Potential Antidiabetic and Antioxidant Effects of Coconut Oil on Streptozotocin-Induced Diabetes in Male Sprague-Dawley Rats

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ABSTRACT

Objectives: This research aims to examine the antidiabetic effect of virgin coconut oil in STZ-induced diabetes and assess its efficacy in alleviating renal and hepatic diseases associated with diabetes.**Material and methods:** Thirty-six male Sprague-Dawley ratswere used in this study. After induction and confirmation of diabetes mellitus (DM) usingSTZ, the diabetic rats were divided into four groups (n=6); the untreated diabetic control group (DC),the metformin-treated diabetic group (DM),the coconut oil-treated diabetic (DCo) group and coconut and Metformin-treated groups (DCoM). In addition to a normal control group (n=6).Biochemical assessment of fasting blood glucose level, liver andkidney functions, lipid profile tests, insulin, glucosetolerance test and glycated HB were all performed.**Results:** Blood glucose level and glycated hemoglobin level significantly reduced,whilst insulin level was significantly elevated in groups treated with either VCO alone or in combination with metformin compared to thecontrolgroup. Glucose homeostasis was improved in coconut treated rats as evidenced by the oral glucose tolerance curve. Although the effect of coconut on controlling the lipid profile was not directly observed in this study, its renoprotective and hepatoprotective effects were evident biochemically. **Conclusion:**Virgin coconut oil possesses antidiabetic effect either alone or in combination with metformin in an animal model of type II diabetes mellitus. It improves the undesirable impact of diabetes on both liver and kidney. These results are promising in terms of the use of virgin coconut oil to reduce the dosage of synthetic drugs as well as to mitigate undesirableeffects to the liver and kidneys.

Key Words: *Coconut oil, diabetes, virgin coconut, oxidative stress, ROS, antioxidants.*

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INTRODUCTION

Diabetes mellitus (DM) is a widespread metabolic disease worldwide. In DM, the underlying feature is the hyperglycemia which is associated with many changes in lipid and protein metabolism and resulted in many long-term complications.[1]

Several studies documented that combination of herbal products with synthetic drug is useful comparing to single treatment as observed in various pathological situations. [2] As more as 400 plants have been assessed for their therapeutic effects and been utilized in controlling blood glucose.[3] Metformin, one of the efficient therapeutic drugs to lowering blood sugar was synthesized from herbal prototype molecule of French Lilac*(Galega officinalis)*. [4]

Cocos nucifera, belonging to family the *Arecaceae*. *C. nucifera*, is one of the favorable herbs to treat high blood sugar.[5]Coconut oil is predominantly used in the tropical and subtropical regions. Coconut oil has been

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well-known for its medicinal and nutritional values. Studies on the biological effects of coconut oil have shown that it ameliorates oxidative stress by enhancing antioxidant defense system, helps in weight loss and increases thyroid function. [6,7] It possesses many useful effects like antitumor, bactericidal, antihelminthic, antioxidant, vasorelaxant and antihypertensive.[8]

The fresh endosperm of the coconut fruit produces virgin coconut oil (VCO) by a "wet" extraction process.[9]The mode of extraction of VCO makes it more useful compared with copra oil, the other type of coconut oil. No chemicals or heat application are utilized through its extraction process. Thus, the activity of polyphenols is retained which play essential role as antioxidants.[6]

Streptozotocin (STZ)-induced high blood sugar in rats, and therefore is commonly used to assess the antidiabetic activity of either natural or synthetic compound. STZ damages the islets of Langerhans beta cells and results in high blood glucose. It also changes normal metabolism, serum glucose, serum insulin, C-peptide in diabetic rats. [10]Therefore, this study aims to study the antidiabetic effect of coconut oil in STZ-induced diabetes and assess its ability to ameliorate the renal and hepatic afflictions.

MATERIALS AND METHOD:

Virgin Coconut Oil (VCO): Coconuts were purchased from the local markets in Jeddah. The nuts were broken, and its meat was scrapped from the shell and cut into small pieces. The pieces were then grinded in an electric blender with distilled water, squeezed through cheese cloth to obtain coconut milk. Coconut milk was transferred to glass container and left overnight to allow the coconut milk and oil to separate into a layer of curd which appears at the top of the container. The container was refrigerated overnight so that the oil could harden. Thereafter, the pure virgin oil was separated out and stored at 4^0C for use in further experiments.

