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Research article

A Cameroon Western Regions high-fat diet (MACAPOS 2) induces visceral obesity in rat

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ABSTRACT

The prevalence of obesity increases yearly in the world. The traditional local diet of the Western Regions of Cameroon was suspected to be the main contributor to the high prevalence of obesity in these Regions. This study aimed to evaluate the effects of a Cameroon-comparable fat diet on visceral obesity in rats.

Two groups of male *Wistar* rats were fed for four months with respectively a normal diet (ND) (3400 kcal/kg of food) and a high-fat diet (HFD) containing maize, cassava, palm oil, and sugar (MACAPOS 2): 35 % carbohydrate, 55 % fat and 10 % proteins (4730 kcal/kg of food). Lee index, body weight, food intake, blood and hepatic lipids, body fat, insulin resistance, glucose tolerance, glycemia, serum insulin, leptin, and adiponectin were evaluated.

HFD significantly ($P < 0.01$) increased body weight and decreased food intake. After four months of diet, 88.8 % of HFD rats were obese (Lee index >30 g/cm), and HFD significantly increased visceral and subcutaneous fats compared to ND. HFD increased triglyceride, total cholesterol, Low-density lipoprotein-cholesterol levels, and the atherogenic index, while the high-density lipoprotein-cholesterol level was decreased. The hepatic triglyceride and total cholesterol levels significantly ($P < 0.01$) increased in HFD, compared to ND. In HFD, the fasting blood glucose, serum insulin, and leptin levels significantly ($P < 0.01$) increased, meanwhile adiponectin decreased. HFD-induced glucose intolerance and insulin resistance in rats.

Based on our findings, we can conclude that HFD MACAPOS 2 can induce central obesity. Therefore, it can be used as a model of diet-induced obesity.

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1. Introduction

Obesity is an excess body fat accumulation due to an excessive imbalance between regular energy intake and expenditure [1]. Factors like economic growth, modernization, urbanization, environmental and genetic mutations, increased consumption of hypercaloric food rich in saturated fats and sugars, and reduced physical activities are the main causes of obesity [2]. Obesity represents a significant public health problem in the world [3], more than 1.9 billion adults were overweight and over 650 million overweight adults were obese in 2016 [4]. Although obesity is more common in developed countries, developing countries have been projected to have a larger proportional increase in the number of obese individuals in 2030 [5]. In Africa, obesity is increasing annually. WHO reported that obesity prevalence increased from 12 % in 2000 to 18.4 % in 2021 in women, and from 4.1 % to 7.8 % in men during the same period [6]. Cameroon is not an exception; here, obesity prevalence increased from 4.9 % (2009) to 9.5 % (2016) [7] with the highest prevalence from the Western Regions [8,9]. Central or visceral obesity is characterized by excessive visceral fat with fat droplets surrounding the abdominal organs. Fat storage is very harmful because it has been strongly linked to metabolic dysfunctions including insulin resistance, dyslipidemia, hepatic steatosis, type II diabetes, some types of cancer, cardiovascular diseases, and metabolic syndrome [10–12]. For a better understanding of obesity and to evaluate potential treatments, various animal models have been developed. Some of them include genetic, surgical, chemical, and diet-induced models of obesity [13]. However, diet-induced obesity represents the one that mimics human obesity. Different hypercaloric diets such as high fat, high sucrose, or high fat/high sucrose diets have been used to induce obesity in rodents since they seem to be the most effective strategy to set up an obesity model close to humans [14,15]. Investigations revealed that the high prevalence of obesity among the natives of Cameroon's Western Regions could mainly be attributed to its traditional high-caloric diet based on natural palm oil, [16,17]. Hence, a typical diet of these Regions was conceived with maize, cassava, palm oil, and sugar (MACAPOS 2) to elucidate its obesogenic potential [18,19]. The present work was undertaken to evaluate the effects of MACAPOS 2 on visceral obesity in *Wistar* rats.

2. Results

During the HFD obesity induction period, no signs of toxicity or adverse reactions were observed. An important number (88.8 % of the animals) was obese according to the Lee Index.

2.1. Food intake and body weight during the diet and Lee index after a four-month diet

During the four-month diet, the food intake remained significantly ($P < 0.01$) low in HFD compared to ND (Fig. 1a). The increase of the body weight gain was significant ($P < 0.01$) in HFD compared to ND on the 1st, 2nd, 3rd, and 4th month; respectively +77.42, +48.09, +62.85, and +78.53 % (Fig. 1b). The Lee index after the four-month diet was significantly ($P < 0.01$) high in the HFD compared to ND: 31.39 ± 0.43 g/cm vs 29.71 ± 0.07 g/cm (Fig. 1c).

