

## Prevention of Oxidative Stress by Taurine in Alloxan-Induced Diabetic Mellitus in Wistar Rats

Jimoh Abdulazeez<sup>1\*</sup>, Tanko Y<sup>1</sup>, Salihu KI<sup>1</sup>, Yusuf R<sup>2</sup>, Tende JA<sup>1</sup>, Abdullahi A<sup>1</sup>, Mohammed A<sup>1</sup> and Ayo JO<sup>3</sup>

<sup>1</sup>College of Health Sciences, Faculty of Basic Medical Sciences, Human Physiology Department, Ahmadu Bello University, Zaria, Kaduna State, Nigeria

<sup>2</sup>College of Health Sciences, Faculty of Basic Clinical Sciences, Chemical Pathology Department, Ahmadu Bello University, Zaria, Kaduna State, Nigeria

<sup>3</sup>Faculty of Veterinary Medicine, Department of Veterinary Physiology, Ahmadu Bello University, Zaria, Nigeria

\*Corresponding Author: Jimoh Abdulazeez, College of Health Sciences, Faculty of Basic Medical Sciences, Human Physiology Department, Ahmadu Bello University, Zaria, Kaduna State, Nigeria.

Received: May 22, 2018; Published: July 20, 2018

### Abstract

Taurine, is a non-protein amino acid, with a unique chemical structure, it is involved in numerous biological and physiological functions with health benefits. In this study, the preventive effect of taurine on body weight, blood glucose level, oxidative stress and lipid peroxidation in alloxan-induced diabetes mellitus in wistar rats. Diabetes was induced by single intraperitoneal injection of alloxan monohydrate (150 mg/kg). Blood glucose level was measured after 72 hours of induction and diabetes was considered in animals with blood glucose level greater than 200 mg/dL with Accu-Check Active Glucometer. The rats were grouped into (5) groups of five animals each n = (5) Group 1: Normal control group, Group 2: diabetes untreated: Group 3: diabetes treated with 100 mg/kg taurine: Group 4: diabetes treated with 200 mg/kg, and Group 5: diabetes treated with glibenclamide (1 mg/ml). blood glucose and body weight of the rats were determined on weekly bases for three consecutive weeks. At the end of the treatment, all animals were sacrificed and blood samples was collected for determination of oxidative stress biomarkers and lipid peroxidation. The result of the present study shows a significant (P < 0.05) decrease in body weight and blood glucose in groups that received 100 and 200 mg/kg of taurine compared with diabetic untreated group alone at 21 days of the experiment. Oxidative stress biomarkers superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) recorded a (P < 0.05) decrease in values in groups that received taurine at doses of 100 mg/kg and 200 mg/kg compared with the diabetic untreated group. Lipid peroxidation activities showed a significant (P < 0.05) decrease in serum malondialdehyde (MDA) concentrations in diabetes groups treated with taurine when compared with diabetes untreated only. those treated with glibenclamide also recorded a significant decrease in serum oxidative stress biomarkers and lipid peroxidation when compared with the diabetic untreated group. In conclusion, this study revealed that taurine could be a preventive supplement against oxidative stress in type 1 diabetic and could be a potent supplement to mitigate against it occurrence.

**Keywords:** Taurine; Oxidative Stress Biomarkers; Body Weight; Blood Glucose; MDA; Diabetes Mellitus

### Introduction

The most abundant free intracellular amino acid in human cells is taurine, a non-protein amino acid present in virtually all animal tissues [1]. Taurine has a very unique chemical structure, and due to this, it is involved in numerous biological and physiological functions which give rise to important health benefits [2]. Diabetes has been associated with a reduction or decline in the levels of taurine, which

is an important antioxidant. This raises the possibility of contributing negatively to the severity of the oxidant-mediated damage present in the diabetic context [3]. Reactive oxygen species (ROS) are produced by living organisms as a result of normal cellular metabolism. They function in physiological cell processes at low to moderate concentrations and produce adverse modifications to cell components such as lipids, proteins and DNA at high concentrations [4]. The shift in balance between oxidant/antioxidant in favor of oxidants is termed "oxidative stress". Oxidative stress contributes to many pathological conditions including diabetes mellitus, acute respiratory disease syndrome and atherosclerosis [5]. Experimental animal models are one of the best strategies for the understanding of pathophysiology of any disease in order to design and develop the drugs for its treatment [6]. Different animal models have been developed for the past few decades for studying diabetes mellitus and testing anti-diabetic agents [7]. One of the most potent methods of inducing experimental diabetes mellitus is chemical induction by alloxan. It is a well-known diabetogenic agent used to induce type 1 diabetes in experimental animals [6]. Alloxan causes selective necrosis of the  $\beta$ -cells of pancreatic islets and has been widely used to produce experimental diabetes in animals such as rats, rabbits, mice and dogs, with different grades of disease severity by varying the dose of alloxan used [8]. The underlying mechanism involves the selective uptake of the compound due to its structural similarity to glucose, as well as highly efficient uptake mechanism of the pancreatic cells [9]. The present study aims at exploring the preventive effect of taurine on oxidative stress in alloxan induced diabetes mellitus in wistar rats.

