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Role of Coconut Oil (*Cocos nucifera*) in Modifying Cancer Risk: A Systematic Exploratory Review of Randomized Controlled Trials

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ABSTRACT

Background: Natural methods and substances as cancer protective aids is taking big stage in current research. Polyphenol rich coconut oil was proved to offer many health benefits. The effect of medium chain fatty acids on cardiovascular health is very well established. But the scientific evidence about the effectiveness of coconut oil in cancer prevention and as supplemented adjuvant in cancer treatment is scarce.
Aim: To evaluate the anti-cancer effects of coconut oil.
Objective: To determine various beneficial effects of coconut oil that may alter microenvironment of human body that can render cancer preventive effects.
Methods: Systematic search was conducted following PRISMA guidelines to include articles that evaluated the effect of coconut oil in cancer prevention in scientific websites. Quality of the studies was independently assessed by all authors. Bias was assessed by The Office of Health Assessment and Translation (OHAT) bias assessment tool for human and animal studies.
Results: 10 studies in total were included in the systematic review from which data is extracted, clustered and tabulated. Virgin coconut oil (VCO) supplementation resulted in antioxidant, anti-inflammatory effects promoting good gut microbial growth and reducing total cholesterol, and reducing insulin resistance.
Conclusion: VCO may provide beneficial effects in cancer prevention by correcting the microenvironment of human body which needs to be established by further evidence from large scale human clinical trials.
Keywords: Virgin coconut oil, anti-inflammatory, Oxidative stress, Insulin resistance, Quality of life.

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INTRODUCTION

The raising burden of cancer cases around the world is of major concern. About 9.7 million deaths from cancer worldwide and 20 million new cases were recorded in 2022. Precisely in low- and medium-income countries, the number of cancer cases is expected to rise to 35 million by 2050, with 9.7 million fatalities and 20 million new cases expected in 2022. It's estimated that 1 in 9 males and 1 in 12 women will pass away from cancer [GLOBACAN]. Cancer therapy with conventional treatment modalities and cancer chemoprevention with synthetic drugs and compounds are on the continuum of research with the recent addition of natural products. Natural goods are thought to include a vast array of bioactive substances with potential applications in medicine. Natural items accounted for almost 25% of all recently authorized anti-cancer medications.[1] Dietary lipids derived from plants like medium-chain triglycerides (MCTs) and low-chain triglycerides that are available in coconut and palm oils continue to be one of the most fascinating and intricate groups of biological substances with distinct physico-chemical characteristics and possible health advantages. Their unique qualities set them apart from other saturated fatty acids and make them an important part of nutrition. The effectiveness of such compounds is found in the treatment of gastrointestinal problems, neuroprotection, antibacterial, and anticancer actions.[2] The fatty acid profiles of the two primary forms of coconut oil, virgin coconut oil (VCO) and copra oil (CO), are comparable; however, the latter has higher concentrations of certain nutrients and dietary bioactive substances. Caffeic acid, syringic acid, p-coumaric acid, vanillic acid, and ferulic acid are the principal phenolic acids present in VCO in increasing order of concentration. Additionally, VCO has higher concentrations of vitamins A and E as well as flavonoids, specifically flavanones and dihydroflavonols.[3] A significant component of coconut oil, lauric acid, has the ability to affect a variety of processes, including inflammation, angiogenesis, metastasis, tumor start, migration, and invasiveness. impact the survival of HepG2, intestinal, breast, endometrial, and oral cancer cells. Induce downregulation of EGFR signaling, upregulation of the CDK inhibitor p21Cip1/WAF1 via the PI3K/AKT pathway, and death of cells.[4] The evidence from in vitro and in vivo studies indicates potential anti-cancer benefits. But there exists a research gap in the literature in translating such benefits for the human population. The present study explores studies that investigated anti-cancer effects both direct and indirect of coconut oil with the aim of reviewing the scientific literature systematically.

