



## **Effect of Antioxidant-rich Nutraceuticals in the Management of High Fat Diet-induced Obesity in Albino Rats**

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### **Authors' contributions**

*This work was carried out in collaboration among all authors. Author JN initiated the research design and carried out the statistical analysis of all results. Interpreted results and generated conclusion. Author IBA dealt with rats' managements to include induction, feeding and sacrificing. Author CMU dealt with general procurement and a major contributor in the writing of the manuscript. Author UUU fine-tuned research methodology and manuscript editorial. All authors read and approved the final manuscript.*

### **Article Information**

DOI: 10.9734/AJBGMB/2021/v8i130184

Editor(s):

(1) Dr. Arulselvan Palanisamy, Muthayammal Centre for Advanced Research (MCAR), Muthayammal College of Arts and Science, India.

Reviewers:

(1) Bao Le, Ton Duc Thang University, Vietnam.

(2) Sara Ebrahimi, Jahrom University of Medical Sciences, Iran.

Complete Peer review History: <http://www.sdiarticle4.com/review-history/68143>

**Original Research Article**

**Received 10 March 2021**

**Accepted 14 May 2021**

**Published 26 May 2021**

### **ABSTRACT**

**Background:** Obesity, a metabolic disorder caused by an imbalance in energy intake and energy expenditure, is a major risk factor for chronic diseases such as type 2 diabetes, hypertension, cardiovascular heart diseases (CHD) and some types of cancer. This research was designed to investigate the effect of antioxidant rich- nutraceuticals in the management of high fat diet-induced obesity in rats.

**Method:** Induction of obesity was achieved by feeding rats with a formulated high fat diet (HFD) for ten (10) weeks. Rats were subsequently group administered 250mg/kg body weight and 500mg/kg

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body weight of the nutraceutical respectively while apple cider vinegar of 5ml/kg body weight was administered to the standard group.

**Result:** Supplementation showed significant ( $P < 0.05$ ) decrease in the glucose, total cholesterol, triglyceride, low density lipoprotein- cholesterol, malondialdehyde and increased in high density lipoprotein-cholesterol and antioxidant status as compared with untreated high fat diet groups. However, there was no significant difference between supplementation of 500mg/kg and the standard group treated with 5ml/kg of Apple cider vinegar.

**Conclusion:** Antioxidant rich nutraceuticals could provide a protective effect against oxidative stress in obesity and remedy complications associated with obesity by reversing the damage to near normal.

**Keywords:** Antioxidant; nutraceuticals; obesity; high fat diet; catalase.

## 1. INTRODUCTION

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems [1]. Obesity transiently puts the individual at the risk of diseases like hypertension, heart disease, type 2 diabetes, obstructive sleep apnoea, certain types of cancer, and osteoarthritis [2].

Many factors are implicated in the exponential rise in cases of obesity. Although there is certainly a genetic predisposition to obesity, several environmental factors are also implicated [3], including excess portion size, dietary macronutrient composition and sedentary lifestyle in the setting of modern-day conveniences.

Since the late 20<sup>th</sup> century, the use of nutraceuticals have been receiving considerable interest due to their potential nutritional, safety and therapeutic effects. Studies have also shown promising results for these compounds in various pathological complications such as diabetes [4,5], atherosclerosis [6,7], cardiovascular diseases (CVDs) [8], cancer [9] and neurological disorders [10]. Many nutraceuticals possess antioxidant properties with the ability to counteract the above situations [11]. Hence, they are considered as healthy sources of health promotion, especially for prevention of life threatening- diseases such as diabetes infection renal and gastrointestinal disorders [12].

The startling rate of increase in overweight or obesity in developing countries like Nigeria and the steady growth in developed countries demands attention. Very few drugs are available for the treatment of obesity, [13]. Many of these drugs used may have potential hazardous effects

over the long-term administration. Behavioural interventions like diet modification and increased physical activity have long been adopted. However, the effectiveness and practice of a strict regimen has been a problem [14]. Furthermore, epidemiological studies suggested that, diets containing significant amount of naturally occurring antioxidants should relieve most of the traits of metabolic syndrome and may reduce obesity and cardiovascular risk [15]. Some locally available foodstuff contains significant number of antioxidants.

