IAA Journal of Scientific Research 13(1):129-134, 2026. www.iaajournals.org

©IAAJOURNALS ISSN: 2636-7319

https://doi.org/10.59298/IAAJSR/2026/131129134 IAAJSR:1311

# Exosome-Mimetic Nanovesicles in Inter-Organ Communication: Implications for Obesity-Driven Diabetes

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## ABSTRACT

Obesity-driven type 2 diabetes is increasingly understood as a disease of dysregulated inter-organ communication rather than isolated defects in single tissues. Exosomes and related extracellular vesicles (EVs) are key mediators of this crosstalk, transporting lipids, proteins and nucleic acids between adipose tissue, liver, skeletal muscle, pancreas and immune cells. In obesity, the cargo and secretion patterns of endogenous exosomes are profoundly altered, contributing to insulin resistance, metaflammation, and β-cell dysfunction. Exosome-mimetic nanovesicles (EMNVs), like synthetic or bioengineered vesicles that replicate key structural and functional features of natural exosomes, have emerged as powerful tools to probe and therapeutically modulate these communication pathways. Produced by top-down cell membrane fragmentation or bottom-up assembly from defined lipids and proteins, EMNVs can be loaded with drugs, RNAs or proteins and tailored for specific tissue tropism, while avoiding some manufacturing and scalability limitations of native exosomes. This review discusses the role of EV-mediated inter-organ communication in obesity-driven diabetes, the rationale for using EMNVs to interrogate and rewire these networks, and key design principles of exosome-mimetic systems. It then examines how EMNVs can be used to modulate adipose–liver–muscle–β-cell crosstalk, summarizes preclinical evidence, and highlights translational challenges, including heterogeneity, safety, and regulatory classification. Finally, it outlines future directions, including precision EMNVs carrying cargo that reprogram pathogenic signaling axes, integration with metabolic drugs, and potential use as "message decoys" to buffer harmful obesity-associated vesicle signals.

**Keywords:** Exosome-mimetic nanovesicles; inter-organ communication; obesity-driven diabetes; extracellular vesicles; metabolic crosstalk

## INTRODUCTION

Obesity-driven type 2 diabetes (T2D) arises from a network of dysfunctional signals linking adipose tissue, liver, skeletal muscle, pancreatic islets, gut and immune organs rather than from isolated defects in any single compartment[1–4]. Adipose tissue expansion and remodeling, particularly in visceral depots, initiate a cascade of events: adipocyte hypertrophy, hypoxia, extracellular matrix stiffening, immune cell recruitment and metaflammation[5–7]. These local changes alter not only classical hormonal outputs such as leptin and adiponectin but also the quality and quantity of extracellular vesicles (EVs) secreted into the circulation.

EVs, including exosomes (30–150 nm endosome-derived vesicles) and microvesicles (100–1000 nm plasma membrane–derived vesicles), are now recognized as major conveyors of inter-organ information[8]. They transport bioactive cargo such as lipids, receptors, cytokines, enzymes, mRNAs, and microRNAs (miRNAs) protected by a lipid bilayer, allowing signals to travel over long distances and withstand enzymatic degradation. In the metabolic context, exosomes derived from adipocytes, hepatocytes, myocytes, β-cells, and immune cells have been shown to influence insulin sensitivity, lipid handling, inflammation, and β-cell survival in recipient tissues[8].