### Materials and Methods

#### Experimental Materials

Alloxan (Sigma chemical company St. Louis U. S. A). Taurine (1g Abcam Plc,330 Cambridge, science park Cambridge CB4 OFL United Kingdom): Glibenclamide tablet (5 mg tablet, nature field U. S. A): Analytical weighing balance, Glucometer (Accucheck active), Glucometer strips, Syringes and needles (1 ml insulin syringe and 5 ml syringe)

#### Experimental Animal

Young adult Wistar Rats weighing between 70 -100g, were obtained from the Department of Human Anatomy. The animals were kept for 2 weeks to enable them acclimatize and adapt to the laboratory conditions in the animal house of the Department of Human Physiology, Ahmadu Bello University, Zaria.

#### Ethical Approval

The principles of ethics guiding the use and handling of experimental animals in Ahmadu Bello University, Zaria, was observed.

#### Induction of Experimental Diabetes Mellitus

The animals were fasted for 6 - 8 hours with free access to water prior to induction of diabetes. Diabetes was induced by single intraperitoneal injection of alloxan monohydrate at a dose of 150 mg/kg body weight dissolved in 5ml of sodium citrate solution [10]. Since alloxan is capable of producing fatal hypoglycaemia as a result of massive pancreatic insulin release, rats were treated with 20% glucose solution orally after 30 minutes. The rats were then kept for the next 24 hours on 5% glucose solution bottles in their cages to prevent hypoglycemia. 72 hours after the induction, the blood glucose level of each rat was measured from the tail vein of each animal using the glucometer and glucometer strips after starving the rats overnight. Rats having fasting blood glucose level greater than 200 mg/kg were considered as diabetes [11].

### Experimental Design

Animals were randomly divided into five groups of five animals each (N = 5) as follows:

- **Group 1 (Negative control):** Normal rats and received distilled water orally.
- **Group 2 (Positive control):** Diabetic untreated wistar rats and were given 1 mL of distilled water orally.
- **Group 3:** Diabetic treated with 100 mg/ kg body weight of taurine orally.
- **Group 4:** Diabetic treated with 200 mg/ kg body weight of taurine orally.
- **Group 5:** Diabetic treated with 1 mg/ ml body weight of glibenclamide orally.

### Determination of Body Weights

The body weight of the rats was determined using electronic weighing scale model: Ek 3052 balance. The weights were taken on a weekly basis for four consecutive weeks (week 0, 7, 14 and 21).

### Determination of Blood Glucose Levels

All blood samples were collected from the tail vein of the rats on a weekly basis for 3 weeks. Fasting blood glucose levels were determined by using glucose oxidase method resided in the unit of mg/dL (Beach and Turner, 1958) using a digital glucometer (Accu- Chek Advantage, Roche Diagnostic, Germany) and the results were expressed in the unit of mg/dL [12].

### Collection and Preparation of Samples

Three weeks after the experimental protocols, the animals were exposed to light anaesthesia (ketamine and diazepam). The heart of the animals was exposed through dissection and via cardiac puncture at the apex of the heart, blood sample of about 3 ml was drawn and placed in a plain test tube for extraction of serum for antioxidant enzymes and lipid peroxidation assay.

### Precautions

1. The drugs were freshly and carefully prepared to obtain standard readings.
2. Air bubbles in the syringe were removed to obtain accurate measurements.
3. The syringe and canula was inserted deep into the pharyngoesophageal junction so that all the dose of the drug will enter the system.

### Estimation of Oxidative Stress Parameters

#### Determination of Superoxide Dismutase Activity

It was determined using the method described by [13]. Absorbance was measured every 30s up for a total of 150s at 480 nm from where the SOD activity was calculated.

#### Determination of catalase Activity

The determination was done in accordance with [14] described method. The absorbance was read at 570 nm and the catalase activity was obtained from the graph of standard curve.

#### Assay of Glutathione Peroxidase activities

The reduced Glutathione peroxidase concentration was measured using the method described by [15]. The absorbance was read at 412 nm.

#### Estimation of Lipid peroxidation

Lipid peroxidation (LPO) as evidenced by the formation of thiobarbituric acid reactive substances was measured by the method of Niehaus and Samuelson [16]. The absorbance of the pink supernatant was measured against a reference blank using a spectrometer at 535 nm.

### Statistical Analysis

All data were expressed as mean SEM. The data obtained were analyzed using on way analysis of variance (ANOVA). The value (P 0.05) was considered significant.

### Results

The result in table 1 shows that at day 21 there was a significant ( $P < 0.05$ ) increased in body weight of the normal control group with the value ( $110.60 \pm 2.77$ ) compared to diabetes untreated with a value of ( $81.60 \pm 1.40$ ). Significant ( $P < 0.05$ ) increase was recorded in diabetes treated groups of with both doses of 100 mg/kg of taurine at a value of ( $95.60 \pm 2.80$ ) and 200 mg/kg of taurine at ( $95.40 \pm 1.47$ ), when compared with the diabetes untreated group alone.

