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Nanoencapsulation of Natural Lipid-Modulating Agents for Obesity-Induced Dyslipidemia

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ABSTRACT

Obesity-induced dyslipidemia, typically characterized by elevated triglycerides, increased small dense LDL, and reduced HDL functionality, is a major mediator of cardiovascular risk in metabolic disease. Natural lipid-modulating agents such as polyphenols, phytosterols, omega-3–rich bioactives, terpenoids, and alkaloids can influence lipid absorption, hepatic lipogenesis, cholesterol transport, bile acid signaling, inflammation, and oxidative stress. However, clinical translation is often limited by poor aqueous solubility, chemical instability, variable intestinal absorption, extensive first-pass metabolism, and inconsistent tissue exposure, especially in the context of obesity-associated gut and hepatic dysfunction. Nanoencapsulation offers a formulation-driven strategy to improve solubilization, protect labile compounds, enhance mucosal residence, promote lymphatic uptake, and enable controlled or region-specific release. Lipid-based systems, polymeric nanoparticles, biopolymer matrices, micelles, and nanocrystals can increase exposure consistency and strengthen pharmacodynamic engagement of lipid metabolism pathways, including reductions in triglyceride-rich lipoproteins and improved hepatic lipid handling. Yet dyslipidemia requires chronic management; therefore, long-term safety, scalable manufacturing, and rigorous characterization are essential, as is clear PK–PD linkage to clinically meaningful endpoints such as triglycerides, apoB-containing lipoproteins, non-HDL cholesterol, and markers of hepatic steatosis and inflammation. This review evaluates nanoencapsulation strategies for natural lipid-modulating agents in obesity-induced dyslipidemia and highlights translational priorities for clinical adoption.

Keywords: dyslipidemia; obesity; nanoencapsulation; natural products; lipid metabolism

INTRODUCTION

Obesity-induced dyslipidemia is a core feature of cardiometabolic disease and a major driver of atherosclerotic cardiovascular risk[1, 2]. In the setting of visceral adiposity and insulin resistance, lipid flux and lipoprotein metabolism are altered at multiple levels. Increased adipose lipolysis and impaired insulin-mediated suppression of free fatty acid release raise hepatic fatty acid delivery[3–5]. The liver responds with increased triglyceride synthesis and secretion of triglyceride-rich lipoproteins, while clearance pathways for these particles are often impaired. The resulting phenotype commonly includes elevated fasting and postprandial triglycerides, increased remnant lipoproteins, reduced HDL cholesterol and HDL functionality, and a shift toward small dense LDL particles that are more atherogenic[6]. This dyslipidemic profile often coexists with non-alcoholic fatty liver disease and systemic inflammation, amplifying vascular injury[7].

Pharmacologic lipid-lowering therapies, including statins, ezetimibe, PCSK9 inhibitors, fibrates, and omega-3 formulations, have improved outcomes, yet gaps remain. Some individuals experience residual hypertriglyceridemia and remnant cholesterol despite optimized therapy, while others face tolerability issues, contraindications, or access barriers[8]. In addition, dyslipidemia in obesity is not only a matter of lipid levels; oxidative modification of lipoproteins, endothelial dysfunction, and inflammatory signaling contribute to cardiovascular risk[8]. These complexities motivate interest in adjunctive strategies that can modulate lipid handling more broadly, including improvements in hepatic steatosis and reductions in inflammatory lipid signaling.

Natural lipid-modulating agents are attractive candidates for adjunctive management because many exert pleiotropic actions across lipid absorption, synthesis, transport, and oxidative pathways[9-13]. Phytosterols can reduce intestinal cholesterol absorption by competing for incorporation into mixed micelles. Polyphenols can

modulate hepatic lipogenesis, improve antioxidant defenses, and influence bile acid signaling and gut microbiota, which can affect lipid metabolism[10–12,14-19]. Omega-3–rich bioactives influence triglyceride synthesis and secretion, while certain alkaloids and terpenoids can modulate AMPK-related pathways, improving lipid oxidation and reducing lipogenesis[13,20-27]. Some natural agents also influence cholesterol efflux and HDL functionality, potentially improving reverse cholesterol transport and anti-inflammatory properties of HDL particles. However, translation of these agents into consistent clinical benefit has been limited by delivery barriers. Many phytochemicals are poorly water-soluble, leading to dissolution-limited absorption and high dose requirements. Chemical instability and oxidation can reduce potency[14,28-33]. Extensive first-pass metabolism can convert parent compounds into conjugates with altered activity, and efflux transport can further reduce net absorption. In obesity, these challenges may be amplified by altered gut physiology, changes in bile acid pools, and hepatic dysfunction, which can change absorption, metabolism, and distribution[15,34-38]. Furthermore, inter-individual variability in diet and microbiome composition can produce heterogeneous exposure and response patterns[15,39-43]. Therefore, improving delivery and exposure consistency is central to making natural lipid-modulating agents clinically tractable in obesity-induced dyslipidemia.

