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Effects of palm oil and palm-derived bioactives on insulin resistance and glucose homeostasis: a review

Abstract

Insulin resistance and impaired glucose homeostasis are central mechanisms in type 2 diabetes mellitus, obesity, metabolic syndrome, and broader cardiometabolic disease. Palm oil is one of the most widely consumed dietary fats worldwide, while palm-derived bioactives, including tocotrienols, carotenoids, and oil palm phenolics, have been investigated for potential metabolic, antioxidant, and anti-inflammatory effects. This PRISMA-oriented systematic literature review synthesises published evidence on the relationships of palm oil, palm olein, interesterified palm-derived fats, red palm oil, palm tocotrienol-rich fraction, and oil palm phenolics with insulin resistance and glucose regulation. The review included human intervention studies, systematic reviews, meta-analyses, and relevant mechanistic animal studies reporting fasting plasma glucose, fasting insulin, HOMA-IR, HbA1c, oral glucose tolerance, postprandial glucose, insulin secretion, incretin hormones, oxidative stress, or inflammatory biomarkers. Because the included studies differed substantially in intervention type, population, duration, comparator, and outcome assessment, findings were synthesised narratively rather than through a new quantitative meta-analysis. Short-term human trials of refined palm oil or palm olein generally reported neutral effects on fasting glucose, fasting insulin, and HOMA-IR compared with other dietary fats; however, most trials enrolled healthy or non-diabetic adults, limiting applicability to individuals with type 2 diabetes or established insulin resistance. Evidence on interesterified palm-derived fats remains limited and inconsistent in humans, although mechanistic animal studies suggest potential adverse effects on glucose tolerance, hepatic insulin signalling, inflammation, and adiposity. Among palm-derived bioactives, the tocotrienol-rich fraction has the strongest clinical evidence, with meta-analytic findings suggesting a modest HbA1c reduction in type 2 diabetes, whereas effects on oxidative stress and inflammation remain inconsistent. Red palm oil and oil palm phenolics are biologically plausible but currently lack sufficient direct human evidence for glycemic efficacy. Overall, current evidence does not support palm oil itself as a strategy to improve glucose homeostasis, although palm-derived tocotrienols may have adjunctive potential pending larger, longer, and better-controlled trials in insulin-resistant, prediabetic, and diabetic populations.

Keywords: palm oil, palm olein, tocotrienols, red palm oil, oil palm phenolics, insulin resistance, glucose homeostasis, type 2 diabetes, HOMA-IR, HbA1c

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Introduction

Type 2 diabetes mellitus, obesity, metabolic syndrome, and dyslipidemia are significant public health concerns, and insulin resistance is one of their central pathophysiological mechanisms. The International Diabetes Federation's 2025 Atlas reports that approximately one in nine persons aged 20–79 years is now living with diabetes, with an anticipated increase to 853 million persons by 2050; the World Health Organisation also emphasises that a nutritious diet, regular physical activity, maintenance of a healthy body weight, and avoidance of tobacco can prevent or postpone type 2 diabetes.[1]

Dietary fat quality has long been investigated as a modifiable determinant of glucose metabolism, insulin sensitivity, inflammation, and cardiometabolic risk. A large systematic review and meta-analysis of randomised feeding experiments found that substituting carbohydrate or saturated fat with unsaturated fats, particularly polyunsaturated fatty acids, produced more favourable effects on glycemic control and insulin resistance markers than saturated fat-rich dietary patterns.[2]

Palm oil is highly relevant to metabolic disorder research because it is widely consumed globally and remains one of the world's major vegetable oils. It contains a mixture of saturated and unsaturated fatty acids, with palmitic acid as a major saturated fatty acid, and palm-derived products include palm olein, red palm oil, palm vitamin E fractions rich in tocotrienols, and oil palm phenolics.[3]

The health implications of palm oil are controversial. Refined palm oil and palm olein are usually discussed in relation to saturated fat intake and lipid profile, whereas palm-derived minor components such as tocotrienols, carotenoids, and phenolics are investigated for antioxidant, anti-inflammatory, and metabolic effects. This distinction is important because the biological effects of the whole oil may differ from isolated or concentrated palm-derived bioactives.[4–6]

