Asian Journal of Research in Biochemistry

Volume 12, Issue 3, Page 30-40, 2023; Article no.AJRB.99375
ISSN: 2582-0516

Modulation of the Expression of Lipogenic Activity by Malted Hungry Rice Flour (*Digitaria exilis*.) on Rats Induced with Obesity

Umerah, Nkemjika Nnedinso [a*] and Asouzu, Ann Ifeoma [b]

[a] *Department of Food Science and Technology, Enugu State University of Science and Technology, P.M.B.-01660, Enugu, Nigeria.*
[b] *Department of Home Economics, Ignatius Ajuru University of Education, Port Harcourt, Nigeria.*

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJRB/2023/v12i3238

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/99375

Original Research Article

Received: 22/02/2023
Accepted: 24/04/2023
Published: 23/05/2023

ABSTRACT

Lipolysis and lipogenesis causes obesity and these can be mediated by hormones. The study was aimed at determining the effect of malted hungry rice flour on lipogenic activity on diet-induced obese rats. The study was conducted between September to January 2021 and 2022. Twenty male adult albino rats were divided into four groups and obesity was induced which resemble mild obesity condition in human. The groups for the experiment were normal control (AIN-93), obese control, and two test diets were used to evaluate lipid profile parameter using a standard assay technique. There is an increase in BMI (from 0.37-0.41 to 0.58-0.72). There are also an increase in other lipid profile with a decrease in HDL ($p<0.05$) after consumption of a palatable diet. Consumption of the test diet led to a significant reduction

**Corresponding author: E-mail: nkemumerah@yahoo.com;*

in the lipid profile parameter, and a significant increase in HDL cholesterol compared to the (group) obese control ($p<0.05$). The result suggests that malted hungry rice flour has an anti-obesity effect.

Keywords: Obesity; lipogenic; hungry rice; insulin; antihyperlipidemia; atherosclerosis.

1. INTRODUCTION

Obesity is a nutritional disorder caused by a disarray of energy balance. Obesity is a public health concern in both developed and developing countries. In developing Country, the affluent are more vulnerable to obesity but in developed Country the reversed is the case. This prevalence of obesity is associated with some health disorders such as cardiovascular diseases, hyperlipidemia, insulin resistance, non-alcoholic fatty liver, hypertension, and osteoarthritis [1,2]. Energy inbalance between intake and expenditure has been implicated as the leading cause of obesity [3,4]. Hyperlipidemia are mainly caused by high blood pressure, diet rich in saturated fatty acid, age, family history, and life pattern.

Studies showed that hyperlipidemia is associated with high level of triglyceride (TG), total cholesterol (TC), low density lipoprotein (LDLc) cholesterol, and low level of high density lipoprotein (HDLc) cholesterol [5]. Hyperlipidemia, over time has led to atherosclerosis and coronary heart diseases, but may be managed through diet modification. There is a strong correlation between low density lipoprotein reduction and decreased cardiovascular disease mortality [6,7]. Diet rich in polyphenols and antioxidants have been reported to exhibit antiatherogenic effects [8]. Dietary management of atherosclerosis can be achieved with a plant-based diet comprising of fruit, vegetables, and legumes and at the same time low in saturated fat [8].

Research has geared towards search for the antilipidemic agents from natural origin. The use of plants for food and medicine has been in existence from time immemorial. More than 30% of drugs presently used in modern medicine are derived from plants because they contained biologically active ingredients [9,10]. There are some studies on plant foods possessing lipid lowering activities [11-14]. 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 is cultivated to provides food for both human and animal feeding [15]. In Nigeria, it is popularly known as "acha" and are produce mainly in (Kebbi, Kaduna, Bauchi, Niger, Plateau) and Abuja [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 a nutritious grains and is among the world's fastest maturing grain [17]. In most community, acha is used in preparation of porridge food and other dishes in Nigeria [14]. It is also used in dietary management of a diabetic patients [18]. Production of beverage (malt drink) with barley (*Hordeum vulgare L*.) can be perfectly reproduced with acha [19]. Nigeria is one of the African Country with a rich heritage of medicinal food crops of wide diversity, which are used by the local population and traditional healers for the treatment of several diseases. This study attempted to determine the effect of malted and unmalted hungry rice flour (*Digitaria exilis*.) on diet-induced obese (DIO) rats.