Drugs:

STZ (Sigma Chemical Company). Metformin hydrochloride (SPIMACO,SAUDI ARABIA). It was dissolved in distilled water including 0.9% (w/v) sodium chloride for oral administration.

Animals:

This study was approved by the biomedical research ethics committee at the faculty of Medicine, King Abdulaziz University (KAU). Thirty-six male Sprague-Dawley rats weighted from 200 to 250 g were obtained from the animal house of King Fahd Medical Research Center (KFMRC), KAU, Jeddah, Saudi Arabia. The rats were left to acclimatized for the laboratory condition for two weeks before starting the experiment. Six rats were assigned as non-diabetic control group (NC) (n=6) and were given daily normal saline. The other thirty rats were injected intraperitoneally with STZ at the dose 40 mg/kg. It was freshly dissolved in 1 ml of 0.05 M citrate buffer (pH 4.5) immediately before used, followed by 10% w/v fructose solution *ad libitum* for 3 days.[11] On the fourth day, blood glucose level was assessed and rats with blood glucose level 250 mg/dl or higher was considered diabetic and were divided into 4 groups (n=6); the untreated diabetic control group (DC) that received normal saline orally,the metformin-treated diabetic group (DM), that daily received metformin at a dose of 300 mg/kg BW [12] through oral gavage for two weeks then the dose of metformin was increased to 500 mg/kg BW[13] for four weeks. The fourth group was the coconut oil-treateddiabetic (DCo) group that received orally coconut oil (1ml /100g BW/day). The fifth group included diabetic rats treated withcoconut oil and metformin (DCoM). They received coconut oil orally for six weeks at the same dose mentioned before and they were treated daily with metformin with the same methods described before for 6 weeks.

Procedure:

The body weight and fasting blood glucose levels were measured weekly during the experiment. At the 30^{th} day of the experiment, the rats were fasted for sixteen hours and given 2 g/kg body weight of D-glucose (200 mg/ml) orally [14]. The blood glucose concentration was measured at 0, 30, 60, 90, and 120 min periods after the administration of the glucose load. Blood glucose concentration was measured using Blood Glucose Monitoring System (Contour). Blood was collected from the tip of tail at the specific time patterns.

At the end of the experiment, rats were fasted for thirteen hours with free access to water before blood collection. Blood was collected from the intraorbital sinus of the rats, using 75 mm heparinized microhematocrit tubes (Clay Adams, Parsippany, New Jersey, USA) under ether anesthesia. The blood was withdrawn into a plain tube for serum preparation and into a lithium heparin tube for plasma preparation. Then centrifuged using the tabletop centrifuge (Sigma, USA) at 4000 rmp for 10 min. The serum and plasma were kept at -80 until needed for biochemical analysis**.**

Biochemical assessment:

Biochemical parameters including blood glucose level, liver function tests (Alkaline phosphatase, Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Albumin (ALB), Bilirubin (BIL) and Total protein (TP), Kidney function tests (Creatinine (CREA), Uric acid (URCA) and Blood urea nitrogen (BUN), Lipid

profile tests (Cholesterol (CHO), High density lipoprotein (HDLC), Low density lipoprotein (LDLC) and, glycated haemoglobin (HbA1c) and Insulin were determined using standard kit methods (Dimension Vista 1500 Intelligent Lab System, USA) in the KAU Hospital, Jeddah, Saudi Arabia.

Statistical analysis:

Statistical Science for Social Package (SPSS version 20, SPSS Inc., Chicago, IL, USA) was used for data analysis. Data were expressed as mean ± standard deviation. One way ANOVA test was used for comparison between different groups. For all tests, $p<0.05$ was considered significant.

