Original Research Article

Evaluation of the Effect of Coconut Oil *(Cocos nucifera)* on Some Biochemical Parameters in Alloxan-Induced Diabetic Rats

Mohammed A[1], Luka CD[2], Gyang SD[2], Ngwen AL[3]

[1]Department of Biochemistry, College of Medical Sciences, Abubakar Tafawa Balewa University, PMB 0248 Bauchi, Nigeria

[2]Department of Biochemistry, Faculty of Medical Sciences, University of Jos, Nigeria

[3]Faculty of Natural and Applied Sciences, Plateau State University Bokkos, Nigeria

*Corresponding Author:

Mohammed A

Email: abdulrash2010@yahoo.com

Abstract: Diabetes mellitus is among the major global public health problems and its prevalence is currently increasing at an alarming rate. According to the International Diabetes Federation, about 366 million people are living with diabetes and this figure is projected to increase to 552 million by the year 2030. The study investigated the effect of oral administration of aqueous extract of coconut oil on blood glucose, total protein, kidney function indices, liver function indices, and lipid profile levels in alloxan-induced diabetic rats. The aqueous extract was administered orally at a dose of 400mg/kg body weight to both normal and alloxan-induced diabetic rats. Twenty adult male rats were divided into four groups of five rats each, two groups were made diabetic and the other two groups were non diabetic. One of the diabetic groups was treated with the extract and the other serves as control. The alloxan was administered intraperitoneal at a dose of 120mg/kg per body weight. The administration of the extract lasted for 21 days. Effect of the extract on blood glucose, total protein, albumin, bilirubin, urea, creatinine, total cholesterol, triglyceride, and high density lipoprotein concentrations were analyzed. The toxic effect of the extract was determined using biochemical enzyme markers. Treatment with the extract showed significantly ($p<0.05$) reduction in elevated blood levels of glucose, cholesterol, and proteins and other biochemical parameters associated with alloxan-induced diabetic rats. The extract possesses no toxic effect as indicated by the lowering of ALP and ALT levels and may be used for the management of diabetes mellitus.

Keywords: *Cocos nucifera,* hypoglycaemic*,* diabetic mellitus, lipid profile, hypocholesterolaemic

INTRODUCTION

Traditional medicine according to the world health organization, refers to the knowledge, skills and practices based on the theories, beliefs and experiences indigenous to different cultures, used in maintenance of health and in the prevention, diagnosis, improvement or treatment of physical and mental illness. Traditional medicine covers a wide variety of therapies and practices which vary from country to country and region to region. [1]. In some countries, it is referred to as "alternative" or "complementary" medicine. Traditional medicine has been used for thousands of years with great contributions made by practitioners, to health, particularly as primary health care providers at the community levels. Complementary medicine has maintained its popularity worldwide. Since the 1990's. Its use has surged in many developing countries [1].

Herbal medicines include herbs, crude plant materials and oil, herbal preparation and finished herbal products that contain as active ingredients, parts of plants or other plant materials or combinations [2].

Traditional Chinese medicine (TCM) is an important example of how ancient and accumulated knowledge is applied in a holistic approach in present day health care. TCM has a history of more than 3000 years [3]. The book *The Devine Farmer's Classic of Herbalism* was compiled about 2000 years ago in China and is the oldest known herbal text in the world, though the accumulated and methodically collected information on herbs has been developed into various herbal pharmacopoeias and many monographs on individual herbs exist [4].

The coconut tree is a member of family Arecaceae (palm family). It is the only accepted species in the genus cocos [5]. The term coconut can be referred to the entire coconut palm, the seed, or the fruit, which botanically is drupe, not a nut found throughout the tropics and subs tropic area, the coconut is known for its great versality as seen in the daily diets of many people. Coconuts are different from any other fruits because they contain a large quantity of "water" and when

immature they are known as tender-nut or jelly-nuts and may be harvested for drinking [6]. When mature, they are still containing some water and can be used as seed nuts or processed to give oil from the kernel, charcoal from the hard shell and coir from the fibrous husk [5].

