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## Research Article

# Therapeutic Effect of White Cabbage (*Brassica oleracea*) Aqueous Extract on Hyperglycemia in Prediabetes-induced Male Albino Rats

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## Abstract

**Background and Objective:** Some plant materials have been found to possess hypoglycemic property which could ameliorate hyperglycemic condition. Thus, this study aimed at determining the effect of cabbage on hyperglycemia. **Materials and Methods:** Extracts from fresh and dried cabbage and thirty male albino rats grouped into six groups were used for the study. The groups were the positive control (fed regular diet), negative control (fed high sucrose feed) and treatment (high sucrose feed and extract for 2 weeks) groups. Each of the treatment groups received 50 mg or 100 mg kg<sup>-1</sup> b.wt., of either of fresh and dried cabbage extracts. The fasting blood sugar (FBS) was determined at baseline and at two weeks interval. The experiment lasted six weeks when the serum insulin, oral glucose tolerance test and liver oxidative stress were determined. Data were subjected to Graph pad prism 5 and the blood glucose was analyzed using analysis of variance (ANOVA) at p<0.05 followed by Duncan multiple range tests. **Results:** There was a reduction in the blood sugar and resistance to insulin by the extracts. The control negative had the highest fasting blood sugar (62 mg dL<sup>-1</sup>) and the result of oral glucose tolerance also showed higher peak at 60 min. There was a reduction in the MDA (0.54±0.08-0.79±0.05) accompanied by significant reduction in the SOD (0.52±0.04-0.57±0.04) and CAT (1.85±0.65-15.75±1.69) of the test groups. **Conclusion:** Potential of cabbage to control blood sugar and ameliorate oxidative stress of the liver was demonstrated in this study.

**Key words:** Cabbage extract, blood sugar, hyperglycemia, prediabetes, oxidative stress, insulin

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**Competing Interest:** The authors have declared that no competing interest exists.

**Data Availability:** All relevant data are within the paper and its supporting information files.

## INTRODUCTION

Hyperglycemia or high blood sugar is a condition in which an excessive amount of glucose circulates in the blood plasma and it is the underlying abnormality characterizing the diabetic condition. Chronic hyperglycemia introduces a plethora of complications such as cardiovascular disease with serious damage to the nerves and blood vessels which is the most frequent cause of death in the diabetic population<sup>1-3</sup>. The increasing rate of diabetes mellitus is becoming a serious threat to human health in all parts of the world. It has been reported that in the world, 347 million people have diabetes with 9% of 18 years and above having diabetes in 2014. In 2012 diabetes was the direct cause of 1.5 million deaths and WHO projects that diabetes will be the 7th leading cause of death in 2030. More than 80% of diabetes related deaths occur in low and middle-income countries<sup>4</sup>.

Physicians typically recommend an anti-diabetic medication as treatment. Treatment with an old, well-understood diabetes drug such as metformin is the safest, most effective, least expensive, most comfortable means of managing the condition. However, these drugs have limited efficacy and sometimes produced severe side effects such as weight gain, hypoglycaemia, liver injury, channel disturbances, cardiopathy and bloating<sup>5,6</sup>.

Food supplement are becoming more popular in managing hyperglycemia in prediabetic condition especially for those whose blood glucose level are on the borderline and pharmacological therapies have not been initiated<sup>7</sup>.

Cabbage (*Brassica oleraceae* var. *Capitata* L.) belongs to the Brassicaceae family and is closely related to the broccoli, cauliflower and brussels sprouts. It is thought to have originated in the Mediterranean region and is in fact one of the oldest vegetables grown. It has high water content, is high in fibre and has significant quantities of protein, calcium and iron. The cabbage is a rich source of vitamin A and vitamin C, in addition to containing some B vitamins<sup>8-11</sup>. It also contains significant amounts of glutamine, an amino acid which has anti-inflammatory properties<sup>12</sup>. Research has shown that cabbage has a number of anti-oxidative compounds that might be beneficial in the prevention of cancer<sup>13</sup>. Red cabbage is especially rich in these antioxidants<sup>14</sup>.

Basic research on cabbage phytochemicals is ongoing to discern if certain cabbage compounds may affect health or have anti-disease effects<sup>15</sup>. Such compounds include sulforaphane and other glucosinolates which may stimulate the production of detoxifying enzymes during metabolism<sup>16</sup>. It was as a result of the numerous benefits of cabbage that this research was undertaken to determine its effect in pre-diabetic condition.