RESULTS:

No significant variation in rat body weight (BW) in different studied groups during the 1st week. At the end of the 6th week, there was considerable($P = 0.02$) decrease in BW of diabetic rats (DC) compared to the normal control (NC) rats. There was significant ($P = 0.016$) increase in BW of diabetic rats treated with coconut oil and metformin (DCoM) compared to the (DC) group Figure (1).

At the end of the experiment, changes observed in weights of the different body organs. There were no significant changes in the weight of the heart, kidneys, pancreas and liver of (DC) compared to (NC). There was a significant increase in the weight of left kidney, right kidney of diabetic rats treated with metformin (DM) than the diabetic control ($p=0.028$, $p=0.025$) respectively, Figure (2 and 3). Table (1) showed that the percentage heart weight in DM and DCoM groups was considerablyincreased than NC ($P = 0.004$ and $P =0.040$). The percentage kidney weight in DM group was significantlyincreased than NC and DC groups ($P =0.0001$ and $P =0.026$), but was decreased in Dco and DCoM groups versus DC group ($P =0.0003$ and $P =0.012$). The percentage liver weight in DC and DM groups was increased than NC group ($P =0.0001$ and $P =0.0001$) but was significantly lower than DCo and DCoM groups versus DC group ($P =0.021$ and $P =0.001$). It was observed that the blood glucose level in the 1st week was significantly higher in DC, DM, DCo and DCoM groups in comparison to NC group ($P = 0.0001$ for all). On the other hand, it was decreased in DCo group compared to DC and DCoM groups ($P = 0.0001$ and $P = 0.019$). The same alterations observed in the blood glucose level during the 2nd, 3rd, 4th and 5th and 6th weeks. In the final week, blood glucose level in DC group was significantly higher than NC group ($p=0.0001$) while it was significantly decreased in DCo and DCoM groups compared to the DC group ($P = 0.0001$ for both). Figure (4).

Glycated hemoglobin level in DC group was higher than NC group ($P = 0.0001$) while it was significantly decreased in DM, DCo and DCoM groups compared to DC group ($P = 0.0001$, $P = 0.001$ and $P = 0.009$). Figure (5A). Insulin serum level in DC group was lower than NC group ($P = 0.001$) and it was significantly increased in DCo and DCoM groups compared to DC group ($P = 0.0001$ and $P = 0.001$). Figure (5B).

Blood glucose level presented in Figure (6) was assessed at 30 minutes periods after taking oral glucose in all of the study groups. In the normal control (NC) rats, the blood glucose level reached its highest peak at 30 min, then it gradually returns to the normal level after 120 min. In diabetic rats, the highest peak reached at 30 min and remained at a plateau for 120 min. Oral administration of the metformin or coconut oil or metformin combined with coconut oil in diabetic rats demonstrated a considerable decrease in blood glucose level at 30 and 120 minutes suggesting an improvement in glucose homeostasis, with the best results observed in DCo group.

Assessment of the lipid profile in all studied groups showed a significant increase in HDL-C serum level in DCo group compared to DC group ($P = 0.011$). Triacylglycerol level was increased significantly in DM group compared to the DC groups ($P = 0.0001$) as well as in DCoM group compared to DC and DCo groups ($P = 0.0001$). Non-significant changes have been observed in other lipid profile parameters in all of the study groups. Table (2).

The liver function tes twas assessed in all groups. A significant increase was seen in ALT and Alkaline phosphatase in the DC when compared to the NC. AST was significantly decreased in DM compared to the DC group. The level of AST, ALT and Alkaline phosphatase were significantly ($P = 0.03$, $P = 0.0001$ and $P = 0.009$) decreased in DCo compared to the DC. Albumin level was significantly ($P = 0.12$) increased in the (DCo) compared to the DC, while the total bilirubin and proteins were not significantly affected by induction of diabetes or by the different therapies used in this study Table (3).

The blood urea nitrogen level in DM group was increasedcompared to the DC group ($P = 0.041$). However, it was decreased in DCo group ($P = 0.026$) and DCoM group ($P = 0.006$) compared to DC group. Uric acid was considerable increased in DM group compared to DC group ($P = 0.006$). Serum creatinine was not significantly changed in the studied groups Table (4).

Table 1: Percentage organ weights (%) in the study groups.