Groups	Day 0	Day 7	Day 14	Day 21
Normal Control	$73.80 \pm 1.36$	$88.80 \pm 3.01$	$104.00 \pm 3.46^a$	$110.60 \pm 2.77^a$
Untreated Diabetes	$73.00 \pm 1.90^{ns}$	$78.40 \pm 0.98$	$81.20 \pm 2.48^b$	$81.60 \pm 1.40^{ac}$
Diabetes + Taurine 100 mg	$72.20 \pm 1.02^{ns}$	$83.20 \pm 4.79$	$87.80 \pm 5.34^b$	$95.60 \pm 2.80^c$
Diabetes + Taurine 200 mg	$71.60 \pm 0.81^{ns}$	$77.80 \pm 2.44$	$86.20 \pm 2.61^b$	$95.40 \pm 1.47^c$
Diabetes + Glibenclamide (1 mg/Kg)	$72.80 \pm 0.86^{ns}$	$74.40 \pm 1.21$	$83.20 \pm 2.01^b$	$87.20 \pm 2.15^c$

**Table 1:** Effect of taurine supplement on body weight of alloxan induced diabetic Wistar rats.

Values are expressed as mean  $\pm$  SEM; n = 5 table with different superscript letters <sup>a,b,c</sup> differ significantly ( $p < 0.05$ ) compared with the untreated diabetes group; and <sup>ns</sup>: non-significant: Day 0 (First day of body weight taken), Day 7 (One week of body weight taken), Day 14 and Day 21 (two and three weeks of body weight taken)

Table 2 shows the result for blood glucose level. Blood glucose level reduced significantly at ( $P < 0.05$ ) from a value of ( $333.80 \pm 27.06$ ) for diabetes untreated group to corresponding values of ( $157.40 \pm 14.09$ ) with 100 mg/kg taurine and ( $137.80 \pm 11.72$ ) with 200 mg/kg taurine supplement respectively. Glibenclamide treated group also showed highest decreased in blood glucose level with value ( $99.00 \pm 9.57$ ), when compared with both taurine supplement treated groups.

Groups	Day 0	Day 7	Day 14	Day 21
Normal Control	$117.40 \pm 4.88$	$95.40 \pm 3.38$	$95.60 \pm 1.47$	$100.80 \pm 3.76$
Diabetes Untreated	$421.00 \pm 21.12^a$	$308.40 \pm 41.16^a$	$358.40 \pm 32.40^a$	$333.80 \pm 27.06^a$
Diabetes + Taurine 100 mg	$410.40 \pm 49.31^a$	$333.00 \pm 36.31^b$	$221.80 \pm 10.83^b$	$157.40 \pm 14.09^b$
Diabetes + Taurine 200 mg	$384.20 \pm 49.31^b$	$249.60 \pm 37.99^c$	$210.20 \pm 29.46^b$	$137.80 \pm 11.72^b$
Diabetes + Glibenclamide (1 mg/Kg)	$384.20 \pm 54.82^b$	$266.20 \pm 35.58^c$	$147.80 \pm 20.09^{ab}$	$99.00 \pm 9.57^{ab}$

**Table 2:** Effect of taurine supplement on blood glucose level in alloxan induced diabetic Wistar rats.

Values are expressed as mean  $\pm$  SEM; n = 5 table with different superscript letters <sup>a,b,c</sup> differ significantly ( $p < 0.05$ ) compared with the untreated diabetes group; and <sup>ns</sup>: non-significant: Day 0 (First day of blood glucose taken), Day 7 (One week of blood glucose taken), Day 14 and Day 21 (two and three weeks of blood glucose taken).

The result for SOD activities as shown in table 3, the result showed a significantly decrease ( $p < 0.05$ ) in superoxide dismutase activities in groups given taurine at a dose of 100 mg/kg with a value of ( $1.40 \pm 0.29$ ), and 200 mg/kg with a value of ( $1.62 \pm 0.86$ ), when compared to diabetic untreated with a corresponding value of ( $2.22 \pm 0.21$ ).

Groups	SOD (IU/L)	CAT (IU/L)	GPx (IU/L)	MDA (nmol/ml)
Normal Control	$1.16 \pm 0.11$	$40.00 \pm 0.71$	$40.00 \pm 1.70^a$	$1.04 \pm 0.81$
Diabetic Untreated	$2.22 \pm 0.21^a$	$62.20 \pm 6.16^a$	$47.00 \pm 1.52$	$2.70 \pm 0.33^a$
Diabetic + Taurine (100 mg)	$1.40 \pm 0.29^b$	$43.25 \pm 1.49^b$	$47.50 \pm 0.29^{ns}$	$1.65 \pm 0.45^b$
Diabetic+ Taurine (200 mg)	$1.62 \pm 0.86^b$	$42.80 \pm 0.86^b$	$49.80 \pm 0.97^{ns}$	$1.96 \pm 0.18^b$
Diabetic+ Glibenclamide (1 mg/Kg)	$1.74 \pm 0.31^{12ab}$	$42.60 \pm 0.87^b$	$48.60 \pm 0.93^{ns}$	$1.24 \pm 0.21^c$