In obesity, adipocyte-derived exosomes are enriched in pro-inflammatory and insulin resistance–promoting cargo. For example, obese adipose exosomes have been reported to carry miRNAs that suppress insulin signaling nodes in the liver and muscle, or proteins that activate macrophages and endothelial cells, thus propagating insulin resistance and vascular dysfunction[9]. Conversely, exosomes from healthy or exercise-

trained tissues can carry protective factors that enhance fatty acid oxidation, improve insulin sensitivity, or support β-cell resilience. This duality underscores the central role of EVs as both mediators and potential modulators of obesity-driven diabetes[9].
However, endogenous exosome populations are highly heterogeneous and difficult to control. Their cargo reflects the complex transcriptional and metabolic state of source cells, which in obesity is already pathological. While there is growing interest in using purified exosomes as therapies for instance, mesenchymal stromal cell–derived exosomes with anti-inflammatory properties, manufacturing challenges, batch variability, and limited scalability have hindered clinical translation[10].
Exosome-mimetic nanovesicles (EMNVs) have emerged to address these challenges. EMNVs are synthetic or semi-synthetic vesicles designed to recapitulate key features of exosomes like size, membrane composition, surface markers and ability to deliver cargo while offering greater control over content and production[10–12].
Two broad engineering strategies predominate. In top-down approaches, cells are physically extruded through filters or microfluidic devices, shearing their membranes into nanoscale vesicles that retain many native proteins and lipids but lack the complex endosomal biogenesis route. In bottom-up approaches, defined lipids and selected membrane proteins are assembled into vesicles reminiscent of exosomes, often with modular insertion of targeting ligands and controlled loading of therapeutic cargo[13–15].
EMNVs bring several advantages to the study and treatment of obesity-driven diabetes. First, they provide a tunable platform to dissect inter-organ signaling. By engineering EMNVs that display specific adipocyte or hepatocyte surface markers and carry defined miRNA or protein payloads, researchers can model how particular messages propagate through metabolic networks[16]. For example, EMNVs mimicking obese adipocyte exosomes but with altered miRNA content can be used to isolate the contribution of individual miRNAs to hepatic insulin resistance. Conversely, EMNVs carrying "corrective" cargos such as miRNAs that enhance insulin signaling or anti-inflammatory proteins can be targeted to metabolic tissues to test therapeutic hypotheses[16].
Second, EMNVs can be mass-produced more efficiently than native exosomes. Extrusion-based generation from cultured cells yields higher vesicle numbers per cell than natural exosome secretion, and synthetic liposomal methods are already well established at an industrial scale for several FDA-approved drugs[17]. Combining these capabilities with advances in microfluidics and high-throughput manufacturing could make EMNVs a practical modality rather than a niche tool[17, 18].
Third, EMNVs allow decoupling of source cell pathology from vesicle cargo. Instead of relying on exosomes secreted by diseased adipose or liver tissue, which may carry harmful contents, one can derive EMNVs from "clean" parental cells genetically engineered to express desired surface markers without unwanted pathogenic cargo. Alternatively, fully synthetic mimetics can incorporate specific lipids and proteins without inheriting the entire molecular history of a donor cell[18].
Within diabesity, inter-organ communication of particular interest includes adipose–liver, adipose–muscle, adipose–muscle, adipose–β-cell, and immune–metabolic axes[19]. Adipose-derived vesicles influence hepatic gluconeogenesis, steatosis, and inflammation; hepatocyte vesicles impact muscle insulin sensitivity and adipose lipolysis; β-cell vesicles can modulate islet immune infiltration and peripheral insulin action; and immune cell vesicles influence all of the above. EMNVs could be deployed to either block pathological messages (e.g., acting as decoys or scavengers for specific ligands and receptors) or deliver compensatory signals (e.g., adiponectin-mimetic peptides, pro-oxidative or anti-oxidative regulators, or miRNAs that reverse insulin resistance)[19].
The conceptual appeal of EMNVs in obesity-driven diabetes thus lies at the intersection of three trends: recognition of EVs as critical metabolic messengers; technological maturation of vesicle-mimetic nanomedicine; and therapeutic focus on systems-level, multi-organ interventions rather than single-receptor pharmacology[20]. Nonetheless, key questions remain. How closely must EMNVs mimic native exosomes to engage the right uptake pathways and intracellular routing? Which cargos and surface signatures are most relevant for correcting obese metabolic crosstalk? How can safety, immunogenicity and off-target effects be managed in a chronic disease context? The following sections address these issues by outlining endogenous EV biology in diabesity, core design principles for EMNVs, and emerging preclinical applications[20].

