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Pharmacological activities of Coconut in Metabolic Diseases: A Review

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ABSTRACT

Cocos nucifera (L.), (*C. nucifera*) Arecaceae, also called the coconut tree, is probably the widely most extensively dispersed fruit plant and supplies all the necessities of life. It is an important economic plant that feeds a million people. All the parts of coconut plant are extensively used for religious practices, culinary purposes, for making household equipment's and is also used as traditional medicine. The goal of the review is to provide an insight into its phytochemical profile and its therapeutic potential in metabolic diseases. The plant as a whole possess plethora of uses such as, neuroprotective activity, antidiabetic activity, anticancer activity, antihypertensive and lipid lowering activity. Various study reports its safety in preclinical and clinical setup.

Keywords: *Cocos nucifera*, Coconut, Chemical constituents, Pharmacological activities.

INTRODUCTION

Cocos nucifera is stated as a crucial component in the palm family Arecaceae commonly called coconut in English, nariyal in Hindi, and karýda in Greek. Other names in different languages are given in (Table 1). In India, it is well known as '*Kalpa vriksha'* which interprets as the palm which supplies all the necessities of life. It is called '*Pokok suribu gunci'* in Malaysia which means a tree of thousand years. Virgin coconut oil (VCO) is safe for human beings and is referred to as a *'Drugstore in a bottle'*. It is linked to better heart health, weight loss, and digestion Each part of *C. nucifera* like a nut, toddy, shell, water, and fiber exhibit diverse functions in religious, culinary, traditional, medicine and has significant economic value [1]. Its culinary uses include making rugs, brooms, utensils, furniture, insect repellents, dye, mouth wash, etc. All parts of the coconut tree- leaves, inflorescence, fruit, trunk, and root have varied uses. Coconut fruit is composed of about 38.5% shell, 51.7% kernel, 9.8% water. Oils removed from *C. nucifera* by the direct pressing method were used for various bone complaints because of their healing nature. It has been the primary source of essential fats and has fibre and medium-chain triglycerides, both of which have been linked to better heart health, weight loss, and digestion. *C. nucifera* is known as a thirst quencher all over the world because of its water properties. Considering the vast literature available on different parts of the coconut, varieties of coconut, the current review highlights the role of *C.nucifera* in combating various diseases. The attempt has been made to discuss various chemical constituents present in coconut and its mechanism of action.

Table 1: Common names of Coconut in various regions

Country	Name
Amharic	Kokonet, Kokas
Arabic	Jooz al-hind
Aramaic	Nargil
Armenian	Hentagagan Engouz
Basque	Koko
Caratan	Coco
Danish	Kokosnod
Georgian	Kokosi
German	Kokos
Greek	Karyda, Karida
Indonesian	Kelapa
Italian	Cocco
Japanese	Kokoyashi
Russian	Kokos, Koksovyj

The plant is an arborescent monocotyledonous, monoecious tree that grows up to 25 m in height (giant coconut) with a dense canopy. The root of the coconut system is fasciculated. The main stem is unbranched, and a tuft of leaves shields a single apical bud at the immature apex. The pinnate leaves are feather-shaped, having a petiole, rachis, and leaflets. The fully grown coconut may produce 12–14 inflorescence spikes per year under ideal environmental conditions, but the mature dwarf coconut may produce 18 spikes in the same time frame. The axillary inflorescence has globular clusters of female flowers. The fruit is made up of exterior epicarp, a mesocarp, and an endocarp. The epicarp, which is the fruit's outer skin, and the mesocarp, which is thick, fibrous, and browned when dried, while the endocarp is the stiff dark center. Inside the fruit, there is solid endosperm (solid white albumen) of various thicknesses regardless of the age of the fruit, as well as an oily pulp consistency [2].

Scientific classification

C. nucifera is distinct from other fruits and is classed based on traits such as the formation of leaves, fruits, and flowers. Since it is a flowering plant, this belongs to angiosperm or Magnoliophyta division which is divided into two major plant groups like Liliopsida (monocots) and Magnoliopsida (dicots). *C. nucifera* belongs to the Liliopsida class which is further subdivided into the order of Arecales and placed in a genus of Cocos with species Nucifera. Thus is botanically classified as *C. nucifera* [3].