Cocos nucifera is a large palm, growing up to 30m (98ft) tall, with pinnate leaves 4-6 (13-20ft) long and pinnae 60-90cm long; old leaves break away clearly, leaving the drunk smooth. Coconuts are generally classified into two general types; tall and dwarf. On very fertile land, a tall coconut palm tree can yield up to 75 fruits per year, but more often yield less than 30, mainly due to poor cultural practices [7]. Coconut oil is an edible oil extracted from the kernel or meat of matured coconut harvested from the coocnut palm *(cocos nucifera).* It has various applications in food, medicine and industry. Because of its high saturated fat content, it is slow to oxidize and thus resistant to rancidity, lasting up to two years without spoilage [6].

MATERIAL AND METHODS
Material
Study Animals

Male and female albino rats weighing about 100g and 120g were purchased from animal house of Pharmacology Department, University of Jos. The animals were housed in well-ventilated cages in the animal house unit of Pharmacology Department, University of Jos. The rats were allowed to acclimatize for one week prior to the experiment and had access to food and clean water.

Plant Material

Coconut were collected from Farin Gada market in Jos north local Government Area of Plateau State-Nigeria. The plant was identified and authenticated at Botany Department, University of Jos.

Preparation of Coconut Oil

$$\text{Volume to be administered in mls} = \frac{weight\ of\ rats\ in\ (kg)\ \times Dose\ (mg/kg)}{Concentration\ of\ Extract\ (mg/ml)}$$

Animals with fasting blood glucose ≥200 mg/dl were considered to be diabetic and are used for the study [8].

Administration Of Oil

The coconut oil was given through oral administration at a dose of 400mg/kg- body weight daily. This was administered for 21 consecutive days after confirming diabetes in the rats.

Experimental Design
Animal Experimentation

Sixteen (16) adult rats were randomly allocated into 4 groups (A-D) of 4 animals each, and allowed to

Extraction

Using the wet mill method for the extraction of oil, a matured, brown coconut is used. The coconut shell is split with a sharp cleaver. The meat of the coconut is scraped out from the shell using a sharp paired knife or a sturdy. The meat of the coconut is then cut to smaller pieces. This is then placed in the food processor (blender). The processor is then turned on to a medium speed and the meat is blended until well shredded. Little water is added if necessary to help it blend proper.

The coconut milk is filtered (with a cheese cloth over a wide-mouth jar). The cloth is wrapped around the coconut mixture and milk is squeezed hard, making sure every last drop is gotten. This process is repeated until all the coconut mixture has been used.

The jar is left unattended to for at least 24hrs. (the jar is refrigerated so that the curd hardens more quickly if needed on time).

As it sets, the coconut milk and oil will separate and a layer of curds will appear at the top of the jar. The curd is scooped out with a spoon and discarded. The pure oil is left in the jar.

Preparation of Alloxan

One (1) gram of Alloxan Hydrate was dissolved in 10ml of distilled water and to give a concentration of 100mg/ml.

Induction of Diabetes Mellitus With Alloxan

Rats induced with Diabetes mellitus were fasted overnight for a period of 12 hours; diabetes was induced by injecting alloxan hydrate intraperitoneally at dose of 120 mg/kg using a sterile 1ml syringe. The volume of the solution containing 100 mg/kg given to each experimental albino rat was determined by the following relationship;

acclimatize for 3 days before the experiment commenced. They were kept in wide normal cage and fed with standard feed and also allowed the access to water and lithium. Diabetes was induced for rats in groups A and D (as diabetic rats and diabetic control respectively. The rats were grouped and fed as follows:

Group 'A' Diabetic Rat

Alloxan induced diabetic rats given coconut oil extract for 21 days.

Group 'B' Normal Control

Rats in this group were fed with normal diet for 21 days. No alloxan induced and no extract administered (poisitive control).