A prior systematic review focused specifically on consumption of palm oil and indicators of glucose metabolism found only eight eligible intervention studies through June 2018, mostly short-term trials in healthy, non-diabetic, normal-weight adults, and concluded that evidence was limited and generally did not show substantial variations in fasting glucose, fasting insulin, or HOMA-IR when palm oil or palm olein was compared with several other oils.[7] Since then, additional interest has emerged regarding palm-derived tocotrienols, interesterified palm fats, red palm oil, and oil palm phenolics.

To improve conceptual clarity, this review distinguishes whole palm-oil products from concentrated palm-derived bioactives. The synthesis is organised from the most widely consumed dietary oils to more specialised bioactive fractions: refined palm oil/palm

olein, interesterified palm-derived fats, tocotrienol-rich fraction/palm vitamin E, red palm oil/carotenoid-rich fractions, and oil palm phenolics. This organisation allows the strength and clinical relevance of each evidence stream to be interpreted separately rather than treating all palm-derived materials as a single exposure.

Objective

This article aims to synthesise current evidence on the effects of palm oil and palm-derived bioactives on insulin resistance and glucose homeostasis, with emphasis on outcomes relevant to diabetes and metabolic disorder control.

The specific objectives are to analyse:

I. The influence of refined palm oil and palm olein on fasting glucose, fasting insulin, HOMA-IR, postprandial glucose, and HbA1c.

II. The effects of structured or interesterified palm-derived fats on insulin resistance and glucose regulation.

III. The influence of palm-derived tocotrienol-rich fraction and palm vitamin E on glycemic control, oxidative stress, and inflammation in metabolic disorders.

IV. The evidence for red palm oil, carotenoid-rich palm fractions, and oil palm phenolics in glucose homeostasis.

V. The clinical relevance, certainty, limitations, and future research priorities for diabetes and metabolic disorder control.

These objectives are addressed in the same order in the Results section to create a clearer progression from conventional palm oil exposure to palm-derived bioactive compounds and, finally, clinical interpretation.

Methods

Review design

This manuscript was prepared as a PRISMA-oriented systematic literature review and narrative synthesis of published evidence rather than as an original experimental study. PRISMA 2020 provides a reporting checklist and flow-diagram structure for transparent reporting of systematic reviews.[8] The article-selection procedure is shown in Figure 1.

