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Plant-Derived Bioactives in Nanoplatforms for Obesity and Cardiometabolic Risk Reduction

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ABSTRACT

Plant-derived bioactives offer pleiotropic actions relevant to obesity and cardiometabolic risk, including modulation of appetite and gut hormone signaling, suppression of adipose inflammation, improvement of insulin sensitivity, regulation of lipid metabolism, and enhancement of antioxidant defenses. However, translation into consistent clinical benefit is frequently limited by poor solubility, chemical instability, extensive first-pass metabolism, efflux transport, and inter-individual variability driven by diet and microbiome. Nanoplatforms can address these barriers by improving dissolution and stability, enhancing mucosal residence, enabling controlled or region-specific release, and reshaping biodistribution toward metabolically relevant tissues such as liver and adipose depots. Lipid-based nanoparticles, polymeric and biopolymeric carriers, micelles, and nanocrystals have been used to stabilize and deliver polyphenols, alkaloids, terpenoids, and phytosterols with the goal of achieving more predictable exposure and stronger pathway engagement. Beyond weight loss, nanoplatform-enabled bioactives may reduce cardiometabolic risk by lowering atherogenic dyslipidemia, improving endothelial function, reducing inflammatory and oxidative stress markers, and ameliorating hepatic steatosis. Yet obesity is a chronic-use indication; therefore, long-term safety, scalable manufacturing, rigorous characterization, and clear PK–PD linkage to meaningful endpoints remain decisive. This review synthesizes how plant-derived bioactives delivered via nanoplatforms can contribute to obesity management and cardiometabolic risk reduction, highlighting mechanism-aligned design strategies and translational challenges that must be addressed for clinical adoption.

Keywords: phytochemicals; nanoplatforms; obesity; cardiometabolic risk; metabolic syndrome

INTRODUCTION

Obesity is a chronic, relapsing metabolic disease that increases risk of type 2 diabetes, atherosclerotic cardiovascular disease, hypertension, chronic kidney disease, and non-alcoholic fatty liver disease[1–3]. While excess adiposity is the defining feature, cardiometabolic risk is driven by deeper biological changes: adipose tissue inflammation and immune infiltration, dysregulated adipokine secretion, insulin resistance, altered lipid flux, endothelial dysfunction, oxidative stress, and ectopic lipid deposition[4–6]. These processes interact through reinforcing loops that make obesity difficult to reverse and contribute to residual cardiometabolic risk even when modest weight loss is achieved. Therefore, therapies that improve metabolic tissue quality and reduce inflammatory and oxidative drivers can be valuable complements to interventions that primarily reduce caloric intake.

Plant-derived bioactives have long been considered candidates for such adjunctive roles. Polyphenols, alkaloids, terpenoids, saponins, and phytosterols can influence multiple targets relevant to obesity and cardiometabolic risk. Many bioactives reduce inflammatory signaling and oxidative stress, improve insulin sensitivity through AMPK-linked and insulin receptor pathway modulation, regulate lipid metabolism through effects on hepatic lipogenesis and fatty acid oxidation, and influence gut microbiota and bile acid signaling that shape systemic metabolism[7–9]. Some also interact with appetite regulation and gut hormone pathways, potentially contributing to reduced energy intake and improved glycemic control. The appeal of plant-derived bioactives lies in this pleiotropy, which aligns with the multi-organ nature of obesity-related cardiometabolic disease[10–12].

However, the clinical translation of plant bioactives remains inconsistent. A central barrier is pharmacokinetic. Many bioactives are poorly soluble in water, unstable in gastrointestinal conditions, and rapidly metabolized, resulting in low and variable systemic exposure. Efflux transporters can further reduce net absorption, while extensive first-pass metabolism produces conjugated metabolites whose activity may differ from the parent compound[13, 14]. In addition, inter-individual variability in microbiome composition can alter phytochemical biotransformation, generating divergent metabolite profiles and variable biological responses. Botanical variability and differences in formulation quality also contribute to inconsistent outcomes in trials and real-world use. Consequently, while mechanistic and preclinical evidence is often strong, effect sizes in clinical studies may be modest or variable, limiting confidence for therapeutic integration.

