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Palm oil intake and lipid profile alterations in patients with type 2 diabetes: a systematic review of clinical and experimental evidence

Abstract

The occurrence of dyslipidemia is a common metabolic complication in patients with Type 2 Diabetes Mellitus (T2DM). Despite extensive scientific attention directed toward the effects of palm oil and other saturated fat sources on lipid metabolic processes, published findings have yet to establish a clear and unified pattern of evidence. Through a systematic appraisal of published clinical and experimental evidence, this review sought to clarify the relationship between palm oil intake and lipid profile changes in individuals diagnosed with Type 2 Diabetes Mellitus (T2DM), focusing on the agreement among study outcomes, the strength of the reported effects, and the mechanistic explanations supporting these observations across different research designs. The investigation was designed as a systematic review consistent with PRISMA 2020 recommendations, utilizing only peer-reviewed published research as the data source while excluding primary data collection and observational fieldwork. Published literature was identified through a predefined search procedure applied to the Scopus database, beginning with 773 records and narrowed through keyword refinement, a 2020–2026 publication-year filter, and open-access screening to 30 eligible articles. Data analysis used thematic and narrative synthesis to categorize findings and identify converging and diverging patterns across study types. Results showed that direct clinical trials (16.7% of the corpus) generally reported no significant short-term change in conventional lipid parameters following palm oil or tocotrienol intake, whereas population-based studies found habitual palm oil consumption independently associated with metabolic syndrome risk (adjusted OR 4.87). Mechanistic studies (33.3%) offered plausible biological pathways, including oxidative stress and hepatic lipogenesis, without directly testing palm oil in T2DM populations. In conclusion, short-term palm oil intake appears to exert limited direct effects on conventional lipid parameters, while cumulative, habitual consumption shows a stronger association with adverse metabolic outcomes. Future research should prioritize longitudinal and mechanistic human studies with standardized reporting of palm oil type and processing method.

Keywords: palm oil, lipid profile, type 2 diabetes mellitus, systematic review, dyslipidemia

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Introduction

As one of the leading non-communicable diseases, Type 2 Diabetes Mellitus (T2DM) imposes a substantial worldwide health burden. Epidemiological projections suggest that the number of affected adults, already numbering in the hundreds of millions, will continue to grow considerably over the coming decades, largely as a consequence of rapid urban development and nutrition transitions occurring across many low- and middle-income nations.[1] T2DM accounts for the overwhelming majority of all diagnosed diabetes cases and is characterized not only by chronic hyperglycemia but also by a constellation of secondary metabolic disturbances, among which dyslipidemia is one of the most clinically consequential.[2] Dyslipidemia marked by increased LDL-C and decreased HDL-C concentrations and elevated triglyceride concentrations, frequently co-occurs with insulin resistance, forming what is commonly referred to as diabetic dyslipidemia, a condition that substantially amplifies the predisposition of patients in this population to atherosclerotic cardiovascular disorders.[3] As cardiovascular disease remains the predominant cause of both morbidity and mortality in individuals living with T2DM, understanding the modifiable dietary determinants of lipid profile alteration in this population continues to represent a central concern in clinical nutrition and endocrinology research.[4]

Among modifiable dietary factors, the type and source of dietary fat has received sustained scientific attention due to its direct biochemical relationship with circulating lipoprotein metabolism.[5] Saturated fatty acids, in particular, have long been implicated in the modulation of LDL-C concentrations, though the magnitude and clinical significance of this relationship vary considerably depending on the specific fatty acid source, food matrix, and population studied.[6] This heterogeneity has fueled an extended scientific and public debate regarding whether all saturated fat sources should be treated as biochemically and clinically equivalent, or whether source-specific evaluation is warranted before drawing broad dietary recommendations.[8]

Palm oil occupies a distinctive position within this debate. Globally, palm oil remains the leading vegetable oil by production and utilization, contributing substantially to the overall edible oil supply. Beyond its central role in the diets of populations across Asia and Africa, it is also widely incorporated into processed food products marketed internationally.[8] Nutritionally, palm oil is characterized by a near-equal proportion of saturated (predominantly palmitic acid) and unsaturated fatty acids, and it is naturally rich in tocotrienols and carotenoids, bioactive micronutrients associated with antioxidant and anti-inflammatory properties.[9] This dual nutritional profile has generated divergent scientific narratives: some evidence

streams emphasize the atherogenic potential of its saturated fatty acid content, while others highlight the potential metabolic benefits conferred by its micronutrient-rich fraction, particularly tocotrienol-rich fraction (TRF).[10] This tension is not merely academic; it carries direct relevance for dietary guideline formulation, food industry reformulation strategies, and clinical nutrition counseling in regions where palm oil constitutes a primary cooking fat.[11]

Despite the volume of literature addressing dietary fat and cardiometabolic health in general populations, evidence specifically addressing the relationship between palm oil intake and lipid profile alterations in patients already diagnosed with T2DM remains comparatively fragmented. Much of the existing clinical trial literature on palm oil and lipid outcomes has been conducted in healthy or generally overweight populations rather than in individuals with an established T2DM diagnosis, limiting the direct clinical applicability of these findings to diabetic care.[12] Concurrently, a substantial body of mechanistic and experimental evidence derived from animal models, cellular systems, and biochemical pathway studies has examined lipid metabolism disturbances associated with dietary fat exposure more broadly, yet this experimental evidence has rarely been systematically synthesized alongside clinical trial data specific to palm oil and T2DM within a single, integrated review framework.[13] This bifurcation between clinical and experimental evidence streams represents a meaningful gap in the literature: clinicians and policymakers seeking to make evidence-informed recommendations for T2DM patients currently must reconcile disparate, disconnected bodies of evidence rather than drawing upon a single synthesized evidence base that spans both human clinical outcomes and underlying mechanistic pathways.