From the foregoing therefore, it is imperative to formulate antioxidant rich nutraceutical from these locally available foodstuffs. Hence, this study is designed to investigate the effect of antioxidant rich nutraceutical in the management of obesity in rats as this would go a long way in replacing anti-obesity drugs and their many side effects.

## 2. MATERIAL AND METHODS

### 2.1 Chemicals and Reagents

All chemicals and reagents used were of analytical grades. They were all purchased from BDH Chemicals, UK, Sigma- Aldrich, UK, Thermo Fisher Scientific, Nigeria, Chemelex Lab kit S.A, Randox Laboratories, UK and Cayman Chemicals, USA.

### 2.2 Experimental Animals

Male Wister albino rats weighing between 150-220g were used for the study. The animals were acclimatized for two weeks before the commencement of the experiment and fed with pelletized growers' feed (Vital feed, Jos, Plateau, Nigeria) during the acclimatization period. They were allowed access to clean water *ad libitum* before and during the experimental period.

### 2.3 Formulation of High Fat Diet (HFD)

HFD was formulated in accordance with Noeman et al. [1]. The formulation consisted of fat (46%), carbohydrate (24%), protein (20.3%), fibre (5%), salt mixture (3.7%) and vitamins mixture (1%).

### 2.4 Formulation of the Antioxidant Rich Nutraceutical

100g antioxidant rich nutraceutical was formulated according to local designs using viz: turmeric (15g), ginger (10g), onions (15g), garlic (10g), tomatoes (10g), lemon (20g), palm oil (10g) and crayfish (10g).

### 2.5 Experimental Design

After acclimatization, all the animals were fed a high fat diet (HFD) for ten weeks. Body weight was measured weekly, body weight change measured from difference between final body weight and initial body weight. Food intake was estimated by subtracting the amount of food left in the cages from the total amount of food provided to each rat [16]. The BMI was recorded weekly. After ten weeks, thirty-two (32) rats confirmed to be obese were used for the study. They were divided into four [4] groups of eight [8] rats each based on equalized body weight and continually fed HFD for the duration of the study. The groups are as below

- Group I: Untreated obese rats to serve as untreated control
- Group II: Obese rats treated with 250mg/kg body weight of nutraceutical
- Group III: Obese rats treated with 500mg/kg body weight of nutraceutical
- Group IV: Obese rats treated with 5mg/kg body weight of apple cider vinegar to serve as treated control.

### 2.6 Determination of Body Mass Index (BMI)

The body mass index was measured on a weekly basis for 10 weeks of the experimental period. Rats were weighed in grams using an electronic weighing balance. BMI was estimated as an index of obesity. The formula below was used to calculate the BMI.

$$BMI = \frac{\text{Weight (g)}}{\text{Length (cm}^2\text{)}}$$

### 2.7 Biochemical Analysis

The following investigation and analysis were carried out after the extraction of the tissue and serum.

#### 2.8 Estimation of Serum Glucose Level

Serum glucose was estimated by glucose oxidase/ peroxidase [17] method using Randox kit.

#### 2.9 Estimation of Serum Lipid Profile

##### 2.9.1 Estimation of serum total cholesterol

Serum total cholesterol (TC) was estimated by cholesterol esterase method [18] using Randox kit.

##### 2.9.2 Estimation of serum HDL- C

This was estimated by enzymatic method of Burstein et al. [19] using Randox Kit:

##### 2.9.3 Estimation of serum triglyceride

This was assayed by the method of Tietz [20], using glycerol phosphate oxidase/peroxidase method using Randox Kit.

##### 2.9.4 Estimation of serum LDL- C

This was calculated using Friedewald formula [21].

$$LDL - C \text{ (mg/dl)} = TC - (HDL - C) - \left( \frac{TG}{5} \right)$$

##### 2.9.5 Estimation of Serum VLDL- C

This was calculated using Friedewald formula [21].

$$VLDL - C \text{ (mg/dl)} = \frac{TG}{5}$$

### 2.10 Estimation of Markers of Oxidative Stress

#### 2.10.1 Estimation of Malondialdehyde (MDA)

Lipid peroxidation as evidenced by the formation of thiobarbituric acid reactive substances (TBARS) was measured by the method of Shah and Walker [22].

