

## **Modulation of the expression of lipogenic activity by malted hungry rice flour (*Digitaria exilis.*) on diet-induced obese (DIO) rats**

### **Abstract**

Lipolysis and lipogenesis are the major causes of obesity which are mediated by hormones. The aim of this study was to determine the effect of malted hungry rice flour on lipogenic activity on diet-induced obese rats. The studies were carried out using Completely Randomized Design (CRD). It was conducted at the Department of Home Science, Nutrition and Dietetics (Animal research house) and Department of veterinary science (laboratory analysis) both at the University of Nigeria, Nsukka. The study was conducted between September to January 2021 and 2022. Four groups of twenty male adult albino rats were induced obesity resembling mild obesity condition in the human population after a one-week acclimatization period. Four experimental groups: normal control (AIN-93), obese control, and two test diets were used to evaluate body mass index, serum cholesterol, serum triglyceride, serum low-density lipoprotein, and serum high-density lipoprotein using a standard assay technique. The data were statistically analyzed using ANOVA and the mean was separated using LSD. There is an increase in BMI (from 0.37-0.41 to 0.58-0.72), total cholesterol, LDL, and triglyceride levels along with a decrease in HDL ( $p < 0.05$ ) after consumption of a palatable diet. Consumption of the test diet resulted in a significant reduction in the BMI, LDL, triglyceride, total cholesterol, and a significant increase in HDL cholesterol compared to the obese control group ( $p < 0.05$ ). The result suggests that malted hungry rice flour has an anti-obesity effect.

**Keywords:** Obesity, lipogenic, hungry rice, insulin, antihyperlipidemia, atherosclerosis.

### **Introduction**

Obesity is a nutritional disorder caused by a disarray of energy balance. Obesity has turned out to be a major public health concern in both developed and developing countries. In developing Country, the affluent have high prevalence of obesity but in developed Country the reversed is the case. This prevalence of obesity is associated with chronic health disorders such as insulin

resistance, hyperlipidemia, hypertension, cardiovascular diseases, non-alcoholic fatty liver and osteoarthritis [1,2]. Excessive energy intake over energy expenditure has been implicated as the leading cause of obesity [3,4]. Although, diet rich in saturated fatty acid, high blood pressure, family history, age, and life pattern has an important role in causing hyperlipidemia.

Hyperlipidemia is characterized by high level of low density lipoprotein (LDLc) cholesterol, triglyceride (TG), total cholesterol (TC) and low level of high density lipoprotein (HDLc) cholesterol [5]. Hyperlipidemia, extensively contributes in the progression of coronary heart diseases and atherosclerosis, but may be managed through alterations in the nutritional pattern. There is a great awareness regarding association between low density lipoprotein reduction and decreased cardiovascular disease mortality [6,7]. Studies show that diet rich in polyphenols and antioxidants have antiatherogenic effects [8]. A plant-based diet rich in fruit, vegetables, and legumes and low in saturated fat is an effective prescription for anyone with more severe atherosclerosis [8].

There is an increasing search for the lipid lowering agents from natural origin. Plants have been used as food and for medicinal purposes for centuries. It is well known that many of these plants contain biologically active components and at least 30% of drugs presently used in modern medicine are derived from plants [9,10]. There are some studies on plant foods possessing lipid lowering activities [11,12,13,14]. Medicinal plants have always been considered as healthy source of treatment due to its therapeutic effect and safety. Moreover, the clinical uses of most synthetic drugs for managing obesity are usually accompanied with some adverse effects. Therefore, there is need to search for more antihyperlipidemic agents with more effectiveness and no side effects.

Hungry rice (*Digitaria exilis*) commonly known as acha" originated in West African where it occupies about 300,000 hectares and provides food for the population [15]. In Nigeria, it is popularly known as "acha" and are produce mainly in (Bauchi, Kaduna, Kebbi, Plateau and Niger) and the Federal Capital Territory [16]. Acha is a good breakfast cereal and is a good cereal for the production of complementary food because of its high fibre (2-4%) and protein (8-12%) content. It is one of the most nutritious of all grains and is the world's fastest maturing grain [17]. Traditionally, acha is used in preparation of unfermented porridge food and other dishes in Nigeria [14]. It is also used in dietary preparations for diabetic patients [18]. Acha could

serve as a better alternative to barley (*Hordeum vulgare* L.) which is being presently used to produce conventional beverage (malt drink) [19]. Africa especially Nigeria has a rich heritage of medicinal plants of wide diversity, which are used by the local population and traditional healers for the treatment of several diseases including lipid disorders. In this study attempt was made to determine the effect of malted and unmalted hungry rice flour (*Digitaria exilis*.) on diet-induced obese (DIO) rats.

## **Materials and Methods**

### **Procurement of sample materials**

The raw seeds of Hungry rice (*Digitaria exilis*.) was supplied by a farmer from Plateau State of Nigeria and authenticated at the Herbarium by a Botanist, Department of Plant Science and Biotechnology, University of Nigeria Nsukka, Nigeria.