## MATERIALS AND METHODS

**Processing of the cabbage extracts:** The identity of the cabbage used for this study was confirmed at Botany Department, Federal University of Agriculture Abeokuta with herbarium number 028. The cabbage was processed into extracts by drying and extracting the fresh cabbage. The dried and ground cabbage (125 g) was extracted with 1500 mL (in 250 mL conical flasks) of water in continuous shaker machine at 40°C for 48 h and filtered using 0.03 mm sieve<sup>17</sup>. For fresh cabbage extract, the blended cabbage was sieved and dried in an air oven at 40°C for 48 h.

**Biological study:** The research was carried out at Babcock University animal facility between 6th of February and 26th of March, 2017. Thirty albino rats weighing 110-120 g were obtained from the facility and housed for 10 days for acclimatization and allowed free access to regular rat pellet before the experiment<sup>18</sup>.

Rats were grouped into six with each group consisting of five rats of which two were control and four treatment groups. The control groups were positive control group A1 and negative control group A2. The positive control group A1 was placed on regular rat feed while the negative control group A2 was fed high sucrose (30% concentration) feed in order to induce type 2 diabetic condition and was not subjected to any treatment<sup>19,20</sup>. Two concentrations (50 and 100 mg b.wt.) of the extracts from the two methods of extraction (dried and fresh) were administered, respectively. The treatment groups were fed high sucrose feed for 4 weeks after which they were subjected to treatment (cabbage extract) for 2 weeks with continued intake of sucrose feed. Group C1 was given 50 mg b.wt., of cabbage fresh extract, group C2 received 100 mg b.wt., of cabbage fresh extract, group C3 was given 50 mg b.wt., of cabbage dried extract and group C4 also received 100 mg b.wt., of cabbage dried extract by oral administration, respectively. Fasting blood sugar was determined before the commencement of the experiment and every 2 weeks during the period of the experiment using glucometer. Water and food were giving freely during the experiment. After 24 h of last dose, the animals were fasted and blood sample was collected from the tail vein for determination of oral glucose tolerance test and blood sample was also obtained from the eye (orbital plexus blood samples) for estimation of serum insulin.

The rats were rendered unconscious by means of cervical dislocation and the liver samples were collected and homogenized in deproteinizing solution for determination of oxidative stress. Lipid peroxidation, hepatic superoxide dismutase (SOD) activity, hepatic catalase (CAT) activity and hepatic reduced glutathione (GSH) level were determined.

### **Biochemical analysis**

**Serum insulin:** All reagents and rats liver samples (stored below 0°C) were brought to room temperature before use and ELISA (enzyme-linked immunosorbent assay) were used for the analysis. Reagents used for ELISA procedure includes; Insulin calibrator (blank and reference), Biotinylated Detection Ab (Avidin-biotin) working solution, HRP (Horseradish Peroxidase) conjugate working solution, substrate solution (H<sub>2</sub>O<sub>2</sub>-TMB 0.26 g L<sup>-1</sup>), stop solution (Sulphuric acid 0.15 mol L<sup>-1</sup>) and wash solution (NaCl 45 g L<sup>-1</sup>; Tween-20 55 g L<sup>-1</sup>).

The samples were centrifuged at 3,000 revolutions for 15 min and again after thawing before the assay. All the reagents were mixed thoroughly by gently swirling to avoid foaming before pipetting. All samples and standards were assayed in duplicate. About 100 µL of standard, blank or sample was added per well. Added to the blank well was the reference standard and sample diluent. Solutions were added to the bottom of micro ELISA plate well, wall touching and foaming was avoided as possible. This was mixed gently with the plate covered with the sealer and incubation was done for 90 min at 37°C.

The liquid of each well was removed (not wash). Immediately 100 µL of Biotinylated Detection Ab working solution was added to each well and covered with the plate sealer. The plate was gently tapped to ensure thorough mixing and incubation was for 1 h at 37°C. Each well was aspirated and washed, repeating the process three times. Washing was done by filling each well with wash buffer approximately 350 µL using a squirt bottle, complete removal of liquid at each step was ensured. After the last wash, the remained wash buffer was removed by aspirating or decanting. The plates were inverted and patted against thick clean absorbent paper. Then 100 µL of HRP conjugate working solution was added to each well, covered with the plate sealer, incubated for 30 min at 37°C and wash process was repeated for 5 times. Also, 90 µL of substrate solution was added to each well and covered with a new plate sealer. The plate was incubated for about 15 min at 37°C and the plate was protected from light. The reaction was terminated, when apparent gradient appeared in standard wells.

Then finally, 50 µL of stop solution was added to each well and there was formation of yellow colouration immediately. The optical density (OD value) of each well was determined at once using a micro-plate reader<sup>21</sup> set to 450 nm.