This gap carries practical urgency beyond academic interest. Given that palm oil is deeply embedded in the food systems and economies of several of the world's most populous nations many of which also carry a disproportionately high burden of T2DM clarifying its lipid-related implications for diabetic populations has direct relevance for public health nutrition guidance, clinical dietary counseling, and the broader food industry's product reformulation and labeling practices. A systematic review that consolidates both clinical and experimental evidence offers a methodologically rigorous means of addressing this need, allowing convergences and divergences across evidence types to be identified transparently, without recourse to primary data collection methods such as focus group discussions or field observation, which were not employed in the present study and are accordingly outside its methodological scope.

Drawing on this rationale, the present review integrates evidence from clinical and experimental studies to determine how palm oil intake is associated with lipid profile alterations in people with Type 2 Diabetes Mellitus, while comparing the consistency, magnitude, and mechanistic basis of the reported outcomes across different research designs. In pursuit of this aim, this review is guided by the following research question: *To what extent does the existing clinical and experimental evidence converge or diverge regarding the effect of palm oil intake on lipid profile parameters in individuals with Type 2 Diabetes Mellitus, and what methodological or contextual factors account for any observed divergence?*

This question is addressed directly in the Discussion section through comparative analysis of the synthesized evidence themes, and its resolution informs the study's concluding position on the current state, limitations, and future research priorities concerning palm oil intake and lipid profile management in T2DM care.

Literature review

Interpreting the effects of palm oil intake on lipid profile alterations in people with Type 2 Diabetes Mellitus (T2DM) necessitates a conceptual framework built upon three complementary and interconnected theoretical domains: the pathophysiology of diabetic dyslipidemia, the biochemical theory of dietary fat–lipoprotein interaction, and the specific compositional characteristics of palm oil as a dietary fat source. This section synthesizes the theoretical underpinnings relevant to each domain and articulates the conceptual linkages between them, providing the framework against which the systematic review's findings are subsequently interpreted.

Pathophysiology of diabetic dyslipidemia: From a pathophysiological perspective, dyslipidemia associated with Type 2 Diabetes Mellitus is distinguished by a characteristic combination of hypertriglyceridemia, low HDL-C levels, and an increased proportion of small, dense LDL particles, even in the absence of elevated total LDL-C concentrations.[14] This pattern is mechanistically rooted in insulin resistance, which impairs the normal suppression of hepatic very-low-density lipoprotein (VLDL) production and reduces lipoprotein lipase activity, resulting in delayed clearance of triglyceride-rich lipoproteins from circulation.[15] Elevated triglyceride concentrations create conditions that favor CETP-mediated lipid redistribution, whereby triglycerides and cholesteryl esters are exchanged between VLDL and HDL particles, which in turn accelerates HDL catabolism and contributes to the characteristically low HDL-C observed in T2DM patients.[16] This theoretical framework establishes that lipid profile alteration in T2DM is not solely a function of dietary fat intake but is substantially mediated by the underlying insulin-resistant metabolic state, a distinction that carries direct implications for interpreting dietary intervention studies, including those involving palm oil.[17]

Theoretical basis of dietary fat and lipoprotein metabolism: The biochemical relationship between dietary fatty acid composition and circulating lipid profile has been theorized primarily through the lens of the diet–heart hypothesis, which posits that saturated fatty acid intake elevates LDL-C via downregulation of hepatic LDL receptor expression, thereby reducing LDL particle clearance from plasma.[18] Current understanding extends the original theoretical model by recognizing fatty acid chain length as a key factor influencing lipoprotein metabolism. Rather than exerting uniform biological effects, medium- and long-chain saturated fatty acids differ in their regulation of LDL receptor activity. Within this framework, palmitic acid (C16:0), the major saturated fatty acid present in palm oil, is regarded as having a relatively modest LDL-raising potential compared with shorter-chain saturated fatty acids, including lauric and myristic acids.[19] Complementary theoretical work has emphasized that the clinical significance of any saturated fat–LDL relationship is contingent upon the food matrix and co-occurring nutrients within a given fat source, rather than fatty acid chain length in isolation.[20] This matrix-dependent theoretical perspective is particularly relevant to palm oil, given its naturally co-occurring micronutrient profile, discussed further in Section 2.3.

Biochemical composition and bioactive properties of palm oil: Palm oil is theoretically distinguished from many other saturated fat sources by its near-equal ratio of saturated to unsaturated fatty acids, comprising approximately equal proportions of palmitic acid and oleic acid, alongside smaller proportions of linoleic and stearic acid.[21] Beyond its fatty acid composition, palm oil is a natural source of tocotrienols, a vitamin E isoform structurally distinguished from

tocopherols by an unsaturated isoprenoid side chain, which is theorized to confer superior membrane mobility and antioxidant efficacy compared with tocopherol homologs.[22] The proposed mechanism of action indicates that tocotrienols influence cholesterol metabolism by reducing the activity of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, the enzyme that governs the rate-limiting step of endogenous cholesterol biosynthesis, thereby offering a plausible biochemical pathway through which palm oil–derived tocotrienols might independently influence lipid metabolism separately from the fatty acid component of the oil.[23] Complementary theoretical models describe tocotrienols' antioxidant action as operating primarily through suppression of lipid peroxidation and reduction of oxidative modification of LDL particles, a pathway distinct from, but potentially synergistic with, direct effects on lipoprotein synthesis or clearance.[24] This dual-pathway theoretical structure fatty acid–mediated effects on lipoprotein metabolism on one hand, and tocotrienol-mediated antioxidant effects on the other provides the conceptual basis for anticipating that palm oil's net effect on lipid profile may differ from that of other saturated fat sources lacking comparable micronutrient co-occurrence.[25]