**2.10.2 Estimation of catalase activity**

Catalase activity was assayed using chemical reactivity method [23].

**2.10.3 Estimation of superoxide dismutase**

This was assayed using Cayman's Superoxide Dismutase Assay Kit [24].

**2.10.4 Estimation of glutathione peroxidase activity**

This was estimated using the method of Petterson and Lazarow, [25].

**2.11 Estimation of Serum Vitamin A**

Serum Vitamin A was assayed by spectrophotometric method of Rutkowski et al. [26]; a modification of Bessey et al. [27].

**2.12 Serum Vitamin-C estimation**

Serum vitamin C was assayed by spectrophotometric method of Baker et al. [28].

**2.13 Serum Vitamin E Estimation**

Serum vitamin E concentrations were estimated using the method of Hashim and Schuttringer [29].

**3. RESULTS**

Table 1 shows effect of antioxidant rich nutraceutical on the body mass index of an obese induced rat when compared with untreated control group. There is a significant increase (P<0.05) in the BMI of the untreated control group as the week increases. Similarly,

there is a significant increase (P<0.05) in the BMI of the group treated with 250 mg/kg bw of nutraceutical as the week increases. However, there is no significant difference (P<0.05) for the first seven weeks in the BMI of the group treated with 500mg/kg bw of nutraceutical. A significant increase (P<0.05) was observed in the eight week of treatment with 500 mg/kg bw of the nutraceutical. Table 1 also show that there is no significant difference (P<0.05) in the BMI of the group treated with Apple Cider vinegar (ACV).

Table 2 shows the effect of antioxidant rich nutraceutical on the Lipid Profile of an obesity induced rats.

There is no significant difference (P<0.05) in the Total cholesterol (TC) of the untreated group and the group treated with 250mg/kg bw of the nutraceutical. There is also no significant difference (P<0.05) in the Total cholesterol of the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV. There is, however, significant decrease in the TC in the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV when compared with the untreated group and the group treated with 250mg/kg bw of the nutraceutical.

Table 2 shows that there is no significant difference (P<0.05) in the triglyceride concentration (TG) of the untreated group and the group treated with 250mg/kg bw of the nutraceutical. There is also no significant difference (P<0.05) in the Total cholesterol of the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV. There is, however, significant decrease in the TG in the group treated with 500mg/kg bw of the

**Table 1. Effect of antioxidant rich nutraceutical on the body mass index on HFD-induced obese rat**

	HFD	HFD + 250mg/kg	HFD + 500mg/kg	HFD + ACV 5mg/kg
WEEK 1	0.0845±0.020 <sup>a</sup>	0.830±0.034 <sup>a</sup>	0.788±0.007 <sup>a</sup>	0.833±0.025 <sup>a</sup>
WEEK 2	0.862±0.020 <sup>b</sup>	0.810±0.033 <sup>ab</sup>	0.765±0.067 <sup>a</sup>	0.775±0.021 <sup>a</sup>
WEEK 3	0.878±0.021 <sup>b</sup>	0.842±0.168 <sup>b</sup>	0.738±0.065 <sup>a</sup>	0.727±0.021 <sup>a</sup>
WEEK 4	0.883±0.020 <sup>c</sup>	0.782±0.037 <sup>b</sup>	0.700±0.0086 <sup>a</sup>	0.672±0.019 <sup>a</sup>
WEEK 5	0.887±0.020 <sup>c</sup>	0.738±0.031 <sup>b</sup>	0.662±0.009 <sup>a</sup>	0.620±0.021 <sup>a</sup>
WEEK 6	0.895±0.019 <sup>c</sup>	0.693±0.028 <sup>b</sup>	0.625±0.007 <sup>a</sup>	0.572±0.021 <sup>a</sup>
WEEK 7	0.902±0.021 <sup>c</sup>	0.663±0.027 <sup>b</sup>	0.587±0.005 <sup>a</sup>	0.530±0.024 <sup>a</sup>
WEEK 8	0.905±0.020 <sup>d</sup>	0.6300±0.024 <sup>c</sup>	0.548±0.007 <sup>b</sup>	0.480±0.018 <sup>a</sup>
WEEK 9	0.911±0.017 <sup>d</sup>	0.593±0.187 <sup>c</sup>	0.523±0.067 <sup>b</sup>	0.442±0.014 <sup>a</sup>
WEEK 10	0.923±0.017 <sup>d</sup>	0.555±0.016 <sup>c</sup>	0.482±0.009 <sup>b</sup>	0.398±0.010 <sup>a</sup>