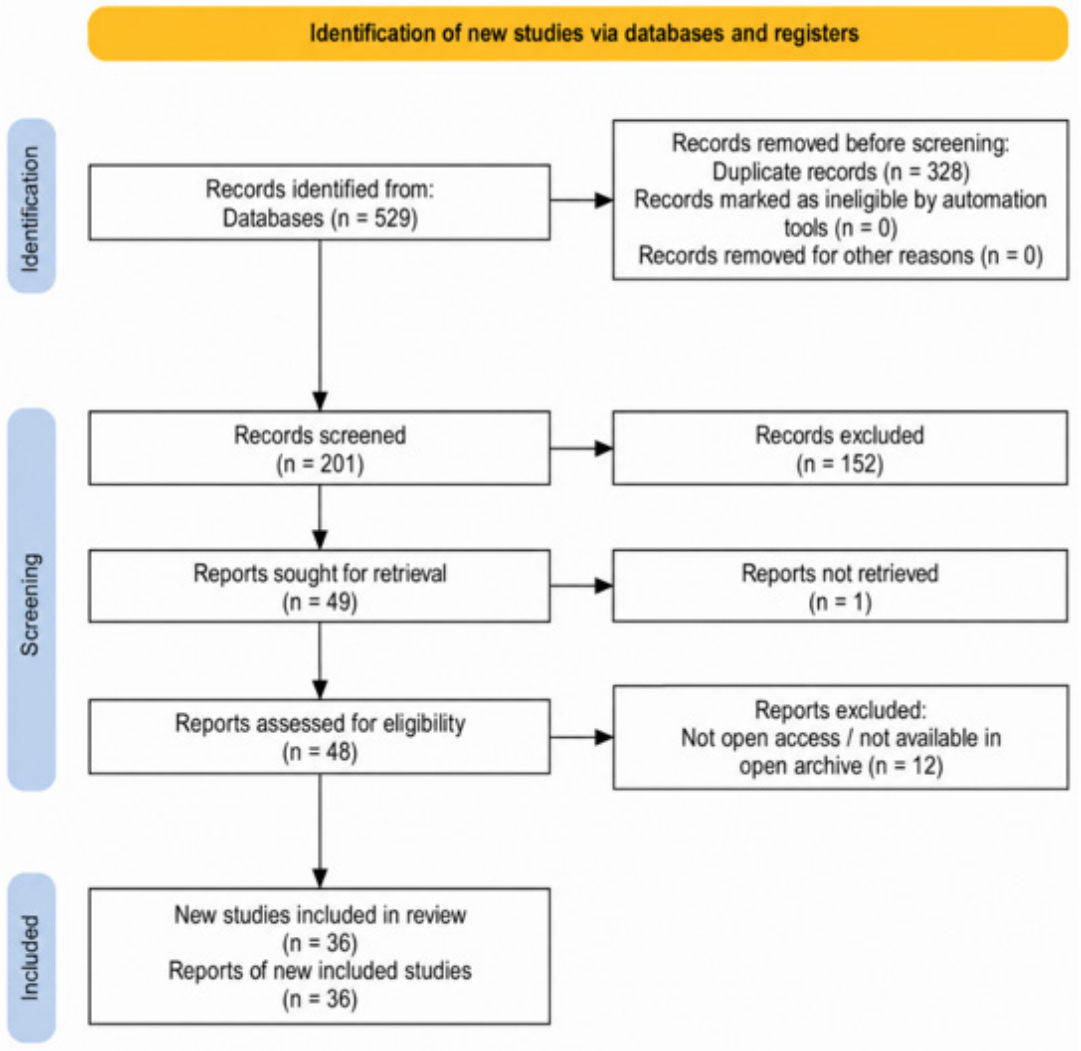

Figure 1 PRISMA flowchart diagram for the selection of articles.

Because intervention types, comparator oils, populations, doses, durations, and outcomes were heterogeneous, a new quantitative meta-analysis was not performed. Instead, evidence was grouped by intervention class and synthesised narratively: refined palm oil/palm olein, interesterified palm-derived fats, palm-derived tocotrienols, red palm oil, oil palm phenolics, and mechanistic preclinical evidence.

Eligibility criteria

The review question was structured using a PICO framework.

Population: Adults with normal glucose tolerance, overweight/obesity, insulin resistance, prediabetes, type 2 diabetes mellitus, dyslipidemia, metabolic syndrome, or elevated cardiometabolic risk. Animal and cell studies were considered only for a mechanistic context and were not treated as direct clinical evidence.

Interventions: Palm oil, palm olein, red palm oil, interesterified palm oil or palm-derived fats, palm vitamin E, tocotrienol-rich fraction, palm-derived tocotrienols, palm carotenoids, and oil palm phenolics.

Comparators: Other dietary oils or fats, habitual diet, placebo, control supplement, or baseline values in crossover studies.

Outcomes: Fasting plasma glucose, fasting insulin, HOMA-IR, HbA1c, oral glucose tolerance, postprandial glucose, insulin secretion, C-peptide, GLP-1, GIP, inflammatory biomarkers, oxidative-stress biomarkers, adiposity, liver-related metabolic outcomes, and lipid profile when relevant to metabolic risk.

Study types: Randomised controlled trials, controlled feeding studies, crossover trials, systematic reviews, meta-analyses, trial registry records, and mechanistic animal studies.

Exclusion criteria

Studies were excluded if they did not evaluate a palm-derived intervention, did not report glucose-metabolism or insulin-resistance outcomes, used only industrial or environmental outcomes, lacked a relevant comparator or interpretable metabolic endpoint, or were not available in English-language scientific records. Narrative reviews were used only for background, where appropriate.