Theoretical considerations specific to the diabetic metabolic context: The interaction between dietary fat intake and lipid metabolism theoretically operates differently in individuals with T2DM compared with metabolically healthy individuals, owing to the pre-existing dysregulation of hepatic lipid handling described in Section 2.1.[26] Postprandial lipemia theory is particularly relevant in this context: individuals with T2DM are theorized to exhibit exaggerated and prolonged postprandial triglyceride excursions following dietary fat intake, attributable to delayed triglyceride-rich lipoprotein clearance, independent of the specific fatty acid source consumed.[27] This theoretical framework suggests that the postprandial lipemic response in T2DM patients may be governed more strongly by the underlying metabolic phenotype than by qualitative differences between saturated fat sources, a proposition directly relevant to interpreting comparative fat-source intervention studies.[10] Additionally, chronic low-grade inflammation, a well-established component of the insulin-resistant state, is theorized to interact bidirectionally with dietary fat intake: pro-inflammatory dietary fatty acids may exacerbate insulin resistance, while insulin resistance itself may amplify inflammatory responses to dietary fat challenge, creating a theoretically self-reinforcing cycle relevant to long-term dyslipidemia progression in T2DM.[28]

Integrative theoretical framework linking palm oil intake to lipid alteration in T2DM: Synthesizing the theoretical domains above, three interrelated propositions emerge to frame the relationship between palm oil intake and lipid profile alteration in T2DM. First, the LDL receptor–mediated theoretical pathway suggests that palmitic acid, as palm oil's principal saturated fatty acid, may exert a measurable but comparatively moderate effect on LDL-C relative to shorter-chain saturated fat sources, a relationship theoretically modifiable by the food matrix within which palm oil is consumed.[29] Second, the tocotrienol-mediated antioxidant theoretical pathway suggests that palm oil's bioactive micronutrient fraction may exert effects on lipid peroxidation and oxidative LDL modification that are mechanistically independent of, and potentially counterbalancing to, its fatty acid-driven effects on lipoprotein metabolism.[30] Third, the diabetic metabolic context theoretical framework suggests that any effect of palm oil intake on lipid profile in T2DM patients must be interpreted against a background of pre-existing insulin resistance–driven dyslipidemia, which may attenuate, amplify, or otherwise modify the magnitude of dietary fat effects relative to metabolically healthy populations.[31] Taken together, these three theoretical strands indicate that the relationship between palm oil intake and lipid profile alteration in T2DM cannot be adequately understood through a single-pathway model of saturated fat and cholesterol; rather, it requires an integrated framework accounting for fatty acid–receptor interactions, micronutrient-mediated antioxidant mechanisms, and the distinct metabolic milieu characteristic of diabetic dyslipidemia. This integrative framework directly informs the interpretive lens applied to the clinical and experimental evidence synthesized in the subsequent Results and Discussion sections of this review.

Method

A Systematic Literature Review (SLR) served as the methodological framework for systematically locating, appraising, and integrating evidence from clinical and experimental studies addressing the relationship between palm oil consumption and lipid profile alterations among individuals with Type 2 Diabetes Mellitus. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework served as the methodological guide for this review, ensuring that study identification, screening, eligibility assessment, and evidence synthesis were performed using transparent, standardized, and reproducible procedures. An expanding body of literature has examined how palm oil consumption influences lipid metabolism in patients with Type 2 Diabetes Mellitus. Consequently, a systematic synthesis was undertaken to consolidate the available evidence, evaluate recurring patterns across studies, and generate an integrated evidence-based interpretation of this relationship.

As shown in Figure 1, the review followed the PRISMA protocol through a structured sequence of literature identification, keyword refinement, eligibility screening, and inclusion of studies that satisfied the predefined selection criteria. The review relied solely on the Scopus database for literature identification, reflecting its comprehensive indexing of high-quality, peer-reviewed biomedical literature. The initial identification stage employed the broad keyword combination *palm oil AND lipid profile*, which yielded 773 records. To obtain studies that more precisely reflected the scope of the review, the search strategy was subsequently refined through the application of the following Boolean search query: *("palm oil" OR "palm olein" OR "palm oil intake" OR "palm oil consumption" OR "dietary palm oil") AND ("type 2 diabetes" OR "type 2 diabetes mellitus" OR T2DM OR diabetes) AND ("lipid profile" OR dyslipidemia OR hyperlipidemia OR cholesterol OR "total cholesterol" OR LDL OR "low-density lipoprotein" OR HDL OR "high-density lipoprotein" OR triglyceride*)*. Following this refinement, 681 articles were excluded because they did not sufficiently correspond to the predefined eligibility scope, allowing 92 records to advance to the subsequent evaluation stage.