Groups	Heart/body weight	Left kidney/ body weight	Right kidney/ body weight	Kidney/ body weight	Pancreas/body weight	Liver / body weight
Non-diabetic control (NC)	0.28±0.06	0.34±0.03	0.38±0.05	0.36±0.02	0.25±0.03	3.09±0.36
Diabetic control (DC)	0.31±0.01 ^{1}P =0.308	0.41±0.03 ^{1}P =0.027	0.40±0.06 ^{1}P =0.583	0.41±0.02 ^{1}P =0.108	0.27±0.06 ^{1}P =0.560	4.15±0.36 ^{1}P=0.0001
Diabetic and Metformin (DM)	0.35±0.05 ^{1}P =0.004, ^{2}P=0.065	0.47±0.05 ^{1}P=0.0001, ^{2}P=0.087	0.47±0.05 ^{1}P=0.006, ^{2}P=0.033	0.47±0.05 ^{1}P=0.000, ^{2}P=0.026	0.30±0.10 ^{1}P =0.194, ^{2}P=0.505	4.47±0.44 ^{1}P=0.000, ^{2}P=0.256
Diabetic and Coconut oil (DCo)	0.33±0.05 ^{1}P =0.075, ^{2}P=0.480	0.31±0.03 ^{1}P =0.333, ^{2}P=0.003	0.32±0.02 ^{1}P=0.058, ^{2}P=0.021	0.32±0.02 ^{1}P=0.114, ^{2}P=0.003	0.26±0.05 ^{1}P =0.873, ^{2}P=0.666	3.49±0.14 ^{1}P=0.137, ^{2}P=0.021
Diabetic, Coconut oil and Metformin (DCoM)	0.33±0.03 ^{1}P =0.040, ^{2}P=0.303,	0.33±0.04 ^{1}P =0.703, ^{2}P=0.014,	0.33±0.03 ^{1}P=0.114, ^{2}P=0.044,	0.33±0.03 ^{1}P=0.267, ^{2}P=0.012,	0.27±0.05 ^{1}P =0.591, ^{2}P=0.965,	4.04±0.37 ^{1}P=0.001, ^{2}P=0.699,

Data are expressed as mean +/- SD. Significance made using OneWay ANOVA test.
^{1}P: significance versus control; ^{2}P: significance versus diabetic control

Table 2:Lipid profiles in the study groups.

Study Groups	Cholesterol (mmol/L)	High density lipoprotein (mmol/L)	Low density lipoprotein (mmol/L)	Triacylglycerol (mmol/L)
Non-diabetic control (NC)	1.37±0.13	1.30±0.10	0.23±0.02	0.63±0.13
Diabetic control (DC)	1.39±0.10 ^{1}P =0.899	1.11±0.21 ^{1}P =0.127	0.33±0.03 ^{1}P =0.097	0.82±0.17 ^{1}P =0.540
Diabetic and Metformin (DM)	1.47±0.23 ^{1}P =0.486, ^{2}P=0.590	1.31±0.19 ^{1}P =0.943, ^{2}P=0.111	0.30±0.06 ^{1}P =0.196, ^{2}P=0.658	1.98±0.65 ^{1}P =0.0001, ^{2}P=0.0001
Diabetic and Coconut oil (DCo)	1.45±0.27 ^{1}P =0.566, ^{2}P=0.674	1.43±0.31 ^{1}P =0.258, ^{2}P=0.011	0.27±0.05 ^{1}P =0.434, ^{2}P=0.349	0.91±0.27 ^{1}P =0.336, ^{2}P=0.758
Diabetic, Coconut oil and Metformin (DCoM)	1.60±0.16 ^{1}P =0.145, ^{2}P=0.200,	1.31±0.21 ^{1}P =0.909, ^{2}P=0.117,	0.31±0.03 ^{1}P =0.190, ^{2}P=0.726,	2.26±0.90 ^{1}P =0.0001, ^{2}P=0.0001,

Data are expressed as mean +/- SD. Significance made using OneWay ANOVA test.
^{1}P: significance versus control; ^{2}P: significance versus diabetic control.

