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LITERATURE REVIEW
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The Potential Benefits and Mechanism of Action of Tropical Nuts Against Metabolic Syndrome: A Literature Review

Potensi dan Mekanisme Kacang Tropis terhadap Sindrom Metabolik: Tinjauan Literatur

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ABSTRACT

Background: Metabolic syndrome is a significant risk factor for both type 2 diabetes mellitus and cardiovascular disease, with a high prevalence in Asia Pacific, particularly in Indonesia. To reduce its prevalence, several studies have recommended the use of tropical nuts, which can be developed as functional foods and complementary treatment. In this context, the bioactivities of tropical nuts can largely be attributed to their rich content of monounsaturated fatty acids, polyunsaturated fatty acids, fiber, minerals, vitamins, phytosterols, and polyphenols.
Objectives: This literature review aims to evaluate the potential benefits and mechanism of action of tropical nuts against metabolic syndrome.
Methods: The study design was a literature review of several articles from 3 online databases, including PubMed, Google Scholar, and ScienceDirect.
Discussions: The results showed that tropical nuts (peanut, sacha inchi, cashew, tropical almond, and Brazil nut) had several biologically active components, such as arginine, fiber, fatty acid, mineral, vitamin, phenolic compounds, resveratrol, and phytosterol. The test samples were reported to have the ability to modulate Nrf2, SOD, MDA, GSH, GPx, and CAT due to their antioxidant activity. In inflammation, tropical nuts had a significant effect on NF-κB, NLRP3, TNF-α, IL-8, IL-1β, IL-6, and IL-10. The results also showed their ability to enhance lipid synthesis, nitric oxide production, advanced glycation end-product, prostaglandin, SIRT3, homocysteine, protein kinase C, adhesion molecules, platelet aggregation, GLP-1, PYY, AGRP, PPARα/β/δ, GLUT4, and insulin receptor.
Conclusions: Tropical nuts had beneficial effects on metabolic syndrome due to their bioactivities, including antioxidants, anti-inflammatory, anti-obesity, antidiabetic, antihypertensive, anti-dyslipidemia, and cardioprotective.

INTRODUCTION

Metabolic syndrome (MetS) is a widely recognized risk factor that increases the possibility of experiencing type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). This condition is characterized by high blood pressure, elevated fasting glucose, and low high-density lipoprotein (HDL) cholesterol. According to WHO, the percentage of individuals in most Southeast Asian countries suffering from elevated blood glucose and blood pressure is higher compared to the United States and the United Kingdom. In addition, the prevalence of metabolic syndrome in Asia Pacific ranges from 11.9% to 37.1%, with Herningtyas and Ng reporting an average value of 21.66% in Indonesia[1]. Recent studies showed that its prevalence in China reached 31.1% for individuals aged ≥ 20 years and 24.4% for those aged ≥ 15 years in Indonesia[2,3].

According to several studies, the underlying cause of metabolic syndrome is the imbalance between caloric requirements and intake[4]. In this context, overeating, lack of physical activity, as well as genetic and epigenetic factors have been reported to be significant contributors to the imbalance. Genetic and epigenetic factors can affect body mass index, waist circumference, abdominal visceral fat, HDL-C, triglycerides (TG), insulin, glucose, and blood pressure. A recent study also showed that insulin resistance, visceral adiposity, and chronic inflammation are significant triggers with the potential to activate metabolic syndrome and its progression to CVD and T2DM[5]. Insulin resistance in fat tissue triggers the mobilization of Free Fatty Acids (FFA)[6], which inhibits insulin's effect on fat and triggers lipolysis, leading to increased FFA production. In addition, these fatty acids can reduce muscle glucose uptake and induction of

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gluconeogenesis and lipogenesis in the liver. In the pancreas, FFA inhibits insulin release but is metabolized by the liver into TG, which some transforming into VLDL[7]. Insulin resistance is known to trigger the sympathetic nervous system, leading to increased regulation of angiotensin II receptors and reduced synthesis of Nitric Oxide (NO), a vasodilator[5]. Individuals with obesity typically experience increased renal tubular reabsorption, causing sodium retention and hypertension[8]. Building on this idea, proinflammatory cytokines inhibit insulin signal transduction, causing resistance in the liver, skeletal muscle, and adipose tissue[9].

In line with these results, nutrition is the most influential risk factor for various diseases and early mortality[10]. For example, pathological disorders, such as hypertension, hypercholesterolemia, obesity, and inflammation are influenced by dietary patterns. In addition, Reactive Oxygen Species (ROS) in the mitochondria are produced in excess when diets heavy in fat and/or sugar are consumed, leading to insulin resistance caused by oxidative stress[11].

Previous reports have shown that the tropics cover approximately 36% of the world's land area, with 30% of the total population living in the region. In addition, tropical foods have the prospect of being developed as functional foods and complementary treatment for metabolic due to their phytochemicals, fiber, minerals, vitamins, and macronutrients content[12]. A typical example of tropical foods is nuts, which have been reported to be rich in bioactive compounds, monounsaturated fatty Acids (MUFAs), and polyunsaturated fatty Acids (PUFAs). Nuts also contain fiber, minerals, vitamins, phytosterols, polyphenols[13], and relatively high protein. Building on this idea, several systematic reviews have been conducted to assess their nutritional benefits and bioactivities. For example, Nunes, et.al. extensively discussed peanuts[14], while Bauset, et.al. (2022) explored almonds, cashews, hazelnuts, peanuts, pecans, pistachios, and walnuts[15]. A review by Eslami*, et.al.* also discussed the potential of nuts related to risk factors for metabolic syndrome but did not address any particular species[16]. Despite the existing literature, there is no information on nuts that grow in tropical areas. Therefore, this review aims to evaluate the potential benefits and mechanism of action of tropical nuts against metabolic syndrome. The results are expected to facilitate the development of further studies on tropical nuts and prompt their commercial cultivation. These efforts are anticipated to provide insights into their health benefits, particularly in tropical countries, such as Indonesia.