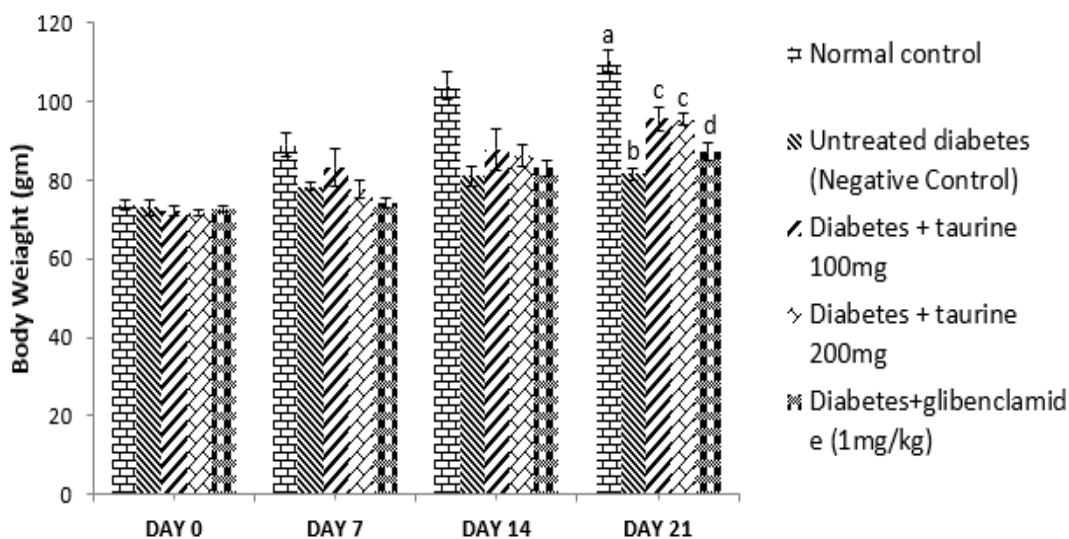
**Table 3:** Effect of taurine on some oxidative stress biomarkers in alloxan-induced diabetic Wistar rats.

Values are expressed as mean  $\pm$  SEM; n = 5 table with different superscript letters <sup>a,b,c</sup> differ significantly ( $p < 0.05$ ) compared with the untreated diabetes group; and <sup>ns</sup>: Non-Significant; SOD: Superoxide Dismutase; CAT: Catalase; GPx: Glutathione Peroxidase; MDA: Malondialdehyde Concentrations

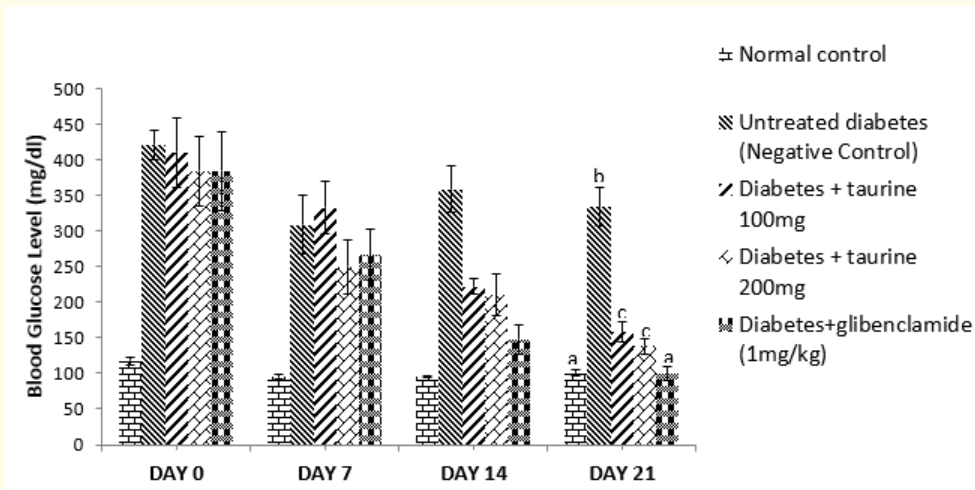
Catalase activities as shown in table 3 recorded a significant ( $p < 0.05$ ) decrease in groups given taurine at a dose of 100 mg/kg ( $43.25 \pm 1.49$ ) and 200 mg/kg ( $42.80 \pm 0.86$ ), when compared to diabetic untreated ( $62.20 \pm 6.16$ ).

GPx activities shows a statistically not significant ( $P > 0.05$ ) change in groups given taurine at a dose of 100 mg/kg ( $47.50 \pm 0.29$ ) and 200 mg/kg ( $49.80 \pm 0.97$ ), when compared to diabetic untreated ( $47.00 \pm 1.52$ ).

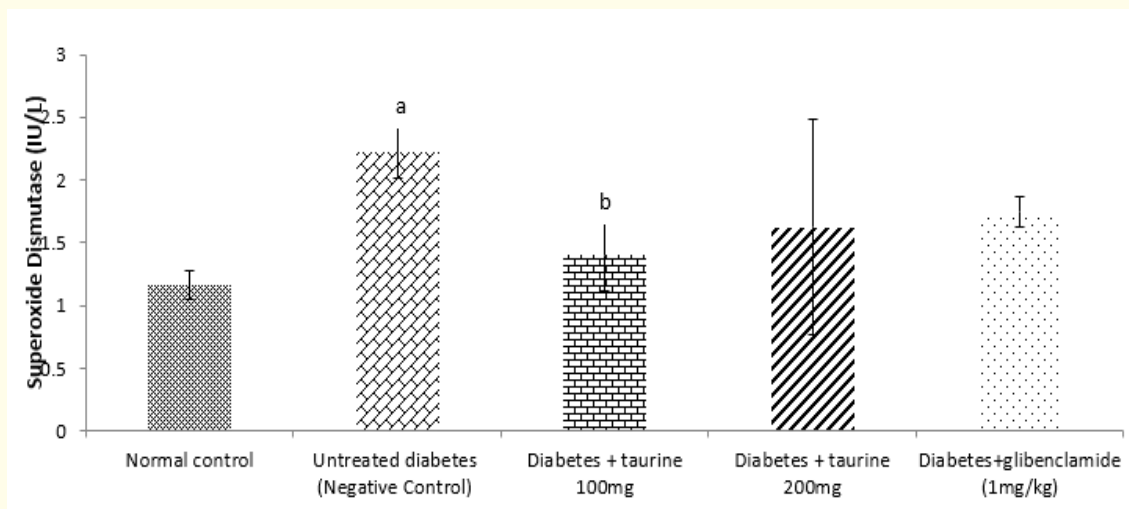
The result for MDA concentrations as shown in table 3 recorded a significant decrease ( $P < 0.05$ ) in groups given taurine at a dose of 100 mg/kg ( $1.65 \pm 0.45$ ) and 200 mg/kg ( $1.96 \pm 0.18$ ), when compared to diabetic untreated group with a value of ( $2.70 \pm 0.33$ ), respectively.