Nanoencapsulation provides a formulation-driven route to address these constraints. Encapsulation in nanoscale carriers can increase apparent solubility and dissolution rate, protect labile compounds from degradation, and reduce food-dependent variability by generating more predictable dispersion in gastrointestinal fluids[16,44-49]. Lipid-based nanoformulations can leverage physiological lipid absorption pathways and may promote lymphatic transport, partially bypassing hepatic first-pass metabolism for highly lipophilic compounds[16,50-55]. Polymeric and biopolymeric nanoparticles can provide controlled release, maintaining steadier exposure and potentially improving adherence by reducing dosing frequency[17,56-60]. Region-specific delivery, particularly distal intestinal release, can modulate bile acid signaling and gut microbiota, potentially improving lipid handling and systemic inflammation[18,61-67]. Nanocrystals offer a carrier-minimal strategy for dissolution-limited but stable compounds, potentially reducing excipient burden for chronic use.

Yet nanoencapsulation is not a universal solution. The clinical value depends on whether the natural agent is truly exposure-limited and whether improved exposure translates into meaningful changes in lipoprotein metabolism. Dyslipidemia management requires long-term therapy, so chronic safety and tolerability are decisive. Nanocarriers must use biocompatible materials and avoid cumulative toxicity, immune activation, or gastrointestinal irritation with repeated dosing[19, 20,68-73]. Manufacturing must be scalable and reproducible, with defined critical quality attributes such as particle size distribution, stability, encapsulation efficiency, and release behavior under biorelevant conditions. Regulatory expectations will also be higher when a nanoformulation is positioned as a therapeutic product rather than a supplement-like intervention, especially if claims include cardiovascular risk reduction. This review examines nanoencapsulation strategies for natural lipid-modulating agents in obesity-induced dyslipidemia. We discuss key pathophysiological targets relevant to triglyceride-rich lipoproteins and atherogenic dyslipidemia, summarize how nanoencapsulation can improve absorption and pharmacodynamics, evaluate platform classes and design considerations, and highlight translational challenges and a pragmatic roadmap toward clinically credible nano-enabled therapies.

2. Obesity-Induced Dyslipidemia: Targets for Natural Lipid-Modulating Agents

The dyslipidemia of obesity is driven by increased free fatty acid flux from insulin-resistant adipose tissue and increased hepatic synthesis and secretion of triglyceride-rich lipoproteins[21, 22,74-76]. Impaired lipoprotein lipase activity and altered apolipoprotein composition reduce clearance of these particles, increasing remnant cholesterol that contributes to atherosclerosis. HDL particles may become dysfunctional, losing anti-inflammatory and cholesterol efflux capacity, while LDL particles shift toward smaller, denser forms that are more prone to oxidation and arterial retention[23, 24]. Hepatic steatosis and inflammation further amplify dyslipidemia by impairing lipid oxidation and increasing de novo lipogenesis[25, 26].

Natural lipid-modulating agents act across these targets. Compounds that reduce intestinal cholesterol absorption can lower atherogenic cholesterol burden. Agents that suppress hepatic lipogenesis or increase fatty acid oxidation can reduce hepatic triglyceride production and improve steatosis[27]. Anti-inflammatory and antioxidant phytochemicals may reduce oxidative modification of lipoproteins and improve endothelial function, affecting risk beyond lipid concentrations alone. Gut-modulating agents can influence bile acid signaling and microbiota-derived metabolites that regulate hepatic lipid synthesis and systemic inflammation. In obesity-induced dyslipidemia, the most valuable natural agents are those that couple lipid-lowering activity with improvements in hepatic fat and inflammatory tone, because these processes reinforce each other[27]. However, these mechanisms require sufficient exposure at the relevant site, whether in the gut lumen, intestinal wall, liver, or systemic circulation. When exposure is inconsistent, metabolic effects become variable and difficult to scale clinically. This is where nanoencapsulation can meaningfully influence therapeutic reliability.