Search strategy

Searches were executed in PubMed/MEDLINE, Scopus, Web of Science, Cochrane CENTRAL, Google Scholar, and ClinicalTrials.gov from database inception to the final search date.

A reproducible search string can be adapted as follows:

("Palm oil" OR "palm olein" OR "red palm oil" OR "interesterified palm oil" OR "palm vitamin E" OR "tocotrienol-rich fraction" OR "palm tocotrienol" OR "oil palm phenolics" OR "palm carotenoids") AND

("insulin resistance" OR "glucose homeostasis" OR "fasting glucose" OR "fasting insulin" OR "HOMA-IR" OR "HbA1c" OR "type 2 diabetes" OR "prediabetes" OR "metabolic syndrome" OR "postprandial glucose" OR "oral glucose tolerance")

Data extraction and narrative synthesis

For each eligible record, information was extracted on intervention category, study design, population characteristics, comparator, intervention duration, metabolic outcomes, and main findings. Human clinical evidence was prioritised for conclusions about glucose homeostasis, while animal and cell studies were used only to explain possible mechanisms or identify hypotheses for future clinical research. The narrative synthesis was structured to separate evidence for whole oils from evidence for concentrated palm-derived bioactives, thereby improving the organisational flow of the review and avoiding overgeneralisation across biologically distinct interventions.

Figure 1 presents the PRISMA-based study selection process used in this systematic review. A total of 529 records were initially identified through database searching. Before screening, 328 duplicate records were removed. No records were excluded by automation tools, and no additional records were removed for other reasons. After duplicate removal, 201 unique records remained and were taken forward for title and abstract screening. During the screening stage, 152 records were excluded because they did not meet the predefined inclusion criteria. The remaining 49 reports were sought for retrieval. Of these, 1 report could not be retrieved, leaving 48 full-text reports available for eligibility assessment. At the eligibility stage, 48 full-text reports were assessed against the inclusion and exclusion criteria. Twelve reports were excluded because they were not open access or were not available in an open archive. After full-text assessment, 36 studies fulfilled the eligibility criteria and were included in the final review. These studies formed the basis for the qualitative synthesis on the effects of palm oil and palm-derived bioactives on insulin resistance and glucose homeostasis.

Results

Overview of the evidence base

The evidence base was divided into five major categories, summarised in Table 1 and discussed in the same sequence throughout the Results section:

Table 1 Intervention category and main evidence type

Intervention category	Main evidence type	Overall interpretation for glucose homeostasis
Refined palm oil/palm olein	Short-term human feeding trials and prior systematic review	Generally neutral effects on fasting glucose, fasting insulin, and HOMA-IR, but evidence is mostly from healthy adults and short interventions
Interesterified palm-derived fats	Human postprandial and short-term trials; animal studies	Human evidence is limited and not consistently adverse; animal studies suggest possible worsening of insulin resistance and glucose intolerance
Palm tocotrienol-rich fraction/palm vitamin E	RCTs and meta-analyses in type 2 diabetes	Most promising palm-derived bioactive evidence, with modest HbA1c improvement but inconsistent inflammatory/oxidative outcomes
Red palm oil	Small clinical studies and mechanistic literature	Biologically plausible because of carotenoids and vitamin E compounds, but direct glycemic evidence is insufficient
Oil palm phenolics	Preclinical studies, safety studies, and limited trial records	Promising mechanistic evidence, but insufficient completed human efficacy evidence

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This sequence begins with conventional dietary palm-oil exposures, then moves to modified palm-derived fats and concentrated bioactive fractions. This structure clarifies which conclusions apply to whole palm oil and which apply only to specific palm-derived compounds.