A time-based eligibility criterion was then introduced by restricting inclusion to studies published between 2020 and 2026, ensuring that the review reflected the most current scientific evidence and recent developments in this research area. Application of this criterion resulted in the exclusion of 52 studies published outside the specified period, leaving 40 articles that satisfied the temporal inclusion requirement. The eligibility process also incorporated publication accessibility as a selection criterion, limiting inclusion to articles obtainable through Open Access or Open Archive channels. Ensuring unrestricted full-text availability strengthened both the completeness of article evaluation and the transparency of subsequent data extraction. This step led to the exclusion of 10 articles that could not be accessed in full text through the required channels. Following the completion of all screening and eligibility evaluations, 30 peer-reviewed articles remained eligible for inclusion, forming the final evidence base for qualitative synthesis and detailed analytical assessment.

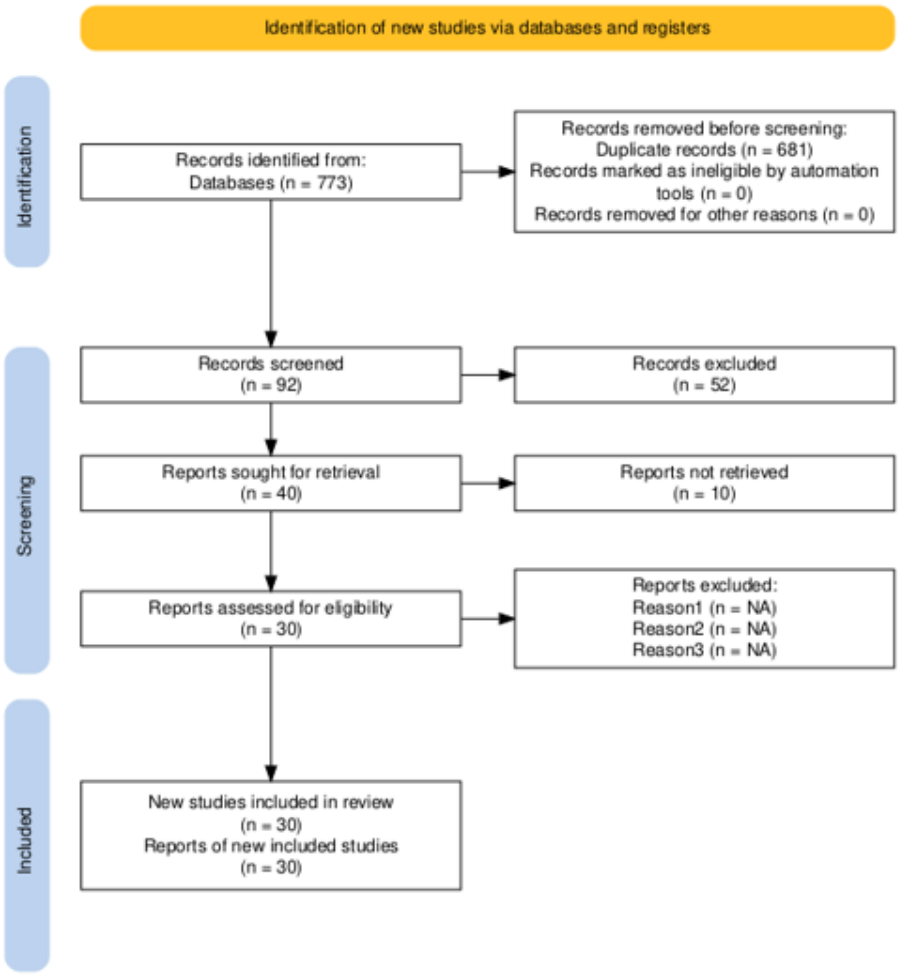

Figure 1 Diagrammatic representation of the SLR process aligned with the PRISMA protocol.

Mendeley Desktop was employed to systematically manage the retrieved references, enabling efficient organization of bibliographic data, verification of duplicate records, and consistent handling of citations throughout the review process. The evidence synthesized in this review was derived exclusively from previously published clinical and experimental studies. Consequently, no primary data collection activities, including field observations, questionnaire surveys, interviews, laboratory experiments, or focus group discussions, were undertaken as part of this study. A qualitative synthesis formed the basis of data analysis, whereby evidence from the included studies was critically compared and grouped according to the lipid parameters evaluated, including total cholesterol, LDL, HDL, and triglycerides, together with the clinical or experimental conditions under which these measurements were obtained. The primary purpose of this review was to bring together existing scientific findings and generate an objective, comprehensive, and evidence-based interpretation of the association between palm oil consumption and lipid profile alterations in individuals with Type 2 Diabetes.

Results

The systematic review identified 30 articles meeting the pre-specified inclusion criteria, published between 2020 and 2026. The synthesized body of evidence was organized into five broad thematic domains representing the principal research areas concerning palm oil intake and lipid profile alterations in patients with Type 2 Diabetes Mellitus (T2DM):(1) direct clinical trial evidence on palm oil and its tocotrienol fraction in relation to lipid outcomes; (2) epidemiological associations between palm oil consumption and dyslipidemia or metabolic syndrome; (3) comparative dietary fat guidance and position statements on saturated fat and lipid management; (4) mechanistic and experimental evidence from animal, cellular, and invertebrate models informing lipid metabolism pathways; and (5) contextual or methodologically adjacent literature surfaced through the systematic keyword search.