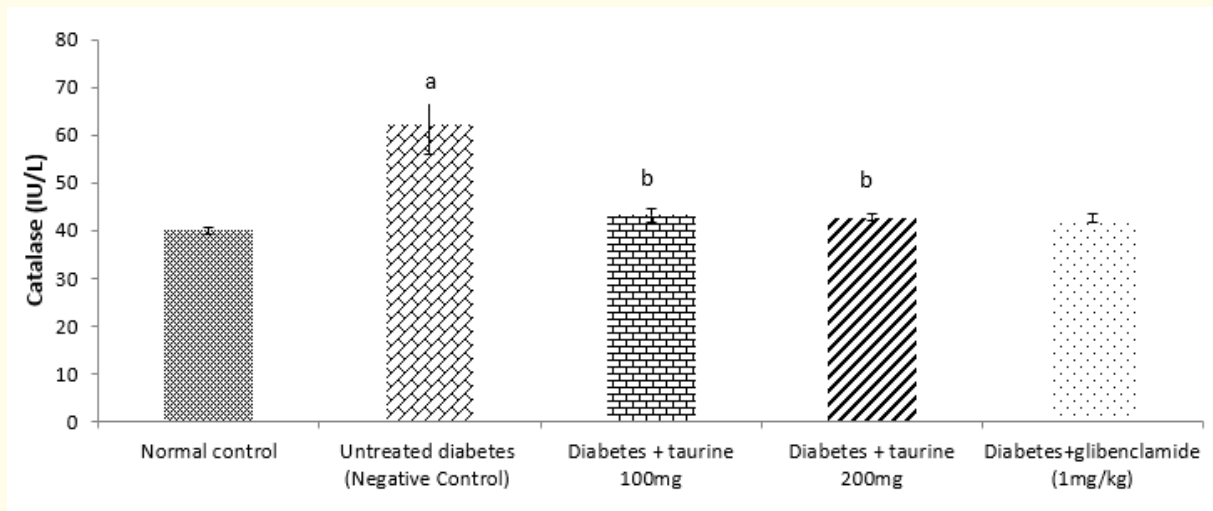
**Figure 1:** Effect of taurine treatment on body weight of diabetic wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b, c, d) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.



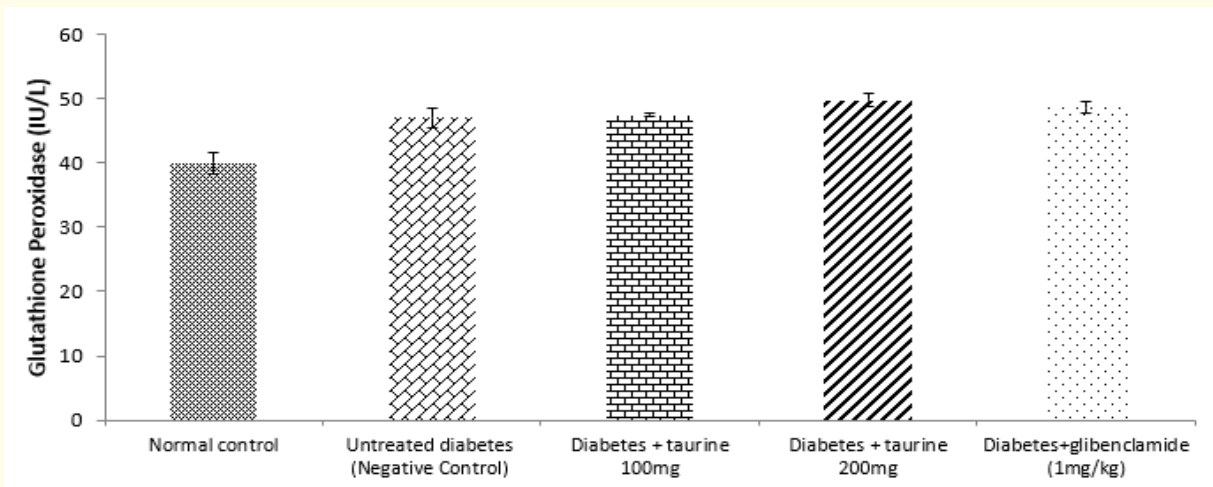
**Figure 2:** Effect of taurine treatment on blood glucose level of diabetic Wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b, c) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.