Distribution

C. nucifera was first found in Southeast Asian countries such as Indonesia, Malaysia, and the Philippines also found between the Indian and Pacific Oceans on the islands. From there it has made its way to India and East Africa [4]. Later, it was distributed across the American and African continents [5].

Cultivation

In perfect weather conditions, it grows to be 100 feet tall with 13-20 feet long pinnate leaves and can produce up to 75 fruits each year. Coconut palms require moisture and warmth to thrive and produce fruit. The most favourable conditions for its cultivation are the temperature of 25°C- 30°C, a sunshine period of 1800–2000 h/year, rainfall of 1800–2000 mm/year well distributed (around 150mm/month), the relative humidity of 75% and an altitude of< 400m above sea level. It may be grown in a variety of soil types, from halophytic to mesophytic. Since coconut is perennial with a continuous flowering habit, it must maintain productivity by continuously replacing the nutrients. It has been found that coconut palms annually require 56 kg Nitrogen, 12 kg Phosphorus, 70 kg Potassium, 34 kg Calcium, and 12.5 kg Magnesium from one hectare. Potassium and nitrogen are most important and should be replaced continuously to maintain yield. Thus, are grown on loams and well-drained clays.

There are two varieties of coconut trees i.e., tall and dwarf. The tall variety germinates early as compared to the dwarf, however, tall variety show late-flowering i.e. 5-6 yrs., while dwarf variety show early flowering i.e. 3 yrs.

The stem of the coconut palm acts as an important water store or capacitor. The differentiation of a leaf to its emergence. New leaves open at intervals of about four weeks (tall) or three weeks (dwarfs). Availability of water increases the rate of frond shedding and slows the emergence of new leaves and also a close linear relationship between yield and cumulative light interception. Seasonal changes in light interception are related to water stress. An inflorescence can start growing up to 44 months before the fruit is picked. Roots are haphazard and can reach more than 2 meters deep and 3 meters lateral. Variations in monthly and annual yields are determined largely by rainfall distribution.

Chemical constituents

The crucial phytoconstituents in coconut are tannin, lignin, pentose, furfural, and cellulose [6]. Miscellaneous phytoconstituents from *C. nucifera* were found to be, phenols, steroids, flavonoids, alkaloids, triterpenes [7]. Phytochemical analysis of an ethanolic extract of coconut fiber [mesocarp] identified the presence of tannins, phenols, flavonoids, leucoanthocyanidins, steroids, alkaloids, and triterpenes, whereas butanol extract retrieved, saponins, condensed tannins, and triterpenes [8]. The ethyl acetate extracts, of *C. nucifera* fiber, are rich in polyphenols like tannins, catechins, epicatechins, and flavonoids [9]. The liquid albumen contains nicotinic acid, vitamin B, biotin, pantothenic acid, biotin, riboflavin, folic acid, with very small amounts of vitamins B1, B6, and C, folic acid, pyridoxine, thiamine, L-arginine, amino acids, plant hormones (auxin, 1,3-diphenylurea, cytokinin), enzymes (acid phosphatase, catalase, dehydrogenase, diastase, peroxidase, RNA polymerases), and growth-promoting factors [10]. The electrolyte composition of coconut water consists of Na (9.17mg/100ml), K (173.5 mg/100ml), Fe (0.15 mg/100ml), Mn (0.4 mg/100ml), Ca (47.8 mg/100g), Mg (15.01 mg/100ml), P (3.92 mg/100g). Coconut water has antioxidants which consist of esters (58.3%), ketones (33.5%), diols (8.0%). The coconut oil derived from the kernel consists of monolauric acid, lauric acid. Alcohols, lactones, aldehydes, and esters having short carbon chains were identified in the essential oil of coconut water. compounds identified in leaf epicuticular wax were lupeol methyl ether, skimmiwallin, (3b-methoxy-25-ethyl-9,19-cyclolanost-24(241)-ene), and isoskimmiwallin [3b-methoxy-24-ethyl-9,19-cyclolanost-25[251]-ene] [11]. Root phenolic compounds were identified as flavonoids and saponins [12]. *C. nucifera*is considered to be the world's principal source of lauric fat (45%- 52%). The processed compounds from it include myristic (16 %- 21%), caprylic (5%- 10%), palmitic (7%-10%), and capric acid (2%-4%), palmitoleic acid. Various types of compounds have been isolated and identified from juice and kernel such as zeotin-o-glucoside and dihydrozeatin-o-glucoside, orthotopolin, kinein, kinetin riboside. VCO contains fatty acid profiles and a higher amount of some nutrients (e.g. Vitamin E) and dietary bioactive compounds such as polyphenols. The unsaturated fats consist of oleic acid (5%-8%), linoleic acid (1%-3%), linolenic acid (0.2%). By-products of coconut, oil breakdown result in the production of medium-chain fatty acids (MCFA) and monoglycerides (MG) [13].

