World Nutrition Journal |eISSN 2580-7013

LITERATURE REVIEW

Nutraceutical potential of propolis for cardiometabolic improvement in type 2 diabetes mellitus: A literature review

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Received 17 November 2025
Accepted 16 April 2026
Published 31 Agustus 2026

Link to DOI:
10.25220/WNJ.V10.i1.0016

Citation: Sentosa W, Gunarti DR, Manikam NRM. Nutraceutical potential of propolis for cardiometabolic improvement in type 2 diabetes mellitus: A literature review. World Nutrition Journal.2026 August 31,10(i1): 185-195.

Abstract

Background: Type 2 Diabetes Mellitus (T2DM) is associated with chronic hyperglycemia and cardiometabolic complications. Nutraceuticals rich in flavonoids, such as propolis, have emerged as potential adjunctive strategies to improve cardiometabolic outcomes in T2DM patients. However, human clinical evidence remains limited and inconsistent. Therefore, this review evaluates the potential effects of propolis supplementation on cardiometabolic outcomes in patients with T2DM.
Methods: Literature searches were conducted in PubMed, ScienceDirect, and Wiley Library for studies published between 2015 and 2025 using keywords related to "Propolis," "Type 2 Diabetes Mellitus," "Cardiometabolic benefits", "oxidative stress", "glycemic control", and "lipid profile". Eight studies were included in this review, including randomized controlled trials published in English.
Results: Propolis, which is rich in flavonoids and phenolic compounds, exerts antioxidant, anti-inflammatory, and lipid-lowering effects. Supplementation improved fasting glucose, postprandial glucose, lipid profile, antioxidant and anti-inflammatory markers in several trials. Although, results remain heterogeneous due to differences in botanical sources and dosages.
Conclusions: Propolis demonstrates potential as a nutraceutical adjunct for cardiometabolic management in T2DM. Standardization of its composition and dosage remain essential for consistent therapeutic efficacy.

Keywords: cardiometabolic health, flavonoids, nutraceutical, propolis, type 2 diabetes mellitus

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Introduction

Type 2 Diabetes Mellitus (T2DM) is one of the leading causes of mortality in Indonesia. Based on the Indonesian Health Survey (2023), the prevalence of T2DM increased from 10.8% in 2018 to 11.7%. As of 2024, diabetes remains the main contributor to both mortality rate and healthcare cost burden in Indonesia.[1–3] Type 2 Diabetes Mellitus is a metabolic disorder characterized by elevated blood glucose levels caused by insulin resistance. Several factors contribute to its complications, including uncontrolled glycemic index, dyslipidemia, increased oxidative stress, and inflammation.[4]

The American Diabetes Association (ADA) recommends that adults with diabetes consume 14 grams of fiber/1000 kcal/day, or at least five portions (approximately 400 grams) of fruits and vegetables daily. However, based on the Individual Food Consumption Survey (SKMI) in 2014, the consumption of fruits and vegetables in Indonesia remains below recommended levels.[5] In addition to being the source of dietary fibers, fruits and vegetables are also the major sources of flavonoids in daily diets. [6]

Flavonoids are natural compounds formed by a phenolic structure and are secondary metabolites derived from plants. Their characteristic varies according to the arrangement of their phenyl and carbon rings.[6] Flavonoids are known to exert antioxidant, anti-inflammatory, and cardioprotective effects.[7]

One emerging nutraceutical rich in flavonoids is propolis, a resinous substance produced through a mixture of resin and bee saliva.[8] Each gram of *Apis mellifera* propolis contains approximately 1990 mg of flavonoid, while *Trigona* sp. contains 1000 mg.[6] The biological properties of propolis vary according to its flavonoid content, which depends on plant origin and extraction method.[9] Compared with other flavonoid-rich nutraceuticals, propolis contains unique bioactives such as caffeic acid phenethyl ester (CAPE) and artepillin C, which show dual hypolipidemic and anti-inflammatory effects.[10]

However, human clinical evidence regarding standardized propolis supplementation and its impact on cardiometabolic biomarkers in T2DM remains limited and inconsistent. This review aims to evaluate the potential effects of propolis supplementation on cardiometabolic outcomes by maintaining glycemic control, improving lipid profiles, and reducing oxidative stress in T2DM patients.