### **Preparation of unmalted Hungry rice**

The unmalted hungry rice flour was produced using standard method. The hungry rice grain of about 5 kilogram was cleaned by winnowing and thoroughly washed. The washed grains were dried in an oven (Model, DHG 9101.1 USA) at 60°C for 6 hours. The dried unmalted hungry rice grains were milled into flour using an attrition mill Model, (Atlas exclusive, Alzico Ltd. mill) and sieved through a 100 mm standard mesh sieve. It was cooled and packaged in a sealed zip lock and stored in refrigerator for further analysis.

### **Preparation of malted Hungry rice**

The malted hungry rice flour was produced following the flow diagram below. The hungry rice grain of 5 kilogram was cleaned by winnowing and thoroughly washed. These grains were steeped for 24 hours in tap water changing water at 6 hours interval (w/v 1:2) at ambient temperature. The steeped grains were drained and spread out on a table and covered with jute bag. Water was sprinkled on it daily at 6 hours intervals until the grains sprouted. The germination was terminated after 72 hours. The sprouted grains were dried in an oven (Model, DHG 9101.1 USA) at 60°C for 6 hours in order to terminate enzyme activities. The plumule were separated from the grain on palm by abrasion. The dried malted hungry rice grains were milled into flour using an attrition mill Model, (Atlas exclusive, Alzico Ltd. mill) and sieved

through a 100 mm standard mesh sieve. It was cooled and packaged in a sealed zip lock and stored in refrigerator for further analysis.

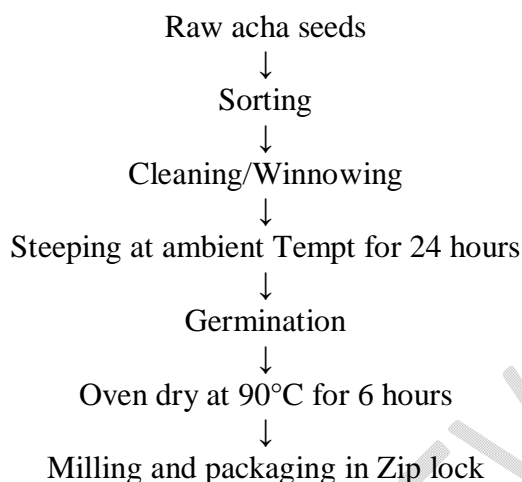


Fig 1: Flow diagram of production of malted acha

#### **Determination of Phenolic Content**

The phenolic content was determined using the Folin– reagent. Approximately 1 mL of this reagent was added to 1 mL of  $\text{Na}_2\text{CO}_3$  7.5% w/v and to 200 mL of sample previously prepared at a concentration of 2 mg/mL in a mixture of acetone/methanol/water/acetic acid (40:40:20:0.1). Two hours later, the absorbance was measured at 726 nm [20]. The results were expressed as mg of chlorogenic acid equivalents per g of dry matter (g CAE/kg DM).

#### **Determination of Flavonoid Contents**

Flavonoid content was determined using an aluminum chloride colorimetric method. About 1 mL of  $\text{AlCl}_3$  2% was added to 1 mL of sample previously prepared at a concentration of 2 mg/mL in EtOH 80%. After 15 min, the absorbance was measured at 430 nm [21]. The results were expressed as mg per g of dry matter (g QE/kg DM).

#### **Free Radical Scavenging Activity (FRSA) Assay**

The antioxidant activity of malted and unmalted sample were determined by DPPH free radical scavenging ability according to the method of [22]. The analysis was based on the reduction of a purple methanolic solution of the free radical 2,2-diphenyl-1-picrylhydrazyl (DPPH). About 0.8 mL of methanolic solution of DPPH (0.1 mM) was mixed with 0.2 mL of various concentrations (5–200  $\mu\text{g/mL}$ ) of sample solutions. After 30 min in the dark at room temperature, the

absorbance of the samples was measured at 517 nm by using a Perkin Elmer Lambda 40 UV–spectrophotometer. The solution containing methanol and DPPH without sample was used as a blank. The percentage inhibition of DPPH free radical was calculated by using the following formula:

$$\text{Percentage of inhibition} = \{1 - [(\text{DPPH absorbance with extract})/(\text{DPPH absorbance without extract})] \times 100\}.$$

Ascorbic acid was used as a positive control.

## **EXPERIMENTAL DESIGN**

The obesity studies were carried out using the Completely Randomized Design (CRD). Rats were randomly assigned to the treatments based on their weights. There were four treatments each replicated five times. The rats were the replicates while the different diets was the treatments.