2. MATERIALS AND METHODS

2.1 Procurement of Sample Materials

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

2.2 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 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.

2.3 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.

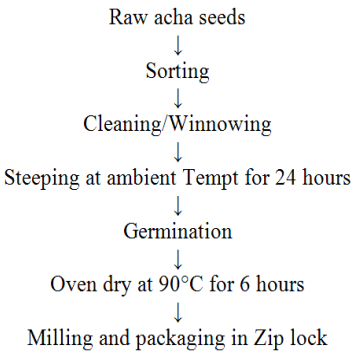

Fig. 1. Flow diagram of production of malted acha

2.4 Determination of Phenolic Content

Folin– reagent was used for the determination of phenolic content of the sample. Approximately 1 mL of this reagent was added to 1 mL of Na_2CO_3 7.5% and to 200 µL of sample previously prepared. The results were expressed as mg of chlorogenic acid equivalents per g of dry matter (mg CAE/g DM).

2.4.1 Preparation of extract

The extract of 200 µL was 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].

2.5 Determination of Flavonoid Contents

The aluminum chloride colorimetric method was used to determine the flavonoid content of the sample. About 1 mL of $AlCl_3$ 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 quercetin per g of dry matter (mg QE/g DM).

2.6 DPPH Assay

The method of Marelli et al. [22] was used to determine the antioxidant activity of malted and unmalted sample by DPPH free radical scavenging ability. 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 µg/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. Blank is the solution containing methanol and DPPH without sample. The percentage inhibition of DPPH free radical was calculated by using the following formula:

Percentage of inhibition = {1 − [(DPPH absorbance with extract)/(DPPH absorbance without extract)] × 100}.

Ascorbic acid is the positive control.

2.7 Experimental Design

The study used a completely randomized design (CRD). Based on their weights, rats were distributed randomly among the treatments. Four treatments were used, and each was reproduced five times. The various diets served as the treatments, while the rats served as the duplicates.

2.7.1 Animal housing

The Faculty of Veterinary Medicine, University of Nigeria Nsukka, Nigeria, sold a total of twenty adult male Albino rats that were from the same colony and weighed between 150 and 153g. The rats were placed in metabolic cages that could separate waste from urine. For the rats, a day consisted of precisely 12 hours of light and 12 hours of darkness. The National Research Council's [23] recommendations for the handling and usage of experimental animals were strictly followed. Animal Experimentation Ethics Committee of University of Nigeria Nsukka approved the study.

2.8 Induction of Obesity

Based on their body weight, the animals were separated into 4 groups of 5 rats each, making sure that the difference between the mean body weights of each group did not exceed 5g [24]. The animal house at the Department of Home Science, Nutrition, and Dietetics at the University of Nigeria Nsukka was used for the rats. Prior to the trial, the animals were acclimated for a period of two weeks. For three weeks, the rats were given a delicious diet to encourage mild obesity. 60% of the energy in rodent pelleted food comes from carbohydrates, 30% from protein, and 10% from fat. The tasty diet was made up of additional water and 33% chow, 33% condensed milk, and 7% sugar by weight. Carbohydrates will make up 65% of the energy, followed by proteins at 19% and fat at 16%. In contrast to standard rat food, this diet was created to encourage weight growth through hyperphagia without significantly altering the content of the macronutrients. Insulin resistance and weight gain can be reliably induced using this technique [25-28]. Rats were kept on a 12:12 h light:dark cycle and given free access to water during the research.

At the conclusion of the three-week period, when the rats given the palatable diet had significantly put on weight. The animals received the designed meals for a period of six weeks. Group 1 was given normal control (AIN-93), Group 2 was given obese control, Group 3 was given unmalted acha flour, and Group 4 was given malted acha flour. The AIN-93G (American Institute of Nutrition) approach was used to formulate the diets [29]. Animal lengths and weights were measured every day. In order to calculate nutritional intake, daily food intake and food extract were also recorded.