Nanoplatforms provide a way to address this translational gap by engineering delivery systems that improve exposure consistency and align the site and kinetics of bioactive release with relevant biology. Nanoplatforms can increase apparent solubility and dissolution rate, protect labile compounds from degradation, and reduce precipitation in intestinal fluids. Some lipid-based nanocarriers can promote lymphatic uptake, partially bypassing hepatic first-pass metabolism and increasing systemic availability for lipophilic bioactives[15–17]. Polymeric nanoparticles can provide sustained release, enabling steady exposure that may be better suited to metabolic reprogramming than transient peaks. Biopolymeric platforms can deliver region-specific release in distal gut segments, amplifying microbiota and bile acid signaling mechanisms that influence insulin sensitivity and lipid metabolism[18–20]. Nanocrystals offer a carrier-minimal approach for stable but poorly soluble compounds, potentially improving dissolution while limiting excipient burden, an important consideration for long-term use.

Importantly, obesity and cardiometabolic risk reduction should not be evaluated solely through weight loss. Reductions in waist circumference and fat mass matter, but risk reduction also depends on improvements in glycemic control, lipid profiles, blood pressure, inflammatory markers, endothelial function, and hepatic steatosis[21, 22]. Plant-derived bioactives may be particularly valuable when they improve these risk mediators, even if absolute weight loss is moderate. Nanoplatforms can help by ensuring the bioactive reaches tissues where these risk pathways are regulated, such as liver for lipid handling and steatosis, adipose tissue for inflammatory signaling and adipokines, and vascular endothelium for oxidative stress and nitric oxide bioavailability. Gut-targeted delivery can also influence cardiometabolic risk indirectly through reduced endotoxemia, altered bile acid pools, and improved incretin signaling[15, 23, 24].

Despite these opportunities, translation into obesity therapy requires high standards. Obesity and metabolic syndrome are chronic conditions, so nanoplatforms must demonstrate long-term safety, minimal immunogenicity, and no cumulative toxicity. Materials and excipients must be suitable for repeated dosing, and formulations must not chronically disrupt gut barrier integrity or microbiome stability in ways that cause unintended harm[25]. Manufacturing must be scalable and reproducible; small changes in particle size distribution or surface chemistry can alter absorption and biodistribution, undermining clinical reproducibility. Regulatory expectations for nanoformulated products are also substantial, especially if therapeutic claims are made. Finally, PK–PD linkage is essential: improved bioavailability must translate into meaningful changes in risk-related biomarkers and clinical endpoints. This review examines plant-derived bioactives delivered via nanoplatforms for obesity management and cardiometabolic risk reduction. We synthesize key mechanistic domains through which bioactives influence risk, evaluate nanoplatform classes and design strategies that align delivery with biology, and discuss translational challenges including safety, standardization, manufacturing, and clinical validation.

2. Cardiometabolic Risk Pathways Addressed by Plant Bioactives

Cardiometabolic risk in obesity is shaped by insulin resistance, atherogenic dyslipidemia, adipose inflammation, endothelial dysfunction, oxidative stress, and hepatic steatosis. Plant bioactives can influence each domain, often through overlapping mechanisms[26–28]. Improvements in insulin sensitivity can reduce hyperinsulinemia-driven lipogenesis and lower triglyceride-rich lipoprotein production. Anti-inflammatory actions in adipose tissue can improve adipokine profiles and reduce cytokine signaling that impairs insulin pathways. Antioxidant and redox-modulating effects can improve endothelial function by preserving nitric oxide signaling and reducing oxidative modification of lipoproteins[29, 30]. Modulation of hepatic lipid metabolism can reduce steatosis and improve lipid profiles, lowering remnant lipoproteins and atherogenic particle burden. Gut-mediated mechanisms, including microbiota remodeling and bile acid signaling shifts, can influence glucose and lipid metabolism and reduce metabolic endotoxemia that contributes to systemic inflammation[31].

