammatory program in adipocytes, macrophages, or tumor cells. Co-loading strategies, such as pairing a prostaglandin pathway inhibitor with nucleic acids that downregulate chemokine axes, enable multi-node interventions that are otherwise limited by systemic tolerability[41–43].

Targeting is enhanced by decorating vesicles with ligands for receptors enriched in obese tissues. Mannose or galactose units can bias uptake toward macrophages that express C-type lectin receptors. Peptide ligands for integrins abundant on inflamed endothelium or nascent vasculature enrich delivery along vascular surfaces where leukocyte trafficking and cytokine exchange are active. Zwitterionic or alternative stealth polymers can

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mitigate anti-PEG immune responses reported in metabolic syndrome and help control the composition of the protein corona when plasma lipids are high[44]. Release timing can be tuned through pH-sensitive lipids that destabilize in the mildly acidic interstitium typical of inflamed adipose and tumors, or through thermosensitive compositions that permit regional activation by mild hyperthermia[44].

A particular opportunity lies at the interface of metabolism and inflammation. Lipid carriers can deliver inhibitors of ceramide synthesis or neutral sphingomyelinase to blunt lipotoxic signaling, while simultaneously transporting agents that favor adiponectin pathways or restore insulin sensitivity in peritumoral adipose[45]. By shifting adipose immunometabolism toward resolution rather than propagation of inflammation, these formulations may reduce the paracrine fuel that encourages malignant growth. In breast and endometrial cancers, where adjacent fat pads are active contributors, peritumoral injections or depot-forming lipid systems provide regional control with a favorable therapeutic index. Across indications, lipid nanotherapeutics pivot from simple drug shuttles to microenvironment editors that nudge the adipose–tumor axis away from metaflammation and toward homeostasis[45].

4 Polymeric and Biomimetic Nanoparticles for Myeloid Re-education and Cytokine Circuit Control

Polymeric nanoparticles based on PLGA, PEG-PLA, or related chemistries offer programmable release over days to weeks and can carry diverse cargo, including small molecules, proteins, and nucleic acids[46–48]. For obesity-linked inflammation, a central use case is the repolarization of macrophages and suppression of myeloid-derived suppressor cells. Delivery of inhibitors of CSF1R or PI3K- γ within carriers that display mannose or folate motifs can selectively accumulate in myeloid compartments and tilt macrophages away from immunosuppression toward antigen presentation and tumoricidal activity. Incorporation of charge-reversal layers masks cationic segments during circulation and unmask them under acidic conditions to promote endosomal escape and cytosolic access for RNA therapeutics directed at STAT3, NF- κ B, or checkpoint ligands such as PD-L1[49–51].

Biomimetic approaches extend these capabilities. Cell-membrane-coated nanoparticles cloaked with macrophage or platelet membranes inherit chemokine receptors and adhesion molecules that favor homing to inflamed vasculature and immune niches while evading phagocytic clearance. Engineered extracellular vesicles derived from adipose stromal cells or macrophages can be stripped of pro-tumor microRNAs and loaded with anti-inflammatory RNA cargos or STING agonists to awaken local innate immunity without the systemic cytokine surge that limits soluble agonists[52–54]. When stromal architecture is a dominating barrier, polymeric carriers can deliver hyaluronidase or TGF- β pathway inhibitors to decompress vessels and lower interstitial pressure, thereby enhancing subsequent delivery of immune effectors. Because obesity alters polymer metabolism and renal handling of fragments, excipient selection favors well-characterized, biocompatible backbones, and release kinetics are calibrated to minimize peak systemic exposure while sustaining local activity[55, 56].

Combination designs are especially potent. Sequential administration that first reduces matrix stiffness and myeloid suppression, followed by vaccination or checkpoint blockade, can transform non-inflamed tumors into responsive ones. Polymeric systems enable such choreography with temporal control embedded in degradation rates and linker chemistry. In colorectal and pancreatic cancers, where desmoplasia and obesity often co-occur, these strategies convert physical and immunologic barricades into transient windows for engagement by adaptive immunity.

5 Inorganic and Hybrid Nanoplatforms for Redox Control, Imaging Guidance, and Stimuli-Responsive Therapy

Obesity-linked inflammation raises reactive oxygen and nitrogen species and oxidized lipid byproducts that both damage tissues and shape immune tone [57]. Inorganic nanoplatforms add catalytic and physical levers to this biology[58]. Ceria and manganese oxide nanoparticles can scavenge reactive oxygen species and modulate redox signaling in macrophages and endothelium, potentially resetting inflammatory set points without the pharmacokinetic liabilities of small-molecule antioxidants[58]. Gold nanoshells and nanorods enable photothermal heating that, when applied gently, can increase perfusion, lower interstitial pressure, and accelerate release from thermo-responsive carriers co-administered at the same site. Iron oxide nanoparticles offer magnetothermal actuation and magnetic resonance contrast, supporting theranostic cycles wherein distribution is verified and microenvironmental access is tuned in real time.