MATERIALS & METHODS

Search strategy: We conducted a thorough search of the databases in PubMed, Elsevier Science Direct, and Wiley Online Library, Google scholar using the MeSH phrases "coconut oil (cocos nucifera)" and "cancer prevention". We followed PRISMA guidelines to conduct the search.

Study selection: Eligibility criteria

Criteria for inclusion were

(i) Original research articles published in the past 20 years.

(ii) Articles for which full text is available.

(iii) Articles testing the relationship between coconut oil and cancer.

Criteria for exclusion were

(i) Studies available in languages other than English.

(ii) Review articles

(iii) Articles those are available only as abstracts.

Quality assessment: All Authors evaluated the quality of the study for appropriate search methods, bias, and standardization in a methodical and independent manner. The Office of Health Assessment and Translation (OHAT) bias assessment for

animal and human studies was employed to evaluate bias.[5]

Data extraction: Data on authors, year, location, samples, study design, exposures, intervention, results, and outcome were extracted and then combined into a narrative synthesis, which was then tabulated and grouped.

RESULT

Search results: A thorough investigation of online databases produced 9,623 research publications about coconut oil and cancer. Following the elimination of duplicate entries, 4,471 unique articles have been identified. After assessing these papers in accordance with pre-established eligibility standards, 136 articles were ultimately chosen for quality evaluation. 10 of those publications passed the strict requirements and were added to the current systematic review [fig 1]. Data relevant to included studies was extracted and tabulated [table 1]. Bias assessment of included studies indicated that all studies except two could be categorized as low and medium risk of bias. [7, 9]