Pharmacological Activities of *C. nucifera* extracts and isolated constituents

Neuro-protective activity

Coconut water comprises *trans*-Zeatin which is a naturally occurring cytokine [14]. Trans-zeatin inhibits acetylcholinesterase therefore attenuate, Alzheimer's disease [AD] and other neurological disorders [15]. Another study found that trans-zeatin may suppress the creation of amyloid protein, which plays a role in the advancement of Alzheimer's [16]. The effects of young coconut juice (YCJ) in ovariectomy induced AD in rats were studied. It was observed that prolonged ovariectomy is related to a substantial loss of memory

which could be overturned by the YCJ administration. YCJ might have certain inevitable implications in the primary prevention of AD in menopausal women [17]. Coconut oil comprising of small chain fatty acid gets converted to ketone, proved to be beneficial in AD patients [18].

Anti-diabetic activity

Coconut water has preventive effects on the kidney and treats urethral stones [19]. Studies have shown the beneficial effect of coconut water on degenerated kidneys in alloxan-induced diabetes in Wistar rats. The glomerular filtration rate is influenced by diabetic nephropathy. Co-administration of coconut water reverses the diabetic nephropathy effect, as well as a considerable drop in urea potassium and bicarbonate. Arginine, a coconut kernel (CKP) was found to reduce blood glucose in alloxan-induced diabetes in rats. CKP affects blood glucose levels as well as the activity of metabolic enzymes. RAGE (receptor for advanced glycated end products) and NFkB activity were down-regulated whereas, iNOS was normal with the CKP-fed diabetic group [20]. Coconut milk exhibits a regenerative effect on pancreatic cells impaired with diabetes, while coconut water has a blood glucose-lowering impact [21]. Using isolated perfused mouse islets, VCO was shown to have an insulinogenic and hypolipidemic action. Anti-diabetic properties of VCO, as well as heat-derived virgin coconut oil (HEVCO), were studied. HEVCO was found to be more beneficial than cold extracted virgin coconut oil (CEVCO) in terms of enhancing antioxidant profile, lowering blood glucose, and lowering lipid levels. HEVCO has a higher polyphenolic profile and possibly higher nutrient bioavailability, both of which may contribute to HEVCO's improved health advantages [22]. VCO (10 mg/kg body) weight alters hyperglycemia and enhances glucose tolerance possibly by its antioxidant effect which in return results in improved insulin secretion [23].

The effects of VCO on gut flora have been studied [24] on alloxan-induced diabetic rats and to improve probiotic bacteria such as *Lactobacillus*, *Allobaculum*, and *Bifidobacterium* types, as well as many other metrics such as food fluid intake and weight increase. The therapeutic potential of mature coconut water (MCW) for its hypoglycemic and antioxidant effects has been investigated on alloxan-induced diabetes in rats [25]. Antioxidant enzyme activity was improved and lipid peroxidation levels, hyperglycemia, and oxidative stress associated with diabetes mellitus were decreased. In another study, the benefits of MCW and glibenclamide in diabetic rats against alloxan (10 mg/kg) induced diabetic rats were compared. Apart from nephrotoxicity and hepatotoxicity, MCW treatment had a favourable effect on diabetic rats [26].