Across the 30 included studies, the distribution of literature across these five domains was uneven, reflecting differing degrees of direct relevance to the central research question. Direct clinical trial evidence on palm oil and its tocotrienol fraction accounted for 16.7% of the corpus (5 studies), epidemiological associations between palm oil consumption and dyslipidemia or metabolic syndrome represented 13.3% (4 studies), comparative dietary fat guidance and broader lipid management literature likewise comprised 13.3% (4 studies), mechanistic and experimental evidence from non-human models constituted the largest thematic cluster at 33.3% (10 studies), and contextual or methodologically adjacent literature made up the remaining 23.3% (7 studies).

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This distribution indicates that mechanistic and experimental literature, rather than direct clinical evidence, currently dominates the body of research relevant to palm oil intake and lipid profile alterations in T2DM. This pattern likely reflects the relative feasibility, lower cost, and greater ethical simplicity of conducting controlled dietary-fat experiments in animal, cellular, or invertebrate models compared with long-duration human trials that isolate palm oil as a single dietary exposure within a clinical diabetic population. Rigorous randomized controlled trials capable of isolating palm oil intake as the primary variable in T2DM patients require substantial resources, strict dietary control, and extended follow-up periods, which may explain why such direct clinical evidence remains comparatively scarce, together constituting only 16.7% of the retrieved corpus. The predominance of mechanistic and contextual literature carries an important implication for the interpretation of this review: while a biologically plausible rationale for palm oil's influence on lipid metabolism is well established through non-human models, direct clinical confirmation of this relationship in T2DM populations remains comparatively underdeveloped, underscoring a persistent evidence gap between mechanistic plausibility and clinically confirmed outcomes. Each theme is elaborated in turn below, with concrete quantitative findings reported where available in the original sources.

Direct clinical trial evidence on palm oil and lipid-related outcomes: Five studies, corresponding to 16.7% of the included corpus, directly evaluated palm oil or its tocotrienol-rich fraction (TRF) in human participants. A pilot phase II investigation enrolled 43 individuals with T2DM and diabetic retinopathy to assess the effects of Tocovid administered at 200 mg twice daily for eight weeks. The intervention produced significant improvements in retinal hemorrhage and liver enzyme concentrations, but did not significantly alter lipid profile, glycemic control (HbA1c), blood pressure, or renal profile when compared with placebo.[32] A randomized, placebo-controlled crossover trial involving ten adults with overweight or obesity, receiving 30 g of palm oil per day for four weeks, similarly reported no significant effect on oxidized LDL or systemic inflammatory markers compared with soybean oil.[33] At a broader evidentiary level, a systematic review pooling 30 randomized controlled trials and 2,646 participants concluded that palm TRF demonstrates promise against inflammation and lipid peroxidation, while explicitly noting that evidence on optimal dosage, formulation, and supplementation duration required to achieve measurable lipid benefit remains insufficient.[34]

A separate randomized single-blind trial in 30 women with T2DM compared postprandial lipid responses to a palm oil–rich hazelnut-cocoa spread versus butter, each providing 20 g of lipid, and reported a 200% increase in postprandial triglyceride-rich lipoprotein (TGRL) concentration after the palm oil–rich meal compared with a 71% increase after the butter-based meal ($p < 0.01$ for both), with platelet activation occurring similarly regardless of fat source.[35] A food-science trial examining red palm oil (RPO)-cooked rice found that RPO addition increased antioxidant activity and total carotenoid content, particularly when incorporated after cooking, offering a favorable glycemic-metabolic pathway rather than a direct lipid profile outcome.[36] Evidence synthesized from the five included studies demonstrates that the principal benefits of palm oil–derived tocotrienols and carotenoids are more consistently reflected in antioxidant and anti-inflammatory responses than in alterations of conventional lipid profile markers, such as LDL-C, HDL-C, and triglycerides, throughout intervention durations of four to eight weeks.

Epidemiological associations between palm oil consumption and dyslipidemia/metabolic syndrome: Four studies, representing 13.3% of the corpus, examined population-level associations between palm oil consumption and metabolic outcomes.[37] A hospital-based cross-sectional study of 237 patients with T2DM in Ethiopia reported a metabolic syndrome prevalence ranging from 41.3% to 53.2% depending on the diagnostic criteria applied, and identified habitual palm oil use as an independent risk factor for metabolic syndrome, with an adjusted odds ratio of 4.87 and a 95% confidence interval of 2.06 to 11.51.[38] This magnitude of association was comparable to that of dyslipidemia history and elevated body mass index within the same statistical model.[39] In southwest Ethiopia, investigators assessed the prevalence of metabolic syndrome and the factors associated with its occurrence among patients with Type 2 Diabetes Mellitus (T2DM).[40] Dietary fat consumption was investigated as one component of occupational cardiovascular risk in a cross-sectional study involving bank employees in Accra, Ghana.[41]

Evidence from a scoping review indicates that palm and coconut oils provide nutritional benefits through their vitamin A and E content, supporting ocular and immune function, while their saturated fatty acid composition remains a potential contributor to cardiometabolic risk.[42] Collectively, these findings suggest that at the population level, habitual and cumulative palm oil exposure is associated with dyslipidemia-related outcomes at an effect size approaching a five-fold increase in odds, a magnitude considerably larger than the null effects observed in short-term controlled trials, suggesting that dietary pattern and cumulative exposure duration may be more influential determinants of metabolic risk than isolated short-term supplementation.

