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Nanomaterial-Based Modulation of Adipokines and Myokines: Implications for Diabetes-Obesity Crosstalk

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ABSTRACT

Adipose tissue and skeletal muscle are central endocrine organs that communicate through a complex network of secreted proteins, including adipokines and myokines, which shape systemic energy balance, insulin sensitivity and inflammation. In obesity and type 2 diabetes, this crosstalk becomes dysregulated: pro-inflammatory adipokines (e.g., TNF-α, IL-6, resistin) and reduced levels of protective factors (e.g., adiponectin, omentin, irisin, myonectin) drive insulin resistance and ectopic lipid deposition. Targeting these signaling axes is therefore an attractive strategy for simultaneously improving obesity and diabetes. Nanomaterials offer powerful tools to modulate adipokine and myokine networks, both as delivery systems for drugs and biologics that act on adipose and muscle, and as active entities that reprogram tissue microenvironments through immune, metabolic and mechanical cues. Engineered nanoparticles, liposomes, polymeric systems and inorganic nanostructures can be designed to accumulate in adipose tissue or skeletal muscle, alter local inflammation, promote browning and thermogenesis, or stimulate exercise-mimetic signaling, thereby reshaping adipokine–myokine profiles. This review outlines the physiological roles of adipokines and myokines in diabetes–obesity crosstalk, summarizes evidence that nanomaterials can modulate these mediators, and discusses mechanistic pathways including targeted delivery, immune modulation and organelle-level interventions. It also considers safety and translational challenges, including off-target effects, nanotoxicology and variability in human adipose and muscle depots. Finally, potential future directions, such as nano–exercise mimetics and combination strategies integrating nanomedicine with lifestyle and incretin-based therapies, are highlighted as routes to more precise and tissue-specific control of adipose–muscle communication in metabolic disease.

Keywords: Adipokines; myokines; nanomaterials; insulin resistance; diabetes–obesity crosstalk

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INTRODUCTION

Obesity and type 2 diabetes mellitus are tightly interwoven epidemics, linked not only by shared risk factors such as energy-dense diets and physical inactivity but also by a complex pathophysiology centered on adipose tissue and skeletal muscle[1–3]. These tissues, long viewed predominantly as energy storage and mechanical organs, are now recognized as dynamic endocrine entities that secrete a broad repertoire of cytokines, hormones and growth factors collectively termed adipokines and myokines that act locally and systemically to coordinate metabolism, inflammation and tissue remodeling[4–6].

In obesity, adipose tissue undergoes remodeling characterized by adipocyte hypertrophy, altered extracellular matrix, local hypoxia and immune cell infiltration. These changes shift the adipokine secretion pattern toward a more pro-inflammatory, insulin-resistance-prone profile[7–9]. Classical pro-inflammatory adipokines such as TNF-α and IL-6 impair insulin signaling in adipocytes, muscle and liver, while chemokines like MCP-1 recruit additional immune cells, reinforcing metaflammation. In parallel, levels of insulin-sensitizing and anti-inflammatory adipokines such as adiponectin and omentin decline, weakening protective counter-regulation. The net result is systemic insulin resistance, dyslipidemia and increased cardiovascular risk[10, 11].

Skeletal muscle, which accounts for a major proportion of insulin-stimulated glucose disposal, also responds to mechanical loading, nutrient status and endocrine signals by secreting myokines. Exercise induces release of factors such as irisin, IL-6 (in a context-dependent anti-inflammatory role), myonectin and BDNF that promote fatty acid oxidation, browning of white adipose tissue, improved insulin sensitivity and neuro-metabolic benefits[12]. In sedentary obesity and diabetes, however, this myokine profile becomes blunted or skewed, with reduced secretion of beneficial factors and possible upregulation of myokines that contribute to insulin resistance or muscle wasting. The crosstalk between adipokines and myokines thus acts as a critical determinant of whole-body metabolic homeostasis[12, 13].

