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Phytochemical Nanoformulations Targeting Adipogenesis, Lipolysis, and Thermogenesis

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ABSTRACT

Phytochemicals with anti-obesity potential modulate adipogenesis, lipolysis, and thermogenesis through pleiotropic effects on transcriptional networks, mitochondrial function, and inflammatory signaling. However, their clinical impact is frequently limited by poor aqueous solubility, chemical instability, extensive first-pass metabolism, efflux transport, and inconsistent tissue exposure. Phytochemical nanoformulations offer a translational strategy to overcome these barriers by improving dissolution, protecting labile structures, enhancing intestinal absorption or gut-local exposure, and reshaping biodistribution toward metabolically relevant tissues. Lipid-based systems, polymeric nanoparticles, biopolymer matrices, micelles, and nanocrystals can stabilize phytochemical delivery and support sustained or region-specific release, thereby aligning exposure with metabolic endpoints such as suppression of adipocyte differentiation, enhanced fatty acid mobilization, and induction of browning and mitochondrial thermogenesis. Beyond bioavailability, nanoformulations enable co-delivery of complementary phytochemicals and potentially amplify pathway-level effects at lower doses. Yet obesity is a chronic-use indication; thus, long-term safety, manufacturing scalability, reproducible characterization, and clear PK–PD linkage are mandatory. This review synthesizes mechanistic targets across adipogenesis, lipolysis, and thermogenesis and evaluates how nanoformulation design can optimize phytochemical engagement of these pathways while addressing translational constraints.

Keywords: adipogenesis; lipolysis; thermogenesis; phytochemicals; nanomedicine

INTRODUCTION

Obesity is a chronic metabolic disorder in which excess energy storage is accompanied by adipose tissue dysfunction, systemic low-grade inflammation, and impaired metabolic flexibility[1–3]. Although body weight is the headline phenotype, obesity-related morbidity is tightly linked to dysregulated adipose biology, including adipocyte hypertrophy, altered adipokine secretion, immune cell infiltration, and mitochondrial dysfunction. These changes impair insulin sensitivity and promote ectopic lipid deposition in liver and skeletal muscle, increasing cardiometabolic risk[4–6]. Contemporary anti-obesity pharmacotherapy increasingly targets appetite and energy intake, yet many patients require complementary strategies that improve adipose tissue quality, reduce inflammatory tone, and increase energy expenditure. Within this landscape, phytochemicals have long been proposed as adjunctive metabolic modulators due to their pleiotropic effects on lipid metabolism, inflammation, oxidative stress, and cellular signaling.

Three adipose-centered processes define key intervention points for phytochemical obesity therapy: adipogenesis, lipolysis, and thermogenesis. Adipogenesis is the differentiation of preadipocytes into mature adipocytes and the transcriptional program that enables lipid storage[7–9]. In obesity, excessive adipogenesis and lipid storage capacity contribute to adipose expansion, yet paradoxically, dysfunctional adipogenesis can worsen ectopic lipid deposition[10]. Therapeutic strategies thus aim less at blocking adipogenesis indiscriminately and more at reducing pathological adipocyte expansion and improving adipose remodeling. Lipolysis is the hydrolysis of triglycerides into free fatty acids and glycerol, primarily regulated by hormone-sensitive lipase and adipose triglyceride lipase and integrated with insulin signaling[9, 11]. Enhancing lipolysis without a parallel increase in fatty acid oxidation can be counterproductive, increasing circulating lipids and ectopic fat; therefore, pro-lipolytic interventions are most valuable when paired with improved mitochondrial oxidation. Thermogenesis, including the browning of white adipose tissue and activation of brown/beige

adipocytes, increases energy expenditure through mitochondrial uncoupling and UCP1-associated pathways. Stimulating thermogenesis is conceptually attractive because it targets the energy balance equation directly, but clinical translation requires sustained, safe activation that does not trigger cardiovascular adverse effects[12–14].

Multiple plant-derived compounds influence these processes. Polyphenols can suppress adipogenic transcription factors and attenuate inflammation that promotes adipose dysfunction. Alkaloids and terpenoids can influence AMPK signaling, mitochondrial biogenesis, and lipid oxidation pathways, shifting adipose metabolism away from storage[15–17]. Several phytochemicals have been associated with increased expression of thermogenic genes and markers of browning in preclinical models, often coupled to improved insulin sensitivity. Importantly, phytochemicals also interact with the gut microbiota and bile acid signaling, indirectly influencing adipose metabolism through systemic inflammatory and endocrine pathways[18].