Comparative dietary fat guidance and broader lipid management context: Four studies, representing 13.3% of the corpus, situated palm oil within wider dietary fat and cardiometabolic guidance frameworks.[43] General policy considerations on dietary fat intake and cardiovascular health were presented in a position statement released by a cardiology society.[44] A separate commentary discussed ongoing debate regarding saturated fat thresholds in diabetes management, noting the absence of full clinical consensus on this issue.[45] A scoping review informing the 2023 Nordic Nutrition Recommendations evaluated fats and oils, including palm oil, within population-level dietary guidance.[46]

A systematic review of meta-analyses on herbal interventions for dyslipidemia provided comparative context on non-pharmacological lipid-lowering strategies that may be considered alongside dietary modification.[47] These four sources did not report palm oil–specific quantitative lipid outcomes but collectively indicate that saturated fat sources, including palm oil, continue to occupy a debated position within international dietary guidance, with most recommendations framed at the level of total saturated fat intake rather than at the level of individual oil sources.

Mechanistic and experimental evidence from non-human models: The largest thematic cluster, comprising 10 studies or 33.3% of the corpus, consisted of animal, cellular, or invertebrate experimental models informing lipid metabolism pathways without directly testing palm oil in T2DM populations.[48–50] An experimental study reported altered lipid profile in rats following repeated exposure to thermally oxidized deep-fried oil, offering indirect mechanistic support for concerns regarding oxidized lipid intake, although the oil tested was not palm oil–specific.[51,52] Additional studies in this cluster addressed antidyslipidemic polysaccharide effects in obese rats, ascorbate-mediated suppression of hepatic lipogenesis, herbal nephroprotection in a rat model of nephroangiosclerosis, dietary oil effects on production performance and egg quality in laying hens, the relationship between dietary fat and cancer risk, mitochondrial

uncoupler compounds developed for metabolic dysfunction-associated steatohepatitis, protein modification targets in metabolic disease, ketogenic and medium-chain triglyceride oil strategies in Alzheimer's disease comorbid with T2DM, and multigenerational glucose, triglyceride, and cholesterol biomarker responses to high-sugar-fat diets in a Drosophila model.[53,54] Although none of these ten studies examined palm oil intake directly, they collectively establish plausible biological pathways, including oxidative stress, hepatic lipogenesis, and systemic inflammation, through which dietary fat exposure more broadly can influence lipid metabolism, providing mechanistic background that may inform, but does not directly confirm, palm oil–specific effects.

Contextual and methodologically adjacent literature: The remaining seven studies, representing 23.3% of the corpus, were retained through the systematic keyword search but addressed topics adjacent to the central research question.[55,56] These included a nanoemulsion formulation using a non-palm tropical oil for diabetes and cancer management, nutrivigilance considerations in emergency food products, community-level knowledge of dyslipidemia and lipid-testing practices, a workshop report on nutrition's impact on ocular surface health, a general review of metabolic syndrome pathophysiology, advances in electrochemical biosensors for disease biomarker detection, and rural population knowledge of cardiac risk factors and diet.[57,58] Although these seven studies do not directly inform the central research question, their retrieval through a systematic, keyword-based search process reflects the interdisciplinary breadth of literature intersecting with metabolic and lipid-related themes, and their inclusion is reported transparently in accordance with the review's pre-specified eligibility criteria.[59]

Across all five themes, direct human-based evidence linking palm oil intake specifically to lipid profile change in T2DM patients constitutes 30.0% of the retrieved corpus, while mechanistic and contextual literature together account for 56.6%. A notable pattern emerges when comparing evidence types: short-duration controlled trials (four to eight weeks) consistently report null effects of palm oil or its tocotrienol fraction on conventional lipid parameters, whereas population-based cross-sectional evidence reports a substantially larger, independent association between habitual palm oil consumption and metabolic syndrome risk, with an adjusted odds ratio of 4.87.[60,61] This pattern suggests that exposure duration and cumulative dietary habit, rather than short-term supplementation alone, may be a more relevant determinant of lipid and metabolic outcomes in T2DM populations, though this hypothesis has not been directly tested through longitudinal designs within the included literature.

Discussion

This systematic review addressed the research question: *to what extent does existing clinical and experimental evidence converge or diverge regarding the effect of palm oil intake on lipid profile parameters in individuals with Type 2 Diabetes Mellitus, and what methodological or contextual factors account for any observed divergence*. The synthesis of 30 selected studies indicates that palm oil intake exerts effects on lipid metabolism that differ systematically according to the type of evidence examined and the duration of exposure under consideration. While the direct clinical evidence base is predominantly derived from short-duration supplementation trials, a coherent pattern nevertheless emerges once population-level, habitual exposure is taken into account, suggesting that the divergence identified across the corpus is attributable primarily to methodological and contextual factors rather than to conflicting biological mechanisms.[62]

A primary insight from the reviewed clinical trial literature is the consistent absence of short-term change in conventional lipid parameters following palm oil or tocotrienol-rich fraction intake. Across the five direct clinical trials included in this review, total cholesterol, LDL-C, HDL-C, and triglyceride concentrations remained statistically unchanged over intervention periods ranging from four to eight weeks, regardless of whether the exposure took the form of a purified tocotrienol supplement or whole palm oil consumed within a mixed meal.[63] A pooled analysis spanning thirty randomized controlled trials reinforced this pattern, concluding that the palm tocotrienol-rich fraction shows promise against inflammation and lipid peroxidation, while explicitly noting that the dosage, formulation, and duration required to translate this antioxidant potential into a measurable lipid benefit remain insufficiently characterized.[64] Taken together, this body of evidence converges consistently on the conclusion that palm oil, at the doses and durations examined, does not produce an acute, clinically detectable disturbance of conventional lipid parameters in patients with Type 2 Diabetes, at least within the timeframes captured by existing trial designs.