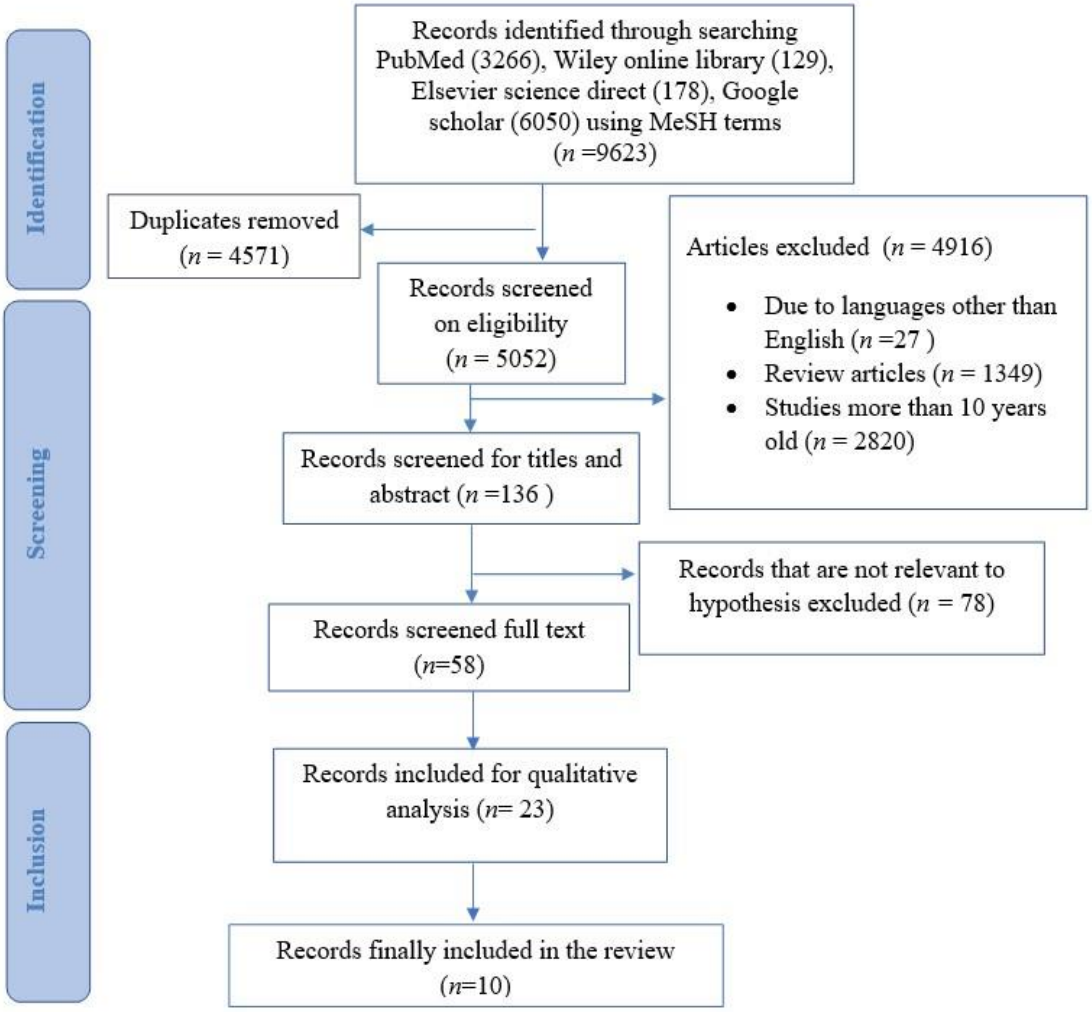

Figure 1: PRISMA flow chart for inclusion of studies.

Table 1: Characteristics of the studies included.

Author, year, area	Design	Sample	Technique	Result	Outcome
Bilge Meral Koc et al. [6] 2022 Istanbul	RCT Cross over design	44 adults, 19–30 years old with BMI 25-29.5 kg/m^2 Divided into 2 groups .	Phase 1: 4 weeks: Group 1 (n = 21) was given 20 mL/day (2 tablespoons per day with coffee and/or tea) of coconut oil in addition to energy containing diet. Group 2 (n = 23) was only given the energy-containing diet. Excretory phase: 2 weeks: no intervention. Phase 2: 4 weeks: Crossed over the intervention.	When subjects were given solely coconut oil, their levels of irisin considerably dropped ($p < 0.05$). The subjects' body weight, BMI, and body fat % significantly decreased ($p < 0.01$). All subjects had significant reductions in their levels of insulin, total cholesterol, LDL, and triglycerides ($p < 0.05$). Body weight loss did not significantly affect the irisin level ($p < 0.05$)	Coconut oil was found to be associated with the hormone irisin. Coconut oil reduced the level of hormone irisin in individuals with overweight.
Elahe Mansouri et al. [7] 2024 Iran	RCT	48 adults with Metabolic syndrome aged 20–50 years.	Control group: usual diet Intervention group: 30 ml of VCO daily to substitute the same amounts of oil in their usual diet for 4 weeks. Total antioxidant capacity (TAC), malondialdehyde (MDA) as well as HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) and QUICKI (Quantitative Insulin Sensitivity Check Index), serum BDNF (neurotrophic factor) were outcome measures.	In comparison to the control group, VCO consumption significantly elevated serum TAC ($p < .01$) and QUICKI index ($p = .01$) and significantly decreased serum levels of MDA ($p = .01$), fasting insulin ($p < .01$), and HOMA-IR index ($p < .01$). When comparing the VCO group to the baseline, serum BDNF levels rose significantly ($p = .02$); however, this change was not statistically significant when compared to the control group ($p = .07$).	Adults with Metabolic syndrome who consumed VCO showed improvements in their oxidative stress state, insulin resistance, and BDNF levels.
Margaret Harris et al. [8] 2017 Colorado Springs	RCT cross over design	12 postmeno-pausal women (45–65 years old) not under hormone replacement therapy, or medicine.	Intervention group and control groups were crossed over with supplementation of 30 mL VCO or safflower oil (SO) for 28 days, with a 28-day washout. LDL, HDL, TC, inflammatory markers were outcome measures. Paired t-test, repeated measures ANOVA	VCO markedly increased LDL (+13.5 – 16.0 mg/dL), HDL (+6.6 – 7.5 mg/dL), and TC (+18.2 – 22.8 mg/dL). Lipid levels were not appreciably altered by SO. One individual experienced elevated inflammation and unfavorable reactions to VCO. IL-1β was lowered by VCO for every subject. Each of the other cytokines was affected differently by VCO and SO.	The study showed VCO had neutral effects on body composition. VCO may be anti-inflammatory for some people.