### **Animal housing**

A total of twenty adult male Albino rats, from the same colony, that weighed between 150-153g were purchased from the Faculty of Veterinary Medicine, University of Nigeria Nsukka, Nigeria. Metabolic cages equipped to separate faeces and urine were used to housed the rats. The rats had exactly 12 h of light and 12 h of darkness in a day. National Research council guidelines on the care and use of laboratory animals [23] were strictly adhered to. The study was approved by Animal Experimentation Ethics Committee of University of Nigeria Nsukka.

## **INDUCTION OF OBESITY**

The animals were divided into 4 groups of 5 rats each on the basis of their body weight such that the difference in mean body weight of each group does not exceed 5g [24]. The rats were housed in the Department of Home Science, Nutrition and dietetics, University of Nigeria Nsukka animal house. The animals were acclimatized for a period of 2 weeks prior to the experiment. The rats were fed a highly palatable diet for 3 weeks to induce mild obesity. The composition of rodent pelleted chow, are 60% of energy as carbohydrate, 30% as protein and 10% as fat. The palatable diet consisted of 33% chow, 33% condensed milk and 7% sucrose by weight, with the

remainder being added water. This will provide 65% of energy as carbohydrate, 19% as protein and 16% as fat. This diet was designed to promote weight gain through hyperphagia, without employing major changes in macronutrient composition, compared with normal rat chow. This is a reliable method of inducing weight gain and insulin resistance [25, 26, 27,28]. Rats were allowed free access to water throughout the study and maintained on a 12 : 12 h light:dark phase schedule.

At the end of the 3-weeks period, when the palatable diet-treated rats had developed significant weight gain. The formulated diets were given for 6 weeks to the animals. The groups were treated as follows- Group 1 received normal control (AIN-93), Group 2- obese control; Group 3 received unmalted acha flour and Group 4 received malted acha flour. The diets were formulated using AIN-93G (American Institute of Nutrition) method [29]. The weights and length of animals were recorded on daily basis. Daily food intake and extract was also recorded for nutrient intake calculation.

### **Blood Sample Collection and Biochemical Indices determination**

Blood was collected from the retro of the medial canthus of the eye of the rats. A nurocapillary tube was carefully inserted into the canthus of the eye to puncture the retro-bulba plexus and thus enable outflow of about 2ml blood into a clean glass test tube. The blood sample was kept at room temperature for 30 minutes to clot. Afterwards, the test tube containing the clotted blood sample was centrifuged at 3,000 revolutions per minute for ten minutes using a table centrifuge to enable a complete separation of the serum from the clotted blood. The clear serum supernant was then carefully aspirated with syringe and needle and stored in a clean sample bottle for the clinical chemistry determination. Blood were collected on weeks 0, 3, and 6 for lipid profile determinations. The serum was used to determine total cholesterol, LDL, HDL and triglyceride.

### **BIOCHEMICAL ASSAY**

Total cholesterol, LDL, HDL and Serum triglyceride were evaluated with standard enzymatic colorimetric kits from a chemical store at Enugu, Nigeria.

#### **Cholesterol**

Cholesterol in the serum was determined by CHOD-PAP method, enzymatic colorimetric test for cholesterol with Lipid Clearing Factor. Approximately, 10  $\mu$ l sample into cuvette was mixed with 1000  $\mu$ l enzyme reagent and incubated for 5 min, and then the absorbance was measured within 60 min [30].

### **Triglycerides**

Triglycerides (TGs) were determined by GPO-PAP method, an enzymatic colorimetric test. About 10  $\mu$ l sample was mixed with 1000  $\mu$ l mono-reagent in a cuvette and incubated for 10 min, then the absorbance was measured within 60 min [30].

### **High-density lipoprotein-cholesterol**

The methods involved careful precipitation and removal of VLDL and LDL, followed by the enzymatic measurement of high-density lipoprotein (HDL) in the supernatant fraction [31]. In this method, HDL-cholesterol, precipitant, and standard was used with human cholesterol Test Kit. About 200  $\mu$ l sample was mixed with 100  $\mu$ l distilled water and 400  $\mu$ l PREC HDL reagent, then incubated for 10 min and centrifuged at 3000 rpm for 10 min. Approximately, 100  $\mu$ l clear supernatant was taken in a separate tube having 1000  $\mu$ l cholesterol reagent and the absorbance was measured within 60 min [30].

### **Low-density lipoprotein-cholesterol**

The LDL-cholesterol was measured by utilizing test kit of Human, Germany. All reagents were warmed at 37°C then pipette 10  $\mu$ l sample + 750  $\mu$ l enzyme into the cuvette. Mixed gently and incubated for 5 min, 250  $\mu$ l substrate was added and incubated at 37°C, and then the absorbance was measured after 5 min [32,33].

### **Statistical Analyses**

Phytochemical and antioxidant analysis were performed in triplicate. Data were expressed as mean  $\pm$  SD. The biological data were fitted through SPSS and Statistical differences between treated groups and the control and among treated group means were estimated by one-way analysis of variance (ANOVA), followed Duncan New multiple range test.

