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Nanoparticle Mediated Modulation of Adipokines in Obesity-Driven Tumor Microenvironments

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ABSTRACT

Adipokines sit at the crossroads of metabolic status and cancer biology. In obesity, elevated leptin and reduced adiponectin converge with chronic low-grade inflammation to remodel the tumor microenvironment toward angiogenesis, immune evasion, fibrosis, and metabolic plasticity. Nanoparticles can intervene directly in this signaling economy by concentrating modulators of adipokine pathways in adipose depots and tumors, shielding labile nucleic acids that edit receptor or downstream effectors, and timing release to microenvironmental triggers such as pH, reactive oxygen species, or matrix proteases. This review synthesizes the rationale and design space for nanoparticle-mediated regulation of leptin, adiponectin, and allied adipokines, from ligand or receptor blockade and biased agonism to gene silencing and mRNA restoration. It details lipid, polymeric, biomimetic, and hybrid platforms that steer payloads to adipocytes, tumor cells, endothelium, and myeloid populations prominent in obese hosts, and it outlines pharmacokinetic, safety, and manufacturing considerations that are specific to dyslipidemia and fatty liver disease. By aligning nanocarrier physicochemistry and targeting ligands with the signatures of the obese microenvironment, adipokine-centric nanotherapy can suppress tumorigenesis and resensitize cancers to standard immuno- and cytotoxic therapies.

Keywords: adipokines; leptin–adiponectin axis; obesity-associated cancer; nanoparticles; tumor microenvironment

INTRODUCTION

Obesity reshapes endocrine and immune tone in ways that tangibly influence cancer risk and therapeutic response. Among the molecular mediators of this reshaping, adipokine hormone-like factors secreted by adipocytes and stromal cells act as both sensors and actuators of metabolic state[1–3]. Leptin levels rise with adiposity and signal through the long isoform receptor LEPR-b to activate JAK–STAT, PI3K–AKT–mTOR, and MAPK pathways that support proliferation, motility, epithelial–mesenchymal transition, and angiogenesis[4–7]. Adiponectin typically declines, removing a brake on inflammation and reducing activation of AMPK and PPARα programs that limit anabolism and oxidative stress. Other adipokines, including resistin, visfatin, omentin, chemerin, and the adiponectin paralog CTRP family, participate in a network that couples nutrient state to stromal architecture and immune composition[7–9]. The sum is a tumor microenvironment biased toward myeloid dominance, dysfunctional vasculature, collagen-dense matrix, and metabolic gradients that privilege malignant survival and impede cytotoxic lymphocytes [10].

Directly drugging adipokine axes with systemic small molecules or biologics is challenging for several reasons. Ligand and receptor expression is distributed across multiple tissues, making on-target but off-tumor activity a driver of toxicity[11, 12]. Many targets reside on the cell surface or signal through hubs such as STAT3 and mTOR that participate in homeostatic processes; broad inhibition is poorly tolerated. Endocrine feedback loops compensate for partial blockade, and the pharmacokinetics of peptide hormones and nucleic acids are inhospitable to sustained exposure[13]. Nanoparticles offer a path around these constraints by increasing local concentration where it matters, adipose depots contiguous with tumors, angiogenic endothelium, and the myeloid and fibroblast compartments that adjudicate immune tone while decreasing systemic exposure [14]. Carriers can be endowed with ligands that recognize receptors enriched by obesity, coated to resist opsonization

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in dyslipidemic plasma, and engineered to release their payloads only after crossing into acidic, oxidative, or enzyme-rich niches[13].

The therapeutic ambitions are twofold and complementary. The first is to dampen pro-tumor leptin signaling by reducing ligand availability, blocking receptor engagement, silencing downstream effectors, or biasing signaling toward less oncogenic branches[15]. The second is to restore adiponectin tone or mimic its protective signaling through receptor agonism or AMPK-centric metabolic reprogramming. In practice, these goals often intersect: reducing leptin–STAT3 activity lowers vascular permeability and myeloid recruitment, while restoring adiponectin–AMPK restrains anabolism and improves mitochondrial quality control in both tumor and stromal cells[16]. Nanoparticle platforms can co-deliver such dual interventions, sequencing release to first soften stromal barriers and then impose durable signaling changes[17–19]. The following sections map adipokine biology in obese microenvironments, survey lipid and polymeric designs that intercept these pathways, explore biomimetic strategies that harness endogenous trafficking codes, and close with translational guidance on targeting, dosing, safety, and manufacturing suited to high-BMI populations.