METHODS

A literature review was conducted from January-February 2024 by searching published literature related to tropical nuts and their potential benefits in metabolic syndrome. This was achieved by retrieving data from journals published from 2014 to 2024. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Statement was used during articles selection process. In addition, the search procedures were carried out using 3 databases, including Google Scholar, PubMed, and Science Direct. The search keywords were “metabolic syndrome”, “diabetes”, “dyslipidemia”, “hypertension”, “inflammation”, “oxidative stress”, “glucose”, “insulin”, “HDL”, “LDL”, “triglyceride”, “cholesterol”, “peanut”, “sacha inchi”, “tropical almond”, “cashew”, “*Bertholletia excelsa*”. The inclusion criteria were 1) articles on nuts grown in tropical areas and their effect on metabolic syndrome and related conditions, 2) published in the last 10 years, 3) available in full text, 4) clinical, pre-clinical, or in vivo studies with human or animal subjects, and 5) published in peer-reviewed journals. The process was limited to articles published in English and Indonesian languages. After duplicates were removed, the remaining were evaluated for inclusion criteria and eligibility, totaling 1056 articles. In the first step, articles published more than 10 years ago, reviews, abstracts only, other than journal articles, and paid articles were excluded. In the second stage, those concerning sections other than nuts seed or kernel (such as root, leaves, bark, etc.), did not have parameters related to metabolic syndrome, did not have significant results related to metabolic syndrome, did not examine the 5 kinds of nuts at all, and had jaded score <3 for trials in humans that did not fulfill the ARRIVE essential 10, were excluded. Conditions associated with metabolic syndrome included insulin resistance, hyperglycemia, dyslipidemia, hypertension, obesity, inflammation, and oxidative stress. However, to evaluate the quality of the samples, the Jaded score was used for human studies, while Arrive was used for animal studies. Based on the inclusion and exclusion criteria, a total of 19 articles were included in this review. Figure 1 showed the search technique used during the selection process.

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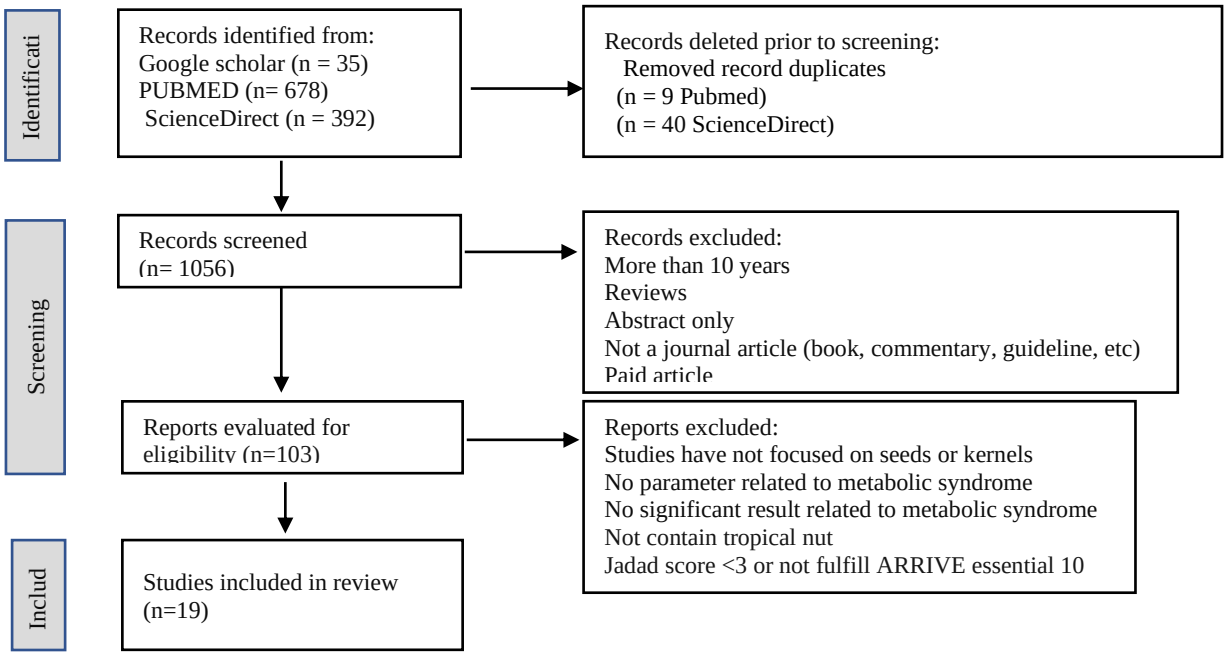

Figure 1. Search strategy for articles

DISCUSSIONS

Nutrition, Bioactive Content, and Potential of Tropical Nuts in Metabolic Syndrome

Nuts are defined as single-drupe dried fruits whose ovarian walls become hard when ripe, such as peanuts and tree nuts. Other sources showed that nuts were fruits with one hard edible seed and were covered in a dry wooden shell that did not split when ripe[17]. Several studies showed that these fruits could grow in tropical climates, including peanuts (*Arachis hypogaea*), Sacha inchi (*Plukenetia volubilis L.*), cashews (*Anacardium occidentale*), tropical almonds (*Terminalia Catappa L.*), and Brazil nuts (*Bertholletia excelsa*)[18]. Other types of tropical nuts were not discussed in this study due to various limitations. Table 1 summarized the nutritional content, including vitamins, minerals, and bioactive ingredients of raw tropical nuts.

Table 1. Nutrients, fatty acids, vitamins, minerals, and bioactive content of tropical nuts, according to several publications in the reference list

Parameters	Peanut	Sacha Inchi	Cashew Nuts	Tropical Almond	Brazil Nuts
Protein (%)	24.70-29.60	23.63	18.8-23	17.66-29.89	13.93-15.52
Fat (%)	42.6-45.5	48.52-56.20	45.05-50.40	54.68-63.65	66.16-66.71
Carbohydrate (%)	8.69-11.55	10.83	18.10-22.20	5.09-7.68	12.36
Dietary Fiber (%)	9.80-13.60	7.90	3.20-3.80	9.97	ND
Crude Fiber (%)	10.6-11.5	13.03	ND	1.98	7.70
Ash (%)	2.08-3.11	2.59	2.40-2.60	3.78-4.60	3.28
Arginine (%)	3.08	ND	2.20	0.70	1.80-2.15
Total SFA (%)	6.60-9.80	7.20	9.71	27.36	16.91
Total MUFA (%)	20.60-45.10	8.52	29.82	20.88	19.37
Total PUFA (%)	1.50-18.40	77.73	8.65	16.04	30.43
Steric Acid (%)	1.00-1.40	2.99	4.31	3.50	6.30-6.60
Palmitic Acid (%)	2.80-5.80	4.21	4.84	23.70	10.10-11.10
Oleic Acid (%)	20.00-44.00	8.52	28.79	20.6	18.9-19.2
Linoleic Acid (%)	1.50-18.40	33.85	8.58	15.7	23.8-30.3
α-Linolenic Acid (%)	ND	43.88	0.06	0.35	0.07-0.12
Folate (µg/100 g)	239.00	ND	39.10	ND	ND
Vitamin C (mg/100 g)	0.00	0.00	0.13	0.003	ND
Vitamin E / (mg/100 g)	6.56	8.99-137.00	5.80	ND	70.70
Vitamin K1	ND	ND	15.26	ND	ND
Cu (mg/100 g)	1.14	0.80-1.29	2.50	1.75	2.00
Se (µg/100 g)	7.20	0.00	0.04	ND	500.00-7150.00
P (mg/100 g)	260.00-430.00	519.70	502.50	2128.00	757.80
Ca (mg/100 g)	30.00-50.00	126.30-240.60	41.00	587.72	256.80
Mg (mg/100 g)	110.00-130.00	321.00-344.20	248.80	19.73	393.50
Fe (mg/100 g)	2.50-4.60	4.00-10.35	5.70	5.32	2.50