Table 3:Liver function parameters in the study groups.

Groups	Aspartate aminotransferase (U/L)	Alanine aminotransferase (U/L)	Alkaline phosphatase (U/L)	Total bilirubin (umol/L)	Total protein (g/L)	Albumin (g/L)
Non-diabetic control (NC)	114.01±8.27	49.20±11.84	134.20±21.79	2.83±0.41	70.00±2.28	11.17±1.72
Diabetic control (DC)	127.00±18.83 ^{1}P =0.274	106.00±14.25 ^{1}P =0.0001	329.25±24.19 ^{1}P =0.0001	2.60±0.89 ^{1}P=0.663	68.40±2.79 ^{1}P=0.505	9.60±1.95 ^{1}P=0.070

Diabetic and Metformin (DM)	153.52±27.24 [1]P =0.001 [2]P=0.029	92.60±18.40 [1]P =0.0001 [2]P=0.105	302.00±73.70 [1]P =0.0001 [2]P=0.506	3.50±1.22 [1]P=0.197 [2]P=0.099	65.83±3.87 [1]P=0.074 [2]P=0.287	10.50±1.87 [1]P=0.411 [2]P=0.291
Diabetic and Coconut oil (DCo)	100.67±7.20 [1]P =0.240 [2]P=0.030	75.00±13.49 [1]P =0.002 [2]P=0.0001	217.67±50.89 [1]P =0.037 [2]P=0.009	2.83±0.41 [1]P=1.00 [2]P=0.663	70.00±6.07 [1]P=1.000 [2]P=0.505	11.83±1.33 [1]P=0.411 [2]P=0.012
Diabetic, Coconut oil and Metformin (DCoM)	106.80±10.89 [1]P =0.542 [2]P=0.107	64.60±9.96 [1]P =0.064 [2]P=0.0001	132.2±39.19 [1]P =0.961 [2]P=0.0001	2.80±0.84 [1]P=0.95 [2]P=0.72	65.20±3.70 [1]P=0.050 [2]P=0.205	10.80±0.45 [1]P=0.665 [2]P=0.180

Data are expressed as mean +/- SD. Significance made using OneWay ANOVA test.
[1]P: significance versus control, [2]P: significance versus diabetic control

Table 4: Kidney function test in the study groups.

Groups	Blood urea nitrogen (mmol/L)	Creatinine (umol/L)	Uric acid (umol/L)
Non-diabetic control (NC)	7.23±0.79	42.83±5.38	55.17±7.83
Diabetic control (DC)	7.84±1.79 [1]P =0.597	49.80±8.90 [1]P =0.055	48.80±4.09 [1]P =0.281
Diabetic and Metformin (DM)	10.25±1.73 [1]P =0.008 [2]P=0.041	47.67±5.54 [1]P =0.159 [2]P=0.549	65.83±11.84 [1]P =0.062 [2]P=0.006
Diabetic and Coconut oil (DCo)	5.20±1.26 [1]P =0.069, [2]P=0.026	46.50±7.31 [1]P =0.282 [2]P=0.355	46.17±5.04 [1]P =0.113 [2]P=0.653
Diabetic, Coconut oil and Metformin (DCoM)	4.40±0.81 [1]P =0.017 [2]P=0.006	46.80±6.26 [1]P =0.268 [2]P=0.420	60.60±11.78 [1]P =0.356 [2]P=0.060

Data are expressed as mean +/- SD. Significance made using OneWay ANOVA test.
[1]P: significance versus control; [2]P: significance versus diabetic control; [4]P: significance versus Diabetic and Coconut oil.