2 Adipokine Biology at the Obese Adipose–Tumor Interface

The leptin–adiponectin balance is a primary determinant of how obesity informs cancer risk[4, 5, 7, 16]. Leptin secreted from hypertrophic adipocytes diffuses through peritumoral fat and enters tumor and endothelial cells expressing LEPR-b[5, 7, 16]. Engagement phosphorylates JAK2 and recruits STAT3, which transactivates c-MYC, cyclin D1, and anti-apoptotic genes; parallel engagement of IRS–PI3K–AKT facilitates mTORC1-driven anabolism, while MAPK branches support motility and invasion[20]. Leptin crosstalks with estrogen signaling in breast and endometrial cancers by upregulating aromatase, intensifying local estrogen biosynthesis. In endothelium, leptin promotes VEGF expression and sprouting, increasing vessel density while degrading perfusion quality. Myeloid cells in obese tissue express LEPR and respond to leptin with enhanced survival and polarization toward immunosuppressive phenotypes[21].

Adiponectin counterbalances these programs. Through AdipoR1 and AdipoR2, it activates AMPK and PPARα to restrain mTORC1 activity, enhance fatty acid oxidation, and reduce oxidative stress. Adiponectin attenuates NF-κB signaling, lowers IL-6 and TNF production, and improves insulin sensitivity in stromal and immune cells. In the tumor context, these actions decrease lactate accumulation, increase mitochondrial coupling efficiency, and create metabolic niches in which cytotoxic lymphocytes can sustain function. In obesity, however, adiponectin abundance falls, and receptor signaling can be blunted by post-receptor defects, allowing leptin-dominant tone to prevail[22].

These hormonal shifts overlay a background of metaflammation driven by saturated lipids, ceramides, and hypoxia. The result is a microenvironment where endothelial cells exhibit increased permeability but chaotic flow, fibroblasts deposit collagen and hyaluronan that elevate interstitial fluid pressure, and macrophages dominate the cytokine economy[23]. This architecture not only welcomes leptin's pro-angiogenic and pro-migratory signals but also makes delivery of conventional therapeutics haphazard. Nanoparticles can exploit the same abnormalities aberrant permeability, acidic interstitium, and elevated protease activity to enter and act preferentially within diseased tissue while carrying interventions that recalibrate adipokine signaling[23].

3 Lipid Nanoparticles and Liposomes to Rebalance the Leptin–Adiponectin Axis

Lipid carriers are natural candidates to host adipokine-modulating therapeutics because their bilayers facilitate endosomal escape and can be tuned for pH responsiveness[24]. One strategy employs liposomes that encapsulate neutralizing antibodies or receptor-decoy fragments directed against leptin or LEPR. By attaching targeting ligands that recognize integrins on angiogenic endothelium or receptors enriched on tumor cells, these vesicles localize antibody action to sites of pathogenic signaling and spare endocrine circuits elsewhere. pH-sensitive compositions using fusogenic lipids enhance cytosolic access for antibody fragments or peptide antagonists that would otherwise be trapped in endosomes[24].

An allied approach uses lipid nanoparticles to deliver siRNA or antisense oligonucleotides against LEP, LEPR, or STAT3 in peritumoral adipocytes, tumor cells, or endothelium[25]. The same chassis can co-deliver mRNA encoding adiponectin or small-molecule AMPK activators, producing a convergent shift that both removes a driver and installs a brake. Because dyslipidemia in obesity favors opsonization and rapid hepatic uptake, surface chemistry merits special attention.[25] Zwitterionic or poly(2-oxazoline) coronas can reduce complement activation and stabilize the protein corona, while inclusion of short fatty-acid anchors promotes albumin hitchhiking that enhances extravasation into inflamed tissues[26].