Anti-cancer Activity

A study suggested the protective use of Coconut cake on 1,2-dimethylhydrazine induced colon cancer [27]. Coconut cake is high in fiber, which has a chemoprotective effect in the genesis of colon cancer by lowering -glucuronidase and mucinase levels thus lowering the activities of anaerobic bacteria. VCO intake along with chemotherapy helps enhance the performance status and quality of life (QOL) of breast cancer patients and lowered the adverse effects of chemotherapy. Consuming VCO enhances body mass index and sexual activity. According to the results obtained, there is certainly a strong possibility of using VCO as a supplement in breast cancer patients in the pursuit of a positive QOL [28]. A significant decrease in DMBA-induced mammary carcinogenesis in Balb/ A mice was studied [29]. Mucin 2, a protein that helps to maintain the overall function of an intestinal barrier. Lauric acid, triggers apoptosis in different colon cancer cells in an in vitro model, primarily via reactive oxygen species [30]. It induces cell cycle arrest in G0/G1 and G2/M phases and also ROS induced activation of Rho-associated kinase-mediated pathway and p21 dependent apoptosis [31]. The anti-apoptotic activity of VCO in the treatment of lung cancer has also been investigated. Changes of two lung cancer cell lines at different doses of VCO over 72 hours were observed. Both cell lines showed alterations such as the emergence of extensive cytoplasmic vacuolization and blebbing of the cell membrane [32]. The effects of coconut water vinegar (CWV) on 4T1 breast cancer cells were studied [33]. It inhibited the growth of 4T1 breast cancer *in vivo* via induction of apoptosis and delayed metastasis and improves the toxicity of immune cells as well as the generation of anticancer cytokines. CWV lowered the evolution of breast cancer via triggering death in breast cancer cells, inhibiting metastasis, or boosting anti-tumor resistance. Coconut kernel fraction (CKf) was reported to inhibit the proliferation of human prostate cancer cells, DU-145, *in vitro* through ROS-mitochondria-driven apoptosis. CKf contains various bioactive compounds with possible anticancer properties, including coumaric acid, myristin, chlorogenic acid, and triterpenoid methyl esters. The expression of pro-apoptotic genes Bax, Bid, Bak, and p53 was also found to be dose-dependently elevated by CKf therapy [34].

Anti-hypertensive activity of coconut

Hypertension is considered to be the main causative factor for cardiovascular complications such as coronary heart diseases, atherosclerosis, and stroke. The effectiveness of frequent use of coconut water in the regulation of hypertension was investigated and significant reductions in average systolic blood pressure were found throughout the coconut water group [35]. In male Wistar rats with NaCl-induced hypertension, the effect of coconut water on heart rate was investigated. Fourteen days of treatment with Coconut water led to lower heart rate frequency than some other isotonic drinks [36]. Hypertensive individuals were given tender coconut water, which is high in the electrolyte, for 3 months. Following 60 and 90 days, the systolic and diastolic pressure dropped significantly [37]. The vasopressor effect of ethanolic extract *C. nucifera* endocarp [CNE] was evaluated in a DOCA (deoxycorticosterone acetate) salt-induced paradigm on isolated aortic rings in rats. A drop in systolic pressure was observed which may be due to the polyphenols (vanillic acid, chlorogenic acid, and ferulic acid) found in the extract [38].

The lipid-lowering activity

In alcohol-fed (3.76 g/body weight/day) rats, the effect of coconut kernel protein on lipid metabolism was investigated.The key factor causing the hypolipidemic effect of coconut protein is a result of the high level of L-arginine [39]. Coconut water administration was found to considerably reduce hyperlipidemia in the study. Tender coconut water (TCW) had a larger lipid-lowering impact than mature coconut water (MCW). This could be due to differences inactive components, particularly L-arginine [40]. In rats fed a fat–cholesterol-rich diet, the hypolipidemic impact of coconut water (4 ml/100 g body weight) was compared to the lipid-lowering medication lovastatin (0.1/100 g diet). Coconut water has the same lipid-lowering impact as lovastatin [41]. Further, a study comparing the effects of VCO on lipid levels and lipid metabolism was performed. VCO treatment significantly lowered total cholesterol, LDL+VLDL cholesterol, Apo B, and serum triglycerides, according to the findings. HMG -CoA reductase, glucose-6-phosphate dehydrogenase, isocitrate dehydrogenase, and

malic enzyme activity were all reduced, indicating hepatic lipogenesis. Furthermore, VCO boosted the activity of lipoprotein lipase, lecithin cholesterol acyltransferase, and bile acid production. Data show that VCO has a hypolipidemic effect by controlling lipid production and breakdown [42].