The convergence of obesity and diabetes can be conceptualized as a breakdown in this endocrine conversation. Pro-inflammatory adipokines released from dysfunctional adipose depots worsen insulin resistance in muscle, while impaired myokine signaling from underactive or insulin-resistant muscle fails to counterbalance adipose dysfunction or promote energy expenditure[14]. Therapies that restore a healthier adipokine–myokine balance, dampening pro-inflammatory signals while boosting insulin-sensitizing and thermogenic cues, could, in principle, address both obesity and hyperglycemia more effectively than approaches targeting glucose alone[14]. Existing pharmacotherapies such as thiazolidinediones and GLP-1 receptor agonists, partially influence adipokine profiles, increasing adiponectin and reducing inflammatory cytokines, and exercise is a powerful physiological modulator of myokines. Yet, translating these insights into precise, tissue-targeted interventions remains challenging[15].

Nanomaterials offer a suite of capabilities that make them well-suited to modulate adipokine–myokine networks. First, nanoparticles can be engineered to preferentially accumulate in adipose tissue or skeletal muscle, leveraging differences in vasculature, permeability, lymphatic drainage and receptor expression[16]. Liposomes, polymeric nanoparticles and inorganic nanostructures can be surface-functionalized with ligands for receptors enriched on adipocytes, endothelial cells in adipose depots, or myofibers. Once localized, they can deliver small molecules, nucleic acids or biologics that directly alter the expression of key adipokines and myokines or their upstream regulators, such as PPARγ, NF-κB, AMPK and PGC-1α[15, 17, 18].

Second, nanomaterials can modulate immune microenvironments within adipose tissue and muscle, thereby indirectly reshaping adipokine and myokine profiles. For example, nanoparticles that reprogram macrophages from pro-inflammatory (M1-like) toward anti-inflammatory (M2-like) states in adipose tissue can reduce secretion of TNF-α and IL-1β while promoting more favorable adipokine secretion[19]. Similarly, nanocarriers delivering anti-inflammatory agents or siRNAs to skeletal muscle can attenuate local inflammatory signaling that suppresses exercise-inducible myokines. Because immune and stromal cells are tightly interwoven with adipocytes and myocytes in these tissues, targeting one compartment can have ripple effects on endocrine output[19].

Third, nanotechnology enables organelle-level interventions that influence adipokine and myokine production. Mitochondrial dysfunction and oxidative stress in adipocytes and myocytes contribute to altered secretion patterns; nanocarriers capable of delivering antioxidants, uncouplers or mitochondrial-targeted peptides can restore healthier organelle function, with consequent normalization of secretory profiles[20–22]. Some inorganic nanomaterials, such as certain metal oxides, possess intrinsic catalytic or redox-modulating activities that can reduce reactive oxygen species or modulate local oxygen tension, indirectly affecting cytokine production[23–25].

Finally, nanomaterials can act as "exercise mimetics" or browning triggers by providing mechanical, thermal or photothermal stimuli. For instance, nanoparticles responsive to near-infrared light or magnetic fields can locally elevate temperature or induce mechanical strain, promoting browning of white adipose tissue and myokine release akin to exercise[26]. While such approaches are still largely experimental, they illustrate how nanotechnology might recreate complex environmental cues that are otherwise achievable mainly through lifestyle changes[26].

Despite this conceptual appeal, the field of nanomaterial-based modulation of adipokines and myokines is still emerging. Evidence comes from diverse preclinical models, often in the context of broader metabolic interventions rather than explicit endocrine profiling[27]. Variability in nanomaterial composition, size, charge and dosing regimens complicates comparisons, and long-term safety remains a concern. Moreover, human adipose and muscle depots are heterogeneous across individuals and anatomical sites, raising questions about targeting precision and reproducibility[27].

Nevertheless, the possibility of steering adipose–muscle endocrine crosstalk using nanotechnology opens an intriguing translational avenue. It suggests that nanomedicine could complement pharmacological and lifestyle interventions by providing tissue-specific, programmable modulation of factors that coordinate energy storage, expenditure and glucose homeostasis[28]. The following sections outline the biology of adipokines and myokines, describe the classes of nanomaterials being studied for metabolic modulation, summarize evidence for nanomaterial-induced changes in endocrine signaling, and consider how these advances might be harnessed for integrated management of obesity and diabetes[28].