Imelda Angeles-Agdeppa, et al. [9] 2023 Philippines	RCT	63 participants over the age 20 with a history of hypertension and slightly elevated liver enzymes	Intervention group: VCO and diet for 28 days. For day 1 to 3 the added VCO was 0.6 mL per kilogram body weight (kg BW) and was increased to 1.2 mL/kg BW for day 4 to 28. Control group: diet alone ANOVA, t tests.	Intervention group subjects showed a significant reduction in the C-reactive protein level, with the mean CRP level normalized to ≤ 5 mg/dL on the 14th day of the intervention.	VCO could be used as an adjunct supplement to probable and suspect cases of covid 19 due to anti-inflammatory properties.
Ademola C. Famurewa et al. [10] 2016 Nigeria	In vivo controlled trial.	24 Male Wistar rats weighing 100 to 120 g divided into 4 groups 6 per group	Group 1 were fed with basal diet Group 2 with basal diet + intra peritoneal Methotrexate (MTX) (20 mg/kg bw) administered on day 10 (to induce hepatotoxicity). Group 3 with 5% virgin coconut oil (VCO) and basal diet + MTX, Group 4 with 15% VCO basal diet + MTX. After 12 days liver damage is assessed by liver serum markers and hepatic levels of malondialdehyde, reduced glutathione, and antioxidant enzymes. Histopathological examination of the liver tissue was also performed. Statistical analysis was done by ANOVA followed by Tukeys' Post Hoc Test.	**Body weight and liver weight**: non-significant difference ($p>0.05$) **Hepatotoxicity**: VCO diet before MTX administration had ALT, AST and ALP activities significantly lower compared to MTX group ($p < 0.01$) and significant increases in serum ALB and TP levels seen in VCO diet group in comparison to MTX group ($p < 0.01$). **Oxidative stress**: VCO + MTX groups significantly prevented decrease in SOD, CAT, GPx and GSH in the liver compared to MTX group ($p < 0.01$). Hepatic MDA content was markedly lower in VCO group compared with MTX group ($p < 0.01$). **Liver architecture:** VCO diet prevented the degenerative and inflammatory histopathological alterations.	The study indicated the hepatoprotective effect of VCO against MTX-induced hepatic damage via inhibition of oxidative stress, lipid peroxidation and improving antioxidant enzyme activities. These beneficial effects may be due to bioactive polyphenols available in naturally obtained virgin coconut oil.
Ademola C. Famurewa et al. [11] 2017 October Nigeria	In vivo RCT	24 Male Wistar rats 7 weeks old, weighing 100 to 120 g randomized into 4 groups 6 per group.	Group 1: Control group, fed with clean water 5mg/kg. Group 2: MTX (20 mg/kg, intraperitoneal) on day 14 to induce renal damagea Group 3: 5% VCO for 17 days and MTX on day 14 Group 4: 15% VCO for 17 days and MTX on day 14 Three days post-MTX injection, kidney function was assessed by measuring serum kidney markers,	**Body weight and liver weight**: non-significant difference ($p>0.05$). **Renal dysfunction**: serum urea, creatinine and uric acid reversed the renal dysfunction ($P < 0.05$). 15% VCO diet markedly reduced creatinine level than 5% VCO diet. **Oxidative stress:** dietary supplementation of VCO (5% and 15%) significantly increased the renal activities of the enzymes (SOD, CAT,	VCO may offer nephroprotective effects through antioxidant and anti-inflammatory properties that might have resulted from polyphenols present. VCO may offer potential benefits for cancer patients undergoing MTX chemotherapy to protect against kidney injury.