Values are expressed as mean ± SEM. Mean values are compared using One-Way ANOVA. Level of significance was evaluated using Duncan's Multiple Range Test (DMRT) at P< 0.05. Values in the same column with the same superscript are not significantly different (P<0.05).

**Table 2. Effect of antioxidant rich nutraceutical on the Lipid Profile on HFD-induced obese rats**

	HFD	HFD + 250mg/kg	HFD + 500mg/kg	HFD + ACV 5ml/kg
<b>TC</b> <sub>(mg/dl)</sub>	106.09±2.23 <sup>bc</sup>	102.26±2.53 <sup>b</sup>	92.42±1.48 <sup>a</sup>	90.83±0.68 <sup>a</sup>
<b>TG</b> <sub>(mg/dl)</sub>	112.97±2.66 <sup>bc</sup>	98.69±1.72 <sup>b</sup>	93.19±0.91 <sup>a</sup>	91.43±0.77 <sup>a</sup>
<b>HDL-C</b> <sub>(mg/dl)</sub>	40.98±1.78 <sup>a</sup>	55.21±4.71 <sup>a</sup>	63.99±1.99 <sup>b</sup>	65.62±2.81 <sup>b</sup>
<b>LDL-C</b> <sub>(mg/dl)</sub>	37.56±2.63 <sup>ab</sup>	27.33±6.83 <sup>b</sup>	19.98±3.05 <sup>a</sup>	11.11±3.15 <sup>a</sup>
<b>VLDL-C</b> <sub>(mg/dl)</sub>	20.29±0.45 <sup>b</sup>	19.44±0.27 <sup>b</sup>	18.53±0.18 <sup>a</sup>	18.21±0.09 <sup>a</sup>

Values are expressed as mean ± SEM. Mean values are compared using One-Way ANOVA. Level of significance was evaluated using Duncan's Multiple Range Test (DMRT) at P<0.05. Values in the same row with the same superscript are not significantly different (P<0.05).

TC- Total cholesterol, TG- Triglyceride, HDL-C-High density lipoprotein cholesterol, LDL-C-Low density lipoprotein cholesterol, VLDL-C- Very low density lipoprotein cholesterol

**Table 3. Effect of antioxidant rich nutraceutical on the Glucose level of HFD-induced obese rats**

	HFD	HFD + 250 mg/kg	HFD + 500 mg/kg	HFD + ACV 5 ml/kg
<b>Glucose level (mg/dl)</b>	122.96±2.68 <sup>c</sup>	110.31±2.92 <sup>b</sup>	103.74±1.66 <sup>b</sup>	96.33±1.93 <sup>a</sup>

Values are expressed as mean ± SEM. Mean values are compared using One-Way ANOVA. Level of significance was evaluated using Duncan's Multiple Range Test (DMRT) at P<0.05. Values with the same superscript are not significantly different (P<0.05).

