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Adipose Tissue-Derived Exosomes as Nanocarriers in Cancer Progression and Therapy

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ABSTRACT

Adipose tissue derived exosomes are nanoscale extracellular vesicles released by adipocytes, stromal cells, and immune cells within fat depots. In obesity, their secretion rate and molecular cargo shift toward pro-inflammatory, lipotoxic, and oncogenic profiles that reprogram cancer cells and their microenvironments to favor growth, invasion, metastasis, and therapy resistance. At the same time, these vesicles possess inherent attributes biocompatibility, immune stealth, membrane complexity, and tissue tropism that can be redirected for therapeutic delivery of small molecules, biologics, and nucleic acids. This review synthesizes current understanding of the dual nature of adipose-derived exosomes in obesity-related malignancies. It outlines how obesity remodels exosome biogenesis and content, explains the mechanisms by which these vesicles drive tumor progression, and details strategies to engineer them as precision nanocarriers. The discussion covers isolation and manufacturing in dyslipidemic contexts, quality and safety controls that minimize pro-tumor risks, and translational frameworks for dosing, imaging, and clinical integration alongside metabolic and microenvironment-normalizing interventions. The goal is to convert a conduit of disease signaling into a clinically reliable delivery system tailored to high-BMI populations.

Keywords: exosomes; adipose tissue; obesity-associated cancer; extracellular vesicles; nanocarriers

INTRODUCTION

Adipose tissue functions as an endocrine, metabolic, and immune organ whose influence extends across the body. Its cellular ecosystem mature adipocytes, adipose-derived stromal cells, endothelial cells, and resident or infiltrating leukocytes communicates through soluble mediators and through extracellular vesicles, especially exosomes measuring roughly 30 to 150 nanometers that arise from multivesicular bodies[1–4]. Exosomal membranes and lumens carry proteins, lipids, RNAs, and metabolites chosen by the donor cell, and they deliver this cargo to recipient cells through endocytosis, fusion, or receptor-mediated uptake. Obesity transforms adipose tissue through hypertrophy, hypoxia, endoplasmic reticulum stress, fibrosis, and chronic low-grade inflammation[4–6]. These conditions increase vesicle biogenesis and bias cargo toward signals that propagate inflammation, metabolic stress, and tissue remodeling. Consequently, adipose tissue–derived exosomes become a system-wide courier of the obese state and a potent amplifier of tumor–host crosstalk[7–9].

In malignancy, the convergence of insulin resistance, altered adipokines, and inflammatory cytokines has long been appreciated, but exosome-mediated communication now appears to be a unifying mechanism that operationalizes these endocrine and metabolic cues at cellular resolution[10–13]. Adipose vesicles can transfer microRNAs that silence tumor suppressors and activate canonical oncogenic pathways, deliver fatty acids and enzymes that promote fatty acid oxidation and anabolic capacity, and present proteins that reorganize adhesion, matrix deposition, and vascular behavior[14–16]. They also influence immune tone by skewing macrophage polarization, dampening antigen presentation, and weakening cytotoxic lymphocyte function. The net result is a tumor microenvironment with higher plasticity, stronger resistance to stress, and improved capacity to seed and colonize distant organs. Clinical observations connecting high body mass index to increased incidence and poorer outcomes in breast, endometrial, colorectal, pancreatic, liver, and renal cancers are consistent with this multimodal vesicle biology[15, 17–19].

Paradoxically, the same traits that make adipose exosomes efficient vectors of pathology offer a path to therapy[20, 21]. Their natural composition supports cellular entry and endosomal escape, their surfaces present adhesion molecules and tetraspanins that can be engineered for active targeting, and their biogenesis pathways can be harnessed to package drugs and genetic cargo with high efficiency[22]. Autologous or standardized allogeneic adipose sources provide a practical starting point for scalable production, while exosome-mimetic nanovesicles generated by extrusion offer improved yields with similar surfaceomes[22]. Turning this potential into clinical reality requires careful control over cargo content, authentic purification that separates vesicles from lipoproteins abundant in dyslipidemic plasma, and assays that confirm potency without unintended pro-

tumor effects[23]. It also invites a theranostic mindset in which labeled vesicles quantify delivery, stratify patients by microenvironmental accessibility, and guide individualized dosing[24–26]. The sections that follow map the landscape from sources and cargo to pathological roles, therapeutic engineering, manufacturing discipline, and clinical translation, with emphasis on the needs of high-BMI hosts.