**Oral glucose tolerance test:** The test was determined according to the method described by Hedrich<sup>22</sup>. Rats were fasted overnight (14 h) followed by placing them in fresh

cages without food. Cages and the rats were identified and the weight of each rat was recorded to within 0.1 g.

The volume of 20% glucose solution required for oral gavage of 2 g glucose kg<sup>-1</sup> at an oral injection volume of 10 µL g<sup>-1</sup> b.wt., was calculated for each rat and recorded. Experiment record sheet, strips for glucose measurement and a 1 mL syringe for each animal containing the calculated volume of glucose to be gavaged were prepared.

The blood glucose monitor was calibrated with the standard strip. The basal glucose concentration (T=0) in each rat was measured by removing one rat at a time from its cage, placing it on top of its cage and making a small incision over the lateral tail vein (1-2 cm from the tail tip) using a scalpel blade or scissors. A small (~3 µL) blood sample was placed directly on to the test strip and inserted into the blood glucose monitor. Direct pressure was applied to the incision until the blood clot and the rat was returned to its cage.

After all the rats have been measured for basal glucose concentrations, the glucose solution was administered by oral gavage to each animal at a 30 sec interval between animals and the timer started with the first rat gavage. All administrations were finished within 15 min in order to perform the next blood glucose measurement at T = 30 min.

At T = 30 min, the blood glucose was measured again, starting with the first rat gavaged and using the same time interval used for gavage, until all the rats in the cohort have been measured. The timing was followed as closely as possible because it is very important. To restart the bleeding, the clot from the first incision was removed and the tail massaged if blood flow is inadequate. This was repeated at T = 60 and 120 min after gavage and it was ensured that no animal bleed excessively. At the end of the experiment, the rats were returned to their original cages and provided plenty of food and water.

### **Oxidative stress (liver sample)**

**Assessment of lipid peroxidation:** This was assayed by measuring the thiobarbituric acid (TBA) reactive products present in the test sample using the procedure of Vashney and Kale<sup>23</sup> expressed as micromolar of malondialdehyde (MDA)/g tissue.

An aliquot of 0.7 mL of the test sample was mixed with 2 mL of 0.375% TBA and the mixture was placed in a water bath for 1 h between 90-95°C. This was then cooled in ice and centrifuge at 3000 rpm for 15 min. The clear pink supernatant was collected and absorbance measured against a reference blank of distilled water at 532 nm in a spectrophotometer. The MDA level was calculated according to the method of Adam-Vizi and Seregi<sup>24</sup>.

**Calculation:** Lipid peroxidation was expressed in units  $\text{mg}^{-1}$  protein, where  $E_{532}$  molar extinction coefficient is  $1.56 \times 10^5 \text{ M}^{-1} \text{ M}^{-1}$ .

Therefore:

$$\text{MDA (units mg}^{-1} \text{ protein)} = \frac{\text{Absorbance of test} \times \text{Volume of mixture}}{E_{532} \times \text{Volume of sample} \times \text{mg protein}}$$

**Hepatic superoxide dismutase (SOD) activity:** The method described by Valerino and McCormack<sup>25</sup> was adopted. 0.1 mL of sample was diluted in 0.9 mL of distilled water to make a 1 in 10 dilution. An aliquot of 0.2 mL of the diluted enzyme preparation was added to 2.5 mL of 0.05 M carbonate buffer (PH 10.2) to equilibrate in the spectrophotometer and the reaction was started by adding 0.3 mL of freshly prepared 0.3 mM adrenaline to the mixture which was quickly mixed by inversion. The reference cuvette contains 2.5 mL of 0.005 M carbonate buffer, 0.3 mL of adrenaline (substrate) and 0.2 mL of distilled water. The increase in absorbance at 480 nm was monitored every 30 sec for 150 sec.

**Calculation:**

$$\text{Increase in Abs per minute} = \frac{A_{2.5} - A_0}{T}$$

where,  $A_0$  is initial absorbance,  $A_{2.5}$  is final absorbance and  $t$  is total time taken (150 sees or 2.5 mins):

$$\text{Inhibition (\%)} = \frac{\text{Final absorbance of sample min}^{-1}}{\text{Increase in the absorbance of blank min}^{-1}} \times 100\%$$

About 1 unit of SOD activity was given as the amount of SOD necessary to cause 50% inhibition of the oxidation of adrenaline to adrenochrome during 1 min:

$$\text{SOD activity (units)} = \frac{\text{Inhibition (\%)}}{50\%}$$

Therefore:

$$\text{Specific activity of SOD (units mg}^{-1} \text{ protein)} = \frac{\text{SOD activity} \times \text{dilution factor}}{\text{Mg protein}}$$

**Hepatic catalase (CAT) activity:** Catalase activity was determined according to the method of Sinha<sup>26</sup>. Different amounts of  $\text{H}_2\text{O}_2$  ranging from 20-160 moles were taken in small test tubes and 2 mL of dichromate/acetic acid was added to each. Addition of the reagents instantaneously produces unstable blue precipitate of perchromic acid.