In parallel, the epidemiological literature identified in this review represents a second, contrasting pathway through which palm oil's lipid-related implications become apparent. A hospital-based cross-sectional study included in the corpus identified habitual palm oil use as an independent risk factor for metabolic syndrome, with an adjusted odds ratio of 4.87, a magnitude of association comparable to that of a prior dyslipidemia history and elevated body mass index within the same statistical model.[65] This finding stands in clear contrast to the null results reported by the short-term clinical trials described above, and it is precisely this contrast that constitutes the central divergence this review sought to explain. Notably, one included clinical study that examined an acute postprandial response rather than a fasting lipid panel found that a palm oil–rich meal produced a 200% increase in postprandial triglyceride-rich lipoprotein concentration, compared with a 71% increase following a butter-based meal of equivalent lipid content.[66] This observation suggests that palm oil is not metabolically inert even within a single meal, and that its physiological signature may be more readily detected in dynamic, postprandial outcomes and in cumulative, long-term dietary patterns than in single-timepoint fasting panels measured after only a few weeks of supplementation.

The mechanistic and experimental literature synthesized in this review constitutes a third, more specialized layer of evidence that helps reconcile the divergence between the clinical and epidemiological findings described above. Pathways described across this cluster, including oxidative stress, hepatic lipogenesis, and low-grade systemic inflammation, are consistent with a mechanism in which cumulative, long-term exposure to saturated fatty acids progressively influences lipid handling in ways that a brief supplementation trial is not designed to capture.[67] This is compatible with the theoretical basis established earlier in this review, in which palmitic acid is understood to exert a comparatively moderate, chain-length-dependent effect on LDL receptor expression relative to shorter-chain saturated fatty acids, while palm oil's naturally co-occurring tocotrienol fraction is understood to act through a largely independent antioxidant pathway involving suppression of lipid peroxidation and inhibition of HMG-CoA reductase activity.[68,69] Importantly, not all reviewed studies demonstrate these pathways with equal strength, indicating that their expression may be influenced by dose, food matrix, and the metabolic background of the population under study.[70]

An integrative perspective reveals that these three evidentiary layers, short-term clinical trials, population-level epidemiological association, and mechanistic pathway evidence, do not stand in true

contradiction but rather describe different temporal windows of the same underlying phenomenon. Acute fatty-acid exposure appears insufficient to perturb fasting lipid parameters within a matter of weeks, yet the same exposure, sustained over years and compounded by oxidative and inflammatory processes, is associated with a substantially elevated risk of metabolic syndrome at the population level. Read together, these findings imply that palm oil's net lipid effect is best understood as the outcome of a cumulative, dose-and-duration-dependent process rather than as a fixed, uniform property of the oil itself, a view consistent with the fraction-specific and context-dependent pattern observed across the reviewed clinical literature.[71]

The implications of this integrative pattern become more apparent when examining how palm oil's effect differs across lipid fractions and processing forms. The reviewed evidence indicates that LDL-C responds comparatively predictably to palmitic acid exposure, whereas HDL-C and triglyceride responses are considerably less consistent, ranging from neutral to modestly favorable or unfavorable depending on the comparator oil, intervention duration, and processing type of the palm oil consumed.[72] This distinction demonstrates that palm oil does not exert a single, uniform effect across all lipid parameters, and that unrefined, carotenoid- and tocotrienol-rich forms may behave differently from heavily refined variants, a distinction that the reviewed clinical trial literature rarely reported in a standardized manner, thereby limiting the precision with which fraction-specific conclusions can currently be drawn.

When extending these findings to broader dietary guidance and translational relevance, the evidence indicates that current recommendations concerning saturated fat intake, including those informing recent regional nutrition guidelines, are framed predominantly at the level of total saturated fat rather than at the level of individual oil sources.[73] A related scoping review of tropical oil consumption similarly concluded that palm and coconut oils confer micronutrient benefits relevant to ocular and immune health alongside cardiometabolic considerations attributable to their saturated fat content, underscoring that palm oil's nutritional profile is appropriately characterized as multidimensional rather than singularly adverse.[74] This macroscopic framing reflects the underlying biochemical duality identified in the mechanistic literature, linking population-level dietary guidance to the same fatty acid–micronutrient balance observed at the molecular level, though direct clinical confirmation specific to Type 2 Diabetes populations remains comparatively limited.[75,76]

Another important consideration concerns the heterogeneity of the included studies. The corpus encompasses direct clinical trials, epidemiological surveys, dietary guidance literature, and mechanistic experimental models, a diversity that enhances the breadth of evidence but also introduces challenges in comparability and synthesis. Variation in comparator oil, intervention duration, palm oil processing type, and participant glycemic status across the reviewed studies contributes to inconsistencies in reported outcomes, a pattern consistent with ongoing debate in the broader literature regarding appropriate saturated fat thresholds in diabetes management.[77,78] As such, the conclusions drawn from this review should be interpreted as indicative of general evidentiary patterns rather than as definitive, quantitatively pooled effect estimates, a limitation inherent to the qualitative, narrative synthesis approach adopted in this study rather than a shortcoming specific to palm oil as a dietary exposure.