Despite compelling mechanisms, phytochemical translation has been inconsistent, primarily because exposure is low and variable. Many phytochemicals are poorly water-soluble, leading to dissolution-limited absorption and high dose requirements. Chemical instability in the gastrointestinal tract, efflux transport, and extensive first-pass metabolism further reduce the fraction reaching systemic circulation[19–21]. Moreover, the active species may be metabolites rather than the parent compound, and microbial biotransformation introduces inter-individual variability. Even when systemic levels improve, distribution to adipose tissue may remain insufficient to reprogram adipocyte biology[22, 23]. These limitations contribute to modest effect sizes, variable clinical signals, and challenges in linking dose to outcome.

Phytochemical nanoformulations address these barriers by engineering delivery systems that improve solubility and stability, control release, and reshape absorption pathways. Lipid-based nanoemulsions and lipid nanoparticles increase apparent solubility of hydrophobic phytochemicals and can reduce precipitation in intestinal fluids[24]. Certain lipid systems may promote lymphatic uptake, potentially bypassing part of first-pass metabolism and increasing systemic exposure[25, 26, 27-32]. Polymeric nanoparticles can protect phytochemicals and provide sustained release, maintaining therapeutic concentrations over longer durations. Biopolymer matrices can deliver region-specific release in the distal intestine or colon, aligning with gut-mediated mechanisms that influence adipose metabolism. Nanocrystals offer a carrier-minimal approach to improve dissolution rate while minimizing excipient burden, an important consideration for chronic obesity therapy.

Yet nanomedicine is not automatically advantageous. Particle size distribution, surface properties, encapsulation efficiency, and stability determine absorption and biodistribution. Obesity is a chronic-use indication, so long-term safety and tolerability are paramount; nanoformulations must avoid chronic gastrointestinal irritation, immune activation, and unintended microbiome disruption[27, 28, 33-38]. Manufacturing scalability and reproducible characterization are also essential, because small formulation differences can produce large pharmacokinetic differences. Finally, mechanistic claims require PK–PD linkage: evidence that nano-enabled exposure changes translate into measurable modulation of adipogenesis, lipolysis, and thermogenesis markers and that these biomarker changes align with clinically meaningful outcomes such as reduced fat mass, improved insulin sensitivity, and improved lipid profiles. This review focuses on phytochemical nanoformulations designed to target adipogenesis, lipolysis, and thermogenesis. We examine pathway-specific rationale, formulation strategies that improve exposure and tissue engagement, emerging co-formulation approaches, and translational challenges that must be resolved to move from preclinical promise to clinically credible anti-obesity nanotherapeutics.

2. Targeting Adipogenesis with Phytochemical Nanoformulations

Adipogenesis is orchestrated by a transcriptional cascade dominated by PPARγ and C/EBP family regulators, supported by insulin signaling, lipid availability, and local inflammatory cues. In obesity, excessive adipogenic drive can expand adipose mass, while inflammatory dysfunction can produce maladaptive adipocyte phenotypes[29, 30, 39-43]. Phytochemicals that modulate adipogenesis often exert effects by suppressing adipogenic transcriptional activation, reducing oxidative stress, and attenuating inflammatory pathways that reinforce adipocyte differentiation and hypertrophy. However, achieving sustained adipose-relevant concentrations is difficult with conventional oral dosing due to solubility and metabolism limitations.

Nanoformulations can improve the practical engagement of adipogenesis pathways by stabilizing phytochemicals and providing sustained exposure. Lipid-based carriers can solubilize hydrophobic anti-adipogenic phytochemicals and reduce dose requirements by improving absorption[31,44-50]. Polymeric nanoparticles can provide controlled release, potentially maintaining submaximal but sustained tissue levels that are better suited to reprogramming transcriptional networks than sharp exposure peaks. This matters because adipogenesis involves time-dependent gene regulation, and intermittent exposure may fail to produce stable phenotypic shifts[32, 33,51-55].

An additional translational consideration is mechanism localization. Some anti-adipogenic effects may be mediated indirectly through improved systemic insulin sensitivity or reduced endotoxemia-driven inflammation via gut mechanisms[34,56-60]. In such cases, nanoformulations designed for distal intestinal release and microbiota interaction may influence adipogenesis secondarily by altering inflammatory tone and endocrine signals. Therefore, nanoformulation design should be aligned to whether adipogenesis modulation is intended

as a direct adipocyte effect or an indirect consequence of systemic and gut-mediated metabolic improvements[34,61-65].
Ultimately, adipogenesis-targeting nanoformulations must demonstrate more than reduced lipid droplet accumulation in cell models. Translational credibility requires linking nano-enabled exposure to modulation of adipogenic markers in vivo and demonstrating that these changes contribute to improved adipose function and reduced ectopic lipid burden without provoking maladaptive lipolysis or energy imbalance[34,66-70].