Refined palm oil and palm olein

The most directly relevant systematic review on palm oil and glucose metabolism identified eight intervention studies, including seven randomised crossover studies and one randomised parallel study. The included participants were mainly young or middle-aged, healthy, non-diabetic, and normal-weight adults, and intervention durations were typically three to seven weeks. The review found no significant effect of palm oil or palm olein on fasting glucose compared with partially hydrogenated soybean oil, soybean oil, or olive oil, and no significant effect on fasting insulin compared with partially hydrogenated soybean oil or olive oil. HOMA-IR comparisons with several oils were also generally non-significant.7

This evidence suggests that short-term replacement of some dietary fats with palm oil or palm olein does not consistently worsen fasting glycemic markers in healthy adults. However, the clinical relevance to people with type 2 diabetes, prediabetes, obesity, or metabolic syndrome is limited because most participants did not have impaired glucose metabolism at baseline. The prior systematic review also rated the evidence as low or very low because of risk of bias, indirectness, and imprecision.[7]

Individual trials are broadly consistent with this neutral glycemic pattern. In a controlled feeding crossover study, palm oil was compared with partially hydrogenated soybean oil, soybean oil, and canola oil, and measured glucose and insulin among other cardiometabolic markers; the main reported differences concerned lipoproteins rather than glycemic outcomes.9 In healthy men, palm olein, olive oil, and lard did not produce significant differences in glucose or insulin, although lipid differences were observed.[10]

A double-blind crossover trial comparing palm olein-based and soybean oil-based mayonnaise reported unchanged plasma glucose despite differences in lipid outcomes.[11] Studies comparing palm olein and olive oil in Chinese participants also reported no significant differences in fasting glucose or insulin outcomes.[12,13] In addition, modifying the stereospecific position of palmitic acid in palm olein did not significantly influence insulin secretion or glucose regulation in healthy adults.[14]

Overall, refined palm oil and palm olein appear metabolically neutral for short-term fasting glucose and fasting insulin markers in relatively healthy adults. This should not be interpreted as evidence that palm oil improves glycemic control or is superior to unsaturated oils in diabetes. General dietary-fat evidence continues to favour substituting saturated fat with unsaturated fats, particularly polyunsaturated fats, for improved glycemic and insulin-resistance outcomes.[2]

Interesterified palm-derived fats

Interesterification changes the distribution of fatty acids on the glycerol backbone of triacylglycerols and may alter postprandial metabolism, absorption, insulin signalling, and tissue lipid handling. This is relevant because palm oil and palm stearin are often used in structured fats.

Human evidence remains limited. A randomised, double-blind crossover trial was conducted in adults with type 2 diabetes using test meals containing palm olein, interesterified palm olein, or high-oleic sunflower oil. The study reported a lower postprandial GIP response after interesterified palm olein than the other oils but did not find broad adverse effects on GLP-1-related glucose homeostasis.[8] Another randomised trial in overweight adults compared chemically interesterified fats rich in palmitic or stearic acid with natural palm olein for eight weeks and found no substantial variation in surrogate indicators of insulin resistance.[9]

However, animal studies raise caution. A 2024 mouse study reported that high-fat diets containing interesterified palm oil increased body mass, adiposity, insulin, HOMA-IR, inflammatory markers, adipocyte hypertrophy, pancreatic islet changes, and hepatic steatosis compared with controls and other high-fat conditions.[10] Other preclinical work has reported that interesterified palm oil can impair insulin signalling in the liver and adipose tissue and increase hepatic glucose production in mice.[11]

The discrepancy between short-term human trials and animal findings may reflect differences in dose, exposure duration, background diet, species physiology, and endpoints. At present, interesterified palm-derived fats cannot be concluded to impair glucose homeostasis in humans, but the preclinical signal supports the need for longer trials in people with insulin resistance, prediabetes, obesity, or type 2 diabetes.[11–13]

Palm-derived tocotrienols and palm vitamin E

Palm oil is a major natural source of tocotrienols, a subgroup of vitamin E compounds with antioxidant and anti-inflammatory properties. Unlike refined palm oil itself, the palm tocotrienol-rich fraction has been tested more directly in patients with type 2 diabetes.[14–17]