## RESULTS

**Table1: Effect of malting on the Phytochemical and antioxidant composition of “hungry rice” flour.**

Samples	Phenol (mg/100g)	Flavonoids (mg/100g)	FRSA (%)
A	3.72±0.03	0.81±0.22	76.78±1.01
B	6.48±0.44	2.56±0.61	93.63±0.35

A=Unmalted hungry rice, B=Malted hungry rice, Result=Mean ± standard deviation.

The results of the phytochemical and antioxidant composition of the samples were shown in Table 1. The result showed phenol 3.72mg/100g and 6.48mg/100g, flavonoid 0.81mg/100g and 2.56mg/100g for unmalted and malted hungry rice flour respectively.

**Table 2: Effect of the diets on the body weight of the rats (g)**

Days	GP1	GP2	GP3	GP4
0 Week	154.30 <sup>a</sup> ±0.71	154.10 <sup>a</sup> ±0.35	153.90 <sup>a</sup> ±0.46	155.00 <sup>a</sup> ±0.16
2 Week	154.45 <sup>b</sup> ±0.23	231.40 <sup>a</sup> ±0.12	232.00 <sup>a</sup> ± 0.98	231.10 <sup>a</sup> ±0.64
6 Week	154.65 <sup>c</sup> ±0.64	232.80 <sup>a</sup> ±0.04	219.20 <sup>b</sup> ±0.03	149.00 <sup>d</sup> ±0.14

Table 2 showed the effect of diets on the body weight of the rats. The results showed that the mean body weight of the rats ranged between 153.90-155.00 on the first day after acclimatization (0 week), 154.45-232.00 (2 week) and 149.00-232.80 on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

**Table 3: Weight gain, Food intake and organ weight of rats fed test diets.**

Parameter	GP1	GP2	GP3	GP4
Food intake (g/day)	12.20 <sup>c</sup> ±0.37	13.40 <sup>b</sup> ±0.06	14.05 <sup>a</sup> ±0.44	13.83 <sup>ab</sup> ±0.12
Weight gain (g/day)	0.04 <sup>c</sup> ±0.01	5.56 <sup>b</sup> ±0.03	6.20 <sup>a</sup> ± 1.05	5.30 <sup>b</sup> ±0.02
FER	0.003 <sup>b</sup> ±0.0	0.41 <sup>a</sup> ±0.67	0.44 <sup>a</sup> ±0.03	0.38 <sup>a</sup> ±0.54
Feaces (g/day)	3.67 <sup>c</sup> ±0.20	6.42 <sup>b</sup> ±0.04	7.28 <sup>a</sup> ±0.16	6.33 <sup>b</sup> ±0.41
Organ weight (mg/g)				
Heart	2.54 <sup>d</sup> ±0.02	3.20 <sup>a</sup> ±0.30	2.96 <sup>b</sup> ±0.13	2.78 <sup>c</sup> ±0.28
Liver	16.31 <sup>d</sup> ±0.45	31.14 <sup>a</sup> ±0.09	20.17 <sup>b</sup> ±0.16	18.10 <sup>c</sup> ±0.23
Kidney	2.39 <sup>c</sup> ±0.74	2.90 <sup>a</sup> ±0.04	2.59 <sup>b</sup> ± 0.02	2.54 <sup>b</sup> ±0.21
Brain	3.21 <sup>b</sup> ±0.0	3.67 <sup>a</sup> ±0.03	2.92 <sup>c</sup> ±0.33	2.24 <sup>d</sup> ±0.01

Table 3 showed the effect of diets on the weight gain, food intake and organ weight of the rats. The results showed that the mean weight gain of the rats ranged between 0.04-6.20 g/day, food intake 12.20-14.05, FER 0.003-0.44g/day, feaces 3.67- 7.28g/day, heart 2.54-3.20g, liver 16.31- 31.14g, kidney 2.39-2.90g and brain 2.24- 3.69g.

**Table 4: Effect of the diets on the BMI of the rats (g/cm<sup>2</sup>)**

Days	GP1	GP2	GP3	GP4
<b>0 Week</b>	0.64 <sup>a</sup> ±0.32	0.62 <sup>a</sup> ±0.51	0.63 <sup>a</sup> ±0.05	0.64 <sup>a</sup> ±0.02
<b>2 Week</b>	0.64 <sup>b</sup> ±0.0	0.90 <sup>a</sup> ±0.76	0.87 <sup>a</sup> ± 0.03	0.86 <sup>a</sup> ±0. 24
<b>6 Week</b>	0.64 <sup>c</sup> ±0.14	0.91 <sup>a</sup> ±0.17	0.76 <sup>b</sup> ±0.11	0.55 <sup>d</sup> ±0.94