2.9 Blood Sample Collection and Biochemical Indices determination

Rats' eyes' retro of the medial canthus were used to collect blood. In order to pierce the retrobulba plexus and allow for the outflow of around 2 ml of blood into a clean glass test tube, a nucrocapillary tube was delicately inserted into the canthus of the eye. The blood sample was allowed to coagulate for 30 minutes at room temperature. The test tube containing the clotted blood sample was then centrifuged using a table centrifuge for ten minutes at 3,000 revolutions per minute to completely separate the serum from the clotted blood. After gently aspirating the clear serum supernant with a syringe and needle, it was placed in a clean sample bottle for the clinical chemical analysis. For the purpose of determining the lipid profile, blood was drawn at weeks 0, 3, and 6. The total cholesterol, LDL, HDL, and triglyceride levels in the serum were measured.

2.10 Biochemical Assay

A chemical store in Enugu, Nigeria delivered standard enzymatic colorimetric kits for the evaluation of total cholesterol, LDL, HDL, and serum triglycerides.

2.10.1 Cholesterol

The CHOD-PAP method, an enzymatic colorimetric test for cholesterol using Lipid Clearing Factor, was used to detect the serum's cholesterol content. The enzyme reagent was combined with approximately 10 l of sample in a cuvette, incubated for 5 min, and the absorbance was measured after 60 min [30].

2.10.2 Triglycerides

The GPO-PAP method, an enzymatic colorimetric test, was used to determine TGs. A cuvette containing 10 l of sample and 1 mL of mono-reagent was incubated for 10 min before measuring the absorbance after 60 min [30].

2.10.3 High-density lipoprotein-cholesterol

The procedures entailed carefully precipitating and removing VLDL and LDL, then measuring high-density lipoprotein (HDL) in the supernatant fraction enzymatically [31]. The human cholesterol Test Kit was utilized in this approach along with HDL cholesterol, a precipitant, and a standard. Approximately200 µl of the sample

were combined with 100 µl of distilled water and 400 µl of the PREC HDL reagent, which was then incubated for 10 min at 3000 rpm for 10 min. Within 60 minutes, the absorbance of approximately 100 µl of clear supernatant in a separate tube containing 1000 l of cholesterol reagent was measured [30].

2.10.4 Low-density lipoprotein-cholesterol

With the aid of a test kit from Human, Germany, the LDL cholesterol was determined. All of the chemicals were warmed to 37°C before the 10 l sample and 750 l enzyme were combined and incubated for 5 min. The absorbance was measured at 234 nm after 5 minutes of incubation at 37°C with 250 l of substrate [32,33].

2.11 Statistical Analyses

Analyses of phytochemicals and antioxidants were carried out in triplicate. The data was presented as mean ± SD. SPSS was used to analyze and fit the biological data, and One-way analysis of variance (ANOVA), followed by the Duncan New multiple range test, was used to evaluate the statistical differences between the treatment groups and the control group as well as between the treated groups' mean values.