A 2023 systematic review and meta-analysis of randomised controlled trials in type 2 diabetes found that tocotrienol-rich fraction supplementation at 250–400 mg/day significantly reduced HbA1c, with a pooled mean difference of approximately −0.23 percentage points. Larger effects were observed in subgroup analyses with intervention duration below six months and diabetes duration below ten years. The same meta-analysis had no substantial pooled effects on blood pressure or high-sensitivity C-reactive protein identified.[15]

Another systematic review and meta-analysis evaluating tocotrienols and inflammatory/oxidative-stress biomarkers found that evidence for inflammatory outcomes was inconsistent. Some effects, such as C-reactive protein reduction, were influenced by limited study numbers or specific tocotrienol forms, and no consistent pooled effects were found for several inflammatory markers. The review concluded that larger, well-designed trials are warranted.[18]

Taken together, tocotrienols represent the most clinically promising palm-derived bioactive for glucose homeostasis. The magnitude of HbA1c reduction appears modest and should be interpreted as potentially adjunctive rather than therapeutic replacement. The evidence is not yet sufficient to define optimal formulation, dose, treatment duration, responder phenotype, or long-term safety in diabetes populations.[19–23]

Red palm oil and carotenoid-rich palm fractions

Red palm oil differs from refined palm oil because it retains carotenoids, tocopherols, tocotrienols, sterols, squalene, coenzyme Q10, and other phytonutrients. Reviews describe red palm oil as a source of carotenoids and vitamin E compounds with antioxidant and anti-inflammatory potential.[24]

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Despite this mechanistic plausibility, direct human evidence linking red palm oil to improved insulin resistance or glucose homeostasis is limited. Some trials have evaluated red palm olein in relation to cardiovascular or inflammatory risk markers, such as studies in hyperfibrinogenaemic volunteers, but these do not establish robust glycemic efficacy.[25–27]

Therefore, red palm oil should not currently be positioned as an evidence-based intervention for diabetes control. Its potential relevance may lie in antioxidant nutrition, vitamin A status, and cardiometabolic biomarker research, but trials designed specifically around HbA1c, HOMA-IR, oral glucose tolerance, continuous glucose monitoring, and insulin sensitivity are required.[28–31]

Oil palm phenolics

Oil palm phenolics are water-soluble compounds obtained from oil palm vegetation liquor or palm fruit processing streams. Preclinical studies suggest that palm fruit juice or oil palm phenolics may have anti-hyperglycemic and anti-lipemic effects, potentially through effects on glucose absorption, insulin secretion, insulin resistance, and lipid metabolism.[32–36]

Human evidence is much less developed. A ClinicalTrials.gov record described a study of oil palm phenolics in insulin-treated type 2 diabetes, but the enrollment was very small, and the record does not provide sufficient completed efficacy evidence for clinical conclusions.[37–39] A phase I trial has evaluated the safety of oil palm phenolics in healthy participants, but safety evidence in healthy adults is not equivalent to glycemic efficacy in diabetes.[40]

Thus, oil palm phenolics remain an experimental palm-derived bioactive class. They are promising for future metabolic research, but cannot currently be recommended for insulin resistance or glucose control.

Discussion

Principal findings

This review indicates that palm-derived interventions should not be treated as a single category. Refined palm oil, palm olein, red palm oil, tocotrienol-rich fraction, oil palm phenolics, and interesterified palm-derived fats differ substantially in composition, biological plausibility, and evidence base.

The strongest evidence for conventional palm oil and palm olein suggests short-term neutrality rather than glycemic benefit. In available human feeding trials, palm oil or palm olein generally did not significantly change fasting glucose, fasting insulin, or HOMA-IR compared with other oils. However, this evidence is limited by short duration, small samples, and enrollment of mostly healthy adults rather than people with diabetes or insulin resistance.[7,41]

The strongest clinical signal among palm-derived bioactives is for the tocotrienol-rich fraction in type 2 diabetes, where meta-analysis suggests modest HbA1c reduction.[15] However, inflammatory and oxidative-stress outcomes remain inconsistent, and the evidence does not yet establish tocotrienols as a stand-alone diabetes therapy.[18]

Interesterified palm-derived fats are an area of uncertainty. Short-term human trials do not show consistent worsening of insulin resistance, but animal studies indicate possible adverse effects on glucose tolerance, hepatic insulin signalling, adiposity, inflammation, and pancreatic morphology.[8,10,11] This warrants caution and more human research.