Table 2 showed the effect of diets on the BMI of the rats. The results showed that the mean BMI of the rats ranged between 0.62-0.64g/cm<sup>2</sup> on the first day after acclimatization (0 week), 0.64-0.90 (2 week) and 0.55-0.91g/cm<sup>2</sup> on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but

there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

**Table 5: Effect of the diets on the Total cholesterol of the rats (mg/dl)**

Days	GP1	GP2	GP3	GP4
<b>0 Week</b>	73.40 <sup>a</sup> ±0.01	73.20 <sup>a</sup> ±0.05	72.68 <sup>a</sup> ±0.04	73.52 <sup>a</sup> ±0.10
<b>2 Week</b>	72.84 <sup>b</sup> ±0.03	99.90 <sup>a</sup> ±0.02	101.10 <sup>a</sup> ± 0.08	102.05 <sup>a</sup> ±0.00
<b>6 Week</b>	73.88 <sup>c</sup> ±0.04	101.03 <sup>a</sup> ±0.012	78.65 <sup>b</sup> ±0.13	73.30 <sup>c</sup> ±0.11

Table 5 showed the effect of diets on the total cholesterol of the rats. The results showed that the mean total cholesterol of the rats ranged between 72.68-73.52mg/dl on the first day after acclimatization (0 week), 72.84-102.05mg/dl (2 week) and 73.30-101.03 on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

**Table 6: Effect of the diets on the Triglycerides of the rats (mg/dl)**

Days	GP1	GP2	GP3	GP4
------	-----	-----	-----	-----

<b>0 Week</b>	80.65 <sup>a</sup> ±0.0	82.50 <sup>a</sup> ±0.01	82.81 <sup>a</sup> ±0.02	81.16 <sup>a</sup> ±0.21
<b>2 Week</b>	81.38 <sup>b</sup> ±0.06	138.90 <sup>a</sup> ±0.11	139.10 <sup>a</sup> ±0.05	138.84 <sup>a</sup> ±0.07
<b>6 Week</b>	81.51 <sup>c</sup> ±0.02	139.20 <sup>a</sup> ±0.03	84.70 <sup>b</sup> ±0.13	80.36 <sup>c</sup> ±0.02

Table 6 showed the effect of diets on the triglyceride of the rats. The results showed that the mean body weight of the rats ranged between 80.65-82.81mg/dl on the first day after acclimatization (0 week), 81.38-139.10mg/dl (2 week) and 80.36-139.20mg/dl on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

**Table 7: Effect of the diets on the LDL of the rats (mg/dl)**

<b>Days</b>	<b>GP1</b>	<b>GP2</b>	<b>GP3</b>	<b>GP4</b>
<b>0 Week</b>	41.60 <sup>a</sup> ±0.26	42.00 <sup>a</sup> ±0.15	42.64 <sup>a</sup> ±0.09	41.98 <sup>a</sup> ±0.14
<b>2 Week</b>	41.86 <sup>b</sup> ±0.18	164.70 <sup>a</sup> ±0.22	165.20 <sup>a</sup> ±0.12	165.00 <sup>a</sup> ±0.06
<b>6 Week</b>	41.83 <sup>c</sup> ±0.06	165.10 <sup>a</sup> ±0.05	49.89 <sup>b</sup> ±0.01	42.40 <sup>c</sup> ±0.10

Table 7 showed the effect of diets on the LDL of the rats. The results showed that the mean body weight of the rats ranged between 41.60-42.64mg/dl on the first day after acclimatization (0 week), 41.86-165.20mg/dl (2 week) and 41.83-165.10mg/dl on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

**Table 8: Effect of the diets on the HDL of the rats (mg/dl)**

Days	GP1	GP2	GP3	GP4
0 Week	32.90 <sup>a</sup> ±0.60	33.10 <sup>a</sup> ±0.0	31.40 <sup>a</sup> ±0.24	32.60 <sup>a</sup> ±0.15
2 Week	33.05 <sup>a</sup> ±0.41	23.69 <sup>b</sup> ±0.07	23.87 <sup>b</sup> ±0.02	24.05 <sup>b</sup> ±0.0
6 Week	33.00 <sup>a</sup> ±0.38	22.90 <sup>c</sup> ±0.01	27.68 <sup>b</sup> ±0.43	32.70 <sup>a</sup> ±0.04

Table 8 showed the effect of diets on the HDL of the rats. The results showed that the mean body weight of the rats ranged between 31.40-32.90MG/DL on the first day after acclimatization (0 week), 23.69-33.05 (2 week) and 22.90-33.00mg/dl on the 6<sup>th</sup> weeks after treatment. There was no significant different among the mean on week 0, the first day after acclimatization. On week 2 which was the day after inducing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 but there was no significant different between the mean body weight of rat in group 2, 3 and 4. There is significant different among the mean body weight of all the groups on the last day of treatment which is week 6.