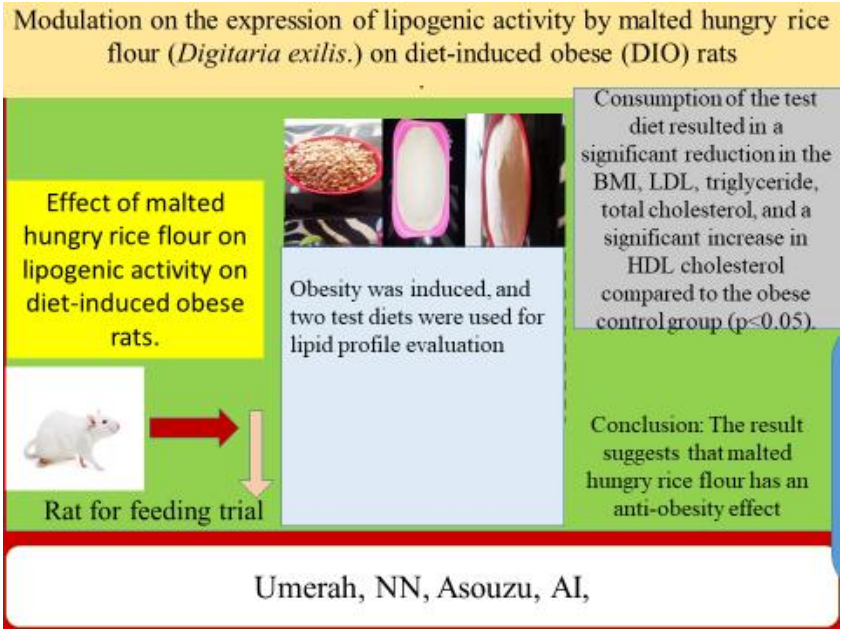

Fig. 2. Modulation on the expression of lipogenic activity by malted hungry rice flour (*Digitaria exilis*.) on diet-indiced obese (DIO) rats

3. RESULTS

Table 1. Effect of malting on the Phytochemical and antioxidant composition of "hungry rice" flour, The units are Phenol (mg CAE/g DM), Flavonoids (mg QE/g DM) and DPPH (%)

Samples	Phenol	Flavonoids	DPPH
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

Table 2. The diets effects on the body weight of the rats (g)

DAYS	GP1	GP2	GP3	GP4
0 WEEK	154.30^{A}±0.71	154.10^{A}±0.35	153.90^{A}±0.46	155.00^{A}±0.16
2 WEEK	154.45^{B}±0.23	231.40^{A}±0.12	232.00^{A}± 0.98	231.10^{A}±0.64
6 WEEK	154.65^{C}±0.64	232.80^{A}±0.04	219.20^{B}±0.03	149.00^{D}±0.14

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 showed the effects of the diets on the rats' body weight. The results showed that the rats' mean body weight on the first day after acclimatization (0 week) ranged between 153.90 and 155.00, 154.45-232.00 on week 2, and 149.00-232.80 on week 6 following therapy. On the first day following acclimatization, or week 0, there was no significant difference in the means. There was a significant difference between the rats in group 1 and the rats in groups 2, 3, and 4 on week 2, which was the day after obesity was induced, but there was no significant difference between the mean body weight of the rats in groups 2, 3, or 4. On the sixth week of treatment, the last day of treatment, there are appreciable differences in the mean body weights of all the groups.

The effects of the diets on the rats' weight gain, food intake, and organ weight were shown in Table 3. According to the results, the rats' mean daily weight gain varied between 0.04-6.20 g, their food intake from 12.20–14.05 g, their FER from 0.003-0.44 g, their feces from 3.67–7.28 g, their hearts from 2.54–3.20 g, their livers from 16.31–31.14 g, their kidneys from 2.39–2.90 g, and their brains from 2.24–3.69 g.

Table 4 showed the effect of diets on the BMI of the rats. The findings revealed that the rats' mean BMI varied between between 0.62-0.64g/cm^2 on the first day after acclimatization (0 week), 0.64-0.90 (2 week) and 0.55-0.91g/cm^2 on the 6th weeks after treatment. There was no significant different among the mean on week 0, the first day following acclimation. On week 2, the following day after causing obesity, there was a significant different between the rats in group 1 and the rats in group 2, 3 and 4 although the mean body weight of the rats in each group did not differ significantly from one another 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 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 6th 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. Rats fed test diets their weight gain, food intake and organ weight

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

Table 4. Effect of the diets on the BMI of the rats (g/cm^2)

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

Table 5. Rats' total cholesterol after dietary changes (measured in mg/dl)

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

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

Days	GP1	GP2	GP3	GP4
0 Week	80.65[a]±0.0	82.50[a]±0.01	82.81[a]±0.02	81.16[a]±0.21
2 Week	81.38[b]±0.06	138.90[a]±0.11	139.10[a]±0.05	138.84[a]±0.07
6 Week	81.51[c]±0.02	139.20[a]±0.03	84.70[b]±0.13	80.36[c]±0.02

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

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

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

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

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 6th 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 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 6th 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 showed how diets affected the rats' HDL levels. The findings demonstrated that the rats' mean body weight varied between 31.40 and 32.90 mg/dl on the first day following acclimatization (0 week), 23.69 and 33.05 mg/dl on week two, and 22.90 and 33.00 mg/dl on week six following therapy. On the first day following acclimatization, or week 0, there was no discernible difference in the means. There was a significant difference between the rats in group 1 and the rats in groups 2, 3, and 4 on week 2, which was the day after obesity was induced, but there was no significant difference between the mean body weight of the rats in groups 2, 3, or 4. On the sixth week of treatment, the last day of treatment, there are appreciable differences in the mean body weights of all the groups.