2.2. Fat mass

After the four-month diet, fat accumulation in HFD animals significantly ($P < 0.01$) increased compared to ND: +116,19, +317,25, +341,56, and +139,10 % respectively for mesenteric, subcutaneous, perirenal, and peri testicular fats (Fig. 2a). Compared with ND (Fig. 2b1), the HFD rat's abdomen morphology showed a significant fat accumulation (Fig. 2b2).

2.3. Serum lipid profile

After the four-month diet, TC, TG, and LDL-C significantly ($P < 0.01$) increased in HFD rats respectively with 1.98 ± 0.01 g/L, 1.61 ± 0.01 g/L, and 1.34 ± 0.02 g/L compared to ND: 1.08 ± 0.16 g/L, 0.71 ± 0.10 g/L, and 0.50 ± 0.14 g/L. HDL-C slightly decreased: 0.32 ± 0.03 g/L vs 0.43 ± 0.04 g/L (Fig. 3a). Compared to the ND, HFD increased the atherogenic index to 6.48 ± 2.48 g/L (against

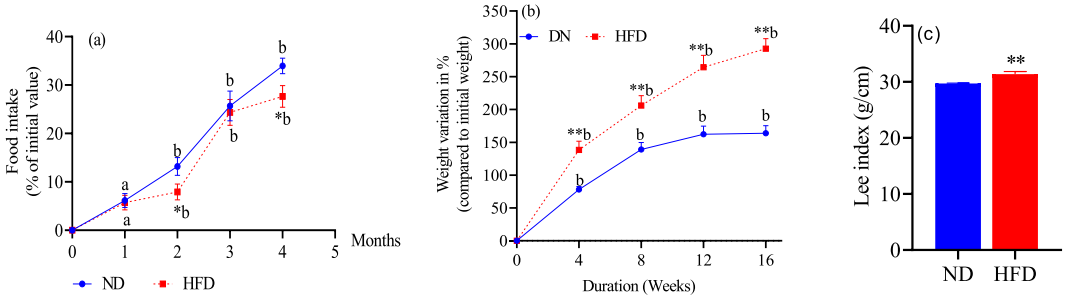

Fig. 1. Food intake (a), Body weight variation or weight gain (b), and Lee index (c) of rats during or after four months of diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each point represents the Mean ± SEM. Significant difference: *P < 0.05, **P < 0.01 compared to ND; ^{a}P < 0.05, ^{b}P < 0.01 compared to the initial value.

2.88 ± 0.09 g/L in ND) (Fig. 3b).

2.4. Fasting blood glucose, serum insulin level, and HOMA index

HFD significantly increased fasting blood glucose and insulin levels, respectively to 106.80 ± 2.08 mg/dL and 218.81 ± 11.59 pg/mL against 91.00 ± 1.67 mg/dL ($P < 0.05$) and 62.49 ± 6.89 pg/mL ($P < 0.01$) in ND (Fig. 4a and b). Similarly, the HOMA index was significantly increased in HFD compared to ND, indicating an insulin resistance status in HFD-fed rats (Fig. 4c).

2.5. Serum leptin and adiponectin

After the four-month diet, the serum leptin level significantly ($P < 0.01$) increased in the HFD animal group compared to ND: 4.48 ± 0.34 ng/mL vs 2.15 ± 0.14 ng/mL (Fig. 5a); meanwhile, the serum adiponectin level remarkably decreased ($P < 0.01$): 143.19 ± 15.67 pg/mL in HFD vs 260.78 ± 14.31 pg/mL in ND (Fig. 5b).

2.6. Oral glucose tolerance test (OGTT)

After oral glucose administration, glycemia in HFD considerably remained high at 30, 60, and 120 min: respectively 156.00 ± 3.59, 143.80 ± 4.87 and 149.80 ± 3.86 mg/dL compared to the ND: 111.60 ± 4.10, 120.40 ± 4.91 and 101 ± 4 mg/dL respectively (Fig. 6a). The HFD regimen increased the area under the curve compared to the ND (Fig. 6b).

2.7. Insulin tolerance test (ITT)

After insulin injection, glycemia in HFD rats increased. Changes in glycemia levels were +27.97, +18.62, and +63.15 % respectively at 15, 30, and 60 min compared to ND which glycemia levels variation at the same period were – 26, - 46, and - 58 % (Fig. 7a). The HFD regimen increased the area under the curve compared to the ND (Fig. 7b).