Group 'C' Non-diabetic

Rats in this group were fed with coconut oil extract and the normal feed for 21days.

Group 'D' Diabetic Control

Alloxan induced diabetic rats with no extract given (negative control). But fed with normal feed for 21 days.

Sample Collection and Preparation

Overnight fasting blood samples were collected from animals induced with diabetes after 72 hours. At the end of 21 days of administration, blood from the animal (both treated and control groups) were collected from the orbital plexus of the rats using non-heparanised haematocrit capillary tubes, the blood samples were allowed to clot at room temperature and then centrifuged. Then Pasteur pipette were used to withdraw the serum (supernatant) into the dry clear Bijou bottles. The serum was then used for necessary assays.

STATISTICAL ANALYSIS

Results were expressed as mean ± standard deviation and analyzed using ANOVA, with p value <0.05 considered significant followed by Tukey´s post hoc test. A component of GraphPad Instat3 Software [9] version 3.05 by GraphPadInc was used to analyze the data.

RESULTS

Table 1: Effect of coconut oil on blood glucose, protein and albumin in alloxan-induced diabetic rats

Treatment Group	Glucose (mmol/L)	Protein (g/L)
Normal Control	9.80±0.22	5.30±2.16
Normal treated	7.60±0.22[ab]	4.00±2.16[b]
Diabetic treated	10.15±0.70[a]	4.25±2.16[a]
Diabetic control	25.10±0.22[a,b]	8.20±2.16[a,b]

Values are expressed as mean ± SD, n= 5 for each group. Values in the same column bearing similar superscripts are significantly different at P<0.05.

Table 2: Effect of Aqueous extract of coconut oil on Liver function indices in alloxan induced diabetic rats

Treatment Group	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	Albumin (g/L)	T. Bilirubin (g/L)	D. Bilirubin (g/L)
Normal Control	105.00±2.16	277.50±2.65	105.00±2.16	35.00±2.16	3.28±0.10	5.35±0.30
Normal treated	109.00±2.16[b]	252.00±2.16[b]	102.00±2.16[b]	36.00±2.16[b]	4.50±0.20[b]	5.26±0.35[a]
Diabetic treated	92.00±2.16[a]	276.00±2.16[a]	92.00±2.16[a]	38.00±2.16[a]	4.40±0.25[a]	5.14±0.23[ab]
Diabetic control	202.00±2.16[a,b]	313.00±2.16[a,b]	202.00±2.16[a,b]	27.00±2.16[a,b]	10.43±0.29[a,b]	15.15±0.17[a]

Values are expressed as mean ± SD, n= 5 for each group. Values in the same column bearing similar superscripts are significantly different at P<0.05.

Table 3: Effect of aqueous extract of coconut oil on Kidney function indices in alloxan induced diabetic rats

Treatment Group	Urea (mmol/L)	Creatinine (mmol/L)	Na^+(mmol/L)	K^+(mmol/L)	Cl^-(mmol/L)	HCO_3^- (mmol/L)
Normal Control	7.50±2.16	66.00±2.16	155.00±2.16	7.30±0.22	124.00±2.16	14.00±2.16
Normal treated	4.80±2.16[b]	56.00±3.06[b]	150.00±2.36[b]	7.90±0.22[b]	120.00±2.16[b]	13.00±2.16[b]
Diabetic treated	6.90±2.16[a]	65.00±2.16[a]	151.06±2.16[a]	4.70±0.22[a]	125.00±2.16[a]	12.00±2.16[a]
Diabetic control	14.30±2.16[a,b]	78.00±2.16[a,b]	139.08±4.03[a,b]	16.88±0.22[a,b]	96.000±2.16[a,b]	22.00±2.16[a,b]

Values are expressed as mean ± SD, n= 5 for each group. Values in the same column bearing similar superscripts are significantly different at P<0.05.