Methods

This study was conducted as a narrative literature review following predefined search and selection criteria. Search was conducted in PubMed, ScienceDirect, and Wiley Library, covering databases from the last ten years (2015-2025). The search included the following keywords: “Propolis”, “Type 2 Diabetes Mellitus”, “Cardiometabolic benefits”, “oxidative stress”, “glycemic control”, and “lipid profile”. Studies were included based on these criteria (1) The study design was a randomized clinical trial, (2) The literature was published in English and full-text accessible, and (3) The study was relevant to the main topic. This review included eight studies investigating the role of propolis, primarily in subjects with Type 2 Diabetes Mellitus (**Figure 1**)

Results

A total of 360 studies were identified through database searching. After removal of duplicates (n=6), 354 articles were screened for eligibility and 338 papers were excluded.

Sixteen full-text articles were assessed for eligibility. Eight articles were excluded for not meeting inclusion criteria, including non-randomized controlled trial design (n=2), non-type 2 diabetes mellitus (n=5), and outcomes not of interest (n=1). Ultimately, eight studies were included in the final review. Study inclusion details are shown in **Figure 1**.

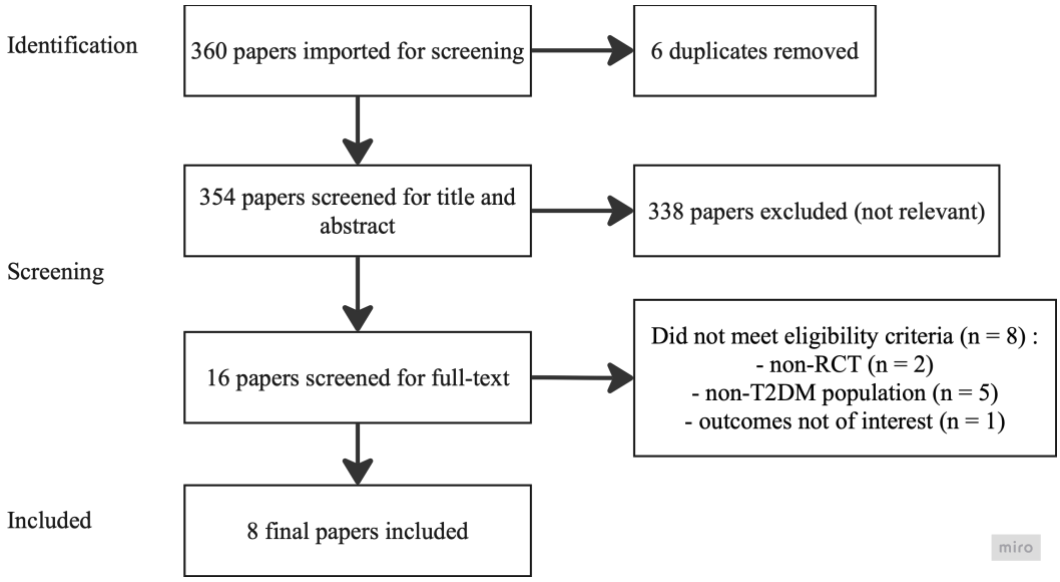

Figure 1. PRISMA Flow Diagram Study

Literature Review

1. Type 2 Diabetes Mellitus and cardiometabolic complications

Persistent hyperglycemia in T2DM promotes de novo lipogenesis, resulting in triglyceride dysregulation, reduced high-density lipoprotein (HDL), and increased low-density lipoprotein (LDL) levels.[11] Insulin resistance further stimulates lipolysis through hormone-sensitive lipase (HSL), increasing free fatty acids that are re-esterified into triglycerides and contribute to hypertriglyceridemia.[12,13] Elevated triglyceride-rich lipoproteins increase hepatic lipase activity and promote the formation of small dense LDL (sdLDL). These particles may undergo structural modifications, including oxidation, desialylation, glycation, and the formation of electronegative LDL—modified LDL particles with increased negative charge—making them highly atherogenic.[11,12]