			renal antioxidant enzyme activities, GSH, lipid peroxidation, inflammatory markers (interleukin-6, nitric oxide, C-reactive protein), histopathological changes. Statistical analysis done with ANOVA followed by post-hoc Tukey test.	TBARS) compared to the MTX-treated group ($P < 0.05$). **Inflammation**: pretreatment with VCO diet prominently reduced ($P < 0.05$) the renal proinflammatory markers in comparison to MTX-treated rats. IL-6, CRP and NO levels between the VCO +MTX groups were comparable, and to normal control ($P > 0.05$). **Histopathology:** pretreatment and concurrent VCO supplementation rats restored MTX-induced histopathological alterations dose-dependently.	
Lilis T, et al. [12] 2017 North Sumatra, Indonesia	In vivo Post-Test Only Control Group design.	A total of 24 Male Wistar rats weighing 150 to 200g split into 4 groups 6 each.	Group 1: Control group, basic diet. Group 2: basic diet and exposed to cigarette smoking. Group 3: basic diet and 0.45 ml VCO and cigarette exposure. Group 4: basic diet and 0.9 ml VCO and cigarette exposure. The cigarette smoke exposure was simulated using a modified smoking pump, with rats exposed to smoke for 28 days, during which VCO was administered to Groups 3 and 4. At the end of the study, blood samples were analyzed for SGOT and SGPT levels using a spectrophotometer. Statistical analyses were performed using one-way ANOVA and Tamhane's Post Hoc Test.	The study evaluated liver function in Wistar rats by measuring ALT and AST levels. Results indicated that rats exposed to cigarette smoke (group P2) had significantly elevated ALT and AST levels, indicating liver dysfunction. In contrast, rats receiving only standard feed (group P1) had the lowest ALT levels. VCO supplementation (groups P3 and P4) effectively reduced ALT and AST levels compared to the cigarette smoke-exposed group, with the 0.9 ml VCO dose (group P4) proving most effective in preventing liver abnormalities. This suggests that VCO can mitigate liver damage caused by cigarette smoke, with higher doses providing enhanced protection.	The current study highlights that smoking induces liver damage and confirms the hepatoprotective effects of Virgin Coconut Oil (VCO) against this damage. VCO appears to mitigate smoking-induced hepatic damage by regulating oxidative stress. The study suggests that bioactive polyphenols in VCO may contribute to its beneficial effects on liver health.
Sinisa Djurasevic et al. [13] Serbia	In vivo RCT	Male Wistar rats 2.5 months old into 4 groups	**Control group:** std food **VCO group:** std food plus cold pressed 20% concentrated VCO. **Aloxan group:** std food, and single I.P. injection of alloxan on day 1	When compared to the control group, the VCO group's intake of food and water, glycaemia, and body mass gain are all considerably increased by supplementing with coconut oil.	VCO did not decrease the diabetes-induced hyperglycemia but increased the abundance of gut probiotic bacteria.