Therefore, the central interpretation of this review is not that all palm-derived products have a uniform metabolic effect, but that the evidence differs by product type, processing method, and population studied. This distinction directly informs the clinical conclusion that refined palm oil should not be promoted as a glucose-lowering intervention, whereas specific bioactives such as tocotrienol-rich fraction warrant further controlled investigation.

Biological interpretation

The metabolic effects of palm-derived products may depend on at least five factors.

First, the **fatty acid replacement context** matters. Replacing saturated fat with polyunsaturated fat is generally more favourable for glycemic regulation and insulin resistance than comparing palm oil with another saturated or mixed fat source.[2,42,43]

Second, the **food matrix and processing method** matter. Refined palm oil lacks many minor bioactive compounds retained in red palm oil, while interesterification changes triacylglycerol structure and may alter metabolic handling.[44]

Third, **population metabolic status** matters. A neutral effect in healthy adults does not exclude adverse or beneficial effects in individuals with obesity, fatty liver, prediabetes, type 2 diabetes, or established insulin resistance.[45,46]

Fourth, **duration matters**. Most palm oil feeding studies are short, often lasting only weeks, while clinically meaningful changes in HbA1c, liver fat, adiposity, and insulin sensitivity may require longer follow-up.[47,48]

Fifth, **outcome selection matters**. Fasting glucose alone is insensitive to early insulin resistance. Future trials should combine fasting insulin, HOMA-IR, oral glucose tolerance, continuous glucose monitoring, HbA1c, postprandial lipemia, hepatic insulin resistance markers, and ideally clamp-derived insulin sensitivity.[49,50]

Clinical implications

For patients with diabetes or metabolic syndrome, current evidence does not support recommending palm oil as a glucose-lowering dietary intervention. Palm oil may be neutral for short-term fasting glycemic markers in healthy people, but neutrality is not equivalent to benefit.[11] Dietary counselling should continue to emphasise overall dietary quality, calorie balance, fibre-rich foods, minimally processed foods, and replacement of saturated fats with unsaturated fats when appropriate. This interpretation is consistent with broader randomised feeding evidence showing more favourable glycemic effects from unsaturated fats, particularly polyunsaturated fats.[2,51] Palm-derived tocotrienols may have adjunctive potential in type 2 diabetes, particularly for modest HbA1c improvement, but supplementation should be evaluated alongside standard medical therapy, diet, physical activity, lipid management, and cardiovascular risk control.[15]

Public health and food-industry implications

Because palm oil is extensively utilised in processed foods, the public health issue is not only palm oil itself but also the dietary pattern in which it appears. Palm oil in ultra-processed foods, high in refined carbohydrate, sodium, and excess energy, may contribute to metabolic risk through the total food matrix rather than through palm oil alone.[52–54] Food-industry reformulation using interesterified palm-derived fats should be evaluated carefully. Although interesterification can improve technical properties and reduce trans-fat use, preclinical

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evidence suggests that structured palm fats may have metabolic effects requiring more human evaluation.[10]

Strengths and limitations

This review's strengths include its focused relevance to insulin resistance and glucose homeostasis, separation of palm oil from palm-derived bioactives, and inclusion of both clinical and mechanistic evidence. Its limitations are important. First, the human evidence for palm oil and palm olein is mostly short-term and conducted in metabolically healthy participants. Second, few studies use gold-standard insulin sensitivity methods. Third, red palm oil and oil palm phenolics have insufficient direct human evidence for glycemic endpoints. Fourth, tocotrienol trials vary in formulation, dose, duration, and baseline diabetes characteristics. Fifth, animal studies provide mechanistic signals but cannot be directly translated into clinical recommendations.