In addition to dyslipidemia, chronic hyperglycemia induces low-grade inflammation through the release of pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). The interaction between oxidative stress, inflammation, and dyslipidemia contributes to endothelial dysfunction and increases the risk of macrovascular complications, including stroke, myocardial infarction, and peripheral artery disease.[11]

The primary macrovascular complication of diabetes mellitus is diabetic dyslipidemia.[14] Newly diagnosed type 2 diabetes has a prevalence of 67.7% high cholesterol, mainly high LDL, which accounts for 91,7%. Adults with diabetes have a 2 to 4 times higher risk of developing cardiovascular disease; the main contributing factors are obesity, hyperglycemia, hypertension, and dyslipidemia.[12,13,15] Main dyslipidemia characteristics were increased triglyceride, remnant lipoprotein, and sdLDL; decreased

HDL, and postprandial dyslipidemia.[12] These changes made dyslipidemia in diabetic patients more atherogenic than other types of dyslipidemia.[16]

Lipoprotein alterations in diabetic dyslipidemia occur both qualitatively and quantitatively. Triglycerides will increase, and HDL will decrease as a result of insulin resistance in diabetes. The changes in lipoprotein particles happen to be increased sdLDL, very low-density lipoprotein (VLDL), and dysfunction of HDL.[12]

Despite pharmacological management, cardiometabolic disturbances in T2DM often persist and contribute to endothelial dysfunction and cardiovascular disease. Therefore, addressing cardiometabolic factors remains essential to reduce cardiovascular mortality and morbidity.[11]

2. *Propolis: Composition and Nutritional Bioactive*

Propolis is a resinous substance produced by bees and used to seal and protect the hive. It was formed by a mixture of resins originating from flowers, leaves, and plants with *Apis mellifera* bee saliva. Most of propolis consists of 50-55% resin, beeswax (30%), and pollen (5-10%), and others such as amino acids, minerals, sugar, vitamins, flavonoids, and phenols. The characteristic and profile of propolis were determined by the amounts of flavone, flavonols, flavonones, dihydroflavonoles, and total phenolic compound.[17] The composition of each propolis varies according to its geographical origin, bee species, botanical source, climatic condition extracted, and extraction method.[18,19]

Generally, propolis has three types based on botanical sources: poplar, Baccharis, and Dalbergia.[10] Different types of propolis based on their geographical origin, botanical source, and main flavonoid composition are presented in the **Table 1**.[17,18,20]

Table 1. Major Types of Propolis and Their Nutraceutical Relevance

Propolis type	Geographical origin	Botanical source	Main composition	Potential Nutraceutical Relevance
Poplar	Europe, North America, Non-tropical area of Asia, New Zealand	*Populus* spp., *Populus nigra* L.	Flavones, flavonones, cinnamic acid and its esters (e.g., chrysin, galangin, pinocembrin, pinobanksin-3O-acetate, quercetin, kaempferol, apigenin, naringenin)	Antioxidant, anti-atherogenic
Green (Alecrim) Brazilian	Brazil	*Baccharis* spp. *(*B. *dracunculifolia)*	Prenylated p-coumaric acid, diterpenoid acid (e.g., Artepilin C)	Antioxidant, anti-inflammatory
Red propolis	Cuba, Brazil, Mexico	*Dalbergia* spp.	Isoflavonoid, (isovlavans and pterocarpans)	Estrogenic modulation, anti-obesity
Mediterranean	Sicily, Greece, Algeria, Malta, Turkey	*Cupressaceae* spp.	Diterpenes such as totarol and totarolone	Antimicrobial, antioxidant
"Pacific"	Pacific region (Okinawa, Taiwan, Indonesia)	*Macaranga tanarius*	C-Prenyl-flavonones (propolins, prokinawan, nymphaeol, isonymphaeol)	Lipid-lowering, glycemic control