Subsequent heating for 10 min in a boiling water bath changed the colour of the solution to stable green due to formation of chromic acetate. After cooling at room temperature, the volume of the reaction mixture was made to 3 mL with distilled water and the absorbance measured with a spectrophotometer at 570 nm.

**Hepatic reduced glutathione (GSH) level:** The total sulfhydryl groups, protein-bound sulfhydryl groups and free sulfhydryl groups like glutathione (GDH) was determined using Ellman's reagent, 5,5-dithio-bis-2-nitrobenzoic acid (DTNB) as describe by Jollow *et al.*<sup>27</sup>.

Serial dilution of the stock GSH was prepared. To each test tube, appropriate volumes of phosphate buffer were added and then followed by the addition of 4.5 mL Ellman's reagent. The absorbance of the yellow colour formed upon the addition of Ellman's reagent was read within 5 min at 412 nm using spectrophotometer. A plot of absorbance versus concentration of reduced GSH was then obtained. About 0.5 mL of the sample was pipette into a test tube and 1.5 mL of phosphate buffer was added followed by the addition of 0.5 mL Ellman's reagent. The absorbance was read within 5 min at 412 nm.

**Calculation:**

$$\frac{\text{Absorbance} \times \text{total volume} \times 10^3}{\text{Molar extinction coefficient} \times \text{volume of sample}}$$

**Statistical analysis:** Results of blood glucose of the control and test animals were subjected to Analysis of variance (ANOVA) to determine significant difference ( $p < 0.05$ ) and Duncan multiple range test was used to separate the means using SPSS version 20.0. While serum insulin, oral glucose tolerance test and liver oxidative stress were analyzed using Graph pad prism 5.

## RESULTS AND DISCUSSION

Type 2 diabetes or non-insulin-dependent diabetes mellitus, is the most common form of the disease, accounting for 90-95% of cases in which the body does not produce enough insulin or properly use it<sup>28</sup>. The efficacy of cabbage aqueous extract in reducing blood sugar was determined and the extracts from both dried and fresh blended cabbage was used for the study.

The blood sugar of the different groups determined over 6 weeks (Table 1) showed that there was significant difference in the blood sugar of the different groups with the

test group administered 50 mg kg<sup>-1</sup> b.wt., of fresh extract having highest blood glucose (109.25 ± 17.68) at baseline. But at the 2nd week there was a drop in the blood sugar of all the animals in the different group as a result of low feed intake during the period the animals were getting familiar with the high sucrose feed. At the 4th week there was no significant difference in the blood sugar of the animals in these different groups with the blood sugar of rats in control negative dropping lower than that of other groups. However, it was found that the blood glucose of the rats in negative control group increased rapidly at 6th week while reduction in the blood sugar of the test groups was recorded<sup>29</sup>. The blood sugar of the test groups was in the same range with the positive control group.

The serum insulin of the test and control groups is as presented in Fig. 1. In hypoglycemic condition, the level of insulin in the blood is usually high, as increased level of glucose in the blood stimulate production of more insulin to take care of the excess glucose and this was observed in the study, as the level of insulin of the animals in the test groups was lower than the serum insulin of the negative control group. However, the results of the serum insulin showed that the positive control group had the highest level of insulin (70 pmol L<sup>-1</sup>) which could be as a result of other factors like quantity of feed consumed or feed conversion ratio which are part of the observations made in this study.

Glucose tolerance test (Fig. 2) which is a measure of resistance to insulin showed that the test groups and control positive attained their peak at 30 min with the group administered 100 mg kg<sup>-1</sup> b.wt., fresh cabbage extract having the highest blood glucose (194.75 ± 24.5 mg dL<sup>-1</sup>). However, the negative control group did not attain its peak until 60 min when the blood glucose of the other groups have already been significantly lowered and its peak (187.33 ± 5.54) was higher than the peak for other groups an indication of higher resistance to insulin<sup>19</sup>. Thus the lowering of resistance to insulin by the cabbage extract was demonstrated in this finding.