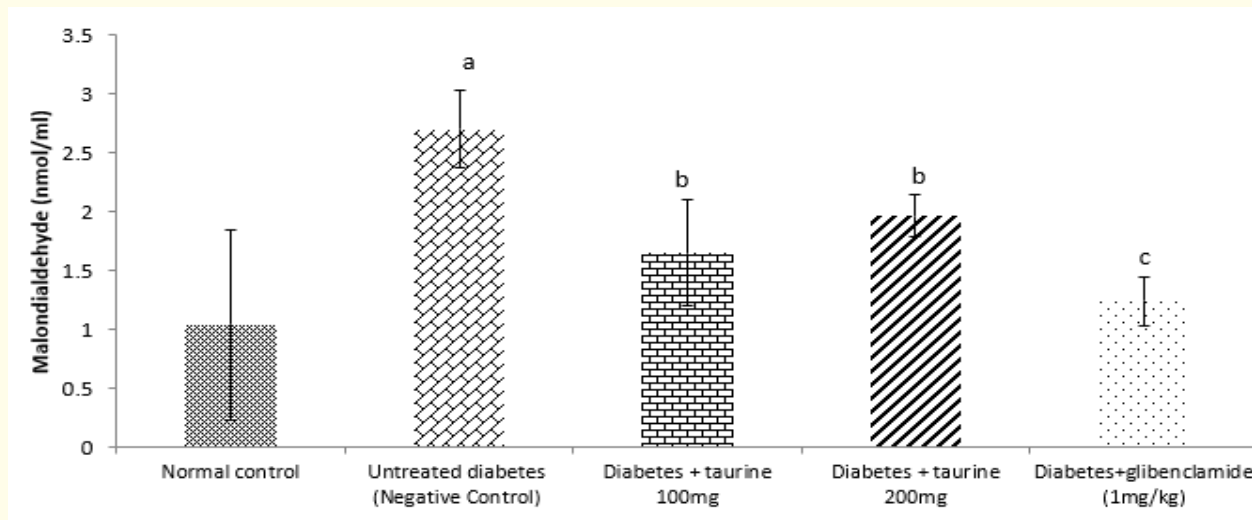
**Figure 3:** Effect of taurine treatment on Serum superoxide dismutase activities on diabetic wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.



**Figure 4:** Effect of taurine treatment on serum catalase activities of diabetic wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b, c) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.



**Figure 5:** Effect of taurine treatment serum glutathione peroxidase activities of diabetic wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b, c) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.



**Figure 6:** Effect of taurine treatment on serum level of malondialdehyde concentrations of diabetic wistar rats. Each bar represents mean of five animals. Bars with different superscript letters (a, b, c) differ significantly ( $P < 0.05$ ) compared with the control group, while bars with the same superscript letters are not significantly different ( $P > 0.05$ ) compared with the control group.

## Discussion

The shift in balance between oxidant/antioxidant in favor of oxidants is termed “oxidative stress” [17]. Oxidative stress contributes to many pathological conditions including diabetes mellitus. One of the most potent methods of inducing experimental diabetes mellitus is chemical induction by alloxan. It is a well-known diabetogenic agent used to induce diabetes in experimental animals. Single intraperitoneal injection of alloxan monohydrate effectively induced diabetes mellitus in rats 72 hrs after administration from our present study [18]. Antioxidant taurine is one of the ingredient in many dietary supplement which perform different function in the body which include conjugation with bile acids, modulation of calcium levels, maintenance of osmolarity, antioxidation and stabilization of membranes [19,20]. This raises the possibility of contributing negatively to the severity of the oxidant-mediated damage present in the diabetic context. Body weight recorded a significant decrease, and blood glucose level was significantly elevated in diabetic induced animal in our study, raising the possibility of stress formation due to production of ROS which are secondary to sustain hyperglycaemia. Since body weight depletion is associated with free radical destruction of visceral adipose mass, with taurine administration, the body weight increase close to normal and blood glucose level also lowers approaching the normal values, demonstrating the antidiabetic property of taurine. This result is in accordance with the work of [21], who demonstrated the hypoglycemic property of taurine on experimental insulin-dependent diabetes mellitus. But the possible antioxidant mechanism of taurine was the gap that our study tends to focus.

There is increasing evidence that oxidative stress plays a major role in the onset and progression of diabetes, and even its complications. High amount of glucose in the blood increases oxygen and releases oxygen radical ( $O^2\bullet-$ ) which easily reacts with the present nitric oxide ( $\bullet NO$ ) disabling its action as endothelial vasodilator [22]. Consequently, there is a reduction in endothelium-dependent relaxation and cell synthesis in the wall of blood vessels, resulting in micro- and macro-pathological changes. Hyperglycaemia, increases the levels of free fatty acids (FFA), and together with hyperinsulinemia lead to increased production of ROS and reactive nitrogen species (RNS) [23].



Various works have reported increased activities of many enzymes of the antioxidant defense system in cells to combat oxidative stress induced by various environmental stresses. Maintenance of a high antioxidant capacity to scavenge the toxic ROS has been linked to increased tolerance of cells to these environmental stresses. Increased activity of SOD is often correlated with increased tolerance of the animal against environmental stresses. The observed increase in SOD and CAT activities in diabetic untreated group may have indicated the response of the animal to stress formation. Taurine administration recorded a decrease in the endogenous antioxidant level [24,25]. From the results obtained, high levels of antioxidants in the serum of untreated diabetic group may probably be due to activation of these endogenous enzymatic antioxidants to counter the effect of hyperglycemia, inflammation and oxidative stress. After taurine administration to the diabetic treated groups, the observed decrease in antioxidants level may probably be due to the used up during the scavenging process. Contrary to our findings, is the work of [26], they reported a significant decrease in endogenous antioxidant activities with the induction of stress. The decrease could also be that taurine down regulate the endogenous antioxidant to do the scavenging itself.