3. *Mechanism Relevant to T2DM*

a. *Propolis and Lipid Modulation*

Some previous studies in type 2 diabetes patients reveal the mechanism in which propolis could lower lipid levels. A possible mechanism shows that flavonoids in propolis may inhibit the hepatic 3-hydroxy-3-methylglutaryl-CoA reductase (HMG CoA reductase), acyl CoA cholesterol-acyltransferase (ACAT), acetyl-CoA carboxylase α (ACAC), and fatty acid synthase (FAS). Therefore, it will reduce cholesterol availability to produce VLDL. In addition, propolis may also decrease the activity of phosphatidylcholine-specific phospholipase C and increase the level of annexin A7.[21]

Propolis also contributes to increasing HDL level. It increases the expression of liver ATP-binding cassette transporter A1 and G1 protein, which will increase the formation of HDL particles.[21] A previous study in mice showed some properties of lipid-lowering effect of propolis, such as increasing the inhibition activity of enzymes involved in cholesterol biosynthesis and reducing hormone-sensitive lipase.[22]

The main bioactive component of propolis that has hypolipidemic activity is caffeic acid. The ester form, caffeic acid phenethyl ester (CAPE), inhibits the differentiation of adipocytes by blocking the expression of PPAR-γ, *adipocyte-specific fatty binding protein* (aP2), CEBP-α, and *fatty acid synthase* (FAS) that lower the triglyceride storage. It also inhibits the phosphorylation of extracellular signal-regulated kinase (ERK) and Akt to decrease the expression of cyclin D1 downstream, therefore suppressing the fat production.[18]

A study by Zakerkish et al.,[23] administered 1000 mg of Iranian propolis supplementation and compared to a placebo group, with a total of 94 participants diagnosed with T2DM aged 35 to 85 years old, analyzed the lipid profile alteration before and after intervention of Iranian propolis 500 mg capsules twice daily as an adjunctive to standard therapy. In the final week of the study, a 90-day period, both glycemic and lipid indicators were measured.Click or tap here to enter text.

Compared to the placebo group, the propolis group showed significant improvement of HDL cholesterol (p=0.024), with basal levels of 44.66 ±8.69 and 43.98 ±10.21 in the propolis and placebo group, respectively. The HDL cholesterol improved by 10.6%. Other cholesterol, such as triglycerides, total cholesterol, LDL cholesterol, and VLDL cholesterol, shows no significant changes.[23]

This result is in accordance with a previous study by Afsharpour et al.,[24] which showed a significant increase of HDL level (p=0.045) and a decrease of LDL (p=0.039), triglycerides (p=0.034), and total cholesterol (p=0.021). On the other hand, Samadi et al.,[25] showed the significant alteration of total cholesterol (p=0.01) and LDL (p=0.04), but not in HDL (p=0.27) and triglyceride level (p=0.17) compared to the placebo group. The propolis used in this study was 300 mg Iranian propolis pills, three times a day for 12 weeks.Click or tap here to enter text.

b. *Glycemic Regulation*

Propolis may improve glycemic control through several mechanisms. Flavonoids present in propolis act as α-glucosidase and α-amylase inhibitors, reducing intestinal glucose absorption and postprandial hyperglycemia.[19,22,26] In addition, compounds such as galangin and pinocembrin modulate insulin signaling through the Akt/mTOR pathway,

while caffeic acid phenethyl ester (CAPE) activates the AMPK pathway, which enhances glucose uptake and improves energy homeostasis.[17]

Clinical trials have demonstrated improvements in glycemic parameters following propolis supplementation. A previous study by Ochoa-Morales et al.,[27] in Mexico showed the supplementation effect of propolis compared to monotherapy metformin and placebo. The propolis used in the study was 600 mg (given in divided doses) produced in Chicago, Illinois, USA, in addition to base therapy of 850 mg metformin twice daily. A total of 36 participants were recruited in the study and were subjects with T2DM aged 30-60 years old, men and women. Participants then were randomly assigned to 3 groups: placebo group, metformin group, and propolis group. Supplementation was given for 12 weeks, and glycemic and lipid alteration was analyzed.