**Table 4. Effect of antioxidant rich nutraceutical on antioxidant status of HFD-induced obese rats**

	HFD	HFD + 250mg/kg	HFD + 500mg/kg	HFD + ACV 5ml/kg
<b>GPX</b> (nmol/min/ml)	37.70 ± 1.08 <sup>a</sup>	86.99 ± 3.15 <sup>b</sup>	120.46 ± 4.24 <sup>b</sup>	156.69 ± 2.50 <sup>c</sup>
<b>SOD</b> (nmol/min/ml)	8.34 ± 0.26 <sup>a</sup>	12.88 ± 0.60 <sup>a</sup>	20.28 ± 0.72 <sup>b</sup>	26.67 ± 0.89 <sup>b</sup>
<b>CAT</b> (nmol/min/ml)	0.95 ± 0.03 <sup>a</sup>	1.64 ± 0.05 <sup>b</sup>	2.96 ± 0.08 <sup>b</sup>	3.64 ± 0.11 <sup>d</sup>
<b>MDA</b> (µmol/l)	0.73 ± 0.009 <sup>b</sup>	0.35 ± 0.013 <sup>a</sup>	0.32 ± 0.005 <sup>a</sup>	0.29 ± 0.005 <sup>a</sup>
<b>VITAMIN A</b> (µmol/L)	1.01 ± 0.01 <sup>a</sup>	1.13 ± 0.03 <sup>b</sup>	1.55 ± 0.17 <sup>b</sup>	2.41 ± 0.04 <sup>c</sup>
<b>VITAMIN C</b> (µmol/L)	37.41 ± 0.87 <sup>a</sup>	42.37 ± 1.01 <sup>b</sup>	53.22 ± 1.11 <sup>c</sup>	60.75 ± 1.61 <sup>c</sup>
<b>VITAMIN E</b> (µmol/L)	17.63 ± 0.76 <sup>a</sup>	22.90 ± 1.16 <sup>b</sup>	25.54 ± 0.92 <sup>b</sup>	32.54 ± 0.83 <sup>c</sup>

Values are expressed as mean ± SEM. Mean values are compared using One-Way ANOVA. Level of significance was evaluated using Duncan's Multiple Range Test (DMRT) at P<0.05. Values in the same row with the same superscript are not significantly different (P<0.05).

**Legend:** TAC-Total antioxidant capacity, GPx-Glutathione peroxidase, SOD- Superoxide dismutase, CAT- Catalase, MDA - Malondialdehyde

nutraceutical and the group treated with ACV when compared with the untreated group and the group treated with 250mg/kg bw of the nutraceutical.

Table 2 shows that there is no significant difference (P<0.05) in the HDL-C concentration of the untreated group and the group treated with 250mg/kg bw of the nutraceutical. There is also no significant difference (P<0.05) in the Total cholesterol of the group treated with 500mg/kg

bw of the nutraceutical and the group treated with ACV. There is, however, significant increase in the HDL-C in the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV when compared with the untreated group and the group treated with 250mg/kg bw of the nutraceutical.

Table 2 shows that there is no significant difference (P<0.05) in the VLDL-C concentration of the untreated group and the group treated with

250mg/kg bw of the nutraceutical. There is also no significant difference ( $P<0.05$ ) in the VLCL-C of the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV. There is, however, significant increase in the VLDL-C in the group treated with 500mg/kg bw of the nutraceutical and the group treated with ACV when compared with the untreated group and the group treated with 250mg/kg bw of the nutraceutical.

Table 3 shows the effect of antioxidant rich nutraceutical on the Glucose level of obesity induced rats. The table shows that there are significant differences ( $P<0.05$ ) in all the groups of rats. The glucose level is significantly higher ( $P<0.05$ ) in the untreated group when compared with the other groups of animals.

Table 4 is the effect of antioxidant rich nutraceutical on antioxidant status of high fat diet induced rats.

Table 4 also shows that there is a significant decrease ( $P<0.05$ ) in the GPX level of the untreated groups of rats when compared with the treated groups of rats. The GPX level is significantly lower ( $P<0.05$ ) in the groups treated with 250mg/kg bw and 500mg/kg bw when compared with the ACV treated group.

Table 4 shows the effect of antioxidant rich nutraceutical on the SOD of obesity induced rats. The table shows that there is no significant difference ( $P<0.05$ ) in the untreated groups of rats when compared with the group fed with 250mg/kg bw of nutraceuticals. The SOD level is significantly higher ( $P<0.05$ ) in the groups treated with 500mg/kg bw and the ACV treated group when compared with the untreated group.

The Table 4 shows that the CAT level is significantly higher ( $P<0.05$ ) in the untreated group when compared with the other groups of animals.

Table 4 also shows that there is no significant difference ( $P<0.05$ ) in the MDA level in all the groups of animals.