3. Enhancing Lipolysis While Preserving Metabolic Safety

Lipolysis is a regulated process that mobilizes stored triglycerides during energy demand. In obesity, insulin resistance and inflammatory signaling can dysregulate lipolysis, increasing basal fatty acid release and contributing to ectopic lipid accumulation[35,71-76]. Therapeutic strategies that stimulate lipolysis must therefore be coupled to improved fatty acid oxidation and mitochondrial function; otherwise, increased lipolysis can worsen dyslipidemia and hepatic steatosis. Phytochemicals linked to improved AMPK signaling, mitochondrial biogenesis, and reduced adipose inflammation may support a safer lipolytic profile by promoting oxidation and limiting inflammatory reprogramming[35,77-80].
Nanoformulations can contribute in two ways. First, by increasing bioavailability, they can enable lower doses that reduce off-target stimulation and adverse effects. Second, by controlling release, they can reduce peak-driven lipolytic surges and promote steadier metabolic modulation. Sustained-release polymeric systems are particularly relevant here, as they may support continuous mild activation of oxidation pathways rather than intermittent spikes that increase free fatty acid flux[36–38,81-87].
Lipolysis modulation may also benefit from macrophage-focused targeting in inflamed adipose tissue. Adipose-resident macrophages contribute to cytokine signaling that drives insulin resistance and dysregulated lipid flux[26, 39,88-93]. Delivering anti-inflammatory phytochemicals to these immune niches can improve insulin responsiveness in adipocytes, thereby normalizing lipolysis regulation rather than simply stimulating lipolysis pharmacologically. This reframes lipolysis targeting away from "turning lipolysis on" and toward restoring physiologic control.
From a translational perspective, lipolysis-targeting nanoformulations should be evaluated using endpoints that capture both mobilization and disposal of fatty acids. Improvements in insulin sensitivity, reductions in triglycerides, improvements in hepatic steatosis markers, and shifts in adipokines provide a more meaningful picture than lipolysis markers alone[40,94-98]. Without demonstrating coordinated oxidation and reduced ectopic lipid burden, lipolysis-focused interventions risk metabolic trade-offs that undermine obesity therapy goals.

4. Inducing Thermogenesis and Browning Through Nano-Enabled Phytochemicals

Thermogenesis increases energy expenditure by dissipating mitochondrial proton gradients, classically through UCP1-dependent pathways in brown and beige adipocytes. Browning of white adipose tissue represents a potential therapeutic approach because it shifts adipose from storage toward energy dissipation[41,99-105]. Phytochemicals reported to influence thermogenic pathways often act through AMPK-related signaling, mitochondrial biogenesis regulators, and anti-inflammatory actions that support mitochondrial function. However, inducing thermogenesis in humans is challenging; it requires sustained and safe activation and may be limited by the quantity and responsiveness of brown/beige adipose depots[41,106-110].
Nanoformulations can strengthen thermogenic engagement by increasing exposure and improving tissue delivery. Lipid-based carriers and micellar systems can increase systemic availability of hydrophobic thermogenesis-associated phytochemicals, while controlled-release nanoparticles can sustain signaling needed for transcriptional reprogramming toward beige adipocyte phenotypes. Because thermogenesis-related gene induction is time-dependent, steady exposure may be more effective than short-lived peaks[42, 43].
A second route is gut-mediated thermogenic signaling. Alterations in microbiota-derived metabolites and bile acid pools can influence energy expenditure and adipose thermogenic programming. Nanoformulations that target distal intestine or colon can amplify such gut–adipose signaling pathways while limiting systemic exposure[44]. This may be advantageous for safety and for chronic use, particularly when thermogenic benefit is mediated through endocrine and immune modulation rather than direct adipocyte receptor engagement.
Nevertheless, thermogenesis claims must be held to rigorous standards. Evidence should include changes in thermogenic gene expression, mitochondrial markers, and energy expenditure metrics, along with confirmation that weight or fat mass reductions are not explained solely by reduced intake or illness behavior. Translationally, the most credible thermogenesis-focused nanoformulations will be those that show consistent exposure, safe long-term use, and measurable improvements in metabolic markers alongside modest but durable reductions in adiposity.

5. Platform Design Choices that Determine Pathway Engagement

The ability of a nanoformulation to influence adipogenesis, lipolysis, and thermogenesis depends on design attributes that determine exposure and biodistribution. For dissolution-limited phytochemicals, lipid nanoemulsions, solid lipid nanoparticles, and nanostructured lipid carriers can markedly improve dispersion and protect against precipitation[45]. For compounds that require sustained signaling, biodegradable polymer nanoparticles can provide extended release profiles that better align with transcriptional reprogramming needed for adipogenesis suppression or thermogenic induction. For gut-local or microbiota-linked mechanisms,

biopolymer matrices enabling region-specific release can amplify endocrine and inflammatory pathways that secondarily influence adipose biology[45].