## DISCUSSION

The present work studied the effect of malted hungry rice on hyperlipidemic activity on diet-induced obese rats. The result showed higher phytochemical and antioxidant values in malted samples than the unmalted samples. The higher phytochemical content of the malted sample could be as a result of the degradation of the enzymes during malting. The result showed that malting improves the phytochemical content of foods. Flavonoids are beneficial to health because of its antimicrobial, [34] antimutagenic,[35] anticarcinogenic,[36] and anti-inflammatory [37] activities. Flavonoids are naturally occurring polyphenols which have been reported to possess biological effects [38] in addition with antihypercholesterolemic action [39,40]. Several in vivo studies have proved the hypocholesterolemic action of flavonoids in rats [28]. Phytochemicals inhibit excessive cholesterol absorption and reduce blood lipid levels [41]. The result of the antioxidant showed 76.78% and 93.63% based on DPPH for unmalted and malted hungry rice flour respectively. Antioxidant seems to lessen the threat of prostate cancer [42]. Antioxidant can scavenge free radical and minimize their impact [43].

Hyperlipidemia is a risk factor for cardiovascular diseases especially atherosclerosis. Coronary artery disease (CAD) is one of the leading cause for premature death worldwide [44]. Poor food

habit and lack of exercise are the major cause of hyperlipidemia and atherosclerosis. Many animal and human studies have established the hypercholesterolemic effect of saturated fatty acids thereby raising total cholesterol and changing lipoprotein pattern [8]. Recently, studies have been geared towards identifying numerous food with antihyperlipidemic activities in order to create variety and minimize monotonous food habit among obese patients. The results of the study showed that consumption of a high fat diet demonstrated a significant increase in BMI, TC, TG, LDL and decrease the HDL levels in rats. However, the elevated lipid levels were significantly reduced and HDL was significantly increased after consumption of malted and unmalted hungry rice. It showed a reduction in abdominal fat accumulation. Thus, indicating the efficacy of the food in preventing atherosclerosis. Studies show that HDL-cholesterol is inversely related to total body cholesterol and a decrease in plasma HDL-cholesterol level may accelerate the growth of atherosclerosis leading to ischemic heart diseases [45]. The increase in the HDL cholesterol could be attributed to the high phytochemical and antioxidant content of both malted and unmalted hungry rice. Several studies have proved that flavonoids increase HDL cholesterol concentration and reduce LDL and VLDL levels in the hypercholesterolemic rats [46]. Therefore, flavonoid content of malted and unmalted hungry rice could be responsible for the elevated HDL and decrease in LDL and total cholesterol in the experimental rats.

The study showed that malted and unmalted hungry rice drastically reduced the weight gain of rats fed with high fat diet. The reduction in body weight could be attributed to the hypolipidemic effect of malted and unmalted hungry rice. Hungry rice has been used among weight watchers because of its high fibre content. The lipid lowering effect of malted and unmalted hungry rice might be due to its flavonoid and antioxidant effects which leads to its inhibition of uptake of oxidized LDL by macrophages, reduced LDL aggregation, and reduced oxidation of LDL cholesterol, this was as a result of macrophages important role [47].

## CONCLUSION

Result of the recent study shows that malted hungry possess antihyperlipidemic effect and this could be attributed to appreciable phytochemical and antioxidant content in malted hungry rice. Phytochemicals and antioxidant are the active ingredient in development of drug for lowering lipid accumulation in the body. Consumption of malted hungry rice will be of great importance

to obese patients and weight watchers where the food is dominant. Hungry rice can be processed into malted flour for protection against atherosclerosis.

## REFERENCE

1. Yuan, H.D., Kim, S.J. and Quan, H.Y. (2010). Ginseng leaf extract prevents high fat diet induced hyperglycemia and hyperlipidemia through AMPK activation. *Journal of Ginseng Research*; 34(4): 369–75.
2. Saravanan, G., Ponmuragan, P. and Deepa, M.A. (2014). Anti-obesity action of gingerol: effect on lipid profile, insulin, leptin, amylase and lipase in male obese rats induced by a high fat diet. *Journal of Science of Food and Agriculture*; 94(14): 2972–2977.
3. Mopuri, R., Idress, H.A., Avinash, P. and Meriga, B. (2010). Evaluation of anti-lipidemic and anti-obesity efficacy of *Bauhinia purpurea* bark extract on rats fed with high fat diet. *International Journal of Plant Science*; 3:104–107.
4. Bhutani, K.K., Birari, R.B. and Kapat, K. (2017). Potential anti-obesity and lipid lowering natural products; a review. *Nature Production Communication*; 2:331–348.
6. Nwangwa E. K, Ekhoye E. I (2013) Anti-hyperlipidemic Activity of aqueous extracts of Carica papaya seeds in albino rats fed with high fat diet. *Current Trends in Technology and Science*, Vol.II, 2279.
7. Anaka Ogochukwu Ngozi, Omonkhelin Josephine, Emenike Chinwendu, (2013) Antihyperlipidemic effect of aqueous leaf extracts of *Emilia praetermissa* (Asteraceae) in rats. *Inter J, of Biosciences*. 356877
8. Neelam M. and Rafeeq A. K. (2016). Antihyperlipidemic effects of *Citrus sinensis*, *Citrus paradisi*, and their combinations. *J Pharm Bioallied Sci*. 2016 Apr-Jun; 8(2): 112–118. doi: 10.4103/0975-7406.171727
9. Mouhssen L (2007): Screening of natural products for drug discovery. *Expert Opin Drug Discovery* 2: 697–705. [Taylor & Francis Online], [Web of Science ®], [Google Scholar]