Table 4 shows the effect of antioxidant rich nutraceutical on the vitamin A level of obesity induced rats. The table shows that there is a significant decrease ( $P<0.05$ ) in the untreated groups of rats when compared with the treated groups off rats. The vitamin A level is significantly higher ( $P<0.05$ ) in the groups treated ACV

treated when compared with the groups treated with the nutraceuticals.

Table 4 shows the effect of antioxidant rich nutraceutical on the vitamin C level of obesity induced rats. The table shows that there is a significant decrease ( $P<0.05$ ) in the untreated groups of rats when compared with the treated groups off rats. The vitamin C level is significantly higher ( $P<0.05$ ) in the groups treated ACV treated when compared with the groups treated with the 250mg/kg bw nutraceuticals.

Table 4 shows the effect of antioxidant rich nutraceutical on the vitamin E level of obesity induced rats. The table shows that there is a significant decrease ( $P<0.05$ ) in the untreated groups of rats when compared with the treated groups off rats. The vitamin E level is significantly higher ( $P<0.05$ ) in the groups treated ACV treated when compared with the groups treated with the nutraceuticals.

#### 4. DISCUSSION

The present study proves that regular intake of antioxidant-rich nutraceuticals is very beneficial in the preventing and management of obesity and its related complications. Dietary fat intake often has been claimed as responsible for the increase in adiposity. Studies on humans have shown that consumption of high-fat diets can induce obesity with ease [30]. The current results in which the BMI of the group that received high fat diet exclusively increased is in accordance with that of Spetter and Hallschmid [31]. This is associated with increased food intake and the fatty content in it. High fat diet has been implicated the development of a positive energy balance and this leads to an increase in visceral fat deposition [31], justifying why consuming it led to obesity. Feeding on a high fat diet leads to high caloric intake and this led to a progressive increase in body mass index (BMI) suggesting that the excess energy led to the build-up of adiposity. This is the source of the increase in body weight [32]. The BMI of the treated groups reduced significantly. This is in support with a research carried out by Rajasekaran et al. [33].

In the present study, feeding on high fat diet resulted in dyslipidaemia changes as revealed by significant increases in low density lipoprotein (LDL) as well as a decrease in serum level of high density lipoprotein (HDL) a finding in accordance with that of Wood et al. [34]. Thus,

obesity led to an increased level of fatty acid implicitly leading to oxidative stress.

Diet supplementation with 500mg/kg of nutraceutical and apple cider vinegar produced significant decreases in serum total cholesterol (TC), low density lipoprotein LDL and very low density lipoprotein VLDL while there were significant increases in HDL cholesterol in the obese rats. Obesity has been implicated in increasing cardiovascular risk through risk factors such as high levels of triglycerides, blood glucose and low levels of LDL cholesterol, HDL cholesterol. This conforms to the results obtained from the group that fed exclusively with high fat diet without treatment. The increased blood levels of total cholesterol, LDL, VLDL as well as lowered levels of HDL in HFD rat have been identified in the development of hypercholesterolemia, which is one of the risk factors for coronary heart disease (CAD).

The significant difference between the nutraceutical treated groups and high fat diet untreated group observed in this research shows that antioxidant rich nutraceutical could serve as an alternative remedy of cardiovascular complications. This finding therefore supports the use of nutraceutical as effective strategies in the management of oxidative stress related conditions like diabetes, atherosclerosis and myocardial infarction.

When there is high intake of carbohydrate, hepatic tissues convert glucose into fatty acids, from which triglycerides are made and transported to the blood stream as very low-density lipoprotein cholesterol (VLDL-C) which is stored as fat in the adipose tissue [35]. Delayed catabolism of VLDL-C because of the competition for the site of lipoprotein lipase between VLDL-C from hepatic origin and chylomicrons from intestinal origin usually lead to the formation of TG-rich lipoprotein remnants. This could lead to insulin resistance which in turn leads to a type 2 diabetes. Consequently, glucose builds up in the system leading to high blood sugar.