## 2. Endogenous Extracellular Vesicles in Obesity-Driven Diabetes

Endogenous EVs arise from virtually all cell types, but in obesity-driven diabetes, adipocytes, hepatocytes, skeletal muscle cells, β-cells, and immune cells contribute disproportionately to the circulating EV pool relevant for metabolic control[21]. Adipose tissue expansion elevates exosome release from both adipocytes and adipose-resident macrophages. These exosomes carry proteins and miRNAs that promote insulin resistance, such as miRNAs targeting insulin receptor substrate or PI3K/AKT pathway nodes in liver and muscle, and factors that activate Toll-like receptors or NF-κB in macrophages. The net effect is the propagation of metaflammation and impaired insulin signaling to distal tissues[5, 6, 16].
Hepatic EVs in nonalcoholic fatty liver disease and steatohepatitis, common comorbidities of obesity, contain lipotoxic signals and inflammatory mediators that exacerbate systemic insulin resistance and vascular dysfunction. Skeletal muscle–derived exosomes, conversely, can transmit exercise-induced benefits by delivering myokines, miRNAs, and metabolites that promote fatty acid oxidation and browning of white adipose

tissue[22, 23]. In obesity and inactivity, this protective exosomal output is diminished, tipping the balance toward pathogenic vesicle signals[24].

Pancreatic β-cells both send and receive EV-based messages. Under glucolipotoxic stress, β-cells release exosomes carrying danger-associated molecular patterns and autoantigens that can recruit immune cells and amplify islet inflammation[14, 25–27]. At the same time, they internalize exosomes from adipose, liver and immune cells that may either support survival or accelerate dysfunction, depending on cargo. Immune-cell–derived vesicles, especially from pro-inflammatory macrophages and T cells, further amplify islet and adipose inflammation. Collectively, these EV exchanges constitute a dynamic "metabolo-exosomal" network that shapes the trajectory from obesity to overt diabetes and its complications[27, 28].

## 3. Engineering Exosome-Mimetic Nanovesicles: Design Principles

Exosome-mimetic nanovesicles are built to recapitulate key exosomal traits while allowing precise control. Core design parameters include vesicle size (typically 50–200 nm), membrane lipid composition, presence of exosome-like surface proteins (tetraspanins, integrins, adhesion molecules), cargo loading strategy, and targeting ligands[13, 18, 29]. Top-down EMNVs produced by serial extrusion of donor cells often retain a broad array of plasma membrane proteins that facilitate cell-type–specific uptake and biodistribution, but may also carry unwanted components. Bottom-up EMNVs assembled from defined lipids and purified proteins provide greater specificity but may lack some uptake-facilitating complexity.

Cargo can be loaded passively (during vesicle formation) or actively (via electroporation, sonication, or membrane-penetrating peptides) and may include small molecules, peptides, mRNAs, miRNAs, siRNAs or CRISPR components[30–32]. For metabolic applications, common goals include enhancing insulin sensitivity (delivery of miRNAs that inhibit negative regulators of insulin signaling), reducing inflammation (anti-inflammatory cytokines, decoy receptors), or modulating lipid metabolism (agonists of PPAR pathways, AMPK activators). Targeting is achieved through surface-functionalized peptides, antibodies or aptamers recognizing receptors enriched on adipose, liver or β-cells, such as integrins, scavenger receptors or specific adhesion molecules.

## 4. EMNVs in Adipose–Liver–Muscle Crosstalk

Adipose–liver–muscle axes are central to obesity-driven insulin resistance. EMNVs can be designed to modulate these axes by delivering corrective cargos to one or more tissues. For example, adipose-targeted EMNVs carrying miRNAs that promote browning and mitochondrial biogenesis could shift white adipose tissue toward a more oxidative, less inflammatory state, indirectly improving hepatic and muscular insulin sensitivity[23, 33–35]. Conversely, hepatocyte-targeted EMNVs delivering inhibitors of gluconeogenic transcription programs or fibrosis pathways could reduce hepatic glucose output and inflammation, benefiting systemic glycemic control and adipose health. Muscle-directed EMNVs carrying exercise-mimetic cargos might restore beneficial myokine profiles and enhance glucose disposal.