Oxidative stress results when reactive forms of oxygen also called free radicals are produced faster than they can be safely neutralized by natural antioxidant mechanisms in the body and it could be as a result of decrease in antioxidant defense<sup>30</sup>. Many diseases are as a result of oxidative stress and Bambolkar and Sainani<sup>31</sup> reported that oxidative stress increased in patients with diabetes mellitus.

Lipid peroxidation determined as MDA concentration (mg mL<sup>-1</sup>) of the control and test rats is as presented in Fig. 3. The MDA concentration of the negative control group was 0.85 ± 0.26 mg mL<sup>-1</sup> which was the highest compared to the positive control and the test groups and the least was that of

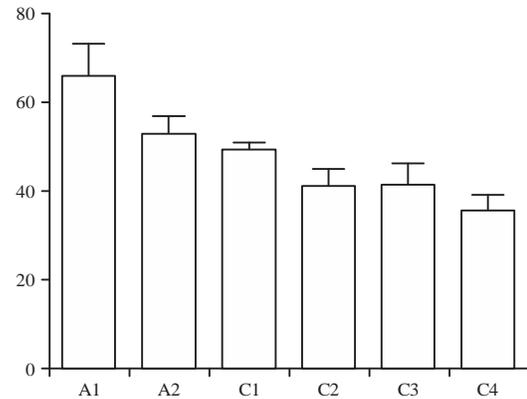


Fig. 1: Serum insulin of the control and test groups

A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group 50 mg kg<sup>-1</sup> b.wt., administered extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage

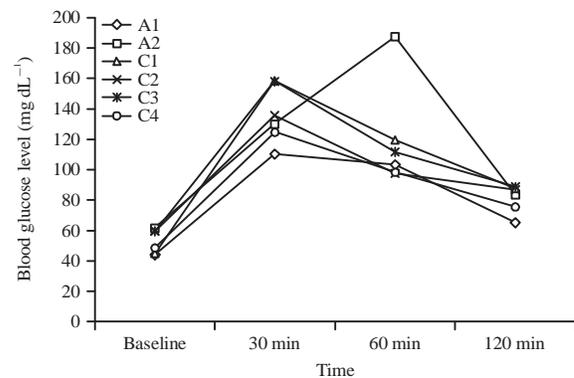


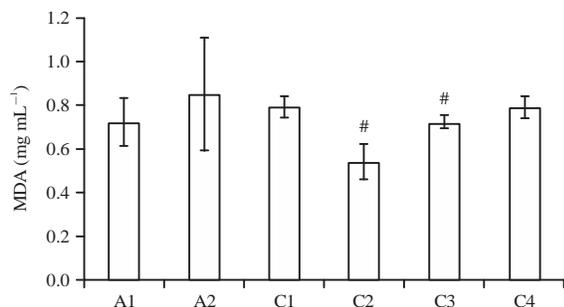
Fig. 2: Blood glucose level of the control and the chemo therapeutic test groups during the oral glucose tolerance test

A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage

Table 1: Blood glucose (mg dL<sup>-1</sup>) of the control and test groups for 6 weeks

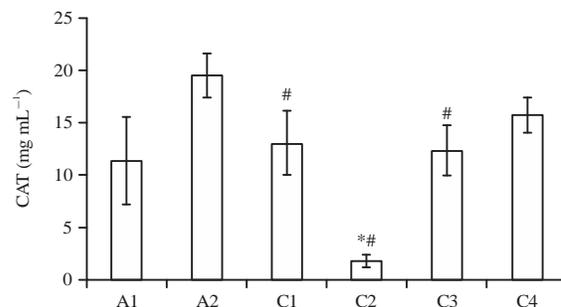
Groups	Baseline	2 weeks	4 weeks	6 weeks
A1	87.25 ± 7.89 <sup>bc</sup>	87.80 ± 5.68 <sup>b</sup>	65.75 ± 6.24 <sup>a</sup>	44.00 ± 9.42 <sup>c</sup>
A2	106.33 ± 14.57 <sup>ab</sup>	53.75 ± 11.15 <sup>a</sup>	48.67 ± 16.26 <sup>a</sup>	62.00 ± 12.00 <sup>a</sup>
C1	109.25 ± 17.68 <sup>a</sup>	55.00 ± 9.09 <sup>b</sup>	65.00 ± 15.59 <sup>a</sup>	44.75 ± 4.349 <sup>bc</sup>
C2	79.75 ± 19.90 <sup>c</sup>	57.25 ± 6.89 <sup>ab</sup>	61.75 ± 12.42 <sup>a</sup>	59.75 ± 8.66 <sup>ab</sup>
C3	98.33 ± 5.03 <sup>abc</sup>	52.67 ± 4.50 <sup>b</sup>	65.00 ± 20.95 <sup>a</sup>	58.33 ± 10.01 <sup>ab</sup>
C4	87.80 ± 5.68 <sup>bc</sup>	54.40 ± 5.37 <sup>b</sup>	60.20 ± 16.20 <sup>a</sup>	51.60 ± 13.07 <sup>abc</sup>