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Parameters	Peanut	Sacha Inchi	Cashew Nuts	Tropical Almond	Brazil Nuts
Mn (mg/100 g)	1.00-2.00	1.03	1.60	2.60	ND
Zn (mg/100 g)	3.00-5.00	4.10-4.90	5.30	0.77	4.70
Saponins (%)	5.50-8.20	0.70	ND	+[a]	ND
Phytates (%)	1.30-2.40	5.94	0.29	ND	0.19
Tannins (mg/100 g)	60.00-200.00	190.00	30.00-40.00	282.80[b]	0.01
Alkaloids (mg/kg)	ND	485.00	ND	+[a]	ND
Phenolic Acid (µg/g)	194.40	ND	ND	ND	248.00
Flavonoids (µg/g)	14.00	0.004	ND	+[a]	54.30
Total Phenol (mg/100 g)	ND	64.60-80.00	68.30	311.00[b]	90.30
Resveratrol (µg/g)	0.10	ND	ND	ND	1.37
Stigmasterol (mg/100 g)	6.70-9.40	21.20-26.90	0.30	ND	32.00
β-sitosterol (mg/100 g)	40.00-103.70	45.20-53.20	1.10	ND	99.10
Campesterol (mg/100 g)	5.60-13.10	7.10-8.80	9.00	ND	2.40
Reference	[19–23]	[20,24–30]	[31–33]	[34–37]	[33,38,39]

A qualitative test, b, in g/kg, ND, No Data, SFA (Saturated Fatty Acids), MUFA (Mono-Unsaturated Fatty Acids), PUFA (Poly-Unsaturated Fatty Acids)

Tropical nuts majorly contained fat, including MUFAs and PUFAs, except in tropical almonds where SFA was the most dominant fatty acid. In addition, the dominant MUFAs and PUFAs were oleic and linoleic acids, except in the sacha inchi where α-Linolenic (ALA) predominated. Arginine was found in high amounts in peanuts and Brazil nuts[20], while minerals, such as Cu, Fe, Zn, Mn, and Se that acted as antioxidants were also found. The results showed that Brazil nuts were a food high in Se and commonly used in Se fortification. Other antioxidants, such as phenol compounds, tannins, and alkaloids were also found and could act as antinutrient agents[31,38]. Tropical nuts contained nutrients and bioactive that had the potential to relieve metabolic syndrome. These components could act as an antioxidant, anti-inflammatory, antidiabetic, anti-obesity, anti-dyslipidemia, antihypertensive, and cardioprotective, while arginine served as an antihypertensive agent[40]. Vitamin B9 had cardioprotective properties[41], and the fiber content served as anti-diabetic, anti-obesity, and serum-lowering lipids agents[42]. Oleic acids had potential as anti-inflammatory, antidiabetic, and anti-hyperlipidemic agents[43]. Cu, Mn, Fe, Zn, and Se were antioxidants[44–47] and resveratrol had anti-inflammatory, antioxidant, anti-obesity, anti-diabetic, and anti-hypertensive properties. Antioxidants, such as phenolic acids and flavonoids in tropical nuts could improve inflammation and diabetic conditions, along with cardioprotective effects[48–53]. Phytosterols, such as β-sitosterol and stigmasterol served as antioxidant, anti-inflammatory, antidiabetic, and anti-dyslipidemic agents, while campesterol had an anti-dyslipidemic effect[54–56]. Tannin and tannic acid were antioxidants and acted as anti-inflammatory and cardioprotective agents[30,57–59].

Peanuts *(Arachis Hypogaea)*

Fats, protein, and fiber are the major components of peanuts, which belonged to the family *Fabaceae*. These fruits contained bioactive components, such as arginine, fiber, vitamin E, vitamin B9, polyphenols, oleic acid, linoleic acid, Cu, Fe, Mn, Se, Zn, luteolin, phenolic acid, flavonoids, resveratrol, stigmasterol, sitosterol, campesterol, and tannin. In addition, 20 different types of amino acids were identified in peanuts, with arginine being dominant[14,60].

A study by Sapp*, et. al*. showed that the consumption of peanuts could increase SCFA producer, *Ruminococcaceae*. The intervention of 28 g peanuts for 6 weeks altered microbiota composition in adults with elevated fasting glucose. However, this study was limited to the Pennsylvania area and needed to be further investigated in larger populations and different areas[61]. Another study proved that peanut consumption for 6 months could decrease weight and systolic blood pressure, and this was superior in duration and number of subjects. The follow-up was carried out well and involved a dietitian. However, there was a limitation such as no assessment of waist circumference and fat-free mass and it could not be ascertained that weight loss was due to a decrease in fat mass or other causes[62]. High-oleic peanut consumption effectively reduced postprandial responses of insulin, TNF-a, and glucose compared to conventional peanut and control. Consuming conventional nuts and high-oleic nuts could reduce TG but there was no change in LDL-c. This could be due to the relatively short duration of the study, which was only 4 weeks, and a longer duration was needed to ensure the effect of high-oleic peanuts[63]. Wang, et. al. stated that some people with central obesity and metabolic disorder could not get the beneficial effect of peanuts. However, this could show that personalized nutrition was still needed for each individual. There was high variability in parameters and anthropometry between individuals in response to peanut consumption. The composition of the gut microbiota was very specific to each person, and the small number of samples could have affected the results of this study[64].

Sacha Inchi (*Plukenetia Volubilis L.*)

Sacha inchi was a group of *Euphorbiaceae*[29], which contained arginine, fiber, tocopherol, oleic acid, linoleic acid, α-Linolenic acid, Cu, Mn, Fe, Zn, phenolic compounds, tannin, stigmasterol, campesterol, and sitosterol[65–69]. In addition, it contained the highest linoleic acid, α-Linolenic, and tocopherol compared to other tropical nuts[70,71]. Linoleic acid and α-Linolenic acid served as anti-inflammatory, anti-obesity, antidiabetic, antihypertensive, and anti-dyslipidemic agents[65–69].