Superoxide dismutase (SOD), plays central role in defense against oxidative stress in all aerobic organisms and catalyzes the dismutation of  $O_2^{\cdot-}$  to  $O_2$  and  $H_2O_2$ . Present in most of the subcellular compartments that generate activated oxygen that result into metabolic oxidative stress [27]. SOD activity has been reported to increase in cells exposed to various environmental stresses, including drought and metal toxicity [27].

Catalase is a tetrameric heme-containing enzyme that catalyzes the dismutation of two molecules of  $H_2O_2$  into water and oxygen. The peroxisomes are major sites of  $H_2O_2$  production. When cells are stressed for energy, they rapidly generate  $H_2O_2$  through catabolic processes.  $H_2O_2$  is degraded by CAT in an energy efficient manner. Environmental stresses cause either enhancement or depletion of CAT activity, depending on the intensity, duration, and type of the stress [28].

Glutathione peroxidase, preferably oxidizes aromatic electron donor such as guaiacol and pyrogallol at the expense of  $H_2O_2$ . It is widely found in animals, plants, and microbes. Radotic and coworkers correlated increased activity of GPX to oxidative reactions under metal toxicity conditions and suggested its potential as biomarker for sublethal metal toxicity in cells [29].

Comparing all the other groups with the control group, the level of these biomarkers are lower in the control group due to absence of diabetes and oxidative stress. However, the lower level of biomarkers activities in the treatment groups compared to the untreated group, signifying the efficiency of taurine as an antioxidant. This shows that taurine is effective in the protection against oxidative stress in diabetes mellitus in Wistar rats.

Lipid peroxidation aggravates the oxidative stress through production of lipid-derived radicals that themselves can react with and damage proteins and DNA. The level of lipid peroxidation has been widely used as an indicator of ROS mediated damage to cell membranes under stressful conditions [30]. The decrease in the cell membrane integrity in diabetic untreated group due to lipoperoxidation evidenced by increased in MDA concentrations may have been a direct response to oxidative stress by the animals in the present study. These results agree with finding of [31], who demonstrated vulnerability of the cell membrane in insulin dependent diabetes mellitus in context of oxidative stress. Also in consonant with our finding is the work of [32], who observed the increase in lipid peroxidation and serum antioxidant enzymes in diabetes subject. Observed decrease MDA concentrations in diabetic taurine treated groups may suggest the ameliorative effect or antioxidant activities of taurine on cell membrane damage associated with diabetes. This observation agrees with the finding of [33], who demonstrated that daily administration of resveratrol an antioxidant decreased MDA concentrations in diabetes animal. Taurine may have cause MDA decrease probably through mopping up of free radical involves in membrane protein and lipid peroxidation destruction.

## Conclusion

1. The present study confirms the involvement of decrease body weight, elevated blood glucose level and oxidative stress in the progression of diabetes mellitus.
2. The findings obtained demonstrated that administration of taurine ameliorated the diabetic-induced alteration in the above parameters.

## Acknowledgement

The authors are grateful to Head, Department of Human Physiology, Ahmadu Bello University, Zaria and Mr A. Bamidele of Human Anatomy Department, Mr O. Ayegbusi of Chemical Pathology Department and Mallam Yau Bello of Department of Human Physiology, Faculty of Basic Health Sciences, College of Health Sciences, Ahmadu Bello University, Zaria, Nigeria, toward the successful completion of this research work.

## Bibliography

1. Kim SJ, *et al.* "Taurine-diabetes interaction: from involvement to protection". *Current Diabetes Review* 3.3 (2007): 165-175.
2. Lorenzo R and Camilo ME. "Taurine: a conditionally essential amino acid in humans? An overview in health and disease". *Nutricion Hospitalaria* 17.6 (2002): 262-270.
3. Bouceknoghe T, *et al.* "Is taurine a functional nutrient". *Current Opinion in Clinical Nutrition and Metabolic Care* 9.6 (2006): 728-733.
4. Valko M, *et al.* "Free radicals, metals and antioxidants in oxidative stress-induced cancer". *Chemico-Biological Interactions* 160.1 (2006): 1-40.
5. Asami S, *et al.* "Cigarette smoking induces an increase in oxidative DNA damage, 8-hydroxydeoxyguanosine, in a central site of the human lung". *Carcinogenesis* 18.9 (1997): 1763-1766.
6. Chatzigeorgiou A, *et al.* "The use of animal models in the study of diabetes mellitus." *In vivo* 23.2 (2009): 245-258.
7. Etuk EU. "Animal models for studying diabetes mellitus". *Agriculture and Biology Journal of North America* 1.2 (2010): 130-134.
8. Iranloye BO, *et al.* "Anti-diabetic and antioxidant effects of Zingiber officinale on alloxan-induced and insulin resistant diabetic male rats". *Nigerian Journal of Physiological Sciences* 26.1 (2011): 89-96.
9. Viswanathaswamy AH, *et al.* "Antihyperglycemic and antihyperlipidemic activity of plectranthus amboinicus on normal and alloxan-induced diabetic rats". *Indian Journal of Pharmaceutical Sciences* 73.2 (2011): 139-145.
10. Kameswara RB, *et al.* "Effect of oral administration of bark extract of Pterocarpus santalinus L. on blood glucose level in experimental animals". *Journal of Ethnopharmacology* 74.1 (2001): 69-74.
11. Stanley P, *et al.* "Antioxidant action of Tinospora cordifolia root extract in alloxan-induced diabetes rats." *Phytotherapy Research* 15.3 (2001): 213-218.
12. Rhenay CC and Kurt KK. "Performance of three blood glucose meters". *Annal of Pharmacotherapy* 34.3 (2000): 317-321.