Means in the same column with the same superscript are not significantly different (p < 0.05), A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage



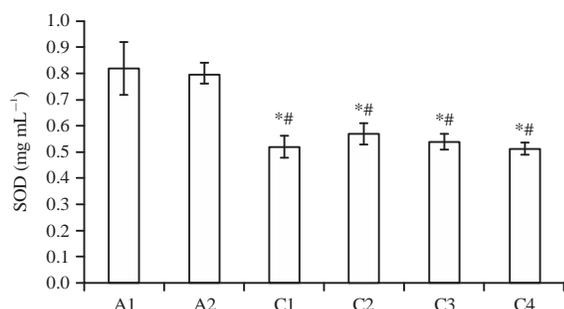
**Fig. 3: MDA concentration (Lipid peroxidation) of the control and the test groups**

\* $p < 0.05$  when comparing with the A1 group, # $p < 0.05$  when comparing with the A2 group, A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage



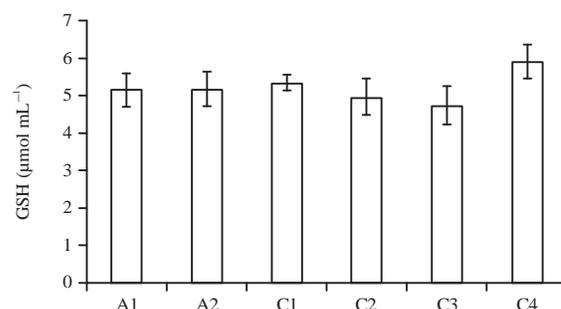
**Fig. 5: Catalase activity of the control and the chemo therapeutic test groups**

\* $p < 0.05$  when comparing with the A1 group, # $p < 0.05$  when comparing with the A2 group, A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage



**Fig. 4: Superoxide dismutase of the control and the test groups**

\* $p < 0.05$  when comparing with the A1 group, # $p < 0.05$  when comparing with the A2 group, A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered extract 100 mg kg<sup>-1</sup> b.wt., from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage



**Fig. 6: Glutathione level of the control and the chemo therapeutic test groups**

\* $p < 0.05$  when comparing with the A1 group, # $p < 0.05$  when comparing with the A2 group, A1: Positive control, A2: Negative control, C1: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C2: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from blended fresh cabbage, C3: Group administered 50 mg kg<sup>-1</sup> b.wt., extract from dried cabbage, C4: Group administered 100 mg kg<sup>-1</sup> b.wt., extract from dried cabbage

the group administered 100 mg kg<sup>-1</sup> of fresh cabbage extract ( $0.62 \pm 0.06$  mg mL<sup>-1</sup>). This observation is in support of the earlier reports that hyperglycaemia contributes to oxidative stress by the direct generation of excessive reactive oxygen species<sup>5,32,33</sup>.

There was reduction in the superoxide dismutase (SOD) activity of groups administered cabbage extract with significant reduction ( $0.59 \pm 0.08$  and  $0.54 \pm 0.02$  mg mL<sup>-1</sup>) in the 2 groups administered 50 and 100 mg kg<sup>-1</sup> b.wt., of dried cabbage extract while the SOD of the positive control group was the highest ( $0.82 \pm 0.10$ ) as presented in Fig. 4.

Reduction in the catalase (CAT) activity of the test groups was also observed with significant reduction in the groups administered 50 mg kg<sup>-1</sup> fresh cabbage

( $11.72 \pm 3.68$  mg mL<sup>-1</sup>) and 100 mg kg<sup>-1</sup> dried cabbage extract ( $12.18 \pm 1.01$  mg mL<sup>-1</sup>) while the CAT of the negative control group was the highest ( $19.53 \pm 2.12$  mg mL<sup>-1</sup>). This is as presented in Fig. 5.

Glutathione level of the control and test groups are as presented in Fig. 6. The glutathione level of the groups administered 100 mg mL<sup>-1</sup> of fresh cabbage ( $5.65 \pm 0.21$  μmol mL<sup>-1</sup>) and 100 mg mL<sup>-1</sup> of dried cabbage extract ( $5.85 \pm 0.24$  μmol mL<sup>-1</sup>) were higher than the glutathione level of the positive ( $5.15 \pm 0.45$  μmol mL<sup>-1</sup>) and negative ( $5.18 \pm 0.46$  μmol mL<sup>-1</sup>) control groups.