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Meanwhile, vitamin E acted as an anti-inflammatory, antioxidant, and cardioprotective agent[44,70].

Giving sacha inchi oil for 5 weeks could reduce fasting blood glucose, HOMA-IR, ALT, AST, TNF-α, and IL-6 while antioxidant enzyme activity increased, and this also improved insulin signaling protein expression and hepatic histopathology [72]. Li, *et. al*. found that in HFD-fed rats, the application of Sacha inchi oil could enhance the beta-oxidation of fatty acids, decrease de novo lipogenesis, and blunt hepatic steatosis and inflammation. Additionally, the combined administration of antioxidants and n-3 PUFAs could enhance the antisteatotic and anti-inflammatory benefits[73]. As both studies used rats for their subjects, further clinical trials must be done to ensure the results. In the human study, giving sacha inchi oil to metabolically healthy person could reduce cholesterol increase and reduce IL-6 levels. Metabolically unhealthy persons who received sacha inchi oil also underwent reduced IL-6 levels. This study was limited to male subjects, and further studies were needed to confirm the results in females. In addition, it was not assessed whether there were any side effects in the study[74]. Giving sacha inchi oil for 4 months was known to lower blood pressure and improve lipid profiles because the duration of administration was quite long, and this also assessed the side effects that appeared. Although there were some minor side effects, giving sacha inchi oil for four months was still safe[75].

Cashews *(Anacardium Occidentale L.)*

Cashews were a plant that belonged to the family *Anacardiaceae* and was native to Brazil but was cultivated throughout the tropics. Cashews contained arginine, magnesium, campesterol, oleic acid, linoleic acid, α-Linolenic acid, Cu, Mn, Fe, Zn, phenolic compounds, tannin, campesterol, vitamins B9, C, E, and K1[76]. Vitamin K1 in cashews acted as an anti-diabetic while magnesium had antihypertensive properties[77,78].

The study on rats showed that cashews could improve inflammatory factors such as TNF-α, IL-1β, IL-6, and IL-10. In addition, cashews also affected antioxidant activity[79]. This report was in line with Fusco, et. al. where cashews could prevent oxidative stress[80]. Based on Caldas, *et.al.* studies on women at risk of metabolic syndrome, giving a mixture of cashews 30 grams and Brazil nuts 15 grams was known to increase plasma Se concentrations and the percentage of lean mass, accompanied by reduced total body fat and VCAM-1 adhesion molecules when compared to the group that was not given a mixture of nuts. This study had limitations as there were many subjects lost to follow-up[81].

Tropical Almonds (*T. Catappa L.*)

T. Catappa Linn. was a plant native to Southeast Asia and South Asia and belonged to the family *Combretaceae*[34,37]. This contained arginine, flavonoids, oleic acid, linoleic acid, α-Linolenic acid, fiber, Cu, Fe, Mn, Zn, phenolic compounds, flavonoids, and vitamin C. *T. Catappa Linn* seed extract could significantly decrease blood glucose, modify lipid profile, restore redox imbalance, and improve liver enzyme in rats[82–84].

T. Catappa could decrease blood glucose, ALT, ALP, and AST and improve lipid profile[82,83]. This nut could also affect antioxidant activity by increasing GSH, CAT, and SOD[84]. Despite its potential against metabolic syndrome, reports on T. Catappa in humans were still limited.

Brazil Nut (*Bertholletia Excelsa H.B.K.)*

Brazil nut was a species native to the Amazon, which belongs to the family *Lecythidaceae*, and was considered a selenium source. Consumption of one Brazil nut each day raised dietary Se intakes. Selenium was a necessary nutrient for human health. The expression of roughly 20 selenoproteins, which had selenocysteine at their active sites, mediated the biological activities of selenium[38]. Brazil nuts contained some bioactive agents such as arginine, fiber, oleic acid, linoleic acid, α-Linolenic acid, Se, vitamin E, Cu, Se, Fe, Zn, flavonoids, phenolic compounds, sitosterol, stigmasterol, campesterol, and resveratrol.

In rats, Brazil nuts showed positive effects on body mass, fat mass, glucose levels, blood pressure, and platelet aggregation[85]. In addition, giving Brazil nuts 10% of the rat's weight showed a higher amount of gastric residue than the other groups which made rats feel full longer[86]. The study of 60 patients with T2DM and overweight/obese showed that there was decreased DNA damage after giving one Brazil nut per day. There was a negative correlation between serum Se and H_2O_2-induced DNA damage, where cells became more resistant to H_2O_2-induced DNA damage after the administration of Brazil nuts over 6 months[87]. Reports in 10 healthy humans showed a single consumption of Brazil nuts (20 or 50 g) which caused decreased levels of IL-1, TNF-a, IL-6, and serum IFN-g, as well as increased levels of IL-10. Moreover, since this study involved few subjects, further reports with more subjects were needed[39]. Consumption of Brazil nuts in hemodialysis patients, increased Se and GPx activity while TNF-α, IL-6, 8-OHdG, and plasma 8-isoprostane decreased, while LDL-c levels decreased and HDL-c levels increased significantly. Although Brazil nuts had the potential to improve the condition of hemodialysis patients, side effects must be monitored considering the decline in kidney function in these patients[88].

In Vivo Study of Tropical Nuts Related to Metabolic Syndrome

Tropical nuts were not only consumed in seed form but could also be in the form of oil such as Sacha Inchi oil[67,72,89]. Several studies examined the consumption of tropical nuts seeds and oil against metabolic parameters associated with metabolic syndromes. These parameters included glycemia, HbA1c, insulinemia, body mass index, plasma lipid levels, body weight, waist circumference, appetite, blood pressure, pro-inflammatory cytokines, anti-inflammatory cytokines, intestinal microbiota, and antioxidant activity. Some in vivo studies with human, rat, and mouse subjects were summarized in Table 2.