The result showed that the fasting plasma glucose (FPG) compared to placebo, both the metformin ($p=0.002$) and propolis groups ($p=0.004$) showed significant reduction. However, no significant differences were observed between the placebo and metformin group for FPG level ($p=0.13$), 2-hour postprandial ($p=0.347$), and HbA1c level ($p=0.617$).[27]

This study showed that propolis supplementation could lower the FPG and 2-hour postprandial blood glucose. The possible mechanism was reduced glucose-6 phosphatase, translocation of glucose transporter 4 (GLUT-4) in skeletal muscle, and increased glucose uptake in peripheral tissue. Propolis also inhibits the alpha amylase, beta glucosidase, and intestinal maltase, which explains the decreased postprandial glucose.[27]

Similarly, Zakerkish et al.,[24] showed that there were also significant decreases of HOMA-IR by 46.6% ($p<0.0001$), HbA1C by 8% ($p=0.006$), FPG by 28.6% ($p<0.0001$), and 2-hour postprandial glucose by 50.8% ($p<0.0001$).[23] Comparable findings were reported by Afsharpour et al., who found significant reductions in fasting and postprandial glucose levels following propolis intake.

c. Anti-inflammatory Effect

Persistent hyperglycemia in T2DM promotes the formation of advanced glycation end products (AGEs), which contribute to oxidative stress and vascular inflammation. This process stimulates the release of pro-inflammatory cytokines, including TNF-α, IL-1β, and IL-6, which accelerate endothelial dysfunction and atherosclerotic plaque development.[28]

Polyphenolic compounds in propolis exert anti-inflammatory effects by inhibiting key signaling pathways such as nuclear factor-kappa B (NF-κB), cyclooxygenase-2 (COX-2), and mitogen-activated protein kinase (MAPK). These mechanisms suppress the production of inflammatory cytokines and may reduce the progression of vascular inflammation.[18]

Cinnamic acid and coumarate in propolis inhibit the production of IL-6; therefore, the production of macrophages decreases, and IL-17 increases as a protective cytokine against myocardial infarction.[17] The bioactive compounds in propolis with anti-inflammatory properties include caffeic acid phenethyl ester (CAPE), terpenoids, steroids, and amino acids.[29]

Yousefi et al.,[29] showed that 8 weeks of propolis supplementation could lower the IL-6 ($p=0.017$) and IL-17 ($p=0.02$) significantly. This study used propolis capsules from Alamut, Qazvin, Iran, with each consumption containing 1500 mg/day, divided into 500 mg capsules three times a day. The study includes participants with T2DM diagnosed less than ten years ago, aged 35-55 years old, in Iran.

Another study by Zakerkish et al.,[23] showed no significant change of IL-6 and TNF-α levels after supplementation of Iranian propolis for 90 days. Zhao et al.,[30] showed that supplementation of 900 mg of Brazilian propolis for 18 weeks shows a significant reduction of IL-6 and TNF-α levels compared to the control group. This study shows that Brazilian propolis intake for 18 weeks results in a favorable anti-inflammatory effect in T2DM patients. IL-1β and TNF-α as pro-inflammatory cytokines were inversely correlated with IL-6 as anti-inflammatory in this study.

d. Antioxidant Mechanism

Oxidative stress plays an important role in the pathogenesis of T2DM and its cardiovascular complications. Persistent hyperglycemia increases the production of reactive oxygen species (ROS), which damage cellular structures and impair endothelial function.[29,31] Propolis contains flavonoids and polyphenols with strong antioxidant properties that scavenge free radicals, inhibit lipid peroxidation, and enhance the activity of endogenous antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx).[22,32] The hydroxyl group of flavonoid has high reactivity to free radicals so that it could bind and form a more stable and less reactive particle.[31]

Polyphenolic compounds in propolis can neutralize reactive species such as superoxide (·O2−), hydroxyl radicals (·OH), and hydrogen peroxide (H2O2), and may inhibit pro-oxidant enzymes including NADPH oxidase and xanthine oxidase while enhancing antioxidant enzyme expression. Certain polyphenols, such as kaempferol, may also form metal-chelating complexes that contribute to antioxidant activity.[18]