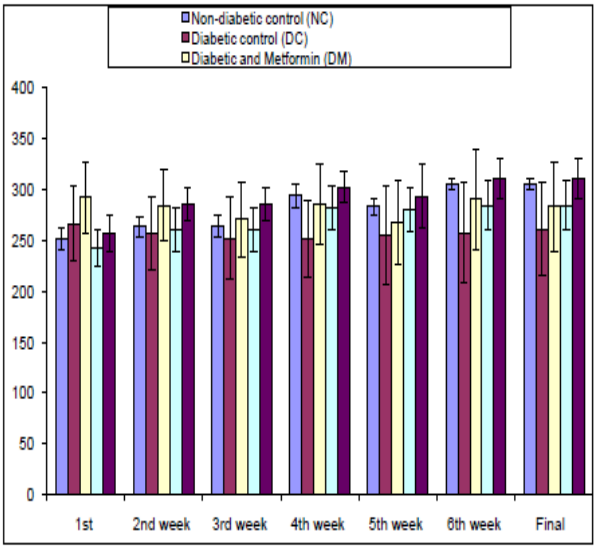


Figure 1: Body weight (grams) in different studied groups during the experiment.

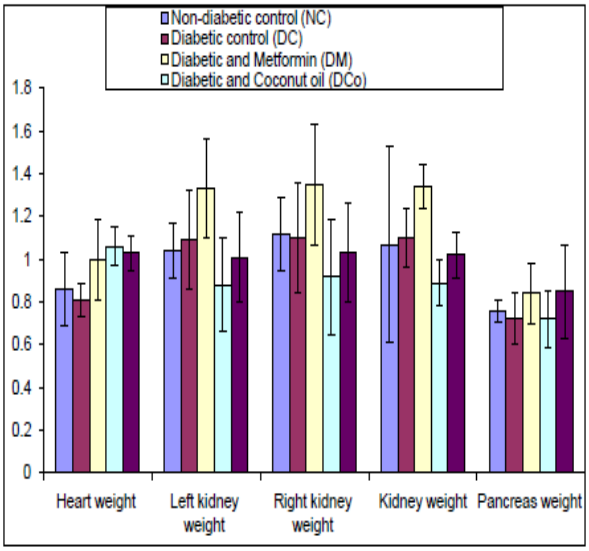


Figure 2: Weight (grams) of different body organs in the different studied groups.

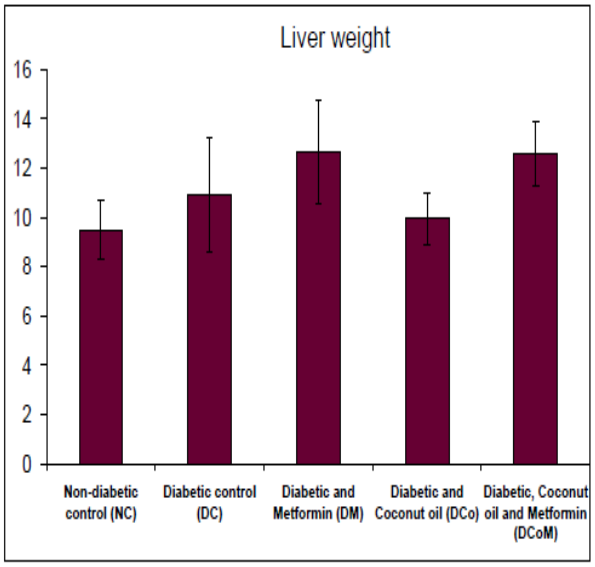


Figure 3:Weight (grams) of liver in the study groups.