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Table 2. Literature data search of tropical peanut studies in vivo with rats, mice, or humans as subjects or populations

No	Types of nuts	Model/Country	Subjects/Population	Intervention/Comparison	Result	Side Effects	Reference
1	Peanuts	Randomized, Crossover Trial/ USA	50 adults (52% men; 42 ± 15 years; Plasma glucose 100 ± 8 mg/dL; BMI 28.3 ± 5.6 kg/m2)	Subjects consumed 28 g/day of dry-roasted nuts or isocaloric snacks for 6 weeks with a 4-week wash-out period.	*Ruminococcaceae* (SCFA-producers) were significantly more abundant (p = 0.027] compared to isocaloric snacks.	NR	[61]
		2-arm Parallel, Randomized, Controlled Trial 6 months/Australia	107 subjects > 18 years, 65% Women, BMI > 26kg/m^2 and with T2DM risk	Subjects were randomized in either the peanut group or the low-fat diet group (control). Limited energy intake was applied in both groups. Measurements are made in the 2nd and 6th month.	Systolic blood pressure decreased in the peanut group (p = 0.008).	No side effects were observed	[62]
		Randomized Parallel Arm Study 4 weeks/ Brazil	76 males, 18-50 years old, BMI 29.86 kg/m2	Subjects were divided into a control group, a regular peanut group (56 g), or a high-oleic peanut group (56 g). Participants ate a low-calorie diet.	Beginning: postprandial response to glucose, insulin, and TNF-α ↓ in a group of high-oleic. TG ↓ in both peanut groups. IL-10 ↑ in all groups.	NR	[63]
		Randomized Clinical Trial 12 weeks/USA	224 subjects; 20 – 65 years, men and women, ≥1 metabolic syndrome risk factor and central obesity	Participants were divided into peanut groups and the control group consumed 82 g/day of isocaloric rice bars.	Improvement in body weight, waist circumference, and glycemia in the peanut group (p = 0.0074, p = 0.0050, and p = 0.0001).	NR	[64]
2	Sacha inchi	True Experimental, randomization for 5 weeks/Thailand	36 Sprague-Dawley male rats; BW 160-180 g	Groups of rats: 1) normal control group; 2) diabetic group; 3) diabetes + SI oil group (0.5 mL/kg bw); 4) diabetes group + SI oil (1 mL/kg bw); 5) diabetes group + SI oil (2 mL/kg bw); and 6) diabetes + pioglitazone group (30 mg/kg body weight).	hyperglycemia index and insulin resistance, ALT, AST, MDA, TNF- α, IL-6, PEPCK, and G-6-Pase ↓ hepatic histopathological improvement; SOD, CAT, GPx, IRS-1, p-Akt, and liver glycogen content↑	NR	[72]
		True Experimental with randomization for 8 weeks/China	70 rats; male; Sprague-Dawley; 8 weeks; 180-220 g	Group of rats: 1) normal food + 10 kcal % fat; 2) HFD with 45 kcal% fat; 3) HFD + SI oil 0.5 mL/kg/day; 4) HFD + SI oil 1.0 mL/kg/day; 5) HFD + SI oil 1.5 mL/kg/day); 6) HFD + fish oil 1.0 mL/kg/day; 7) HFD + simvastatin (10 mg/kg/day).	SI oil reduces liver fat, ALT, AST, TC, LDL-c, and TG and increases HDL-c and gut microbiota diversity. Expression of genes related to TLR, Jak-STAT, NF-κB, NOD-like receptor, and T/B cell receptor signaling pathways was reduced.	NR	[73]
		Randomized Crossover Clinical Trial/Colombia	42 subjects; 21 WC ≥ 92 cm (metabolic unhealthy, MU), 21 WC < 92 cm (metabolic healthy, MH);	Subjects were given breakfast treatment with high saturated fat (HFM) and HFM+ SI oil. Between interventions, there is a 2-week washout period.	The addition of SI oil to HFM subjects prevents an increase in TC. In MH, SI oil intake improved cholesterol and lowered serum IL-6. At MU, SI oil intake lowers LPS and IL-6.	NR	[74]

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No	Types of nuts	Model/Country	Subjects/Population	Intervention/Comparison	Result	Side Effects	Reference
		Randomized, Double-Blind, Placebo-Controlled Study for 4 months/Peru	34 non-vegetarian subjects, male and female, BMI > 20 ≤35 kg/m2, 25–55 years, no history of hyperlipidemia, no consumption of fatty acid, lipid or antioxidant supplements or drugs that interfere with the study	The subjects are divided into the SI oil group and the sunflower oil group. The oil is consumed daily in the morning at a dose of 10 or 15 ml. Blood tests and anthropometry began at the beginning of the study and continued every 4 weeks.	TC, LDL-c, arterial blood pressure↓; HDL-c, and serum albumin ↑ in the SI oil group.	nausea, headache, fatigue, sleep, vomiting, flatulence, belching, heartburn, constipation.	[75]
3	Cashews	True Experimental, randomization for 3 days/Italy	18 Male Sprague–Dawley Rats, BW 200–230 g, induced paw edema with carrageenan (CAR)	Rats were divided into 3 groups: (1) CAR saline; (2) CAR + cashews (100 mg/kg); (3) Sham Group. Not given CAR but treated with saline or cashew nuts.	nitrate/nitrite, MPO, MDA, TNF-α, IL-1β, IL-6 ↓; IL-10, SOD, GSH, CAT↑	NR	[79]
		True Experimental, randomization for 3 days/Italy	18 Male rats, Sprague–Dawley, BW 200–230 g	Intestinal injury was induced in mice by ischemia/reperfusion (I/R) surgery. Rats were divided into 3 groups: (1) I/R +saline; (2) I/R + cashew nuts; (3) Sham Group. Animals had surgery.	CAT, SOD, GST, GPx, GSH ↑; IL-1β, TNF-α, IL-6, creatinine, ALT, and AST ↓	NR	[80]
		Randomized Controlled Trial for 8 weeks/Brazil	40 women; 20–55 y, overweight, WC ≥ 80 cm & fat percentage ≥ 32 %; with ≥1 component of metabolic syndrome; with/out metabolic complications	Subjects were randomly allocated to (1) control group: energy-restricted diet without nuts, n 19 or (2) Brazil and cashew nuts group (BN-Group): energy-restricted diet with daily 45 g of nuts (15 g of Brazil nuts & 30 g of cashew nuts), n 21	BN-group: improved body composition; VCAM-1, endothelial inflammation marker↓; Se status↑	NR	[81]
4	Tropical almonds	True Experimental, randomization for 28 weeks/Nigeria	36 male albino wistar rats	Rats are divided into 6 groups: Group A: saline + rat feed. Groups B – F were given cigarette smoke for 90 minutes. Group B is not treated. Groups C - E + TCSO 2.5, 5.0, and 7.5 ml/kg body weight, respectively. Group F + ascorbic acid .	MDA ↓ while GSH, CAT, and SOD ↑ in serum in mice given TCSO.	NR	[84]
		True Experimental for 28 days/Nigeria	15 male wistar rats, 160-180 g	Rats were divided into 3 groups: 1) control (aquades); 2) T. Catappa seed extract (TCE) 500 mg/kg; 3) TCE 1000mg/kg orally gavage.	The level of ALP significantly dropped in group 3 compared to the control and there was a dose-dependent decrease in LDL-c compared to the control group.	NR	[83]