3. Why Nanoencapsulation Is Strategically Relevant for Lipid-Modulating Phytochemicals

Many natural lipid-modulating compounds are hydrophobic and dissolution-limited, making nanoencapsulation an efficient strategy to increase apparent solubility and enhance absorption. Nanoencapsulation can protect molecules susceptible to oxidation or pH-dependent degradation, preserving bioactivity during gastrointestinal transit[28]. By stabilizing dispersion, nanocarriers can reduce the dependence of absorption on meal

composition and bile secretion, which is particularly relevant in obesity where dietary patterns vary and bile acid signaling may be altered[28].
A unique advantage for lipid-related targets is that lipid-based nanoformulations may promote lymphatic uptake. Lymphatic transport can increase systemic exposure for lipophilic agents and reduce first-pass hepatic metabolism, potentially enabling lower doses and improved predictability[29]. Controlled release, achievable with polymeric systems, may also be valuable in dyslipidemia because lipoprotein metabolism is continuous and postprandial lipid excursions contribute to vascular risk. Sustained exposure may therefore produce more stable modulation of hepatic lipid synthesis and clearance pathways than intermittent peaks[29].
Nanoencapsulation can also be designed for gut-local actions, which are highly relevant for cholesterol absorption inhibition and bile acid signaling modulation. If a natural agent's main effect is luminal, maximizing systemic absorption may be unnecessary; instead, prolonging luminal residence and optimizing release location can improve efficacy while limiting systemic exposure and potential off-target effects. This mechanism-aligned design is central to translating nanoencapsulation into clinically meaningful lipid improvements.

4. Nanoencapsulation Platforms and Their Fit to Dyslipidemia Mechanisms

Lipid-based systems, including nanoemulsions, self-emulsifying formulations, liposomes, solid lipid nanoparticles, and nanostructured lipid carriers, are particularly well suited for hydrophobic lipid-modulating agents[30, 31]. They enhance solubilization in intestinal fluids and can align with physiological lipid transport pathways. Solid lipid nanoparticles and nanostructured lipid carriers add protective matrices and can support controlled release, potentially smoothing exposure profiles during chronic dosing.
Polymeric nanoparticles provide controlled release and strong protection against chemical degradation, which is useful for agents requiring sustained systemic signaling, such as those modulating hepatic lipogenesis or inflammation. Biopolymer matrices can support region-specific release, especially in the distal intestine, which may be valuable for compounds acting through bile acid signaling and microbiota-mediated pathways that influence lipid metabolism and inflammation[32–34].
Micellar systems are efficient solubilizers with small particle sizes that can maintain dispersion, though dilution stability must be ensured[35]. Nanocrystals offer a carrier-minimal approach when the compound is stable but poorly soluble, improving dissolution rate with relatively low excipient load, a potential advantage for long-term dyslipidemia therapy.
Platform choice should reflect the dominant limitation and the target locus. Agents aimed at reducing intestinal cholesterol absorption benefit from formulations that optimize luminal exposure and micellar interaction[36]. Agents aimed at hepatic lipid metabolism benefit from absorption enhancement, controlled release, and potentially lymphatic uptake. Compounds aimed at reducing oxidative modification and inflammation may require systemic exposure and sustained antioxidant presence. Matching platform to mechanism is essential to achieve predictable lipid outcomes.

5. Evidence Expectations: Linking Nano-Enabled Exposure to Lipoprotein Outcomes

For nanoencapsulation to be clinically meaningful in obesity-induced dyslipidemia, it must demonstrate that improved exposure translates into improved lipoprotein biology. This requires careful PK–PD linkage[37]. Measuring only total cholesterol or fasting triglycerides may miss important changes in remnant cholesterol and apoB-containing lipoprotein burden. Clinically relevant lipid endpoints include triglycerides, non-HDL cholesterol, apoB proxies, and measures of postprandial lipemia, along with HDL function metrics where feasible. Because obesity-associated dyslipidemia is often driven by hepatic triglyceride overproduction and NAFLD, endpoints reflecting hepatic fat and inflammation are also important, including liver enzyme patterns and imaging-based steatosis measures when available[37].
Nanoformulations should be evaluated not only for higher systemic concentrations but for reduced variability across individuals and across meal conditions. Consistency is itself a therapeutic advantage because it improves predictability of lipid response and supports adherence[38]. Mechanistic biomarkers can further strengthen translational claims, such as changes in inflammatory markers, oxidative stress indicators, and bile acid profiles for gut-targeted systems.
Without demonstrating improved exposure consistency and meaningful shifts in atherogenic lipid fractions, nanoencapsulation risks becoming a technological refinement without clear clinical value[39]. The translational threshold is therefore higher than in short-term indications and must be met with robust evidence linking formulation attributes to lipid outcomes.