The results of oxidative stress showed increased concentration of MDA and the antioxidant enzymes (SOD

and CAT) in the negative control group while the level of MDA and the antioxidant enzymes were significantly lowered in the test groups. This finding is contrary to earlier reports that increased MDA levels is associated with decrease antioxidant levels in serious hepatic problem<sup>30,34</sup>. This observation could either be that the condition was still a mild situation where the increased level of anti-oxidant enzymes in the negative control group is to prevent onset of liver disease<sup>29</sup> or that the cabbage extract had a negative effect on SOD and CAT while the GSH was not seriously affected.

Increase in GSH can be attributed to the fact that its function is to protect erythrocyte membrane against oxidation which is achieved by breaking down hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) into nontoxic compounds in the body tissues. Thus, this neutralizes toxic radicals which propagate oxidative process and compensate for the loss of defense capability against oxygen radicals by the low SOD and CAT<sup>34</sup>.

### CONCLUSION

Cabbage extract exhibited lowering effect on blood sugar level and also reduced resistance of the cells to insulin meaning that the uptake of glucose by the cells was improved by the cabbage extracts. Oxidative stress of the liver was also ameliorated by the aqueous extract though this need to be further investigated.

### SIGNIFICANCE STATEMENT

This study provided information on the effect of fresh and dried cabbage on the blood sugar and showed that cabbage could be beneficial in managing the sugar level in hyperglycemia. There is possibility of it reducing the severity of diabetes due to its hypoglycemic effect and being an alternative to pharmacological therapies.

### REFERENCES

1. Yendamuri, S.M., G.J. Fulda and G.H. Tinkoff, 2003. Admission hyperglycemia as a prognostic indicator in trauma. J. Trauma Acute Care Surg., 55: 33-38.
2. DCCT Research Group, 1993. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N. Engl. J. Med., 329: 977-986.
3. American Diabetes Association, 2014. Diagnosis and classification of diabetes mellitus. Diabetes Care, 37: S81-S90.
4. World Health Organization, 2015. World Health Statistics 2015. World Health Organization, Geneva, Switzerland, ISBN:978-92-4-156488-5, Pages: 161.
5. Wright, Jr.E., J.L. Scism-Bacon and L.C. Glass, 2006. Oxidative stress in type 2 diabetes: The role of fasting and postprandial glycaemia. Int. J. Clin. Pract., 60: 308-314.
6. Ceriello, A., L. Quagliaro, B. Catone, R. Pascon and M. Piazzola *et al.*, 2002. Role of hyperglycemia in nitrotyrosine postprandial generation. Diabetes Care, 25: 1439-1443.
7. Deng, R., 2012. A review of the hypoglycemic effects of five commonly used herbal food supplements. Recent Patents Food Nutr. Agric., 4: 50-60.
8. Balkaya, A., R. Yanmaz, A. Apaydin and H. Kar, 2005. Morphological characterisation of white head cabbage (*Brassica oleracea* var. *capitata* subvar. *alba*) genotypes in Turkey. N. Z. J. Crop Hort. Sci., 33: 333-341.
9. Adeniji, O.T., I. Swai, M.O. Oluoch, R. Tanyongana and A. Aloyce, 2010. Evaluation of head yield and participatory selection of horticultural characters in cabbage (*Brassica oleracea* var. *capitata*). J. Plant Breed. Crop Sci., 2: 243-250.
10. Meena, M.L., R.B. Ram, R. Lata and S.R. Sharma, 2010. Determining yield components in cabbage (*Brassica oleracea* var. *capitata* L.) through correlation and path analysis. Int. J. Sci. Nat., 1: 27-30.
11. Hasan, M.R. and A.H.M. Solaiman, 2012. Efficacy of organic and organic fertilizer on the growth of *Brassica oleracea* L. (Cabbage). Int. J. Agric. Crop Sci., 4: 128-138.
12. Caunii, A., R. Cuciureanu, A. Zakar, E. Tonea and C. Giuchi, 2010. Chemical composition of common leafy vegetables. Stud. Univ. Vasile Goldis Ser. Stiintele Vietii, 20: 45-48.
13. Barbara, K., B. Agnieszka, W. Lidia, D. Jerzy, G. Shela and J.N. Niik, 2008. Partial characterization of white cabbages (*Brassica oleracea* var. *capitata* f. *alba*) from different regions by glucosinolates, bioactive compounds, total antioxidant activities and proteins. LWT-Food Sci. Technol., 41: 1-9.
14. Tendaj, M. and K. Sawicki, 2012. The effect of the method and time of seedling production on red cabbage (*Brassica oleracea* L. ssp. *oleracea* convar. *capitata* (L.) Alef. var. *capitata* L. f. *rubra* DC.) yield. Acta Agrobot., 65: 115-122.
15. Ederer, S., 1927. Beeinflussung des blutzuckers durch einen peroral wirksamen pflanzenextrakt. Klinische Wochenschrift, 6: 72-72.
16. Dinkova-Kostova, A.T. and R.V. Kostov, 2012. Glucosinolates and isothiocyanates in health and disease. Trends Mol. Med., 18: 337-347.
17. Yeasmen, N. and M.N. Islam, 2015. Ethanol as a solvent and hot extraction technique preserved the antioxidant properties of tamarind (*Tamarindus indica*) seed. J. Adv. Vet. Anim. Res., 2: 332-337.
18. Reeves, P.G., F.H. Nielsen and G.C. Fahey Jr., 1993. AIN-93 purified diets for laboratory rodents: Final report of the American institute of nutrition Ad hoc writing committee on the reformulation of the AIN-76A rodent diet. J. Nutr., 123: 1939-1951.