			Aloxan and VCO group. Stool samples, DNA isolation, 2-way ANOVA with Tukey's post hoc test.	Glycaemia did not significantly differ between the Alx and Alx + VCO groups. The fecal microbiota was significantly impacted by virgin coconut oil, resulting in a notable rise in the quantity of probiotic bacteria, including Lactobacillus, Allobaculum, and Bifidobacterium species.	
Alana A. Arnone et al. [14] 2024 North Carolina.	In vivo RCT	90 female 3-week-old BALB/c mice divided into 6 groups 5 in each.	(n ¼ 15 per group), 10% fat/10% sucrose control diet (Control; TD.08806), 10% fat/60% sucrose diet [HS; TD.160065], 60% kcal from fat/10% sucrose lard-based diet (Lard diet; TD.06414), **60% kcal from fat/10% sucrose coconut oil-based diet [CO; TD.08500]**, 60% kcal from fat/10% sucrose lard + flaxseed oil-based diet (FO); TD.160066], or 60% kcal from fat/10% sucrose lard + safflower oil-based diet (SO); 160067.	Food changed the fecal micro-biome populations and controlled macrophage infiltration of the mammary gland. Media conditioned by feces changed immunity and polarity of macrophages.	Coconut oil supplementation showed neutral evidence and so inconclusive in reducing the risk of progression in breast cancer.
Kim Sooi Law et al. [15] 2014 Malaysia	RCT	60 patients with stage III and IV breast cancer randomized in 2 groups.	Intervention group (n = 30) were given VCO (10 ml twice daily) after 1 week each chemotherapy cycle from the third until the sixth cycle. Patients in the control group did not receive any supplement. QOL was assessed throughout six cycles of chemotherapy using a validated Bahasa Malaysia version of the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ-C30) and its breast cancer-specific module (QLQ-BR23)	Significant differences in mean scores were seen between groups for both functional and global QOL ($\alpha < 0.01$). Additionally, the intervention group scored higher.	The functional status and overall quality of life (QOL) of individuals with breast cancer improved when VCO was consumed during treatment. Furthermore, it lessened the symptoms associated with the adverse effects of chemotherapy.

Table 1: study characteristics. VCO=virgin coconut oil; LDL=low density lipoprotein; HDL=high density lipoprotein; TC=total cholesterol; ALT=alanine transaminase; AST=aspartate transaminase; ALP=alkaline phosphatase; CAT=catalase; GPx=glutathione peroxidase; GSH=reduced glutathione; QOL=quality of life.

Table 2: Bias assessment for included studies using OHAT bias assessment tool.

Author Name	Bilge Meral Koc et al. [6]	Elahe Mansouri et al. [7]	Margaret Harris et al. [8]	Imelda Angeles-Agdeppa et al. [9]	Ademola C. Famurewa et al. [10]	Ademola C. Famurewa et al. [11]	Lilis T et al. [12]	Simisa Djurasevic et al. [13]	Alana A. Arnone et al. [14]	Kim Sooi Law et al [15]
Randomization	++	++	++	++	-	++	--	++	-	++
Allocation Concealment	-	-	-	-	-	-	-	-	-	+
Comparison Group	--	++	++	++	++	++	++	++	-	++
Confounding And Modifying Variables	-	-	--	-	-	-	-	--	+	-
Identical Study Groups	++	++	++	+	++	++	++	++	-	++
Blinding Of Participants and Personnel	-	-	-	++	-	-	-	-	-	--
Incomplete Outcome Data	-	-	++	+	-	-	-	-	-	-
Exposure Characterization	++	++	++	+	++	++	++	++	++	++
Outcome Assessment	++	++	++	+	++	++	++	++	++	++
Reporting Of Outcome	++	++	++	+	++	++	++	+	+	+
Other Potential Threats	-	-	+	-	-	-	-	-	-	+

Table 2: OHAT bias assessment results for included studies. Definitely Low risk Probably Low risk Probably high risk Definitely high risk

Study results:

Randomized controlled trials that intervened with virgin coconut oil supplementation indicated that coconut oil intake reduced the level of irisin hormone, total cholesterol, significantly reduced body fat[1] whereas changes in LDL, HDL is not favorable.[3] The supplementation also resulted in improvement in total antioxidant capacity, decreased serum MDA levels and reduced fasting insulin levels.[2] The supplementation is found to reduce interleukin 1 beta among all the participants significantly.[3] Also, chronic systemic inflammatory marker CRP levels was found to be normalized by dietary intervention with coconut oil among patients infected with covid 19.[4] Intervention with coconut oil among breast cancer patients was found to be effective in improving the quality of life significantly.[10] Animal studies conducted intervening with coconut oil indicated that it is effective in reducing hepatotoxicity, oxidative stress and prevented liver damage, inflammation, real dysfunction due to adverse effects of cancer chemotherapy[5, 6]. It was also tested to be effective in reducing hepatotoxicity caused

by cigarette smoking by normalizing the levels of liver functional enzymes.[7] The coconut oil supplementation also found to be encouraging the abundance of good gut microbiota like Lactobacillus, Allobaculum, Bifidobacterium species and it also increased the permeability and macrophage infiltration thereby supporting immunity [8, 9].