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No	Types of nuts	Model/Country	Subjects/Population	Intervention/Comparison	Result	Side Effects	Reference
		True Experimental for 28 days/Nigeria	16 albino rats	Rats were divided into 4 groups: A) normal control + equates; B) diabetes control + aquades; C) diabetic rats + 200 mg/kg body weight TCE/day; D) normal + 200 mg/kg body weight TCE/day.	blood glucose levels, TC, LDL-c, TG, ALT, AST, ALP ↓ and HDL-c, total protein, and albumin ↑ compared to controls.	NR	[82]
5	Brazil nuts	True Experimental, randomization for 12 weeks/Brazil	32 male Wistar rats, 2 months old	There were 2 groups of rats randomized by weight: 1) the control group (n = 16) and 2) Hypersodic group (+1% NaCl). After 4 weeks, the group was divided into 4 subgroups: 1) control; 2) Brazil nut; 3) hypersonic; 4) Hypersodic + Brazil Nut.	Less body mass and fat mass gain, lower serum glucose levels, lower blood pressure increase, and lower platelet aggregation than the hypersonic group without Brazil nuts.	NR	[85]
		Clinical Trial One-Group Pretest-Posttest Design for 6 months/Brazil	60 patients (31 men, 29 women), 43-81 years, type 2 diabetes min. 5 years, overweight/ obese	All patients were given 1 nut per day. Anthropometric data and blood samples were assessed from the start of the study until 1 month after the study.	Serum Se ↑ and DNA damage ↓. There is a negative correlation between serum Se and H2O2-induced DNA damage.	NR	[87]
		True Experimental, randomization for 8 weeks/Brazil	27 Male Wistar rats, 2 months old	There were 3 groups of rats: (control group), standard feed + 5% Brazil nuts (BN5), and standard feed + 10% Brazil nuts (BN10). The mice underwent constipation and gastric emptying tests to assess motility.	The BN5 group: BW↑, while the BN10 group did not ($p<0.0001$). The BN10 group showed a higher amount of gastric residue than the other group ($p = 0.0008$).	NR	[86]
		Randomized Crossover Study/Brazil	10 healthy subjects, ages 23-34 years, 60% men	Each participant was tested four times with Brazil nuts 0, 5, 20, and 50 g. There was a washout period of 30 days before the treatment.	serum IL-1, IL-6, IFN-g, and TNF-a levels ($P <0.05$) ↓ while serum IL-10 levels ↑($P <0.05$).	NR	[39]
		Clinical Trial One-Group Pretest-Posttest Design for 3 months/Brazil	40 hemodialysis patients, >18 years old, dialysis for min.6 months	All patients were given 1 nut per day. Blood serum was taken at the beginning and end of the study.S	Plasma Se and GPx activity ↑, plasma levels of TNF-α, IL-6, 8-OHdG, and plasma 8-isoprostan ↓ significantly. LDL-c levels ↓ and HDL-c levels ↑ significantly.	NR	[88]

ACE (*Angiotensin-Converting Enzyme*), ALP *(Alkaline Phosphatase),* ALT *(Alanine Transaminase),* AST *(Aspartate Transaminase),* BW (Body Weight), CAT (*Catalase*), GSH (*Glutathione*), HDL (High-Density Lipoprotein Cholesterol), HFD (High-Fat Diet), BMI (Body Mass Index), LDL (Low-Density Lipoprotein Cholesterol), LFHC (Lower-Fat Higher-Carbohydrate), MDA (Malondialdehyde), VLDL (Very Low-Density Lipoprotein Cholesterol), NR (Not Reported), SCFA (Short Chain Fatty Acid), SI (Sacha Inchi), SOD (*Superoxide Dismutase*), TC (Total Cholesterol), TCSO (*Terminalia Catappa* Seed Oil), TG (Triglycerides), TPC (Total Phenolic Capacity), and WC (Waist Circumference).

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Proposed Mechanisms of Tropical Nuts Against Metabolic Syndrome

Peanuts *(Arachis Hypogaea)*

Peanuts were beneficial in weight management because of their protein and fiber. Vitamin E and polyphenols in peanuts could lower blood glucose and inflammatory markers[14]. The high Zn content in peanuts stimulated tyrosine kinase receptors, thereby increasing insulin sensitivity. The content of MUFAs could improve insulin sensitivity and reduce the glycemic response in insulin-resistant individuals by secreting more GLP-1. Nuts contained energy, but not all of it was readily available. This happened as a result of intracellular fat encapsulated in cell walls that were resistant to microbial and enzymatic breakdown in the digestive system, and the amount of energy lost due to this mechanism ranged from 5–18%. Peanut consumption also increased resting energy expenditure[60], while Fiber and MUFAs contributed to microbiota variation, and this could ferment fiber into SCFAs[64]. When propionate and butyrate bind to G-protein-coupled receptors (GPR41 and GPR43) in the colon, the hormones PYY and GLP-1 were released, which affected satiety and glucose balance. SCFAs could reach the circulation and affect substrate metabolism in peripheral tissues. Adipose tissue's lipid buffering ability was impacted by acetate and propionate, which prevented intracellular lipolysis. The presence of acetate, propionate, and butyrate could decrease cytokines and proinflammatory chemokines, and this could increase ß cell activity. SCFAs affected the generation of insulin in response to glucose[90] and by lowering NF-κB activation and the incorporation of cellular stearic acid, oleic acid could lessen the symptoms of inflammation. Additionally, It had the power to raise postprandial IL-10 concentrations and counteract TNF-a's inhibitory influence on insulin production[63]. Peanuts contained folic acid which lowered homocysteine levels and raised the risk of heart disease[41]. Luteolin and resveratrol could modulate Advanced Glycation End-product (AGE) and inhibit arterial stiffening[48]. Resveratrol could influence SIRT1, which in turn activated FOXO and raised the expression of the gene encoding endothelial NO synthase (eNOS), thereby increasing NO production[91].