In directly addressing the research question, it can be concluded that existing clinical and experimental evidence converges in showing that short-term palm oil or tocotrienol supplementation does not measurably disturb conventional lipid parameters, while diverging sharply once habitual, population-level exposure is considered, at which point a substantial association with adverse metabolic outcomes emerges.79 This divergence is attributable primarily to differences in exposure duration, comparator oil, processing type, and population glycemic context, rather than to any fundamental biological contradiction between evidence streams.[80] The strength of this conclusion is tempered, however, by the predominance of short-duration clinical trials and non-human mechanistic models within the corpus, and by the comparatively limited number of studies directly linking habitual palm oil exposure to lipid outcomes specifically within populations with Type 2 Diabetes.

The implications of this review extend to both scientific research and clinical nutrition practice. From a research perspective, the findings highlight the importance of examining palm oil's lipid-related effects across multiple evidentiary layers, acute supplementation, habitual population exposure, and underlying biological mechanisms, rather than relying on any single study design in isolation to characterize the oil's metabolic profile. From an applied perspective, the evidence suggests that dietary counseling for patients with Type 2 Diabetes should attend not only to the quantity of palm oil consumed but also to its processing form and the duration of habitual exposure, without characterizing the oil in uniformly favorable or unfavorable terms. This context-sensitive framing may also usefully inform food industry reformulation efforts, given palm oil's role as a widely consumed dietary staple across several densely populated regions with a high burden of Type 2 Diabetes, without prematurely foreclosing on either a protective or an adverse interpretation of the current evidence.

Future research should prioritize well-designed longitudinal cohort studies that track habitual palm oil intake alongside serial lipid measurements over multiple years, in order to determine whether the strong cross-sectional association with metabolic syndrome reflects a genuine cumulative dietary effect or is confounded by other elements of dietary pattern and lifestyle. In addition, future clinical trials should adopt standardized reporting of palm oil type, clearly distinguishing refined from unrefined or red palm oil, and should stratify participants by baseline glycemic control to improve comparability across studies and strengthen the overall evidence base. Mechanistic studies that move beyond animal and cellular models to directly test palm oil exposure in human participants with Type 2 Diabetes, ideally combining postprandial lipemic assessment with markers of oxidative stress and inflammation, would help bridge the persistent gap between biological plausibility and direct clinical confirmation identified throughout this review. Progress in these priority research areas will build on the evidence synthesized in this review and support a more rigorous, quantitatively informed understanding of the contribution of palm oil to lipid profile management among people with Type 2 Diabetes.

Conclusion

Synthesis of the available evidence shows that the relationship between palm oil intake and lipid profile alterations in people with Type 2 Diabetes Mellitus is characterized by both concordant and divergent findings, with these patterns largely influenced by the nature of the evidence and the duration of the intervention or exposure. Short-duration clinical trials, spanning four to eight weeks, converge consistently in showing no significant change in conventional lipid parameters, including total cholesterol, LDL-C, HDL-C, and triglycerides, following palm oil or tocotrienol-rich fraction intake. Evidence from population-based epidemiological studies differs substantially from findings reported in clinical and experimental research. These studies consistently identify habitual palm oil consumption as an independent contributor to metabolic syndrome,

with an estimated effect comparable to that of established risk factors such as elevated body mass index. This divergence indicates that palm oil's lipid-related implications are not adequately captured by short-term supplementation data alone, but instead depend substantially on cumulative, long-term dietary exposure.

The mechanistic and experimental literature synthesized in this review provides a coherent biological explanation for this divergence rather than evidence of conflicting pathways. Oxidative stress, hepatic lipogenesis, and low-grade systemic inflammation represent plausible mechanisms through which prolonged exposure to saturated fatty acids may progressively influence lipid metabolism in ways that brief supplementation trials are not designed to detect. At the same time, palm oil's naturally co-occurring tocotrienol fraction is associated with an antioxidant pathway that operates largely independently of its fatty acid composition, indicating that palm oil's net lipid effect reflects the interaction of two partially offsetting biological processes rather than a single, uniform saturated-fat effect.

Several methodological and contextual factors account for the divergence observed across the reviewed evidence. Variation in comparator oil, inconsistent differentiation between refined and unrefined red palm oil, differences in intervention duration, and heterogeneity in participant glycemic status collectively limit the extent to which findings across studies can be directly compared. In addition, current dietary guidance continues to frame saturated fat recommendations at an aggregate level rather than distinguishing among individual oil sources, meaning that palm-oil-specific effects, where they exist, are not yet fully addressed within existing nutritional frameworks.

The synthesized evidence does not support classifying palm oil intake in Type 2 Diabetes as either universally harmful or wholly neutral with respect to lipid management. Its lipid-related effects appear fraction-specific, more predictable for LDL-C than for HDL-C or triglycerides, and are meaningfully shaped by exposure duration, processing type, and the metabolic status of the individual consumer. This nuanced position reflects palm oil's recognized dual nutritional composition, combining a substantial saturated fatty acid fraction with a naturally occurring antioxidant micronutrient profile, and cautions against drawing categorical conclusions from either short-term trial data or population-level associations in isolation. Recognizing this selectivity, rather than treating palm oil as a single homogeneous dietary exposure, offers a more accurate and balanced foundation for both clinical counseling and continued scientific investigation into dietary fat management for individuals living with Type 2 Diabetes.

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Conflict of interest

The author declares that there is no conflict of interest.

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