Hybrid systems that fuse inorganic cores with lipid or polymer shells marry responsiveness with biocompatibility. Catalase-loaded shells around inorganic cores can degrade hydrogen peroxide in inflamed tissues, reducing oxidative stress while releasing anti-cytokine drugs or nucleic acids through redox-labile linkers[59]. Enzyme-cleavable coatings keyed to matrix metalloproteinases enable local payload deployment in fibrotic stroma common in obese tumors. For immune activation, nanoscale delivery of cyclic dinucleotides to stimulate STING benefits from co-packaging with redox buffers that prevent premature degradation and from imaging labels that confirm nodal trafficking[59]. Safety considerations are primary when using inorganic materials; dissolution rates, ion release, and long-term tissue retention must be quantified, and doses adjusted for the altered mononuclear phagocyte activity and hepatic steatosis that often accompany obesity.

Imaging guidance is a force multiplier. Positron or near-infrared labels on nanoparticles enable quantification of delivery to adipose depots, liver, spleen, and tumors, allowing dose individualization that accounts for body composition rather than total body weight alone[60]. In trials, this feedback mitigates the heterogeneity that has historically obscured the benefits of nanoscale interventions and provides mechanistic biomarkers of inflammatory modulation, such as changes in macrophage density or perfusion following photothermal priming.

6 Targeting, Pharmacokinetics, Dosing, and Translation in Obese Hosts

Clinical translation of anti-inflammatory nanotherapeutics in obesity requires alignment between device physics and host physiology. Dosing strategies anchored to lean body mass or allometric scaling reduce overexposure relative to total body weight-based schemes[61]. Because dyslipidemia and glycation reshape the protein corona and hasten hepatic and splenic uptake, stealth density and chemistry are tuned to maintain near-neutral surface potentials and resist opsonization; alternatives to PEG, such as poly(2-oxazoline) or phosphorylcholine, can mitigate complement activation and preexisting anti-PEG antibodies. Size windows between roughly seventy and one hundred nanometers balance evasion of renal filtration with penetration into inflamed tissue, though smaller, cross-linked micelles can be useful where dense stroma dominates[61].

Target selection prioritizes receptors and enzymes upregulated by metaflammation, including scavenger receptors, mannose receptors, folate receptor- β on macrophages, angiogenic integrins on endothelium, and matrix metalloproteinases in fibroblast-rich regions. Release triggers mirror local cues: acid-labile and ROS-responsive linkers for inflamed interstitium, protease-cleavable peptides for fibrotic stroma, and thermo-responsive motifs when adjunct heat is planned[62]. Short courses of vascular normalization or matrix modulation timed before nanoparticle infusion improve perfusion and lower interstitial pressure, extending the reach of carriers into otherwise excluded zones. Image-guided dosimetry verifies delivery and supports adaptive scheduling when hepatic sequestration is excessive or adipose sinks dominate[62].

Safety management reflects comorbidities. Infusion reactions and complement activation are minimized with graded infusion, appropriate premedication, and surface chemistries that avoid strong cationic charge[63]. Hepatotoxicity surveillance is heightened in fatty liver disease, and renal monitoring attends to polymer metabolite clearance. Manufacturing follows quality-by-design principles that define critical quality attributes such as size distribution, ligand density, trigger sensitivity, and batch-to-batch reproducibility under stress conditions relevant to dyslipidemic matrices[63]. Release testing includes potency assays linked to mechanisms like macrophage repolarization indices, cytokine suppression readouts, or imaging-verified accumulation to ensure the intended inflammatory rewiring occurs without collateral immunosuppression.

CONCLUSION

Obesity-induced inflammation is both a spark and a scaffold for cancer initiation, progression, and therapeutic resistance. Nanotherapeutics provide a means to extinguish that spark and remodel that scaffold with spatial and temporal precision. Lipid platforms deliver anti-inflammatory drugs and gene silencers to the adipose-tumor axis while tempering systemic toxicity. Polymeric and biomimetic systems re-educate myeloid cells, decompress stroma, and choreograph combination regimens that unlock adaptive immunity. Inorganic and hybrid materials add redox control, imaging visibility, and on-demand actuation that sharpen dosing and expand therapeutic windows. Translation hinges on engineering choices that respect obese pharmacology, on imaging-guided individualization that tames heterogeneity, and on manufacturing discipline that delivers consistent function in dyslipidemic environments. With these guardrails, nanotechnology can convert chronic metaflammation from a diffuse, treatment-resistant adversary into a set of actionable targets, advancing prevention and therapy for the growing population of patients with obesity-associated cancers.

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