Sacha Inchi (*Plukenetia Volubilis L.*)

Administration of sacha inchi oil improved lipid dysmetabolism and reduced intestinal microbiota dysbiosis in HFD-fed rats. This effect occurred primarily through improved regulation of FXR-RXR signaling by enhancing bile acid biosynthesis and uptake thereby reducing de novo lipogenesis, improving fatty acid oxidation, and improving dysregulation of TG, glycerophospholipids, and sphingolipid metabolism, hence improving HFD-induced lipid dysmetabolism. By reacting to excess bile acids, FXR helps to maintain the signal bridge that controlled bile acid production and enterohepatic circulation between the liver and small intestine, and this pathway also involved MUFAs and PUFAs. In HFD-fed mice, Sacha inchi reduced the expression of genes related to lipogenesis (Scd2), hepatic lipid absorption (CD36 and Fabp5), and fatty acid β-oxidation (Acaa1a, Acadm, and Acox2)[73]. By inhibiting the transcription factor expression, SREBP, ALA prevented the creation of fatty acids and cholesterol. Additionally, ALA had the potential to lower blood pressure by inhibiting ACE and modulating calcium release in smooth muscle cells[75].

Sacha inchi contained the highest LA and ALA compared to other tropical nuts. Proprotein convertase subtilisin/kexin type 9 (PCSK9) was reduced in plasma levels by LA. Through control of the degradation of LDL receptors, PCSK9 was identified as a major regulator of plasma LDL cholesterol. LA could inhibit SREBP-1c which in turn inhibited hepatic lipogenesis and triggers lipid catabolism in vitro. In addition, there was a decrease in LDL-c caused by a decrease in apolipoprotein B100 synthesis[66,92]. Toll-like receptor 4 (TLR4), agouti-related protein (AgRP), and the phosphorylation levels of c-Jun amino-terminal kinases (JNK) could all be suppressed by LA. AgRP encouraged eating, while JNK1 deficiency could result in reduced body fat and increased insulin sensitivity. Bioactive metabolites of LA increased PPARγ activation while decreasing NF-κB activation and gene transcription. However, in some cases, LA could also have proinflammatory properties[93]. By acting as a substrate for vasoactive prostaglandins and encouraging the relaxation of vascular smooth muscle cells, LA could lower blood pressure[65], and ALA could also be able to inhibit SREBP-1c. An increase in ALA intake led to an increase in PPARα's capacity to bind to DNA and express itself. NF-κB was inactivated with PPAR-α activation, and ALA could enhance the action of insulin in skeletal muscle and adipose tissue by sensitizing the activity of IGF-1 through activation of PPAR-α. This improved antioxidant status with the increase of GSH and Nrf2[67], and it had cardioprotective properties since it decreased levels of AA in platelets and increased levels of EPA. However, this could result in modulation of platelet aggregation[68].

Sacha inchi could be able to lessen atherosclerotic lesions caused by cholesterol by inhibiting CD36, PKC signaling, phosphorylating MMP-1 and -9, c-jun phosphorylation, inducing Nrf2, PPARdz, LXRa, and ABCA-1 levels. Adjusting PKC activity and p47 phosphorylation also prevented cell growth and LDL oxidation. These nuts played a role in reducing inflammation by inhibiting the NF-kB signaling pathway[70].

Cashews (*Anacardium Occidentale L.*)

Magnesium in cashews increased the synthesis of prostacyclin, formed NO, and modified endothelium-dependent vasodilation. However, lower Mg levels increased contraction triggered by ET-1. The blood artery became stiffer and blood pressure increased with decreasing magnesium[78]. The NF-kB pathway, pro-inflammatory TNF-α, and IL-1β, as well as the expression of ICAM-1 and p-selectin, were all reduced by cashew nuts. Additionally, it reduced inflammation most likely via modifying 5-LOX and COX-2[79]. In human hepatocytes, oleic acid stimulated PPARβ/δ expression through a calcium-dependent and FFAR1/GPR40-mediated pathway. The increase in PPARβ/δ caused by oleic acid negatively regulated phosphatase and tensin homologous (PTEN), a phosphatase that negatively modulated Akt and thereby increased insulin sensitivity[43]. Stigmasterol in cashews could inhibit

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cholesterol, and sitosterol also inhibits thyroid hormones involved in gluconeogenesis and glycogenolysis, and this phytosterol could increase CAT, SOD, and GSH[54]. β-sitosterol reduced IL-8, TNF-a, IL-1ß, and IL-6, as well as ROS and macrophages. Additionally, it inhibited NF-kB, lowered the initiation of NLRP3, stopped caspase-1 activation, and in hepar, it increased the antioxidant enzyme. β-sitosterol could have an anti-diabetic effect since it activated insulin receptors and GLUT4 in adipocyte tissue[56]. Campesterol and sitosterol competed with cholesterol for micellization and sterol uptake transporter and also inhibited pancreatic cholesterol esterase, hence, reducing cholesterol level[55].

Tropical Almonds (*T. Catappa L.*)

Feeding tropical almonds to rats could lower MDA as well as increases GSH, CAT, GPx, and SOD in serum[84]. In addition, tropical almonds could reduce Alkaline Phosphatase (ALP) and LDL-c, and this ability was dose-dependent[83]. When compared to controls, the administration of tropical almond extract in diabetic rats resulted in significant increases in total protein, albumin, and HDL-c and significant decreases in blood glucose, LDL-c, TG, Alanine Aminotransferase (ALT), Aspartate Transaminase (AST), and ALP[82]. When free radicals interacted with the lipids in cell membranes, a process known as "lipid peroxidation" took place, damaging the membranes and releasing toxic byproducts. Flavonoids and phytochemicals contained in tropical almonds prevented lipid peroxidation by neutralizing free radicals and inhibiting the formation of ROS. By either upregulating the expression of LDL receptors or decreasing the production of cholesterol, flavonoids were known to impact lipid metabolism. Furthermore, it was showed that saponins inhibited the rate of intestinal liver and intestinal cholesterol production, raised lipid peroxidation, and inhibited the absorption of cholesterol[83,84]. Even though this value had a variety of bioactive ingredients and nutrients that played a positive role in metabolic syndrome, studies on nutritional content and in vivo experiments on tropical almonds were still limited. There was a further recommendation of nuts to be studied considering their potential effect.