13. Martin JP, *et al.* "Negative and positive assays of superoxide dismutase based on hematoxylin autooxidation". *Archives of Biochemistry and Biophysics* 255.2 (1987): 329-336
14. Beers RF Jr and Sizer IW. "A Spectrophotometric Method for Measuring the Breakdown of Hydrogen Peroxide by Catalase." *Journal of Biological Chemistry* 195.1 (1952): 133-140.
15. Takebe G. "A comprehensive study on the hydroperoxide and thiol specificity of the glutathione peroxidase family and seleno-protein P". *Journal of Biological Chemistry* 277.43 (2002): 41254-41258.
16. Janero DR. "Malondialdehyde and thiobarbituric acid-reactivity as diagnostic indices of lipid peroxidation and peroxidative tissue injury". *Free Radical Biology Medicine* 9.6 (1990): 515-540.
17. Lazarova M, *et al.* "The antioxidant acetylcysteine reduces oxidative stress by decreasing level of AOPPS". *Biomedical Papers* 148.2 (2004): 131-133.
18. Jimoh A, *et al.* "Anti-diabetic effect of methanolic leaf extract of *Cissus cornifolia* on alloxan-induced hyperglycemic in wistar rats". *Annals of Biological Research* 4.3 (2013): 46-54.
19. Schaffer SW, *et al.* "Role of antioxidant activity of taurine in diabetes". *Canadian Journal of Physiology and Pharmacology* 87.2 (2009): 91-92.
20. Annad P, *et al.* "Effect of oral administration of taurine on some hematological parameter in wistar rat". *Pakistan Journal of Biological Science* 13.16 (2010): 785-793.
21. Gavrovskaya L, *et al.* "Protective effect of taurine on rats with experimental insulin-dependent diabetes mellitus". *Bulletin of Experimental Biology and Medicine* 146.2 (2008): 226-228.
22. Kennedy G, *et al.* "Oxidative stress levels are raised in chronic fatigue syndrome and are associated with clinical symptoms". *Free Radical Biology and Medicine* 39.5 (2005): 584-589.
23. Chandra K, *et al.* "Protection against FCA induced oxidative stress induced DNA damage as a model of arthritis and in vitro anti-arthritis potential of *costus speciosus* rhizome extract". *International Journal of Pharmacognosy and Phytochemical Research* 7.2 (2015): 383-389.
24. Puerta CD, *et al.* "Taurine and glucose metabolism: a review". *Nutrition in Hospital* 25.6 (2010): 910-919.
25. Seneff S. "Taurine: a mysterious molecule with intriguing possibilities". *Wise Tradition Workshop* (2012): 5-70.
26. Abd El Kader MA, *et al.* "Evaluation of taurine role on some biochemical and histological alterations in  $\gamma$  - irradiated rats". *International Journal of Pharmaceutical Sciences Review and Research* 30.1 (2015): 263-271.
27. Sies H and Stahl W. "Nutritional protection against skin damage from sunlight". *Annual Review of Nutrition* 24 (2004): 173-200.
28. Campanella L, *et al.* "Protective action of antioxidants against amino acids degradation caused by free radicals". *International Journal of Environmental Health* 1.1(2007): 98-119.
29. Kaute D, *et al.* "Antioxidant characteristics of *Dichrostachys glomerata* spice extracts". *CyTA- Journal of Food* 8.1 (2010): 23-37.
30. Dieudonne K, *et al.* "Effectiveness of *Dichrostachys glomerata* spice phenolics in reduction of oxidative stress associated with obesity and type 2 diabetes; a randomized, double-blind placebo-controlled clinical trial". *Journal of Food Research* 2.2 (2013): 887-895.

31. Gauri SV and Vijaya AH. "RBC membrane composition in insulin dependent diabetes mellitus in context of oxidative stress". *Indian Journal of Clinical Biochemistry* 23.3 (2008): 223-226.
32. Ahmed FN., *et al.* "Lipid peroxidation and serum antioxidant enzymes in patients with type 2 diabetes mellitus". *Annals of the New York Academy of Sciences* 1084 (2006): 481-489.
33. A Jimoh A., *et al.* "Protective effect of resveratrol co-administration with cholesterol diet on erythrocyte osmotic fragility and malondialdehyde concentration in rabbits". *British Journal of Pharmaceutical Research* 6.1 (2015): 14-21.

**Volume 6 Issue 8 August 2018**

**©All rights reserved by Jimoh Abdulazeez, *et al.***