Diuretic activity of Coconut

An Ayurvedic formulation, coconut husk *Mashi,* was prepared by *Anterdhum Padhati* (APM; the process by which coconut husk is packed in between 2 earthen posts which is sealed by laomy clay and is subjected to heating for 50 minutes) and *Bahirdhum Padhati* (BPM; dried coconut husk was heated in earthen pot at 145 to 155 °C with continuous stirring till white fumes ceases to come out). BPM showed a dose-related diuretic effect at 500 mg/kg and caused a marked increase in NA^+, K^+, Cl^- levels. It could be inferred that BPM caused diuretic action as well as a rise in electrolyte concentration [43]. The diuretic action of *Bahirdhum Padhati Mashi* (BPM) at different doses (100, 200, and 400 mg/kg) in normal rats was investigated. The maximum dose of BPM (400mg/kg) increased urinary output and may infer that continuous treatment with BPM caused diuresis and increased electrolytes urine output in rats [44]. *C. nucifera's* diuretic potency was tested in albino rats at three distinct doses: 100 mg, 200 mg, and 300 mg/kg body weight. A dose-dependent increase in total urine volume and electrolyte output was observed [45].

Anti-oxidant activity

Coconut milk is made by compressing coconut meat mechanically or manually, with or without the addition of water. coconut milk has the highest antioxidant activity. The study proved that coconut milk does have a high protein content while being low in fat and thus could be a promising food element for reducing the risk of degenerative disorders and preventing oxidative damage [46]. The antioxidant effects of VCO obtained through cooling and fermenting were examined. The VCO produced through fermentation showed the most strong 1,1-diphenyl-2-picrylhydrazyl scavenging action as well as increased antioxidant activity. The most prevalent phenolic acids discovered have been ferulic acid and p-coumaric acid [47].

Immunomodulatory activity of Coconut

In Swiss albino mice, the immunomodulatory action of coconut protein was investigated by suppressing the immune system with cyclophosphamide (CP). In CP-treated mice, oral feeding of coconut protein raised RBC, WBC, and platelet counts. As a result, coconut protein does have an immunostimulatory impact in terms of IgE-mediated hyperreactivity as well as cell-mediated hyperreactivity [48]. VCO inhibits *S. aureus* growth and enhances the ability of phagocytic immune cells to attack *S. aureus* produced from goat milk. VCO can be utilized as an antibiotic replacement and a cellular immune system modulator. The medium-chain fatty acids in VCO help to improve phagocytic activity [49].

The anti-stress activity

The study evaluates the antistress and antioxidant effects of VCO (10 ml/kg) body weight*in vivo* using mice with stress-induced damage. VCO was able to reduce immobility time and restore oxidative stress in mice and exhibited higher amounts of brain antioxidants, lower levels of 5-hydroxytryptamines in the brain, and smaller adrenal glands. Serum cholesterol, triglyceride, glucose, and insulin levels all dropped as a result. These results supported VCO's use as a stress-relieving functional oil. The potential anti-stress impact could be attributed to the polyphenols and medium-chain fatty acids contained in VCO [50].

Renal protective activity

The study tested the effect of coconut water as a prophylactic agent in a rat model of experimentally induced nephrolithiasis. Treatment with coconut water reduced the number of crystals in urine while inhibiting crystal accumulation in renal tissue. Furthermore, coconut water protected the kidneys from decreased renal function and the production of oxidative stress. The findings suggest that coconut water may be used for urolithiasis phytotherapy [51].