2 Sources, Biogenesis, and Cargo Landscape of Adipose Exosomes

Adipose exosomes originate from white, beige, and brown adipocytes as well as stromal and immune constituents of both subcutaneous and visceral depots[27, 28]. Obesity changes the cellular proportions and the chemical milieu, increasing hypoxia and inflammatory signaling and accelerating biogenesis through ESCRT components, tetraspanin-rich microdomains, ceramide-dependent budding, and Rab GTPases that mediate vesicle trafficking and release. The resulting particles display a molecular repertoire that includes microRNAs such as miR-21, miR-27a, miR-130b, and miR-155, which frequently increase in obesity and target regulators of PI3K–AKT, JAK–STAT, and DNA damage responses[29]. Tumor-suppressive microRNAs often decline, further shifting signaling balance. Proteins such as tetraspanins, heat shock proteins, integrins, and matrix modulators co-travel with enzymes involved in lipid metabolism. Lipid cargo encompasses ceramides, diacylglycerols, cholesterol, and externalized phosphatidylserine that facilitates uptake via TIM receptors, while small metabolites like lactate and free fatty acids support metabolic coupling with cancer cells[29].

Heterogeneity aligns with anatomical site and tumor proximity. Visceral depots typically yield exosomes with more pro-inflammatory and oncogenic content than subcutaneous tissue, and peritumoral fat exhibits profiles shaped by tumor education[30]. Donor variables such as diet, sex, age, and glycemic control influence vesicle yield and content. Analytical rigor is crucial because low- and high-density lipoproteins overlap with exosomes in size and density[30]. Robust isolation therefore relies on orthogonal workflows that combine tangential-flow filtration, size exclusion, and density gradients, followed by particle counting, imaging, immunophenotyping for positive and negative markers, and lipidomic and transcriptomic profiling. Uptake occurs through endocytic pathways and is guided by an address code of surface molecules that confers organ tropism, a property that can be exploited therapeutically if properly understood and controlled[30].

3 How Adipose Exosomes Drive Tumor Progression and Therapy Resistance

Exosomes from obese adipose tissue rewire cancer metabolism by transferring fatty acids and enzymes such as CPT1A that enhance fatty acid oxidation and fuel ATP and NADPH production[21, 30–32]. This metabolic advantage supports survival under hypoxia and nutrient scarcity and contributes to resistance against chemotherapy and radiotherapy. On the signaling front, increased delivery of microRNAs that suppress PTEN, SOCS, and FOXO activates proliferative and survival pathways, while proteins that remodel the extracellular matrix and alter integrin profiles facilitate epithelial–mesenchymal transition, invasion, and colonization. Angiogenesis intensifies when vesicles deliver vascular growth factors, angiogenic microRNAs, and pro-coagulant lipids to endothelial cells already primed by metabolic syndrome, yielding vasculature that is disorganized yet permissive to tumor expansion and dissemination[31].

Immune editing proceeds as vesicles tilt macrophages toward alternatively activated phenotypes, inhibit dendritic cell maturation, and restrain cytotoxic lymphocytes through checkpoint ligand cargo or transforming growth factor signaling. Myeloid-derived suppressor cells and regulatory T cells expand, reinforcing a suppressive microenvironment that undermines checkpoint blockade and conventional cytotoxics[33]. Therapy interference extends beyond immune mechanisms: exosomes can export drugs, carry metabolizing enzymes, and deliver nucleic acids that downregulate genes essential for DNA damage responses, thereby reducing the efficacy of DNA-targeting agents[34]. They also participate in preparing pre-metastatic niches by educating stromal and immune cells, increasing vascular permeability, and depositing matrix templates in organs such as the liver and lungs[34]. These combined actions portray exosomes as mobile microdomains that coordinate metabolism, structure, vasculature, and immunity in ways that elevate tumor fitness, with the magnitude of these effects amplified by obesity.

4 Reprogramming Adipose Exosomes into Therapeutic Nanocarriers

Therapeutic designs can draw on native exosomes purified from adipocytes or stromal cells, on exosome-mimetic vesicles generated by mechanical extrusion that preserve membrane signatures while boosting yield, or on hybrids that fuse exosomes with liposomes or polymeric nanoparticles to merge stability and targeting[35]. Cargo loading may be passive, relying on membrane partitioning of hydrophobic small molecules, or active, employing electroporation, sonoporation, or saponin permeabilization, while endogenous loading can be achieved by engineering donor cells to overexpress desired RNAs or proteins[35]. Emerging efforts incorporate stimuli-responsive motifs that trigger release under acidic or oxidative conditions characteristic of tumor microenvironments, and hybrids can include photothermal or magnetic components for externally guided release.

Targeting precision follows two complementary routes. One relies on genetic fusion of targeting peptides or antibody fragments to exosomal scaffolds such as Lamp2b or CD63 to address receptors like HER2, EGFR, folate receptor alpha, integrins present on angiogenic endothelium, or CD206 on tumor-associated macrophages[36]. The other modulates organ tropism by adjusting integrin codes or by adding fatty-acid anchors that promote albumin hitchhiking and accumulation at the adipose–tumor interface. Immuno-oncology applications include delivery of small interfering RNA against PD-L1, nucleic acid agonists of STING, and

inhibitors of CSF1R or PI3K-γ to reprogram myeloid cells, as well as antigen and adjuvant combinations that prime T-cell responses in lymphatic tissues accessible to vesicles[37–40]. Combination regimens that pair exosome therapy with metabolic interventions such as GLP-1 agonists or metformin, and with vascular or matrix normalization strategies like brief anti-VEGF exposure or hyaluronidase, promise to enhance perfusion, reduce interstitial pressure, and deepen tissue penetration. Safety by design depends on stripping pro-oncogenic microRNAs in donor cells, embedding molecular safeguards, and adopting surface chemistries that minimize complement activation while preserving functionality.