Reduced glutathione (GSH) is the first line of defence against free radicals caused by reactive oxygen species and is also responsible for the maintenance of protein thiols and acts as a substrate for Glutathione peroxidase (GPx) [36]. This research shows that GSH was reduced in the rats with obesity induced with HFD. Enzymatic antioxidants, such as superoxide

dismutase and catalase usually scavenge reactive oxygen species and free radicals or prevent their formation. The results of present study showed that antioxidant enzyme activities in the group that received high fat diet were significantly reduced as compared to the normal healthy control group. High fat diet generates oxidative stress in obese rats as shown by a marked increase in the levels of MDA which is a marker of oxidative stress and a distinct diminution in hepatic GSH, as well as activities of the antioxidant enzyme catalase and antioxidant status in cancerous cases.

Superoxide dismutase (SOD) is present in almost all cells and also serve as a first line defense against reactive oxygen species by converting  $O_2^-$  into  $H_2O$  and  $O_2$ . Mitochondrial and bacterial SOD contain Mn, while cytosolic SOD is a dimer containing Cu and Zn. As the  $H_2O_2$  may still react with other ROS, it needs to be degraded by either one of the other two antioxidant enzymes, GSH-Px or catalase [36, 37]. GSH catalyzes degradation of  $H_2O_2$  by reduction, where two glutathione (GSH) molecules are oxidized to glutathione disulfide (GSSG). Regeneration of GSH by GSH-reductase, requires NADPH, which is oxidized to  $NADP^+$ . A decline in cellular level of GSH has been considered to be indicative of oxidative stress. Catalase, on the other hand, is localized primarily in peroxisomes, and so it detoxifies the  $H_2O_2$  that diffuses from the mitochondria to the cytosol, converting it into water and molecular oxygen [37].

Heyes et al. [38] said that ROS detoxification agents also includes non-enzymatic antioxidants such as vitamin E ( $\alpha$ -tocopherol), vitamin A (retinol), and vitamin C (ascorbic acid) they act in concert to protect against the free radical-induced damage. The present study showed decreased concentration of Vitamins E, C and A in the liver. Vitamin E is considered as a mean of correcting plasma antioxidant status and attenuating the cardiovascular disease that accompanied kidney failure. Vitamin E could prevent or delay coronary heart disease (CHD) which comes from several sources [39]. In the present study, since there was significant change in Vitamin E when the control was compared with other groups, it could be that it has been depleted. Humans cannot synthesize ascorbic acid de novo as such, bodily concentrations of Vitamin C are maintained through intake in diet [40]. Vitamin C decreases the malondialdehyde content in the skin, which is a marker of oxidative

stress [41]. The reduction in Vitamin C concentration in the untreated group and the progressive increase in the treated group with concomitant could be an indication of the role of vitamin C in the lowering obesity by playing antioxidant roles. The reduction in MDA level, elevation of GSH, Vitamin C and E, enhanced activities of SOD, GPx, and CAT in the liver homogenate of the treated groups might be to the inhibitory effect on lipid peroxidation and ability to antagonize the formation of free radical damage caused by HFD [42].

## 5. CONCLUSION

Ideal nutrition with proper intake of nutraceuticals in maintaining health to combat against acute and chronic diseases due to nutritional disorders can promote optimal health, longevity, and quality of life. This highlights the importance of nutraceuticals and basically why this research was carried out. The result of this study showed that treatment decreased the rising BMI of the rats as compared with the high fat diet untreated control. Alterations in BMI were associated with dyslipidaemia profile and oxidative stress in serum of rats; therefore, BMI may predict these adverse consequences of the obesity in rats. There was also an increase in the blood glucose level of the high fat diet untreated group and a decrease in other antioxidant parameters. Thus there synergy and potency in more concentration of antioxidants in the formulated composition as against individual components. Also, the nutraceutical should be encouraged to form part of the daily diet in food preparation as the antioxidant will help regulate oxidative stress in the system.

## NOTE

This study draws its strength on emphasis on the effects of the antioxidant-rich nutraceuticals on serum parameters. However, more studies are required to know the effects of antioxidant-rich nutraceuticals on the organs e.g. the brain, heart, liver, kidney, spleen e.t.c.

## ETHICAL APPROVAL

Animal Ethic committee approval has been taken to carry out this study.

## ACKNOWLEDGEMENTS

The authors are acknowledging the efforts of the Chief Technologist, Mr. Sikiru Oyewo for his

laboratory assistance and time during the research.

## DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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