Nanocrystals provide a simpler route when the compound is stable but poorly soluble, offering improved dissolution with limited excipient burden. This can be attractive in obesity therapy, where long-term tolerability and affordability are essential. However, nanocrystals may not protect against chemical degradation, and they generally lack the release control achievable with polymeric systems[46, 47].

Co-formulation can further refine pathway engagement. Co-delivering phytochemicals that complement each other mechanistically can support coordinated modulation, such as pairing an anti-inflammatory antioxidant with an AMPK-associated oxidation modulator to reduce adipogenesis while supporting safe lipolysis and thermogenesis. Yet co-delivery increases complexity in stability, release synchronization, and quality control, so it requires strong justification and careful development[46].

Across platforms, the translational priority is not maximal complexity but predictable performance. The formulation should deliver consistent exposure, demonstrate stability under biorelevant conditions, and show reproducible modulation of adipose-relevant biomarkers in vivo. Without these, pathway-level claims remain speculative and difficult to translate into clinical outcomes.

6. Translational Challenges: Chronic Safety, Standardization, and PK–PD Evidence

Obesity interventions are used for months to years, so chronic safety dominates translation. Nanoformulations must demonstrate no cumulative toxicity, minimal immune activation, and acceptable gastrointestinal tolerability. For orally delivered systems, maintaining gut barrier integrity is crucial, particularly for formulations using mucoadhesive or permeability-modulating polymers[48, 49]. Because many phytochemicals influence the microbiome, nanoformulations that alter local exposure gradients can produce unintended microbial shifts; therefore, microbiome stability and symptom monitoring are relevant safety dimensions.

Standardization is another decisive factor. For single-compound phytochemicals, purity and stability-indicating assays are essential. For extracts, consistent chemical fingerprinting and batch-to-batch reproducibility become central. Without consistent inputs, nanoengineering cannot produce consistent outputs, and clinical trial results become uninterpretable[50, 51].

The most critical translational gap remains PK–PD linkage. Many studies report metabolic improvements without demonstrating that nano-enabled exposure increases drive pathway modulation in adipose tissue. Translation requires measuring parent compounds and key metabolites, quantifying tissue exposure where feasible, and linking these data to mechanistic markers of adipogenesis, lipolysis regulation, and thermogenesis. In obesity, clinical endpoints should prioritize fat mass, waist circumference, insulin sensitivity, lipid profiles, inflammatory markers, and hepatic steatosis indicators rather than body weight alone[50]. Only when biomarker changes and clinical endpoints align with exposure can a phytochemical nanoformulation be considered a credible therapeutic advance.

7. Future Directions: Mechanism-Aligned Nanoformulation and Clinical Validation

Future progress will depend on treating pathway biology as a formulation design input. For adipogenesis modulation, sustained low-level exposure that reprograms transcriptional networks may be more valuable than high peaks. For lipolysis, the goal should shift from stimulation to normalization, emphasizing improved insulin signaling and oxidation capacity to prevent ectopic lipid spillover. For thermogenesis, strategies that combine direct adipose signaling with gut-mediated endocrine modulation may provide safer, more durable effects.

Development programs should adopt a stepwise translational strategy. Early-stage work should clarify the active species, determine whether effects are gut-local or systemic, and choose the simplest platform that addresses the dominant barrier. Biorelevant dissolution and release testing should guide optimization, followed by in vivo PK with metabolite mapping and biomarker confirmation. Trials should be phenotype-aware, accounting for NAFLD, baseline inflammation, and insulin resistance severity, and should compare nanoformulations against optimized non-nano formulations to justify added complexity.

As the field matures, the strongest candidates will likely be biodegradable lipid and polymer platforms with conservative excipients, scalable manufacturing, and strong mechanistic evidence connecting exposure to adipose pathway modulation. If these principles are applied, phytochemical nanoformulations could evolve from inconsistent supplements into credible adjuncts that target adipogenesis, lipolysis, and thermogenesis with measurable cardiometabolic benefit.

CONCLUSION

Phytochemical nanoformulations provide a translational route to target adipogenesis, lipolysis, and thermogenesis—three central processes that shape adipose expansion, lipid flux, and energy expenditure in obesity. By improving solubility, protecting labile compounds, enabling controlled or region-specific release, and reshaping biodistribution, nanomedicine can deliver more consistent exposures and more durable pathway engagement than conventional formulations. The most clinically credible strategies are those that align formulation design with biology: sustained exposure for transcriptional reprogramming of adipogenesis and browning, normalization of lipolysis through anti-inflammatory and insulin-sensitizing actions, and thermogenic modulation coupled to mitochondrial support and gut–endocrine signaling. Translation will depend on long-term safety, standardized botanical inputs, scalable manufacturing, and rigorous PK–PD

evidence linking exposure to adipose biomarkers and meaningful endpoints such as reduced fat mass, improved insulin sensitivity, and improved lipid and inflammatory profiles.

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