Table 4: Effect of aqueous extract of coconut oil on serum lipid profile of alloxan induced diabetic rats

	Lipid Profile (mmol/L)			
Treatment Group	TC	TG	HDL	LDL
Normal control	1.40±0.22	0.63±0.22	1.60±0.22	1.20±0.12
Normal treated	1.42±0.22[b]	0.74±0.22[b]	1.38±0.22[b]	1.13±0.26[b]
Diabetic treated	1.50±0.22[a]	0.82±0.22[a]	1.50±0.22[a]	1.14±0.32[a]
Diabetic control	1.70±0.29[a,b]	1.20±0.22[a,b]	0.30±0.22[a,b]	1.60±0.22[a,b]

Values are expressed as mean ± SD, n= 5 for each group. Values in the same column bearing similar superscripts are significantly different at P<0.05.

DISCUSSION

Diabetes mellitus as in the case with most diseases is present as mild or acute. In the acute diabetes mellitus, the β-cells of the pancreas are completely destroyed or non- functional while the mild form of the disease is characterized by partial β-cells failure. Consequently, there is total lack of endogenous insulin production in acute or severe diabetes mellitus, whereas some limited amount of insulin is synthesized and released into circulation in mild or moderate diabetes mellitus [10]. Experimentally, the severity of the disease is dependent on the dose of alloxan monohydrate administered to the animals; Low or high dose produce incomplete or complete destruction of the β-cells of the pancreas respectively [11].

The Wister rats used were strictly males because it was reported that female sex hormones (17ß estradiol) has a lowering effect on the plasma cholesterol concentration [5]. Thus, using female rats may interfere with the accuracy of the serum cholesterol level, since it was one of the parameters analysed. Glucose is important in the body because it is stored in mainly the liver and muscles as glycogen; it is distributed and utilized in tissues as free glycogen. Protein is also important in the function of growth and cell maintenance; proteins are responsible for muscle contraction. Scientists can speculate on the reasons why glucose and not another monosaccharide such as fructose, is so widely used in organisms. One reason might be that glucose has a lower tendency, relative to other hexose sugar to react non-specifically with the amino groups of protein. The reaction (glycation) reduces or destroys the function of many enzymes. The low rate of glycation is due to glucose preferences for the less reactive cyclic isomer [5]. Nevertheless, many of the long term complications of diabetes e.g. blindness, renal failure and peripheral neuropathy are probably due to the glycation of the proteins or lipids. However, enzyme regulates addition of glucose to protein by glycosylation which is often essential to their function [12]. Treatment with coconut oil shows significant (P<0.05) decrease in both glucose and protein levels. This finding is in accordance with the research of [5].

Changes in serum enzymes levels are often early determinant of tissue damage either by toxicant or in disease conditions. Serum alkaline phosphate, alanine aminotransferase and aspartate aminotransferase are liver biochemical markers. Therefore, increases in ALT activity are always due to hepatocellular damage and it is usually accompanied by AST activity [5]. However, the difference observed in the activities of these enzymes at the dose employed (400mg/kg body weight) showed statistically significant (P<0.05) decrease in ALT, AST, ALP, total bilirubin and direct bilirubin levels when administered with coconut oil with significant (P<0.05) increase in Albumin level when compared with diabetic control group. This implies that coconut oil at that concentration employed has no toxic effect on the liver of the rats.

Kidneys are the major organs in metabolizing toxic compound besides liver. It receives about 1200ml of blood per minute containing a lot of chemical compounds [13]. Therefore, damage to the kidneys can be determined by measuring the level of urea, electrolyte and creatinine in blood as an indicator of kidney damage. There was significant (P<0.05) increases in sodium (Na^+), and chloride (Cl^-) levels, with concomitant reduction in potassium (K^+) and bicarbonate (HCO_3^-) levels in diabetic administered with the extract. Also, diabetic administered with 400mg/kg body weight of extract showed significant (P<0.05) decrease in urea, and creatinine levels when compared with diabetic control group. This finding support the report of [5] on effect of coconut oil on some biochemical parameters in albino rats.