Effect on weight loss

The effect of dietary medium- and long-chain triacylglycerols (MLCT) on the aggregation of body fat in healthy humans was investigated. The consumption of the MLCT diet daily can result in a reduction in body weight and fat accumulation. In a 12-week double-blind trial, volunteers consumed research bread with 1.7 g MCFA daily at breakfast, while the control group ate bread made with long-chain triacylglycerols (LCT). In the research group, there was a substantial reduction in body weight and fat mass, as well as a significant reduction in serum total cholesterol. Another research looked at the impact of coconut oil supplementation on the biochemical and anthropometric profiles of women [52]. The pharmacological uses of *C. nucifera* is shown in Figure 1.

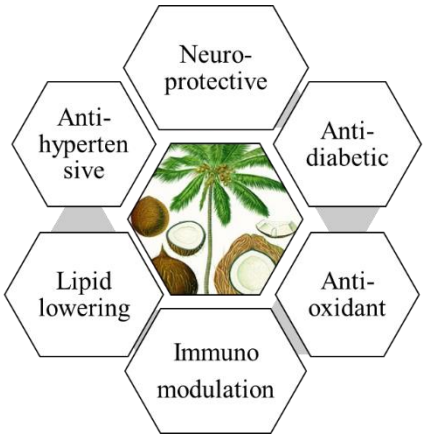

Figure 1: The various pharmacological properties of *C. nucifera*

Toxicity and safety Profile

Many researchers explored the noxious properties of *C. nucifera*. A toxicity study considered the efficacy of ethyl acetate extract of *coconut* fiber on topical inflammation and physiological parameters by a xylene-induced animal model. Concerning the physiological parameters and macroscopical aspects of the lymphoid organs in this trial, neither mortality nor any symptom of toxicity was seen in the animals. The probable toxic effects of crude extract, of *C. nucifera* mesocarp, were assessed in mice. An oral administration of single-dose (500mg/kg) over 5 days caused no behavioral changes and No injury or bleeding stomachs were observed. Methanolic extract of *C.nucifera* endocarp was evaluated for its toxicity [54]. A single dose (5000mg/kg) orally was given to 5 male and 5 female mice and there wasn't any indication of toxicity and mortality in the two groups. All animals gained weight over the testing period. Acute, subchronic, and chronic toxicity from liquid mesocarp of green coconut (LMGC) and butanol extract obtained from the LMGC were examined in mice and

rats [8]. No acute lethal effects were seen in mice at a dose of 3000 mg/kg orally of either extract. In However, no animal survived on intraperitoneal administration of LBGC and butanol extract at a dose of 500 and 700 mg/kg. In subchronic toxicity tests, the rats treated with LBGC had significantly higher white blood cell, neutrophil, red blood cell, hematocrit, and platelet counts. In the chronic toxicity test, the group treated with LBGC showed higher values for neutrophils, white blood cells, basophils, and platelets ($P < 0.05$). However, in the subchronic and chronic toxicity tests, no hematological parameters differed significantly in the group treated with butanol extract ($P > 0.05$). Only triglycerides were higher ($P < 0.05$) in the group treated with LBGC during the chronic toxicity test. Rats treated with both extracts had no histopathological changes related to toxicity, nor did weight gain differ between treated and control groups ($P > 0.05$). In conclusion, both extracts showed low toxicity for these parameters.

CONCLUSION

C. nucifera is a widely scattered plant that has significant pharmacologic properties and low toxicity. The pharmacologic activity varies by the plant part used. Coconut water emerges as a protectant, e.g., on the heart and kidney, and shows antioxidant activity, also hypoglycemic effect. Few limitations on the research of *C. nucifera* must be acknowledged. Primarily, the studies have concentrated on the activity of various plant parts but the mode of action is not known. Later, preparations of the plant parts should be designed to perform clinical trials. Taking into account the variety of pharmacological properties, future investigation into *C. nucifera* should be promoted. The main aim must be to separate particular composites, to explicate the mechanisms concerned with the pharmacological effects, and to study the possible toxic action to establish safe phytotherapy. Many factors can restrict such research such as environmental and recurrent alterations amongst countries and regions that can affect the chemical composition. This review may facilitate the research community to focus on *C. nucifera*-mediated biological applications in the future.

Conflict of Interest

None declared.

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