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

Dyslipidemia management is chronic, so nanoencapsulated natural agents must demonstrate long-term safety and tolerability. Excipient selection matters because daily dosing over years can amplify subtle gastrointestinal irritation or immune effects[40]. Lipid-based carriers must avoid excessive surfactant loads that may cause GI symptoms. Polymeric systems must demonstrate safe degradation products and lack of cumulative tissue effects. Because obesity is often accompanied by hepatic steatosis and sometimes impaired renal function, safety evaluations should consider altered clearance and metabolism[40].
Manufacturing scalability and reproducibility are also decisive. Particle size distribution, stability, encapsulation efficiency, and release behavior must remain consistent across batches, since these attributes determine absorption and biodistribution[41]. For botanical agents, standardization of the active component is essential;

batch variability in phytochemical content can undermine clinical reproducibility even when the nanoformulation is stable.

Drug–nutrient and drug–drug interactions are another translational concern. Many patients with obesity-induced dyslipidemia are taking statins, antihypertensives, and glucose-lowering agents. Nanoencapsulation could alter absorption kinetics and potentially interact with transporters and metabolic enzymes. Therefore, interaction assessments are necessary, especially when the nanoformulation aims to increase systemic exposure substantially[28, 42, 43].

Regulatory expectations will depend on positioning, but therapeutic claims require pharmaceutical-grade characterization, stability, and clinical efficacy evidence. These requirements are manageable but must be planned early to avoid late-stage failure.

7. Future Directions

Future progress will depend on selecting natural agents with strong lipid-modulating rationale and then designing nanoencapsulation strategies that solve the dominant barrier while remaining simple, safe, and scalable. For cholesterol absorption–targeted agents, gut-local delivery that optimizes micellar competition and minimizes systemic exposure may offer the best risk–benefit balance. For triglyceride-rich lipoprotein reduction and NAFLD-linked dyslipidemia, formulations that enhance absorption and provide controlled release may produce steadier suppression of hepatic triglyceride production and improved oxidation, particularly when paired with lifestyle interventions and standard pharmacotherapy[44].

Clinical development should move toward phenotype-aware trials that recognize heterogeneity in obesity-induced dyslipidemia. Patients with predominant hypertriglyceridemia and NAFLD may respond differently from those with mixed dyslipidemia driven by genetic background or medication effects[45]. Trials should incorporate measures of variability and postprandial lipemia where feasible, since these better reflect the remnant-driven risk profile common in obesity.

The most promising translational designs will likely be biodegradable lipid or polymer systems with conservative excipients and robust quality control. Demonstrating reduced exposure variability, clear PK–PD linkage, and meaningful improvements in atherogenic lipid fractions will be essential for adoption[45, 46]. If these criteria are met, nanoencapsulated natural lipid modulators could become credible adjuncts for residual dyslipidemia in obesity, complementing standard therapies and addressing inflammatory and hepatic components that contribute to cardiovascular risk.

CONCLUSION

Nanoencapsulation of natural lipid-modulating agents offers a practical strategy to address obesity-induced dyslipidemia by improving solubility, stability, and exposure consistency while enabling controlled or mechanism-aligned delivery. By enhancing absorption, protecting labile compounds, and in some cases promoting lymphatic uptake or distal gut targeting, nanoformulations can strengthen engagement of pathways governing triglyceride-rich lipoprotein production, cholesterol absorption, hepatic lipogenesis, and inflammatory lipid signaling. However, dyslipidemia requires long-term management, so clinical translation depends on chronic safety, scalable manufacturing, reproducible characterization, and rigorous PK–PD linkage to meaningful lipid endpoints including triglycerides, non-HDL cholesterol, apoB-containing lipoproteins, and markers of hepatic steatosis and inflammation. Minimal-complexity, biodegradable platforms that reduce exposure variability and demonstrate clinically relevant improvements beyond conventional formulations are most likely to succeed as adjuncts to standard lipid-lowering and cardiometabolic therapies in obesity.

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