

# ANTIDIABETIC AND ANTILIPIDEMIC ASSESSMENT OF SOYA BEAN OIL ON LIPID PROFILE AND GLUCOSE LEVEL IN ALLOXAN-INDUCED DIABETIC MALE WISTER RATS.

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## ABSTRACT

**Purpose:** Experimental studies in alloxan-induced diabetic animals have demonstrated several abnormalities upon induction such as physiological, biochemical and histological alterations. In this present study, the influences of graded doses of soya bean oil supplementation for 4weeks on some biochemical parameters in alloxan-induced diabetes in rats were determined. **Methodology:** Sixty (60) male albino rats weighing about 240-260g were divided into 6 experimental groups of 10 rats per group viz: I, II, III, IV, V and VI. Group I serve as male control, group II serve as diabetes control which were fed with normal rat chow while rats in the test group were fed with graded doses of soya bean oil supplements and water *ad libitum* for 4weeks, at the end of the supplementation, plasma lipid profile and glucose level were determined. **Results:** All the diabetic induced rats had significantly higher ( $p < 0.01$ ) Glucose, Total cholesterol, Triglyceride, LDL-C, VLDL-C, but lower HDL-C levels than all the non-diabetic rats. The data obtained after supplementation revealed that soya bean oil significantly reduced the glucose level and ameliorated the lipid profile with attendant decrease in glucose, total cholesterol, triglyceride, LDL-C, VLDL-C levels, but with a corresponding increases in HDL-C levels respectively when compared to alloxan-induced diabetic rats.

**Conclusion:** The observation of significant changes in plasma lipid profile, glucose level might be as a result of supplementation with soybean oil. It is therefore concluded that Soya bean oil possess significant antidiabetic activity that can improve the physiological and biochemical parameters which are abnormally altered due to diabetes mellitus.

## 1.0 INTRODUCTION

Diabetes Mellitus (DM) is a group of metabolic disorder caused as a result of inherited or acquired deficiency in production of insulin by the pancreas, or by the ineffectiveness of the endogenous insulin produced [1]. Diabetes mellitus has been shown to have a common manifestation which is hyperglycemia [2] (Jennifer *et al*, 2010). Although the etiology of this disease is not well defined, viral infection, autoimmune disease and environmental factors have been implicated and it is usually accompanied by alteration in physiological parameters, increased production of free radicals and or impaired antioxidant defense [3](Maritim *et al*, 2003). Diabetes mellitus is a significant healthcare concern worldwide that affects more than 165million individuals leading to cardiovascular disease, nephropathy, retinopathy, and widespread disease of both the peripheral and central nervous systems. Diabetes mellitus has been tagged one of the main threat to human health in the 21<sup>st</sup> century because of changes in human behaviors and lifestyle during the last century which resulted in an increase in the incidence of diabetes worldwide, hence the global number of people with diabetes is estimated to rise up to 366millions in 2030 [4] (Khushk *et al*, 2010).

The incidence of undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose levels raises future concerns in regards to the financial and patient care resources that will be necessary to care for patients with diabetes mellitus (Kenneth *et al*, 2007). Worthy of note is that disease of the nervous system has become one of the most debilitating complications of diabetes mellitus and this affect sensitive cognitive region of the brain, such as the hippocampus that modulates memory function, resulting in significant functional impairment and dementia [5]. (Kenneth *et al*, 2007).

Cardiovascular complications have been established as the leading cause of morbidity and mortality associated with diabetes mellitus [6](Jeanette *et al* 2008), there is now a growing evidence that excess generation of highly reactive radicals, largely due to hyperglycemia causes oxidative stress which further

exacerbates the progression of diabetes and its complication, worthy of mentioning is that the excess generation, overproduction and/or insufficient removal of these free radicals result in vascular dysfunction, damage to cellular proteins, membrane lipids, disruption to nucleic acids to mention but a few [7] (Kris *et al*, 2007). Numerous studies have therefore demonstrated that oxidative stress mediated mainly by hyperglycemia-induced generation of free radicals, contributes to the development and progression of diabetes and related contributions, it therefore became clear that ameliorating oxidative stress might be an effective strategy for reducing diabetic complication [8]. (Maritim *et al*, 2003).

Dyslipidemia is a frequent complication of diabetes mellitus and is characterized by low levels of high-density lipoprotein-cholesterol (HDL-C) and high levels of low density lipoprotein cholesterol (LDLC) and triglyceride (TG) (Baynes, 1991). Hyperlipidaemia is a metabolic complication of both clinical and experimental diabetes [9](Gandhi, 2001). Normally; Low-Density Lipoprotein (LDL) carries cholesterol to parts of the body that need it. Oxidized LDL-C can stick to blood vessel walls. Cholesterol on Blood vessel wall leads to occlusion and thus coronary artery disease. The less LDL in your blood, the better thus it is literally called the Bad Cholesterol. The High Density Lipoprotein (HDL) carries cholesterol away from the blood vessel walls to the liver. Liver breaks the cholesterol down and sends it out of the body. The more HDL in your blood, the better thus it is literally called the good Cholesterol, increase low-density lipoprotein in diabetic patients leads to abnormal metabolism and is associated with increase in very low-density lipoprotein (VLDL) secretion and impaired VLDL catabolism. Ultimately this leads to atherosclerotic plaque [10](Howard, 1987).