2. Adipokines in Obesity and Diabetes: Targets for Nanomaterial Intervention

Adipokines represent a diverse group of secreted proteins derived predominantly from adipocytes and adipose-resident immune and stromal cells. In obesity, expansion and remodeling of white adipose tissue shift the adipokine profile toward a pro-inflammatory and insulin-resistant state[29]. Elevated levels of TNF-α, IL-6, IL-1β, resistin, leptin (in the context of leptin resistance) and plasminogen activator inhibitor-1 are commonly observed, while adiponectin and omentin decline. This pattern contributes to systemic insulin resistance, impaired lipid handling, endothelial dysfunction and increased cardiovascular risk[29].

From a nanomedicine perspective, these adipokines can be conceptualized both as biomarkers of depot health and as functional targets. Strategies that increase adiponectin or restore more physiological leptin sensitivity could improve insulin sensitivity and lipid handling[30–32]. Conversely, reducing TNF-α, IL-6 or resistin secretion or activity could relieve the inflammatory inhibition of insulin signaling. Nanomaterials can support these aims by delivering small molecules, peptides, antibodies or nucleic acids that modulate adipokine gene expression or signaling pathways specifically within adipose tissue[33, 34]. For example, polymeric or lipid nanoparticles carrying PPARγ agonists or AMPK activators targeted to adipose depots might increase adiponectin and promote a more insulin-sensitive adipocyte phenotype while limiting systemic exposure and off-target effects that complicate conventional formulations.

Nanoparticles can also be designed to interact directly with adipose-resident immune cells that influence adipokine secretion. Reprogramming macrophages toward anti-inflammatory phenotypes using nanoparticle-delivered agents could reduce local production of TNF-α and IL-1β, enabling adipocytes to shift toward greater adiponectin and lower pro-inflammatory adipokine output. Similarly, targeting dendritic cells or T cells with tolerogenic nanomaterials could attenuate adipose inflammation and indirectly normalize adipokine profiles[35, 36]. Although many of these strategies have been tested primarily in models of atherosclerosis or general inflammation, their adaptation to metabolic tissues is a growing area of interest.

3. Myokines and Skeletal Muscle Endocrinology in Metabolic Disease

Skeletal muscle is both the largest sink for postprandial glucose and a vigorous endocrine organ that communicates with adipose tissue, liver, bone and brain. Myokines such as irisin, IL-6, IL-15, myostatin, myonectin and fibroblast growth factor 21 respond to contractile activity, nutrient availability and hormonal cues[37]. Exercise acutely elevates several beneficial myokines that enhance fatty acid oxidation, promote browning of white adipose tissue, improve insulin sensitivity, support muscle growth and exert anti-inflammatory effects. In contrast, chronic inactivity and obesity are associated with altered myokine profiles that may favor insulin resistance, sarcopenia and low-grade inflammation[37].

Targeting myokines is attractive because they can act as systemic mediators of exercise benefits. Nanomaterials could modulate myokine networks in multiple ways. First, nanoparticles can deliver agents that activate key transcriptional regulators of myokine expression, such as PGC-1α and AMPK, within muscle fibers, effectively amplifying exercise-induced myokine responses or partially mimicking them in sedentary states. Second, nanocarriers might stabilize exogenous myokines or myokine-mimetic peptides, improving their pharmacokinetics and facilitating tissue targeting[38, 39]. Third, nanomaterials that improve mitochondrial function or reduce oxidative stress in myocytes could restore healthier myokine secretion patterns by reversing cellular stress signals that suppress beneficial myokines and promote catabolic factors like myostatin[38–40].