In addition, flavonoids inhibit lipid peroxidation, platelet aggregation, and cyclooxygenase and lipoxygenase activity. These compounds may also suppress LDL oxidation through activation of the nuclear factor erythroid-2 related factor-2 (Nrf2) pathway, thereby reducing oxidative stress and the risk of atherosclerosis.[22,33]

Several propolis constituents, including 3,5-dicaffeoylquinic acid and 1,4,5-tricaffeoylquinic acid (art-C), further contribute to antioxidant activity by scavenging superoxide and hydroxyl radicals in vascular tissues.[17]

Afsharpour et al.,[31] conducted a randomized, double-blind study in Iran involving 62 T2DM patients, who were randomly assigned to a propolis or placebo group. Subjects were male and female patients aged 33 to 55 years old. The propolis used in this study was in capsule form with a 500 mg dose each, three times a day for 8 weeks. Oxidative stress parameters that were analyzed in the study are plasma levels of total antioxidant capacity, glutathione (GPx), and superoxide dismutase. In the study, after 8 weeks of propolis supplementation, there was a significant increase of total antioxidant capacity ($p=0.042$), superoxide dismutase ($p=0.034$), and glutathione ($p=0.028$).

Hesami et al.,[34] also showed the effect of propolis supplementation on oxidized LDL and catalase activity as an antioxidant, using the same dose of capsule propolis for 8 weeks in an Iranian population. The result showed that catalase activity increased significantly ($p=0.041$) and oxidized LDL decreased significantly ($p=0.031$). This could lower the risk of atherosclerotic plaque formation. A similar result was obtained by Moayedi et al.,[35] who analyzed the result of propolis supplementation compared to standard, exercise and placebo groups.

Sixty women participated in the study and were given 4 treatments: a control group, which was given nothing; an exercise group, which was asked to perform combined aerobic and resistance training; a propolis-only group, which was given supplementation only; and a combined exercise and propolis supplementation group. Propolis was given

in encapsulated form three times daily with a total of 1500 mg/day for 8 weeks. The result shows significant upregulation of SOD ($p<0.05$) and TAC ($p<0.05$), while MDA ($p<0.05$) was downregulated in the propolis supplementation group compared to the control. A more significant result was shown in the exercise combined with the propolis group, which shows an increase in SOD and TAC and reduced MDA.[35]

Strength and limitation

This literature review has several strengths. It focuses on randomized controlled trials evaluating propolis supplementation in patients with type 2 diabetes mellitus and integrates clinical findings with mechanistic explanations, including lipid modulation, glycemic regulation, anti-inflammatory, and antioxidant effects. In addition, multiple cardiometabolic outcomes were evaluated, providing a comprehensive overview of the potential role of propolis as a nutraceutical adjunct in T2DM management. However, this review has limitations, including the absence of a formal risk-of-bias assessment and heterogeneity among the included studies in terms of propolis composition, dosage, and intervention duration.

Conclusion

Type 2 Diabetes Mellitus (T2DM) is a metabolic disease characterized by elevated blood glucose levels resulting from insulin resistance. Despite the availability of T2DM treatment, nutraceuticals such as propolis have shown promising potential as adjunctive therapies for improving metabolic control and preventing complications.

Propolis has demonstrated measurable benefits on glycemic control, lipid modulation, and antioxidant balance among patients with type 2 diabetes mellitus when used as adjunct to standard therapy. However, current clinical studies are characterized by heterogeneity in propolis composition and the absence of standardized formulation, which limit clinical translation. Current evidence also showed limited intervention period and relatively small sample size, therefore limiting conclusions on long-term cardiometabolic outcomes. Future research should prioritize comprehensive bioactive profiling and well-designed long-term intervention to establish clinically relevant therapeutic thresholds.

Conflict of interest

The authors declared no conflict of interest regarding this article.

Ethic Statement

Ethical approval was not required for this study because it is a literature review based exclusively on previously published study and does not involve human participants or animals.

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Author Contributions

WS : Conceptualization, data acquisition, formal analysis, interpretation of results, drafting the manuscript, and final approval of the version to be published; DRG, NRMM: Supervision, critical revision of the manuscript, interpretation of data, and final approval of the version to be published.

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