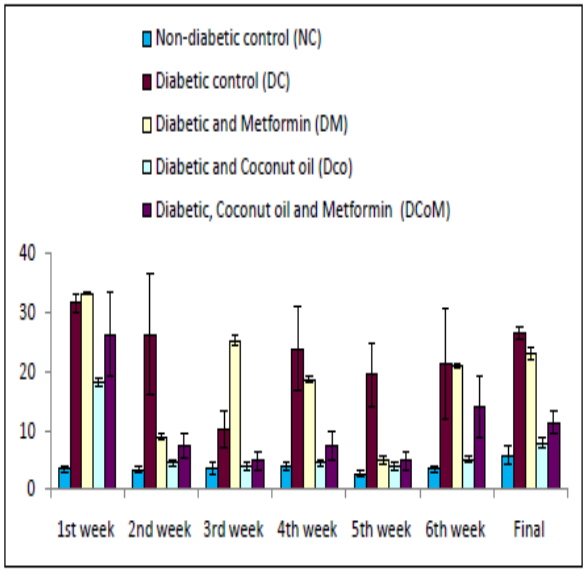
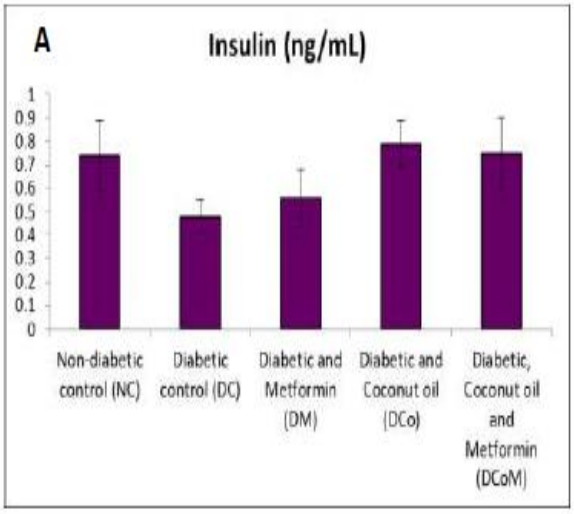


Figure 4: Blood glucose level in different groups.



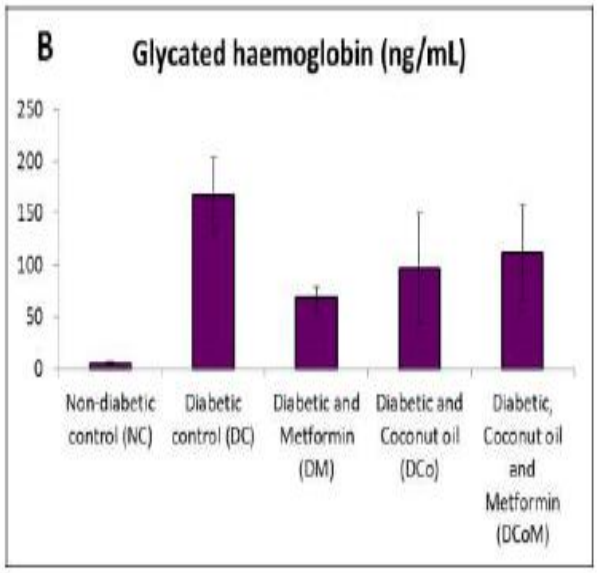


Figure 5: Insulin level (A) and glycated haemoglobin (B) in different groups.

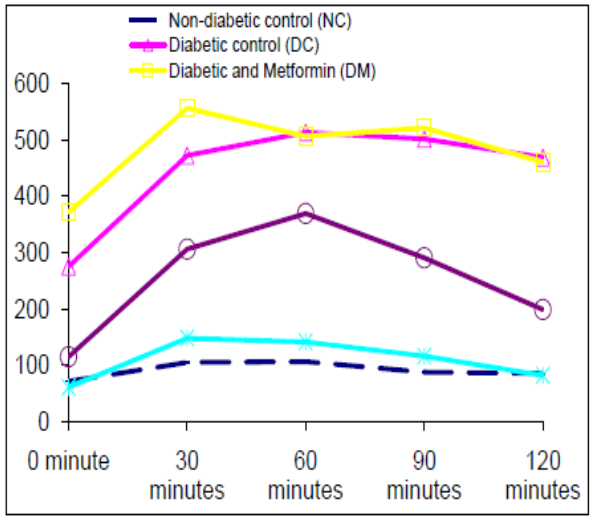


Figure 6: Blood glucose tolerance curve.

DISCUSSION

There is a considerable evidence that diabetes mellitus is induced the production of free radicals through oxidative stress that eventually affecting various organs in the body. Coconut was reported to ameliorate oxidative stress by improving the antioxidant defense system. [7]This study was carried out to estimate the abilityof virgin coconut oil in reducing the blood glucose level in type II DM induced by STZ treatment, and to assess the coconut oil effect on diabetes-induced renal and hepatic afflictions.

In this study, during the final week of the experiment, the blood glucose level of the DC group was considerable increased compared to the NC group while it significantly decreased in diabetic groups treated with either VCO alone on in combination with metformin.