10. Mukherjee PK, Rai S, Kumar V, Mukherjee K, Hylands PJ, Hider RC (2007): Plants of Indian origin in drug discovery. *Expert Opin Drug Discovery* 2: 633–657. [Taylor & Francis Online], [Web of Science ®], [Google Scholar]
11. Khaled M. and Mohamed K. (2014). Antihyperlipidemic activity of the medicinal plants among Kadazan and Dusun communities in Sabah, Malaysia: a review. *Asian Pacific Journal of Tropical Biomedicine* Volume 4, Issue 10, October 2014, Pages 768-779
13. Muhammad, B.A. and Hseed, A. (2021). Recent molecular mechanism and beneficial effect of phytochemicals and plant-based whole foods in reducing LDL-c and preventing cardiovascular disease.
14. Sudha, R.P. and Sushma, A.M. (2009). Antihyperlipidemic effect of aqueous extract of phembago zeylemia roots in diet induced hyperlipidemia rat. *Pharmaceutical Biology*, 47:10.
15. E.H.Kwon-Dung, S.M.Misari; Over view of re- search development of acha (*Digitaria exilis* kippis staff) and prospects of genetic improvement in Nigeria. In: *Genetics and food security in Nigeria*, genetic Society of Nigeria, 71-76
16. J.C.Anuonye, G.I.O.Badifu, C.U.Inyang, M.A.Akpanunam, C.U.Odumodu, V.I.Mbajika; Protein dispersibility index and trypsin inhibitor activity of extruded blends of acha/soyabean. A re- sponse surface analysis. *American Journal of Food Technology*, 2(6), 502-511 (2007).
17. NAS, *Lost crops of Africa, Grains*. NationalAcad- emy press Washington D.C., 1, (1996).
18. J.T.Victor, D.B.James; Proximate chemical com- position of acha (*Digitaria exilis*) grain. *Journal Science Food Agriculture*, 56, 561-563 (1991).
19. E.U.Okon; Effect of mash constitution on sugar production in malted sorghums. *Nigeria Food Journal*, 6, 54-60 (1988).
20. Perri, M.R.; Marrelli, M.; Statti, G.; Conforti, F. *Olea europaea* buds extracts: Inhibitory effects on pancreatic lipase and  $\alpha$ -amylase activities of different cultivars from Calabria region (Italy). *Plant Biosyst.* 2020. [Google Scholar ([https://scholar.google.com/scholar\\_](https://scholar.google.com/scholar_) [CrossRef (<https://doi.org/10.1080/11263504.2020.1857868>)]

21. Marrelli, M.; Argentieri, M.P.; Avato, P.; Conforti, F. Lobularia maritima (L.) Desv. Aerial Parts Methanolic Extract: In Vitro Screening of Biological Activity. *Plants* 2020, 9, 89. [Google Scholar (<https://scholar.google.com/scholar>).
22. Marelli, M.; Russo, N.; Chiochio, I.; Statti, G.; Poli, F.; Conforti, F. Potential use in the treatment of inflammatory disorders and obesity of selected wild edible plants from Calabria Region (Southern Italy). *South Afri. J. Bot.* 2020 128, 304-311.
23. National Research Council,(2010) *Guide for the Care and Use of Laboratory Animals*, National Academies Press, Washington, DC, USA, 2010
24. AOAC. Association of Official Analytical Chemist. Official methods of Analysis, Washington, D.C; 1995. 25. Widdowson PS, Upton R, Buckingham R, Arch J, Williams G. Inhibition of food response to intracerebroventricular injection of leptin is attenuated in rats with diet-induced obesity. *Diabetes*. 1996;46: 1782–1785. [PubMed]
26. Wilding JPH, Gilbey SG, Mannan M, Aslam N, Ghatei MA, Bloom SR. Increased neuropeptide Y content in individual hypothalamic nuclei, but not neuropeptide mRNA, in diet-induced obesity in rats. *J. Endocrinol.* 1992;132:299–304. [PubMed]
27. Pickavance LC. Therapeutic index for rosiglitazone in dietary obese rat: separation of efficacy and haemodilution. *British Journal of Pharmacology* ([www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov)); 1996.
28. N. N. Ngwa and N. M. Nnam (2019). Antiobesity Activity of Flavonoids Isolated from *Solanum macrocarpum* in Wistar Rats. *Annual Research & Review in Biology*.31(2)34-43.<https://doi.org/10.9734/arrb/2019v31i230043>.
30. Trinder P. Enzymatic calorimetric determination of triglycerides by GOP- PAP method. *Ann Clin Biochem.* 1969;6:24–7. [Google Scholar]
31. Warnick GR, Wood PD. National Cholesterol Education Program recommendations for measurement of high-density lipoprotein cholesterol: Executive summary. The National Cholesterol Education Program Working Group on Lipoprotein Measurement. *Clin Chem.* 1995;41:1427–33. [PubMed] [Google Scholar]
32. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem.* 1972;18:499–502. [PubMed] [Google Scholar]