The interplay between myokines and adipokines is bidirectional. Muscle-derived irisin, for instance, induces browning of white adipose tissue, increasing UCP1 expression and thermogenesis, while adiponectin from adipocytes enhances muscle insulin sensitivity and fatty acid oxidation[41]. By tuning myokine secretion through nanomaterial interventions in muscle, one can indirectly reshape adipokine profiles and vice versa. In the context of diabetes–obesity crosstalk, such coordinated modulation could shift the system from a feed-forward insulin-resistant state toward a more insulin-sensitive, energy-expending phenotype[41].

4. Classes of Nanomaterials for Adipose and Muscle Targeting

Several classes of nanomaterials are particularly suited for the modulation of adipokines and myokines. Lipid-based nanoparticles, including liposomes and solid lipid nanoparticles, are biocompatible and can be formulated with surface ligands that recognize receptors enriched on adipocytes or muscle cells[25, 42, 43]. Polymeric nanoparticles, constructed from biodegradable materials such as PLGA, PEGylated polymers and natural polysaccharides, offer tunable size, release kinetics and functionalization capacity. Inorganic nanomaterials such as gold, iron oxide and certain metal oxides provide unique optical and magnetic properties enabling imaging-guided delivery, photothermal or magnetothermal stimulation and catalytic modulation of local redox states[44, 45].

Adipose targeting can be enhanced using peptides that bind to markers of angiogenic vasculature in expanding adipose depots, antibodies against adipocyte-specific proteins or lipophilic coatings that favor partition into fat-rich tissues. Muscle targeting may exploit ligands for receptors expressed on myofibers or endothelial cells in muscle microvasculature, as well as magnetically guided nanoparticles that respond to external fields applied over muscle groups[10, 46, 47]. Once localized, nanocarriers can release their payloads in response to environmental triggers such as pH, redox gradients or enzymatic activity characteristic of inflamed or insulin-resistant tissues, providing spatially and temporally controlled modulation of adipokines and myokines.

5. Mechanisms of Nanomaterial-Induced Modulation of Adipokines and Myokines

Nanomaterials can alter adipokine and myokine networks through several mechanistic routes. One is direct gene expression modulation via nucleic acid delivery. siRNA, miRNA mimics or antisense oligonucleotides loaded in nanoparticles can downregulate or upregulate specific adipokines or myokines or their upstream regulators[32, 42, 48]. For instance, silencing of negative regulators of adiponectin expression in adipocytes or of myostatin in muscle could shift endocrine outputs toward more insulin-sensitizing profiles. Similarly, nanocarriermediated delivery of miRNAs that promote browning or mitochondrial biogenesis could indirectly enhance the secretion of beneficial adipokines and myokines[49, 50].

Another mechanism is immune microenvironment reprogramming. Nanoparticles taken up by macrophages can carry anti-inflammatory drugs, nuclear receptor ligands or tolerogenic signals that promote M2-like polarization, thereby reducing secretion of pro-inflammatory cytokines that suppress insulin-sensitizing adipokines and healthy myokine responses[51]. In muscle, reducing local inflammatory cytokines can restore exercise-induced myokine patterns and improve insulin signaling.

A third mechanism involves organelle targeting. Mitochondria-targeted nanoparticles delivering antioxidants or mild uncouplers can normalize reactive oxygen species and improve oxidative phosphorylation in adipocytes and myocytes, which in turn influences transcriptional programs for adipokine and myokine production. Endoplasmic Reticulum–targeted nanocarriers could relieve ER stress, another driver of maladaptive cytokine secretion in obesogenic conditions[51].

Finally, some nanomaterials exert physical stimuli that mimic aspects of exercise or cold exposure. Photothermal nanoparticles activated by near-infrared light can elevate local temperature in subcutaneous fat, promoting browning and altering adipokine expression. Magnetothermal or piezoelectric nanomaterials exposed to external fields or mechanical forces can modulate ion channels and mechanosensitive pathways in muscle, potentially triggering myokine release. While these strategies are experimental and far from clinical application, they underscore the breadth of mechanisms through which nanomaterials might reshape adipose–muscle endocrine communication.