19. Santure, M., M. Pitre, A. Murette, Y. Deshaies and C. Lemieux *et al.*, 2002. Induction of insulin resistance by high sucrose feeding does not raise mean arterial blood pressure but impairs haemodynamic responses to insulin in rats. *Br. J. Pharmacol.*, 137: 185-196.
20. Arikawe, A.P., A.O. Daramola, A.O. Odofin and L.F. Obika, 2006. Alloxan-induced and insulin-resistant diabetes mellitus affect semen parameters and impair spermatogenesis in male rats. *Afr. J. Reprod. Health*, 10: 106-113.
21. Csont, T., 2007. Determination of serum insulin level by ELISA: A practical manual. Cross-Border Co-operation Programme 2007-2013, Hungary- Romania.
22. Hedrich, H., 2004. *The Laboratory Mouse*. 2nd Edn., Elsevier Academic Press, London, pp: 222-255.
23. Varshney, R. and R.K. Kale, 1990. Effects of calmodulin antagonists on radiation-induced lipid peroxidation in microsomes. *Int. J. Radiat. Biol.*, 58: 733-743.
24. Adam-Vizi, V. and A. Seregi, 1982. Receptor independent stimulatory effect of noradrenaline on Na, K-ATPase in rat brain homogenate: Role of lipid peroxidation. *Biochem. Pharmacol.*, 31: 2231-2236.
25. Valerino, D.M. and J.J. McCormack, 1971. Xanthine oxidase-mediated oxidation of epinephrine. *Biochem. Pharmacol.*, 20: 47-55.
26. Sinha, A.K., 1972. Colorimetric assay of catalase. *Anal. Biochem.*, 47: 389-394.
27. Jollow, D.J., J.R. Mitchell, N. Zampaglione and J.R. Gillette, 1974. Bromobenzene-induced liver necrosis. Protective role of glutathione and evidence for 3,4-bromobenzene oxide as the hepatotoxic metabolite. *Pharmacology*, 11: 151-169.
28. Li, W.L., H.C. Zheng, J. Bukuru and N. de Kimpe, 2004. Natural medicines used in the traditional Chinese medical system for therapy of diabetes mellitus. *J. Ethnopharmacol.*, 92: 1-21.
29. Amnah, M.A.A., 2013. Hypoglycemic and hypolipidemic activities of red cabbage and manganese in diabetic rats. *J. Am. Sci.*, 9: 13-19.
30. Favier, A., 2006. [Oxidative stress in human diseases]. *Ann. Pharm. Fr.*, 64: 390-396.
31. Bambolkar, S. and G.S. Sainani, 1995. Evaluation of oxidative stress in diabetics with or without vascular complications. *J. Assoc. Phys. India*, 43: 10-12.
32. Rains, J.L. and S.K. Jain, 2011. Oxidative stress, insulin signaling and diabetes. *Free Radical Biol. Med.*, 50: 567-575.
33. Piconi, L., L. Quagliario and A. Ceriello, 2003. Oxidative stress in diabetes. *Clin. Chem. Lab. Med.*, 41: 1144-1149.
34. Chrobot, A.M., A. Szaflarska-Szczepanik and G. Drewa, 2000. Antioxidant defense in children with chronic viral hepatitis B and C. *Med. Sci. Monitor*, 6: 713-718.