4. 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]. The hypocholesterolemic effect of flavonoids in rats has been demonstrated in a number of in vivo experiments [28]. Phytochemicals lower blood lipid levels and prevent excessive cholesterol absorption [41]. The result of the antioxidant showed 76.78% and 93.63% based on DPPH for unmalted and malted hungry rice flour respectively. Prostate cancer risk appears to be reduced by antioxidants [42]. Antioxidant can scavange free radical and minimize their impact [43].

Atherosclerosis in particular is at increased risk due to hyperlipidemia. One of the primary causes of early death worldwide is coronary artery disease (CAD) [44]. Hyperlipidemia and atherosclerosis are primarily brought on by poor eating habits and inactivity. Numerous investigations on both humans and animals have shown that saturated fatty acids have a hypercholesterolemic effect, increasing total cholesterol and altering the distribution of lipoproteins [8]. Recently, studies have been geared towards identifying numerous food with antihyperlipidemic activities in order create variety and minimize monotonous food habit among obese patients. According to the study's findings, eating a high-fat diet caused rats' BMI, TC, TG, and LDL levels to significantly rise while their HDL levels dropped. 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, demonstrating the food's effectiveness in reducing atherosclerosis. A drop in plasma HDL cholesterol levels may hasten the development of atherosclerosis, which results in ischemic heart disorders, according to studies that demonstrate an inverse relationship between HDL and total body cholesterol [45]. The high phytochemical and antioxidant content of both malted and unmalted hungry rice may be to blame for the rise in HDL cholesterol. Several studies have proved that flavonoids increase HDL cholesterol concentration and reduce LDL and VLDL levels in the hypercholesterolemic rats [46]. Therefore, flavonoids 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 weigh 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. Malted and unmalted hungry rice may have a lipid-lowering effect due to their flavonoid and antioxidant properties, which inhibit macrophage uptake of oxidized LDL, reduce LDL aggregation, and reduce LDL oxidation [47]. Macrophages play a crucial role in these processes.

5. CONCLUSION

The findings of a recent study indicate 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 patience and weight watcher where the food is dominant. Hungry rice can be processed into malted flour for protection against atherosclerosis.