Lipids profile also known as coronary risk panel, or lipid panel the collective term to the estimation of typically total cholesterol, high density lipoprotein and triglycerides are used to assess risk of coronary heart disease [14]. Lipid profiles have been shown to be the important predictors for the metabolic disturbances including dyslipidemia, hypertension, diabetes, cardiovascular disease, hyperinsulinemia etc. [15]. Administration with coconut oil shows significant (P<0.05) decrease in total cholesterol, triglyceride, LDL levels with significant (P<0.05) increase in HDL level.

CONCLUSION

Treatment with coconut oil decreased the concentration of glucose and protein in alloxan-induced diabetic rats. Therefore, the plants have hypoglycaemic effects. The study also showed that coconut oil may not have toxic effect on the liver at the employed dosage since it produced no significant effects on the enzymes activities as biochemical enzymes makers of liver damage.

REFERENCES

1. WHO. (2012). Traditional medicine strategy: 2002-2005. World Health Organization press, p. 1-6.
2. Luka, C. D., & Mohammed, A. (2012). Effect of aqueous extract of *Dioscorea bulbifera* on some biochemical parameters in alloxan-induced diabetic rats. *Journal of medical and Applied Biosciences, 4*, 53-60.
3. Xutian, S., Zhang, J., & Louise, W. (2009). New exploration and understanding of traditional Chinese medicine. *Am J Chin Med, 37*, 411–26.
4. Benzie, I. F. F., & Wachtel-Galor, S. (2011). *Herbal Medicine: Biomolecular and Clinical Aspects.* 2nd edition. Boca Raton (FL): CRC Press/Taylor & Francis.

5. Mohammed, A., & Luka, C. D. (2013). Effect of Coconut oil, coconut water and palm kernel oil on some biochemical parameters in albino rats. *IORS journal of pharmacy and biological sciences, 6* (3), 56-59.

6. Boateng, L., Ansong, R., Owusu, W. B., & Steiner-Asiedu, M. (2016). Coconut oil and palm oils role in nutrition, health and natural development: A review. *Ghana medical journal, 50*(3), 189-196.

7. Shasshikumar, S., & Chandrashekar, H. M. (2014). An analysis of production and marketing of coconut in Tumkar District, India. *Int. J. curr. Res. Aca. Rev, 2*(10), 167-175.

8. Muhammad, I. U., Alhassan, A. J., Sule, M. S., Idi, A., Mohammed, A., El- ta'alu, A. B., Dangambo, M. A., & Abdulmumin, Y. (2016). Anti-Hyperglycemic Activity of Solvents Extract of *Khaya senegalensis* Stem Bark in Alloxan Induced Diabetic Rats. *JABB*, *6*(2), 1-8.

9. GraphPad Instat3 Software (2000). Available: www.graphpad.com. Retrieve on 23rd February 2015.

10. American Diabetes Association. (2009). Standards of medical care in diabetes. *Diabetes Care*, *32* (1),13-61.

11. Kumar, S., Singh, R., Vasindeva, N., & Sherma, S. (2012). Acute and Chronic animal models for the evaluation of antidiabetic agents. *Cardiovascular Diabetology, 11*, 9.

12. Bieberich, E. (2014). Synthesis, processing and function of N-glycans in N-glycoproteins. *Advances in neurobiology, 9,* 47-70.

13. Dollah, M. A., Parhizkar, S., & Izwan, M. (2013). Effect of *Nigella sativa* on the kidney function in rats. *Avicenna J Phytomed Spring*, *3*(2), 152-158.

14. Harchaoui, K. E., Visser, M., Kastelin, J. J., Stores, E., & Dallinga-Thie, G. (2009). Tryglycerides and Cardiovascular Risk. *Current cardiology reviews, 5*(3), 216-222.

15. Kaur, J. (2014). A comprehensive review on metabolic syndrome. *Cardiology research and practice, 943162*. http://doi.org/10.115/2014/943162.