33. Brisson GJ. Netherlands: Springer; 1981. Lipids in Human Nutrition. The Enigma of the Trans Fatty Acids, An Appraisal of Some Dietary Concepts; pp. 41–71. [Google Scholar]
34. Kim DH, Jung EA, Sohng IS, Han JA, Kim TH, Han MJ. Intestinal bacterial metabolism of flavonoids and its relation to some biological activities. *Arch Pharm Res.* 1998;21:17–23. [PubMed] [Google Scholar]
35. Higashimoto M, Yamato H, Kinouchi T, Ohnishi Y. Inhibitory effects of citrus fruits on the mutagenicity of 1-methyl1,2,3,4-tetrahydro-beta-carboline-3-carboxylic acid treated with nitrite in the presence of ethanol. *Mutat Res.* 1998;415:219–26. [PubMed] [Google Scholar]
36. Le Marchand L, Murphy SP, Hankin JH, Wilkens LR, Kolonel LN. Intake of flavonoids and lung cancer. *J Natl Cancer Inst.* 2000;92:154–60. [PubMed] [Google Scholar]
37. Tsai SH, Lin-Shiau SY, Lin JK. Suppression of nitric oxide synthase and the down-regulation of the activation of NFkappaB in macrophages by resveratrol. *Br J Pharmacol.* 1999;126:673–80. [PMC free article] [PubMed] [Google Scholar]
38. Havsteen B. Flavonoids, a class of natural products of high pharmacological potency. *Biochem Pharmacol.* 1983;32:1141–8. [PubMed] [Google Scholar]
39. Choi JS, Yokozawa T, Oura H. Antihyperlipidemic effect of flavonoids from *Prunus davidiana*. *J Nat Prod.* 1991;54:218–24. [PubMed] [Google Scholar]
40. Kurowska EM, Spence JD, Jordan J, Wetmore S, Freeman DJ, Piché LA, et al. HDL-cholesterol-raising effect of orange juice in subjects with hypercholesterolemia. *Am J Clin Nutr.* 2000;72:1095–100. [PubMed] [Google Scholar]
41. Cerda JJ, Robbins FL, Burgin CW, Baumgartner TG, Rice RW. The effects of grapefruit pectin on patients at risk for coronary heart disease without altering diet or lifestyle. *Clin Cardiol.* 1988;11:589–94. [PubMed] [Google Scholar]
42. Holzapfel NP, Holzapfel BM, Champ S, Feldthusen J, Clements J, Hutmacher DW. The potential role of lycopene for the prevention and therapy of prostate cancer: From molecular mechanisms to clinical evidence. *Int J Mol Sci.* 2013;14:14620–46. [PMC free article] [PubMed] [Google Scholar]

43. Rahman, M., Islam, B.,.....Alam, K. (2015). In vitro antioxidant and free radical scavenging activity of different parts of *Tabebuia pallida* growing in Bagladesh. BMC Research Notes.
44. Verlecar XN, Jena KB, Chainy GB. Biochemical markers of oxidative stress in *Perna viridis* exposed to mercury and temperature. *Chem Biol Interact.* 2007;167:219–26. [PubMed] [Google Scholar]
45. Kanungo SK, Panda DS, Swain SR, Barik BB, Tripathi DK. comparative evaluation of hypolipidemic activity of some marketed herbal formulations in triton induced hyperlipidemic rats. *Pharmacol Online.* 2007;3:211–21. [Google Scholar]
46. Patel DK, Patel KA, Patel UK, Thounaoja MC, Jadeja RN, Ansarullah, et al. Assessment of lipid lowering effect of *Sida rhomboidea*. Roxb methanolic extract in experimentally induced hyperlipidemia. *J Young Pharma.* 2009;1:233–8. [Google Scholar]
47. Anoosh E, Mojtaba E, Fatemeh S. Study the effect of juice of two variety of pomegranate on decreasing plasma LDL cholesterol. *Procedia Soc Behav Sci.* 2010;2:620–3. [Google Scholar]