These results indicate the useful effects of coconut oil in lowering blood sugar.Serum insulin level increasedin DCo and DCoM groups compared to the DC group. On examining the results of oral glucose tolerance curve, it was observed that metformin, coconut oil or metformin combined with coconut oil in diabetic rats observed a considerable lowering in blood glucose at 30 and 120 minutes indicating advancement in glucose homeostasiswith the best results observed in DCo group. It has previously been publishedthat the high content of fatty acid; lauric acid, in VCO can mimic the effect of insulin and that VCO polyphenols may increase sensitivity to insulin and decrease insulin resistance. It was observed that the glycated hemoglobin level significantly decreased in DM, DCo and DCoM groups compared to the untreated diabetic group. These results are in accordance with some previous studies performed on coconut[15]where VCO improves the fasting blood glucose level, the oral glucose tolerance, serum insulin compared to the untreated diabetic rats. The hypoglycemic effect of VCO was attributed to the antioxidant properties of VCO.[15, 16]In another study, coconut kernel protein (CKP) induced a significant hypoglycaemic effect when given to diabetic rats as reported by Salil and his group.[17] They attributed this effect to an arginine-NO pathway which leading to pancreatic beta cell regeneration. In other research, coconut water was the power to lowering plasma HbA1C and urea levels in an alloxan-induced rat model of diabetes mellitus. It also maintains blood sugar levels and elevates body weight.[18]In a more recent study, extract of Cocos nucifera inflorescence (CnI) alone and along with metformin significantly decreased plasma glucose level.[19]

Among the observations in this study was the influence ofcoconut oil on the body weight. There was a considerable increase in body weight of diabetic rats exposed tococonut oil and metformin compared to the untreated diabetic group while coconut-treated group showed insignificant increase in body weight. This finding was supported by the finding of Maidin and Ahmad. [20]

In this study, HDL-C serum level in DCo group was increased than DC group and this was in agreement with the study of Nevin and Rajamohan. [6] They reported that coconut is capable to elevate the level of high-density lipoprotein cholesterol and to lowering the level of low-density lipoprotein (LDL) in serum. However, a considerable decrease in triacylglycerols in DCo-treated group observed comparing to DM and DcoM. This finding was supported by that of Aljumayi and Ali. [21]

In this study, serum urea level increased in metformin-treated diabetic rats than the untreated diabetic groups while it is significantly decreased in rats treated with coconut alone and with metformin. On the other hand, serum creatinine was not significantly changed in the studied groups. These findings were partially in agreement with the study of **Nwangwaet al**. who concluded that coconut water was influential in improving diabetic nephropathy. [22]**Kaur** found that the CnI was influential in improving the elevated creatinine levels in animal model of diabetes.[19]In this study elevated liver enzymes, AST, ALT and Alkaline phosphatase, showed in untreated diabetic rats were lower in coconut-treated diabetic rats compared to the untreated diabetic group. In addition, albumin level was significantly increased in this group. This indicates that hepatoprotective effect of coconut on the liver of diabetic rats. In another study performed on rats with carbon tetrachloride induced liver injury, the tender coconut water exerted a hepatoprotective effect.[23]

CnI was reported to contain proteins, carbohydrates, natural antioxidants and resins.[24]The hypoglycaemic effect of CnI was attributed to the antioxidant activity of bioflavonoid content. A potential demonstration of the anti-diabetic action of CnI might be caused by elevated glycolysis at peripheral tissues by potentiating the action of insulin at the target cell comparable to biguanides.[25]CnI can help, through its high arginine contents, regeneration of pancreatic cells and improving its function and thus returning the carbohydrate metabolism to the normal levels. [17]

CONCLUSION

This study suggests that virgin coconut oil possesses antidiabetic effect alone and in combination with metformin in an animal model of type II diabetes mellitus. This means that the use of this oil could be an influential and substitution method of lowering the dose of synthetic drugs and improving its undesirable side effects. A planned future study will be conducted to explore the mechanism behind the effectiveness of coconut in reducing the blood glucose level.

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