## 5. EMNVs and Islet–Immune Communication in Diabesity

Islet dysfunction in obesity-driven diabetes involves β-cell stress, dedifferentiation and immune attack[36]. EMNVs offer avenues to protect islets by modulating local and systemic signaling[37]. For example, EMNVs engineered to resemble anti-inflammatory mesenchymal stromal cell exosomes but with enhanced β-cell tropism could deliver cytokines, growth factors or miRNAs that support β-cell survival and insulin secretion. Immune-targeted EMNVs could act as tolerogenic agents, delivering signals that shift macrophages and T cells toward regulatory phenotypes, dampening islet and adipose inflammation[37]. Decoy EMNVs displaying β-cell antigens or inflammatory ligands might sequester pathogenic antibodies or receptors, attenuating damaging signaling without global immunosuppression.

## 6. Preclinical Evidence and Safety Considerations

Preclinical work with exosome-mimetic vesicles in metabolic disease is still limited but growing. Studies in other inflammatory or fibrotic conditions show that EMNVs can recapitulate many therapeutic effects of parent-cell exosomes with improved scalability and potency[38]. In obesity and diabetes models, early reports suggest that EMNVs derived from adipose-derived stem cells or engineered cells can reduce inflammatory markers, improve insulin sensitivity and ameliorate hepatic steatosis, though detailed mechanistic attribution to specific inter-organ pathways is often incomplete[38]. Safety studies indicate that EMNVs generally exhibit favorable biocompatibility and low immunogenicity, but chronic administration, off-target accumulation and potential for unintended gene regulation remain concerns, especially when carrying nucleic acid cargos.

## 7. Translational Challenges and Future Directions

Translating EMNVs into therapies for obesity-driven diabetes requires addressing several issues: standardizing manufacturing and characterization to manage heterogeneity; ensuring reproducible targeting and cargo delivery; defining dosing regimens compatible with chronic disease; and navigating regulatory frameworks that straddle biologics and nanomedicine[39, 40]. Future work will likely focus on precision EMNVs tailored to specific metabolic endotypes, combination strategies with GLP-1 receptor agonists and SGLT2 inhibitors, and theranostic designs that combine imaging of inter-organ EV traffic with corrective messaging. EMNVs may also be used experimentally to map and "edit" metabolic communication networks, identifying which vesicle-mediated signals are causal drivers of diabesity and therefore high-value targets.

## CONCLUSION

Exosome-mimetic nanovesicles sit at an emerging interface between extracellular vesicle biology, nanotechnology, and metabolic disease research. By reproducing and refining the inter-organ messaging capacity of natural exosomes, EMNVs offer powerful tools to interrogate and therapeutically modulate the pathological communication networks that link adipose tissue, liver, muscle, islets and immune cells in obesity-driven diabetes. Early conceptual and preclinical work suggests that these vesicles can be engineered to deliver corrective cargos, act as decoys for harmful signals or reshape inflammatory crosstalk, with potential benefits for insulin sensitivity, adipose health and β-cell function. Realizing this promise will require rigorous attention to design, safety, manufacturing and patient-centered implementation, but exosome-mimetic nanovesicles may ultimately become an important component of multi-organ, systems-level strategies to prevent and treat diabetes arising from obesity.

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**CITE AS: Muhindo Edgar (2026). Efficacy of Insecticide-Treated Nets and Community Strategies in Malaria Prevention During Pregnancy. IAA Journal of Scientific Research 13(1):129-134. https://doi.org/10.59298/IAAJSR/2026/131129134**