## 2.0 MATERIALS AND METHODS

Materials used include wooden animal cage, spectrophotometer, glucometer, Grower's feed, manual weighing balance, digital weighing balance, Stainless Steel Plates, Plain bottle, anticoagulated bottles like Fluoride oxalate bottle and Lithium heparine bottle, Reagent kits, Spectrophotometer, Blood lancet, Cotton wool, Methylated spirit, Morning fresh, Syringe and Needleless, Stainless steel plates and so on.

### 2.1 Experimental animal and design

Healthy male albino rats of the wistar strain of at least 4weeks weighing approximately 240g-260g were obtained from the animal house of Ladoke Akintola University of Technology, housed in a cage and allowed for two weeks of acclimatization. Apart from the control groups which were fed with normal rat chow, others were fed with graded doses of soya beans oil dietary supplements and water *ad libitum* throughout the experimental period and kept in a well ventilated environment between 21°C-31°C and had free access to water. All animals were made to receive humane care in accordance with the principle of laboratory animals care of the National Society of Medical Research. A total of 60 healthy male wistar rats were used for this study and are divided into 6 groups each; 10 per group via; Group I: served as positive control group (non-diabetic) rat fed with normal rat chow for 4 weeks. Group II: served as negative control group (diabetic rat) fed with normal rat chow for 4 weeks. Group III: served as diabetic rat fed with 10g/kg of soya beans oil dietary supplements on daily basis for 4 weeks. Group IV: serve as diabetic rat fed with 5g/kg of soya beans oil dietary supplements on daily basis for 4 weeks. Group V: served as diabetic rat fed with 7g/kg of soybean oil dietary supplements on daily basis for 4 weeks. Group VI: served as diabetic rat fed with 10g/kg of soya beans oil dietary supplements on daily basis for 4 weeks.

### 2.2 Induction of diabetes

After 18 hours animals fasting, dilute solution of alloxan (50mg/kg body weight) was administered to the rats intraperitoneally using 1ml sterile disposable hypodermic needles and this was used to induce diabetes into the rats except in the positive control group which was non diabetic group. After injection, the rats had free access to food and water. Diabetes was allowed to develop and stabilize in these alloxan-treated rats over a period of time. Diabetes mellitus was defined in these rats using the Glucometer for determination of fasting blood glucose levels. Rats that showed fasting blood glucose more than 150 mg/dl were considered diabetic and selected for the experimentation.

### 2.3 Sample collection and storage

After 4weeks of supplementation, animals were anaesthetized using chloroform and ultimately sacrificed by cervical dislocation method and 10ml of blood samples were taken from the heart region into Lithium heparin and fluoride oxalate bottles, 5ml in each bottle, gently mixed then it was centrifuged at 4000g for 10min and the supernatant was collected into a plain bottle for analysis, after which the plasma sample stored at 20°C.

**2.4 Statistical analysis:** Data were statistically analyzed using Package for Social Sciences (SPSS) for windows version1.0 software. All experimental data were expressed as MEAN± STANDARD ERROR OF MEAN (SEM). Statistical analysis was performed by one-way analysis of variance (ANOVA). Significance was taken at P<0.05 and highly significant if P<0.001

### 3.0 RESULTS

Diabetes induced groups had significant higher ( $p < 0.01$ ) glucose, total cholesterol, triglyceride, LDL-C, VLDL-C, but lower HDL-C level compared to non-diabetic groups. The data obtained after supplementation with soya bean oil showed significant reduction in glucose, total cholesterol, triglyceride, LDL-C, VLDL-C levels, but with attendant increase in HDL-C levels respectively when compared to alloxan-induced diabetic rats.

### 4.0 DISCUSSION

Alloxan acts as cytotoxin on beta-cells of the islet of Langerhans, thus causing necrosis; increased reactive oxygen species formed as results of oxidative stress mediates this cytotoxic action with an increase in cytosolic calcium concentration, leading to rapid beta-cells destruction which ultimately leads to decreased insulin (a glucose regulating hormone which aids glucose transport, storage and uptake by almost all body cells) secretion and thus an elevated blood glucose [4](Khushk *et al.*, 2010).