Lipid carriers can also enact localized metabolic repair that restores adiponectin sensitivity. Delivering ceramide synthesis inhibitors or neutral sphingomyelinase blockers to peritumoral fat reduces lipotoxic amplification of leptin signaling and improves AdipoR coupling to AMPK. In breast and endometrial cancers where fat pads are anatomically proximate, regional administration peritumoral injection or depot-forming gels studded with adipokine-modulating[27–30] liposomes offers prolonged local action with limited systemic spillover. Thermosensitive liposomes triggered by mild hyperthermia can further refine the spatial footprint, releasing cargo in tumor-adjacent adipose and within vascular hot spots that guide malignant cell trafficking[31–34].

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4 Polymeric Nanoparticles and Nucleic-Acid Therapeutics for Pathway Editing

Polymeric nanoparticles built on PLGA, PEG-PLA, or related backbones offer programmable degradation that matches the tempo of signaling change[35–37]. They can carry small-molecule inhibitors that interrupt leptin crosstalk, such as JAK2 or PI3K antagonists, at doses that would be intolerable systemically. Charge-reversal designs keep surfaces neutral in circulation and expose cationic segments in acidic interstitium to aid endosomal escape of nucleic acids. In this format, siRNA or antisense oligonucleotides against STAT3, SOCS-modulating microRNAs, or aromatase transcripts can be directed to tumor cells, adipocytes, and myeloid compartments using ligands for LEPR, folate receptor-β, or scavenger receptors.

Restoring adiponectin signaling can be attempted with polymeric carriers that deliver AdipoR agonists, stabilized adiponectin-mimetic peptides, or mRNA encoding adiponectin variants engineered for sustained secretion[7, 8, 22]. Pairing these with AMPK activators such as A-769662 analogs or metformin derivatives creates a two-tier intervention: receptor-level restoration and intracellular metabolic biasing. For endothelium, polymeric systems that co-deliver VEGF pathway modulators and leptin-axis silencers can rationalize vessel growth and reduce leakiness, lowering interstitial pressure and enhancing subsequent drug penetration[8, 22]. Because stromal barriers and myeloid dominance characterize obese tumors, a sequential regimen is attractive. First, nanoparticles supply hyaluronidase or TGF-β inhibitors to decompress the matrix and temper fibroblast activation. Next, a wave of adipokine-targeted nucleic acids is released to reset signaling[38]. Finally, a maintenance layer of AMPK-biased small molecules preserves the new equilibrium. Microfluidic manufacturing supports tight control over size and polydispersity, while quality-by-design frameworks define critical attributes such as ligand density and trigger sensitivity that govern biodistribution in dyslipidemic hosts.

5 Biomimetic and Extracellular Vesicle-Inspired Platforms for Precision Homing

Biomimetic nanoparticles coated with adipocyte or macrophage membranes inherit receptors and adhesion molecules that traffic naturally to adipose depots and inflamed vasculature. Such cloaks afford immune stealth and deliver homing codes without extensive synthetic ligand decoration[34, 39, 40]. Within this chassis, payloads that neutralize leptin or that express adiponectin can be ferried to peritumoral fat and stromal junctions with high efficiency. Engineered extracellular vesicles derived from adipose stromal cells are an adjacent strategy. By editing donor cells to deplete pro-tumor microRNAs and to overexpress adiponectin or AdipoR agonist peptides, the resulting vesicles act as both carriers and ligand sources. Their intrinsic membrane proteins assist uptake by adipocytes, endothelium, and macrophages, which are the key arbiters of adipokine tone[41–43].