These mechanisms are often interdependent. Reducing hepatic fat can improve insulin sensitivity; improving insulin sensitivity can reduce hepatic triglyceride production; reducing systemic inflammation can improve endothelial function. Therefore, plant bioactives are appealing not because they act on a single risk factor but because they can shift the overall network toward lower cardiometabolic risk. The challenge is delivering consistent, pathway-relevant exposure to realize these network effects in clinical settings.

3. Nanoplatforms as Enablers: Improving Exposure and Aligning Mechanism to Site of Action

Nanoplatforms address the core barriers that limit plant bioactive translation. For hydrophobic bioactives, nanoscale encapsulation increases apparent solubility and dissolution, enabling improved absorption at lower doses[32]. Encapsulation can protect labile compounds from oxidation and pH-driven degradation, improving

stability across gastrointestinal transit. By stabilizing dispersion, nanoplatforms can reduce variability driven by meals and gut transit, improving exposure consistency.

Mechanism alignment is equally important. If a bioactive's main action is within the gut lumen, such as influencing cholesterol absorption or modulating microbiota-derived signaling, nanoplatforms can be designed to prolong local exposure and deliver the payload to distal gut regions without necessarily increasing systemic absorption[16, 33]. If systemic actions are central, such as improving hepatic lipid handling or reducing adipose inflammation, nanoplatforms can be optimized for absorption enhancement and, when feasible, for biodistribution toward relevant tissues. Lipid-based carriers may support lymphatic uptake for highly lipophilic compounds, potentially reducing first-pass metabolism and increasing systemic availability.

In obesity, where chronic intervention is required, the added value of nanoplatforms is often best framed as improved consistency and durability of biological engagement rather than simply "higher bioavailability." Stable, predictable exposure is what enables meaningful shifts in insulin sensitivity, lipid profiles, and inflammatory tone over time.

4. Platform Classes and Their Relevance to Obesity and Cardiometabolic Outcomes

Lipid-based nanoplatforms, including nanoemulsions, liposomes, solid lipid nanoparticles, and nanostructured lipid carriers, are widely used for hydrophobic plant bioactives and can integrate well with physiological lipid absorption pathways. They are particularly relevant for bioactives targeting dyslipidemia and hepatic lipid metabolism, where improved solubilization and potential lymphatic uptake can support systemic effects[34, 35]. Polymeric nanoparticles provide controlled release and strong protection, making them useful when sustained systemic exposure is needed to modulate inflammatory and metabolic pathways. Biopolymeric platforms are especially valuable for gut-focused mechanisms, because they can be engineered for pH- or enzyme-responsive release and distal intestinal delivery. Micellar systems offer efficient solubilization but must maintain stability upon dilution[19]. Nanocrystals provide a carrier-minimal approach for stable, poorly soluble bioactives, potentially lowering excipient burden for chronic use while improving dissolution.

Across platforms, key determinants of clinical viability include the ability to manufacture reproducibly, maintain stability during storage, and demonstrate safe long-term tolerability. In cardiometabolic risk reduction, the platform should be selected not only for absorption performance but for its ability to support sustained, mechanism-relevant exposure while minimizing systemic risks[36].

5. Evidence Priorities: From PK to Risk Reduction Biomarkers

For nanoplatform-enabled plant bioactives to credibly reduce cardiometabolic risk, evidence must connect formulation performance to meaningful outcomes. PK studies should quantify parent compounds and relevant metabolites and demonstrate reduced variability across individuals and meal conditions[37]. PD studies should then show improvements in biomarkers that mediate risk, including insulin sensitivity indices, atherogenic lipid fractions such as triglycerides and non-HDL cholesterol, inflammatory markers, and indicators of oxidative stress. Because obesity-associated NAFLD is tightly linked to dyslipidemia and insulin resistance, hepatic steatosis markers and liver-related endpoints provide important context for risk reduction[37].

Weight loss alone is insufficient as proof of cardiometabolic benefit. Reductions in visceral adiposity, improvements in lipid handling, improved endothelial function, and reduced inflammatory tone are more directly linked to vascular risk[38]. Therefore, well-designed studies should incorporate these endpoints and demonstrate that nanoformulation improves the magnitude or consistency of these changes compared with conventional formulations or standardized non-nano preparations.