Impaired antioxidant defenses with concomitant hyperglycaemia, hypercholesterolemia low levels of high density lipoprotein –cholesterol (HDL-C), high levels of low density lipoprotein cholesterol (LDL-C) and high triglyceride (TG) is a recurrent decimal of diabetes episodes in both humans and experimental studies by [11, Sena 2008, 4 Khushk 2010 and 12 Baynes 1991]. This present research was in agreement with these authors' speculation and earliest work ([11 Sena 2008; 4 Khushk *et al* 2010; 12 Baynes *et al*, 1991). Abnormalities in lipid profile that accompany diabetes are usually secondary to insulin deficiency in which case, lipolysis is enhanced and plasma non esterified fatty acid (NEFA) concentration was elevated. In the liver, NEFAs are converted to acetyl CoA and ketones and re-esterified to form endogenous triglycerides and incorporated into VLDLs; the latter accumulates in plasma because lipoprotein lipase which is necessary for VLDL catabolism requires insulin for optimal activity. Consequently, there is increased rate of cholesterol synthesis and also low levels of high density lipoprotein –cholesterol (HDL-C) and high levels of low density lipoprotein cholesterol (LDL-C) and triglyceride (TG), Chylomicronaemia may also follow if insulin deficiency is severe [13](Martin, 2006). In actual fact, the abnormal alteration in the lipid profile in diabetics mellitus is mainly due to increase mobilization of free fatty acids from peripheral fats depots [4].

The glucose lowering ability of soya beans oil in alloxan-induced rats as evidenced in this experiment and confirmed by some author [11] may be due to the presence of high amount of coenzyme Q and some antioxidants. Also [4] asserts that there is a possibility that a few of the  $\beta$ -cells are still surviving following alloxan administration, and are therefore stimulated by soya oil component(s), thus causing the B-cells to release insulin again. Although there were slight changes in  $\text{Na}^+$  and  $\text{K}^+$  levels in the experimented groups which were not significant. This is contrary to the findings of [14], they documented that in diabetes mellitus, and there is significant increase in the electrolyte (especially  $\text{K}^+$ ) level which was not seen when diabetic rats were compared with non-diabetic mellitus control rats

### 5.0 Conclusion

The results obtained from this investigation revealed that soya beans oil may have antidiabetic and antilipidemic properties that may ameliorate dreaded effect of diabetics and high blood pressure that is ravaging human population all over the globe.

### 6.0 Acknowledgement:

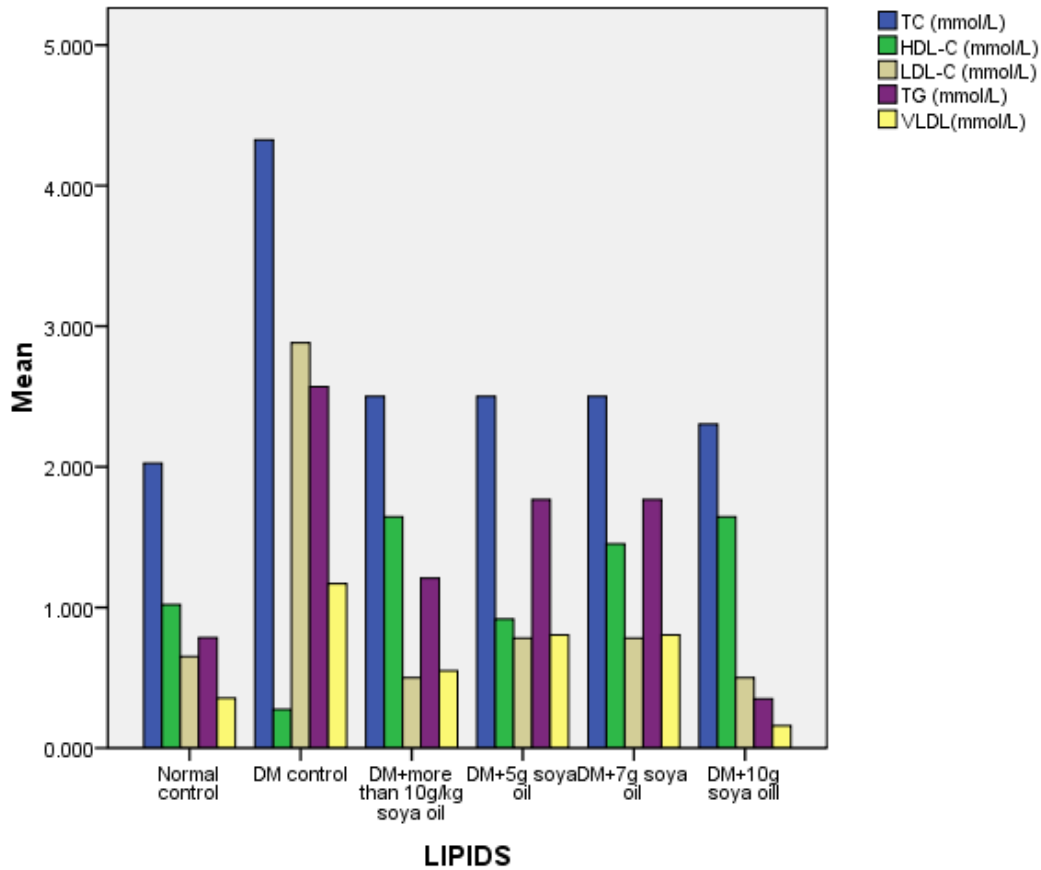
We sincerely express our gratitude to the entire staff of Biochemistry Department, College of Natural Sciences, Joseph Ayo Babalola University Ikeji- Arakeji, Osun State Nigeria for their moral support during this research work.

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**FIGURES**



**Graph**