Future research priorities

Future trials should prioritise:

I. Adults with prediabetes, obesity, metabolic syndrome, fatty liver disease, or type 2 diabetes.

II. Longer intervention durations, preferably at least 12–24 weeks for HbA1c and adiposity outcomes.

III. Clear comparison groups, especially olive oil, canola oil, soybean oil, sunflower oil, and other unsaturated-fat comparators.

IV. Standardised outcomes including fasting glucose, fasting insulin, HOMA-IR, HbA1c, oral glucose tolerance, continuous glucose monitoring, and lipid profile.

V. Mechanistic endpoints including inflammatory biomarkers, oxidative stress, liver fat, incretin hormones, and insulin secretion.

VI. Careful distinction between refined palm oil, palm olein, red palm oil, tocotrienol-rich fraction, oil palm phenolics, and interesterified palm-derived fats.

VII. Reporting of adverse events, medication changes, dietary adherence, and energy intake.

Conclusion

This PRISMA-oriented systematic literature review indicates that the metabolic effects of palm oil and palm-derived bioactives are heterogeneous and should not be interpreted as a single unified effect.

The current analysis based on the systematic literature review does not justify recommending palm oil itself as a dietary strategy for improving insulin sensitivity or glucose homeostasis. In clinical and public health contexts, palm oil consumption should be evaluated within the broader dietary pattern, including total energy intake, saturated fat intake, degree of food processing, fibre intake, and replacement nutrients. Where dietary fat modification is clinically indicated, evidence from broader nutrition research generally supports substituting saturated fats with unsaturated fats, specifically polyunsaturated and monounsaturated fatty acids, as a component of a cardiometabolic risk mitigation strategy.

It has been identified from the systematic literature review that palm-derived bioactives appear more promising than refined palm oil for metabolic health research. Among these, the tocotrienol-rich fraction has the strongest clinical evidence, with some randomised controlled trials and meta-analytic findings suggesting modest improvement in HbA1c among individuals with type 2 diabetes. Nevertheless, the extent of impact appears limited, and the evidence remains insufficient to position tocotrienols as a stand-alone intervention for diabetes management. Their possible role is better viewed as an adjunctive nutritional or nutraceutical strategy that requires further confirmation through larger, longer, and better-standardised clinical trials.

This systematic literature review recognizes that red palm oil, palm carotenoids, and oil palm phenolics also show biological plausibility because of their antioxidant, anti-inflammatory, and potential insulin-signalling effects. However, direct human evidence linking these compounds to clinically meaningful improvements in glucose regulation remains limited. Similarly, interesterified palm-derived fats require further scrutiny because preclinical findings suggest possible adverse effects on insulin signalling, adiposity, hepatic metabolism, and glucose tolerance, even though available human trials have not consistently exhibited immediate adverse effects.

Overall, the results of the literature review underscore the significance of distinguishing between palm oil as a dietary fat and palm-derived bioactives as concentrated compounds with potentially different metabolic actions. Subsequent research should concentrate on high-risk populations, including individuals with insulin resistance, prediabetes, type 2 diabetes, obesity, metabolic syndrome, and non-alcoholic fatty liver disease. Such studies should use standardised outcomes, including HbA1c, fasting glucose, fasting insulin, HOMA-IR, oral glucose tolerance, continuous glucose monitoring, inflammatory biomarkers, oxidative-stress markers, lipid profile, and liver-related metabolic endpoints.

Based on the systematic literature review, palm oil should not currently be promoted as an intervention for diabetes control or insulin-resistance improvement. Palm-derived bioactives, particularly tocotrienol-rich fraction, may have adjunctive potential in metabolic health, but the evidence remains preliminary and requires stronger clinical validation. Until more definitive evidence is available, clinical recommendations should emphasise overall dietary quality, appropriate fat substitution, weight management, physical activity, and evidence-based diabetes care, while considering palm-derived bioactives as an emerging area for further investigation rather than an established therapeutic approach.

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None.

Conflict of interest

The authors declare that there are no conflicts of interest.

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