DISCUSSION

In the current scenario of increasing cancer burden, the constant search for protective factors that can modify risk factors is of utmost importance. Prevention of cancer took its big stage in current research. Microenvironmental changes in human body like chronic systemic inflammation, inadequate immune response, dysbiotic microbial environment, obesity, inflammatory bowel disease, insulin resistance, dyslipidemia and lipid peroxidation, oxidative stress, toxicity, metabolic syndrome, hormonal imbalances might lead to tumorigenesis.[15] Addressing and correcting such changes can prevent the initiation of cancer. Diet can be used as a natural and safe method to halt such changes and normalize the physiological systems. Coconut oil is one such compound with low and medium chain fatty acids. The role of coconut oil in health and disease is well explored and recently supplementation with virgin coconut oil is tested in cardiovascular disease elaborately. But evidence regarding the role of coconut oil in cancer incidence and treatment is scarce. The present systematic review of 10 research studies intended to find evidence regarding such role of coconut oil in prevention of cancer.

The study by Bilge Meral Koc et al indicated that VCO supplementation reduced Irisin hormone, reduced insulin and cholesterol levels, among overweight individuals. Irisin elevation, insulin resistance is seen in case of tumor environment. [16, 17] Another study by Elahe Mansouri et al indicated that VCO reduced oxidative stress, decreased MDA and fasting insulin levels, and increased BDNF levels. Antioxidant capacity of human body is responsible to fight DNA damage due to free radicals and prevent the initiation of cancer due to DNA damage.[18] Margaret Harris et al tested the anti-inflammatory effect of VCO among post-menopausal women and found that VCO was able to reduce the level of IL-1β. Numerous solid tumors, such as melanoma, colon, lung, breast, or head and neck cancers, have been found to have elevated levels of IL-1β, which is linked to a worse prognosis. For certain cancer forms, its role in carcinogenesis is well-described; however, its implications for other types of cancer are less clear.[19] A similar anti-inflammatory effect of VCO was seen by reduction of levels of CRP among Covid-19 patients in a study conducted by Imelda Angeles-Agdeppa, et al. VCO supplementation by Ademola C. Famurewa et al indicated the alleviation of adverse effects caused due to chemotherapy. Similarly, it was found that VCO supplementation improved QOL among breast cancer patients in a study conducted by Kim Sooi Law et al. Microbial environment was found to be positively affected by growth of good bacterial species when animals were fed with VCO according to Sinisa Djurasevic et al. Gut microbiome as well as oral microbial environments are very important in health and disease. Chronic dysbiosis can lead to tumor initiation especially in gastrointestinal system. [20, 21] A study by Lilis T, et al confirms the hepatoprotective effects of VCO among animals that were exposed to cigarette smoke which indicated its protective effect against hepatic damage caused by smoking.

The researchers from all studies used naturally extracted virgin coconut oil for supplementation for a duration of at least 4 weeks. Other forms of coconut oil is not as effective as VCO in providing such health benefits. The beneficial effects of VCO were due to medium chain fatty acids, lauric acid, polyphenols contained in it. To be established as a cancer protective agent,

VCO should be further tested in large scale human randomized controlled trials. Further studies also should investigate the effect of different dosages, duration of intake on human microenvironment. The present study had several limitations the most important being less number of included studies.

CONCLUSION

In conclusion, coconut oil is found to be effective in modifying microenvironments of human body that may lead to initiate carcinogenesis as well as in improving quality of life among patients undergoing chemotherapy. But further evidence from large scale randomized controlled studies is needed to establish the above benefits of coconut oil.

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