Finally, cardiometabolic benefit must be durable. Controlled or sustained release profiles may support durable biomarker improvements, but this must be demonstrated in longer-duration trials consistent with chronic metabolic disease management.

6. Translational Challenges: Safety, Standardization, Manufacturing, and Interactions

Long-term safety is the central challenge because obesity and cardiometabolic risk reduction require prolonged use. Nanoplatform materials must be biocompatible, biodegradable or safely cleared, and tolerable with daily administration[39, 40]. Oral nanoplatforms must not chronically irritate the gastrointestinal tract or disrupt barrier integrity. Since many plant bioactives modulate the microbiome, formulations that alter gut exposure patterns may also shift microbial ecosystems; unintended dysbiosis must be monitored and minimized[41, 42].

Standardization of plant bioactives is also essential. Variability in botanical sources and extraction methods can alter active composition, complicating dose-response interpretation and product reproducibility[43]. For therapeutic products, standardized extracts or defined compounds with validated analytical fingerprints are required. Manufacturing must ensure consistent particle size distribution, encapsulation efficiency, and release behavior, because these attributes drive absorption and biodistribution.

Interactions with concurrent therapies are common in obesity, where patients may be taking statins, antihypertensives, and glucose-lowering agents. Nanoplatforms that increase systemic exposure could alter transporter and enzyme interactions, so interaction assessments are necessary for safe clinical integration. Regulatory expectations for nanoformulated therapeutics further require robust characterization and evidence of clinical benefit relative to simpler formulations[43].

7. Future Directions: Mechanism-Aligned Nanoplatform Design and Phenotype-Aware Clinical Trials

Future progress will likely focus on minimal-complexity nanoplatforms that deliver measurable improvements in exposure consistency and risk biomarker engagement while remaining scalable and safe for chronic use. Distal gut-targeted systems are particularly promising for bioactives that act through microbiota and bile acid signaling, potentially reducing systemic exposure while improving insulin sensitivity and lipid handling. Sustained-release systemic formulations may be prioritized for bioactives that target hepatic lipid metabolism and adipose inflammation, especially in obesity phenotypes characterized by NAFLD and high inflammatory burden.

Clinical trials should be phenotype-aware because obesity is heterogeneous. Individuals with predominant insulin resistance and NAFLD may respond differently than those with milder metabolic dysfunction. Stratification by baseline inflammation, hepatic steatosis markers, and lipid phenotype can clarify who benefits most. Trials should emphasize cardiometabolic endpoints including triglycerides, non-HDL cholesterol, insulin sensitivity, inflammatory markers, and hepatic steatosis indicators, alongside body composition measures. Comparative studies against optimized non-nano formulations are necessary to justify nanoplatform complexity and cost.

If these priorities are addressed, plant-derived bioactives delivered via nanoplatforms could evolve into credible adjuncts for reducing cardiometabolic risk in obesity, complementing lifestyle and pharmacologic therapies while targeting inflammatory and hepatic pathways that drive residual risk.

CONCLUSION

Plant-derived bioactives have strong mechanistic potential to improve obesity-related cardiometabolic risk by modulating insulin sensitivity, lipid metabolism, inflammation, oxidative stress, endothelial function, and gut–metabolic signaling. Nanoplatforms can translate this potential into more reliable clinical effects by improving solubility and stability, reducing exposure variability, enabling controlled or region-specific release, and reshaping biodistribution toward key metabolic tissues. However, the chronic nature of obesity requires stringent long-term safety, standardized botanical inputs, scalable manufacturing, and rigorous characterization. Translational credibility depends on PK–PD linkage demonstrating that nanoplatform-enabled exposure improvements translate into meaningful reductions in atherogenic lipid fractions, improved insulin sensitivity, reduced inflammatory and oxidative stress markers, and improved hepatic steatosis indicators, not merely short-term weight changes. Minimal-complexity, evidence-led nanoplatform designs supported by phenotype-aware clinical trials offer the most credible path for plant bioactives to contribute to durable cardiometabolic risk reduction in obesity.

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