Hybrid vesicles that fuse exosomes with liposomes or polymeric particles combine the targeting surfaceome with the formulation range of synthetic systems. They can house CRISPR ribonucleoproteins to correct LEPR overexpression or to install transcriptional repressors on leptin-responsive enhancers, while retaining thermosensitive or pH-sensitive shells for on-demand release[44]. Theranostic capability comes from embedding near-infrared dyes or positron labels within the membrane, allowing noninvasive verification that vesicles reach adipose–tumor interfaces[30, 45, 46]. In hepatocellular and colorectal cancers, where visceral adipose signaling and portal circulation interface tightly, liver-tropic vesicles must be tuned to avoid excessive Kupffer cell capture; short PEG tethers or albumin-binding motifs help balance residence time and parenchymal access.

Safety is paramount when co-opting biologically active membranes. Release assays confirm the absence of residual pro-angiogenic factors, and potency assays link dose to reductions in STAT3 phosphorylation or restoration of AMPK activity in target cells. Stability is maintained through lyoprotectants and cryopreservation buffers validated to preserve ligand orientation and fusion competence.

6 Targeting, Pharmacokinetics, Dosing, and Translation in High-BMI Populations

Translation of adipokine-centric nanotherapies must respect the altered pharmacology of obesity. Expanded plasma volume, dyslipidemia, and fatty liver disease accelerate mononuclear phagocyte clearance and complicate dose–exposure relationships[47]. Dosing anchored to lean body mass or allometric scaling better predicts exposure than total body weight. Surface chemistries that resist lipid-rich corona formation reduce hepatic sequestration; near-neutral zeta potentials and dense but biocompatible stealth layers mitigate complement activation that may be heightened in metabolic syndrome[47]. Hydrodynamic diameters between roughly seventy and one hundred nanometers balance renal escape with tissue penetration, while smaller cross-linked micelles can navigate desmoplastic regions at the cost of shorter circulation unless stabilized[48].

Target selection centers on receptors and enzymes that report on or regulate adipokine tone. LEPR and AdipoR isoforms are obvious handles, but endothelial integrins, scavenger receptors on macrophages, and hyaluronan receptors on fibroblasts provide additional entry points. Release triggers mirror the obese microenvironment: acid-labile linkers for perivascular interstitium[49], ROS-cleavable tethers where oxidative stress is high, and protease-responsive coatings in collagen-rich stroma. Short courses of vascular normalization or matrix modulation scheduled before infusion can improve perfusion and reduce interstitial pressure, enhancing nanoparticle access to adipocytes and endothelium[50]. Imaging plays a central role: near-infrared or positron

labels on carriers quantify delivery to adipose depots, liver, spleen, and tumor, enabling adaptive dosing or formulation switches if hepatic uptake dominates.

Safety monitoring emphasizes hepatic and cardiometabolic risks[50]. Gradual infusion and premedication limit infusion reactions; alternative stealth polymers avoid anti-PEG responses. Hepatotoxicity surveillance is intensified in steatosis, and renal function is followed when polymer metabolites accumulate[51]. Manufacturing adopts a quality-by-design discipline that defines critical quality attributes size distribution, ligand density, trigger kinetics, and ties them to potency readouts such as reductions in p-STAT3, increases in p-AMPK, normalized microvascular flow, and decreased leptin: adiponectin ratios in regional interstitial fluid[51]. These pharmacodynamic anchors support dose finding and patient selection in early trials, increasing the probability that signaling correction translates into clinical benefit.

CONCLUSION

Adipokines translate metabolic state into tumor behavior, and in obesity, that translation skews decisively toward malignancy. Nanoparticles provide the spatial, temporal, and cellular precision needed to rewrite this script. Lipid systems can deliver antibodies, decoys, and nucleic acids that mute leptin while restoring adiponectin and AMPK tone. Polymeric carriers enable pathway editing and sequenced release that decompresses stroma and stabilizes endocrine rebalancing. Biomimetic and extracellular vesicle–inspired platforms supply homing codes and immune stealth that raise effective dose at the adipose–tumor interface. When these designs are tuned for the pharmacology of obese hosts and coupled to imaging-guided dosing, the leptin-dominant, inflamed microenvironment becomes tractable. Rebalancing adipokine signaling through nanotechnology is therefore not only mechanistically sound but also practically attainable, offering a route to suppress tumorigenesis and improve treatment responses in the growing population of patients with obesity-associated cancers.

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