Brazil Nut (*Bertholletia Excelsa H.B.K.)*

The fiber content in Brazil nuts could slow down gastric emptying. Due to their high content of L-arginine, a powerful vasodilator precursor to NO, Brazil nuts could have had a role in the significant rise in total plasma NOx levels. The connection between serum Se levels and T2D episodes was U-shaped. Se functioned as a selenoprotein and eliminated H2O2 and organic peroxides[87]. Selenium could modulate NF-kB, reduce adipocyte hypertrophy and adipogenesis, and affect decreasing hypertension[45,47]. Se and coenzyme Q10 could modulate TNFr1, TNFr2, osteoprotegerin, osteopontin, and copeptin which had beneficial effects on fibrosis and myocardium[46]. However, compared to SFA, MUFA and PUFA could be more quickly oxidized, had a stronger thermogenic impact, and caused less fat to accumulate[81]. By enhancing postprandial thermogenesis and suppressing lipid synthesis signaling, MUFA and PUFA also functioned as modulators of energy metabolism. Brazil nut could be linked to glucose metabolism through the Selenoprotein P (SePP) module gene rs3877899. The transcription factor Forkhead box (FOX) protein O1, which was involved in gluconeogenesis, had a binding site on SePP. This protein, which was a member of the selenium-proteome, could be impacted by the selenium found in Brazil nuts. This included a substance called catechin that could lower vascular adhesion molecules, which were linked to the activation of inflammatory processes in the endothelium environment. The increased activation of the AKT PI3K pathway, which was also in charge of activating the eNOs, could have contributed to the increased absorption of glucose. Brazil nuts could also increase Nrf2[88].

Chlorogenic acid and caffeineic acid could reduce fatty acid oxidation and block the function of fatty acid synthase (FAS). In addition, triglyceride production and fatty acid lipogenesis were regulated by FAS. Several studies had also shown that nuclear factor PPAR α could be activated by polyphenols. In addition to maintaining nutritional and energy balance and regulating lipid metabolism, PPAR also prevented fat accumulation in the liver. For preserving cellular energy homeostasis, polyphenols could activate AMPK, an energy sensor. Malonyl-CoA levels were lowered by AMPK phosphorylation at Thr172, which further activated CPT1, causing increased acyl-CoA transport to mitochondria and increased oxidation. As a result, there were fewer lipid vacuoles in the liver tissue and FAS expression was downregulated while Cpt1 expression was upregulated. Polyphenols could serve as direct or indirect antioxidants, and giving electrons to ROS was the direct method. Indirectly, polyphenols altered the interaction between Nrf2 and Keap1, which was a negative regulator of this protein[89].

The proposed mechanism of tropical nuts in metabolic syndrome was summarised in Figure 2. The content of each tropical nut tended to be the same. Two special features slightly distinguished were 1) the dominant linolenic content in sacha inchi, while in other tropical nuts linolenic did not dominate. α-Linolenic acid was known to have more positive effects on metabolic syndrome, 2) resveratrol was only found in peanuts and Brazil nuts, although this had not been confirmed in other nuts because there was no supporting report data.

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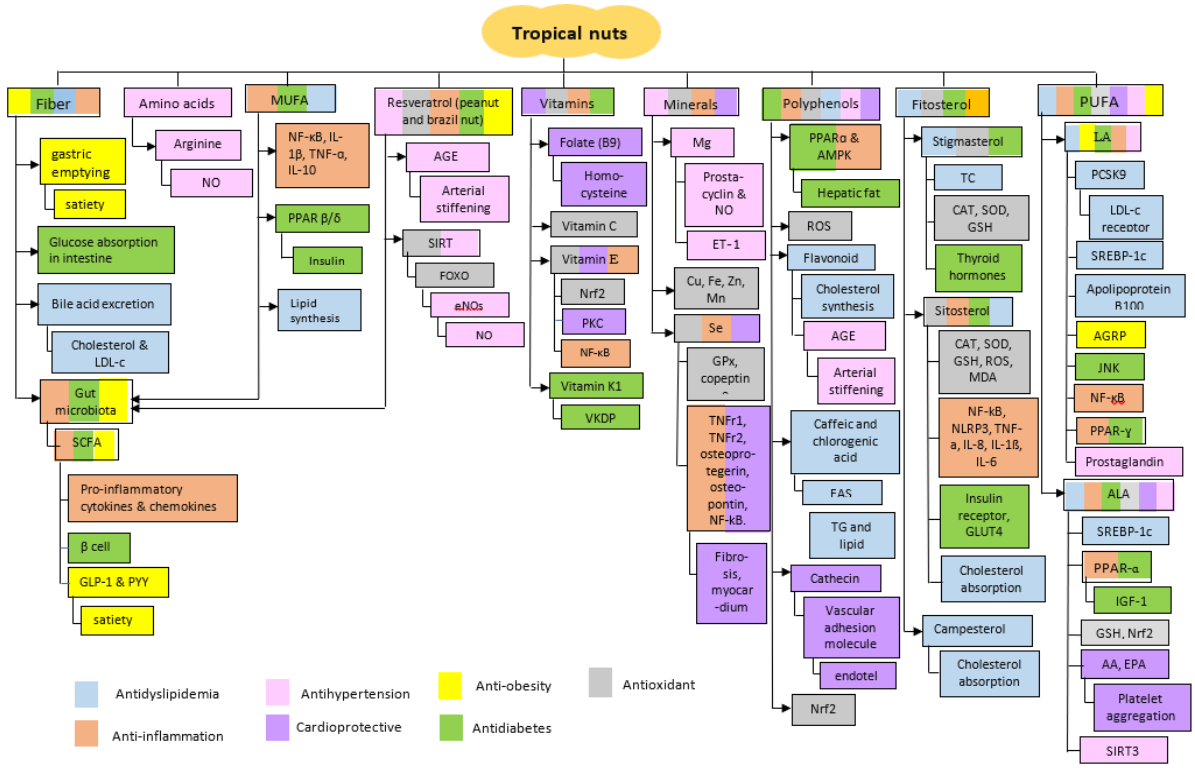

Figure 2. Proposed mechanism of tropical nuts against metabolic syndrome

CONCLUSIONS

In conclusion, tropical nuts contained bioactive ingredients that could have a positive influence on metabolic syndrome. These bioactive ingredients included arginine, fiber, PUFAs (α-linolenic acid, linoleic acid), MUFAs (oleic acid), vitamins (A, B, C, E), minerals (Se, Cu, Mg, Fe, Zn), phytosterols, and polyphenols. In addition, tropical nuts could act as antioxidants, anti-inflammatory, antiobesity, antidiabetic, antihypertensive, anti-dyslipidemia, and cardioprotective. The bioactivities were through mechanisms related to gastric emptying, glucose metabolism, gut microbiota, inflammatory cytokines, antioxidant activity, insulin signaling, and lipid metabolism.

Human studies for tropical almonds were still limited, and it recommended clinical trials to enrich the data. Moreover, given the potential and benefits of tropical nuts, these fruits must be consumed 1-2 times a day as in the Mediterranean diet[94]. Tropical areas could also increase their cultivation due to the suitability with the climate and health benefits.

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AUTHOR CONTRIBUTIONS

ARS: conceptualization, investigation, methodology, writing–original draft, writing-review, and editing; GA: conceptualization, supervising, validation, and writing-review; EM: supervising, validation, and writing-review.

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