6. Preclinical Evidence and Metabolic Outcomes

Preclinical studies investigating nanomaterial effects on adipokines and myokines often report improved systemic metabolic outcomes in parallel with endocrine changes[52]. In rodent models of diet-induced obesity and insulin resistance, adipose-targeted nanoparticles carrying anti-inflammatory agents or PPARγ modulators have been shown to reduce adipose macrophage infiltration, lower TNF-α and IL-6 levels, and increase adiponectin, accompanied by improved insulin sensitivity and reduced hepatic steatosis[53]. Muscle-targeted nanoformulations of AMPK activators or mitochondrial protectants have enhanced exercise capacity, increased expression of PGC-1α and browning-related genes, and upregulated beneficial myokines such as irisin and IL-15, resulting in increased energy expenditure and improved glucose tolerance[53].

Nanomaterial-enabled browning strategies, including photothermal or cold-mimetic nanoparticles localized to white adipose tissue, have induced UCP1 expression, elevated energy expenditure and reduced weight gain, often with concurrent shifts in adipokine profiles toward higher adiponectin and lower leptin and inflammatory cytokines[54]. Similarly, systemic administration of nanoparticles designed to reprogram immune cells has decreased systemic inflammatory markers and improved glycemic control, with changes in both adipokines and myokines suggesting a rebalanced adipose–muscle axis[54].

Although these studies are heterogeneous in design, they collectively support the notion that nanomaterial-based interventions can simultaneously improve metabolic parameters and reshape adipokine–myokine networks. However, detailed longitudinal profiling of endocrine changes is often limited, and the causal interplay between specific adipokine or myokine shifts and metabolic improvements remains to be fully disentangled.

7. Translational Challenges and Opportunities in Diabetes–Obesity Management

Translating nanomaterial-based modulation of adipokines and myokines into clinical practice faces several challenges[55]. Human adipose and muscle depots exhibit substantial interindividual variability in size, distribution, vascularization and inflammatory status, which may affect nanoparticle targeting and endocrine responses[55]. Safety concerns, particularly related to long-term nanomaterial accumulation, immune reactions and off-target effects, must be rigorously addressed, especially as interventions would likely be chronic in metabolic disease. Regulatory pathways for complex nanomedicines that aim to modulate endocrine networks are still evolving and will require robust characterization of pharmacokinetics, biodistribution, endocrine effects and metabolic outcomes.

On the other hand, the opportunities are considerable. Nanomedicine could be integrated with existing therapies such as GLP-1 receptor agonists, SGLT2 inhibitors and lifestyle interventions to enhance their impact on adipose–muscle crosstalk[2, 56, 57]. For example, adipose-targeted nanoparticles might augment the adiponectin-raising and weight-lowering effects of GLP-1 agonists, while muscle-directed nanomaterials could potentiate exercise-induced myokine responses in individuals with limited physical capacity. Personalized approaches, guided by imaging and circulating adipokine and myokine profiling, could tailor nanomaterial-based interventions to specific depot pathologies and endocrine signatures.

CONCLUSION

Nanomaterial-based modulation of adipokines and myokines offers a promising yet still nascent avenue to influence diabetes–obesity crosstalk at its endocrine roots. By targeting adipose tissue and skeletal muscle with precision, nanomaterials can reprogram local inflammation, organelle function and transcriptional networks, thereby reshaping adipokine and myokine profiles toward more insulin-sensitizing, energy-expending states. Preclinical studies suggest that such interventions can improve insulin sensitivity, promote weight loss or weight stabilization, and ameliorate obesity-associated complications, although detailed endocrine profiling and mechanistic dissection remain incomplete. Translational progress will depend on resolving safety and regulatory challenges, understanding interindividual variability in depot targeting and endocrine responses, and integrating nanomedicine with established pharmacological and lifestyle strategies. If these hurdles can be overcome, nano-enabled control of adipose–muscle endocrine communication may become an important component of next-generation, tissue-specific approaches to the prevention and treatment of obesity-associated diabetes.

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