ETHICAL APPROVAL

Animal Ethic committee approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Yuan HD, Kim SJ, Quan HY. Ginseng leaf extract prevents high fat diet induced hyperglycemia and hyperlipidemia trough AMPK activation. Journal of Ginseng Research. 2010;34(4):369–75.
2. Saravanan G, Ponmuragan P, Deepa MA. 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. 2014;94(14):2972–2977.
3. Mopuri R, Idress HA, Avinash P, Meriga B. Evaluation of anti lipedemic and anti-obesity efficacy of *Bauhinia purpurea* bark extract on rats fed with high fat diet. International Journal of Plant Science. 2010;3:104–107.
4. Bhutani KK, Birari RB, Kapat K. Potential anti-obesity and lipid lowering natural products; a review. Nature Production Communication. 2017;2:331–348.
5. Bauman WA, Spungen AM, Zhong YG, Rothstein JL, Petry C, Gordon SK. Depressed serum high density lipoprotein cholesterol levels in veterans with spinal cord injury. Spinal Cord. 1992;30(10):697-703.
6. Nwangwa EK, Ekhoye EI. Anti-hyperlipidemic Activity of aqueous extracts of Carica papaya seeds in albino rats fed with high fat diet. Current Trends in Technology and Science. 2013;II:2279.
7. Anaka Ogochukwu Ngozi Owolabi, Omonkhelin Josephine, Emenike Chinwendu, Antihyperlipidemic effect of aqueous leaf extracts Emilia praetermissa niline-redh (Asteraceae) in rats. Inter J, of Biosciences. 2013;356-877.
8. Neelam M, Rafeeq AK. Antihyperlipidemic effects of Citrus sinensis, Citrus paradisi, and their combinations. J Pharm Bioallied Sci. 2016;8(2): 112–118.
DOI: 10.4103/0975-7406.171727
9. Mouhssen L. Screening of natural products for drug discovery. Expert Opin Drug Discovery 2007;2:697–705. [Taylor & Francis Online], [Web of Science ®], [Google Scholar]
10. Mukherjee PK, Rai S, Kumar V, Mukherjee K, Hylands PJ, Hider RC. Plants of Indian origin in drug discovery. Expert Opin Drug Discovery. 2007;2:633–657. [Taylor & Francis Online], [Web of Science ®], [Google Scholar]
11. Khaled M, Mohamed K. Antihyperlipidemic activity of the medicinal plants among Kadazan and Dusun communities in Sabah, Malaysia: a review. Asian Pacific Journal of Tropical Biomedicine. 2014;4(10):768-779.
12. Anwar F, Latif S, Ashraf M, Gilani AH. Moringa oleifera: a food plant with multiple medicinal uses. Phytotherapy Research: An International Journal Devoted to Pharmacological and Toxicological Evaluation of Natural Product Derivatives. 2007 Jan;21(1):17-25.
13. Muhammad BA, Hseed A. Recent molecular mechanism and beneficial effect of phytochemicals and plant-based whole foods in reducing LDL-c and Preventing Cardiovascular Disease; 2021.
14. Sudha RP, Sushma AM. Antihyperlipidemi effect of aqueous extract of phembago zeylemia roots in diet induced hyperlipidemia rat. Phamaceutical Biology. 2009;47:10.
15. Kwon-Dung EH, Misari SM. 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. Anuonye JC, Badifu GIO, Inyang CU, Akpapunam MA, Odumodu CU, Mbajika VI. Protein dispersibility index and trypsin inhibitor activity of extruded blends of acha/soyabean. A re- sponse surface analysis.American Journal of Food Technology. 2007;2(6):502-511.
17. NAS, Lost crops of Africa, Grains. NationalAcad- emy press Washington D.C. 1996;1.
18. Victor JT, James DB. Proximate chemical com- position of acha (*Digitaria exilis*) grain. Journal Science Food Agriculture. 1991;56:561-563.
19. Okon EU. Effect of mash constitution on sugar production in malted sorghums. Nigeria Food Journal1988;6: 54-60.
20. Perri MR, Marrelli M, Statti G, Conforti F. Olea europaea buds extracts: Inhibitory effects on pancreatic lipase and α-amylase activities of different cultivars from Calabria region (Italy). Plant Biosyst; 2020.
[Google Scholar (https://scholar.google.com/scholar_
[CrossRef (https://doi.org/10.1080/11263504.2020.1857868)]
21. Marrelli M, Argentieri MP, 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, Chiocchio 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. 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 rosigitazone in dietary obese rat: separation of efficacy and haemodilution. British Journal of Pharmacology (www.ncbi.nlm.nih.gov); 1996.
28. Ngwa NN, Nnam NM. Antiobesity Activity of Flavonoids Isolated from Solanum macrocarpum in Wistar Rats. Annual Research & Review in Biology. 2019; 31(2):34-43. Available:https://doi.org/10.9734/arrb/2019 v31i230043.
29. Hintze KJ, Benninghoff AD, Ward RE. Formulation of the Total Western Diet (TWD) as a basal diet for rodent cancer studies. Journal of agricultural and food chemistry. 2012 Jul 11;60(27):6736-42.
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. Lipids in Human Nutrition. The Enigma of the Trans Fatty Acids, An Appraisal of Some Dietary Concepts. 1981;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. *In-vitro* antioxidant and free radical scavenging activity of different parts of *Tabebuia pallida* growing in Bagngladesh. BMC Research Notes; 2015.
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]

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/99375