5 Isolation, Manufacturing, and Quality Control in Dyslipidemic Contexts

Scalable manufacturing begins with accessible tissue sources and expands them in closed, xeno-free systems that limit contaminants. Donor selection and, where applicable, genome editing of master cell banks improve cargo consistency and reduce oncogenic risk[41]. Purification strategies must go beyond ultracentrifugation to avoid lipoprotein co-isolation and should combine size exclusion, density separation, and controlled filtration. Characterization employs particle counting, electron or atomic force microscopy, immunoprofiling of exosome markers and exclusion markers, lipidomics, proteomics, and small RNA sequencing, as well as sterility, endotoxin, and mycoplasma testing. Exosome-mimetic vesicles produced by serial extrusion achieve markedly higher yields and integrate smoothly with tangential-flow filtration and chromatography, while perfusion bioreactors hosting three-dimensional adipocyte or stromal cultures sustain production at clinical scale[41, 42]. Formulation stability depends on cryopreservation or lyophilization with appropriate protectants and on buffers that preserve membrane integrity. Stability-indicating assays should track particle integrity, cargo retention, and biological potency over time. Release testing requires mechanism-linked bioassays that quantify target knockdown, macrophage polarization, or T-cell activation, along with predefined limits for particle count per dose, RNA and protein content, residual DNA, sterility, endotoxin, and absence of lipoprotein contamination[43]. Because dyslipidemia is common in obesity, matrix studies that examine opsonization and complement activation in relevant plasma environments are essential for predicting in vivo behavior[43]. Regulatory alignment follows consensus reporting standards, maps critical quality attributes and process parameters within a quality by design framework, and anticipates bridging analytics when process changes or source transitions occur.

6 Clinical Translation, Dosing, and Safety in Obese Hosts

Clinical deployment in high-BMI populations must acknowledge altered pharmacokinetics resulting from expanded plasma volume, remodeled lipoprotein landscapes, and heightened hepatic and splenic phagocytic activity[44]. Image-guided dosimetry with positron, single-photon, or near-infrared labels can verify accumulation at target sites and support dosing based on lean body mass or allometric principles rather than total weight alone[44]. Short courses of vascular normalization or matrix modulation can precede dosing to improve perfusion and reduce interstitial pressure. Infusion reactions remain uncommon but possible, particularly in metabolic syndrome; mitigation relies on slow infusion, premedication when indicated, and surface chemistries that reduce complement activation. Oncogenic risk management begins with engineered donor cells that minimize loading of microRNAs associated with tumor promotion, continues with release tests that enforce absence of such cargo, and extends into clinical monitoring with circulating vesicle profiling to detect inadvertent pro-tumor signals[45].

Therapeutic indications likely to benefit include breast and endometrial cancers linked to obesity, where exosomes can deliver checkpoint silencers or pathway inhibitors while microenvironment normalization improves penetration[45]. Colorectal and pancreatic cancers, which are often desmoplastic, may respond to vesicles carrying stromal modifiers alongside cytotoxics or RNA therapeutics directed at canonical drivers[45]. Hepatocellular carcinoma presents a special case in which liver-tropic vesicles can deliver immune agonists or anti-fibrotic agents but demand careful hepatotoxicity monitoring in the context of fatty liver disease. Companion diagnostics based on circulating exosome signatures can stratify patients by risk and by expected vesicle responsiveness and can synchronize therapy with improvements in metabolic control. Ethical and manufacturing choices balance the immediacy of autologous products with the standardization of allogeneic lines, both of which require rigorous donor screening, infectious disease testing, and long-term post-approval surveillance with emphasis on high-BMI cohorts that have historically been underrepresented.

CONCLUSION

Adipose tissue derived exosomes occupy a pivotal position at the intersection of obesity and cancer. They propagate oncogenic signals by reshaping metabolism, structure, vasculature, and immunity, yet they also offer an adaptable and biocompatible chassis for targeted therapy. Transforming them from mediators of disease into vehicles of treatment hinges on precise control over cargo and purity, validated potency assays, formulations that remain stable and safe in dyslipidemic milieus, and imaging-enabled dosing strategies suited to obese hosts. When combined with metabolic therapies and microenvironment normalization, engineered adipose exosomes can turn the liabilities of obesity into levers for selective delivery, with the potential to improve efficacy and safety across obesity-associated malignancies.

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