2.8. Hepatic lipids

After the four-month diet, TC and TG levels in the liver tissue of ND animals were 1.19 ± 0.04 mg/dL and 0.79 ± 0.09 mg/dL respectively. HFD significantly ($P < 0.01$) increased hepatic TC to 2.50 ± 0.07 mg/dL (+109.09 %), and TG to 1.84 ± 0.08 mg/dL (+131.38 %) compared to ND (Fig. 8).

3. Discussion

This study aimed to evaluate the effects of a high-fat diet regimen inspired by a traditional diet of Cameroon Western Regions MACAPOS 2 on visceral obesity in rats. Previous studies showed that MACAPOS 2 could induce hyperglycemia associated with glucose intolerance, and dyslipidemia in rats [18,19]. *Wistar* rats were used in our investigation because of their susceptibility to produce diet-induced obesity close to the human obesity model. Most of the metabolic effects caused by high-fat diets were early detected or more pronounced in *Wistar* rats than in other strains and several studies use this strain for experimental diet-induced obesity [15]. Lee index was developed to identify obesity in rats compared to the body mass index (BMI) used to diagnose human obesity. Lee indexes over 300 g/cm were seen as a sign of obesity in rats [29]. Our findings revealed an increase in the Lee index in MACAPOS 2 rats after the four-month diet. This indicates that MACAPOS 2 might generate obesity. The high level of the Lee index could be related to increased weight gain and visceral fat mass. Some studies indicated that rats fed with a HFD show higher weight gain compared to the

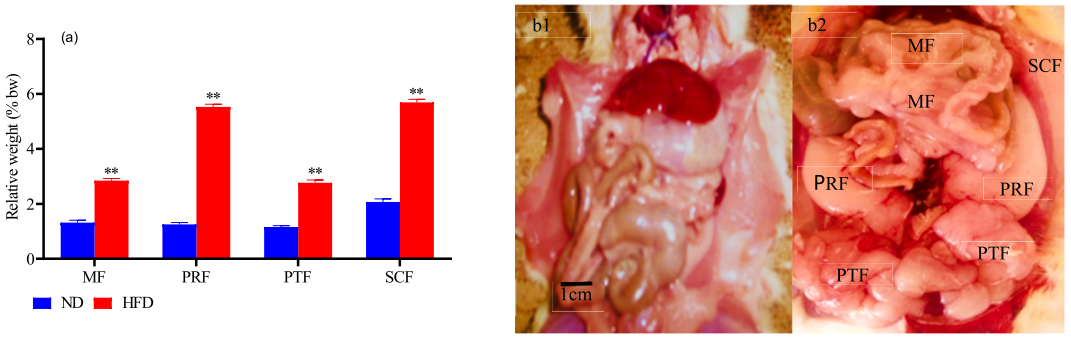

Fig. 2. Mesenteric (MF), peri-renal (PRF), Peri-testicular (PTF), and subcutaneous (SCF) relative weight fat accumulation (a) expressed as % of rat body weight and Abdomen morphology of normal (b1) and HFD (b2) rats after four months diet. ND: Normal diet, HFD: High-fat diet, MF: Mesenteric fat, SCF: subcutaneous fat, PRF: peri-renal fat, PTF: peri-testicular fat. n = 5. Significant difference: **$P < 0.01$ compared to ND. Each column represents the Mean ± SEM.

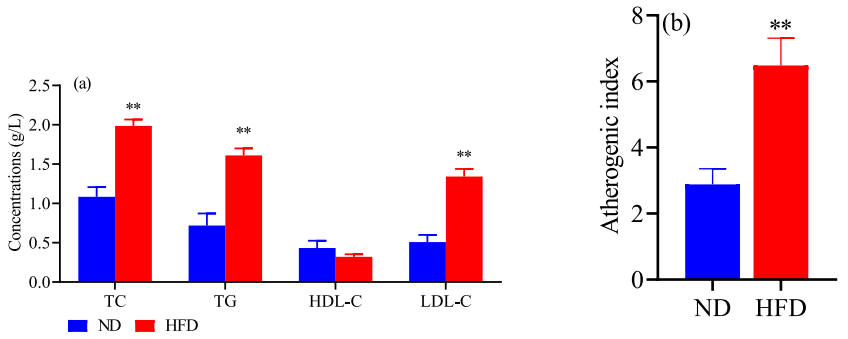

Fig. 3. Serum lipids (a) and atherogenic index (b) of rats after four months of diet. ND: Normal diet, HFD: High fat diet, TC: Total cholesterol, TG: Triglycerides, HDL-C: HDL-cholesterol, LDL-C: LDL-cholesterol. n = 5. Each column represents the Mean ± SEM. Significant difference: *P < 0.05, **P < 0.01 compared to ND.

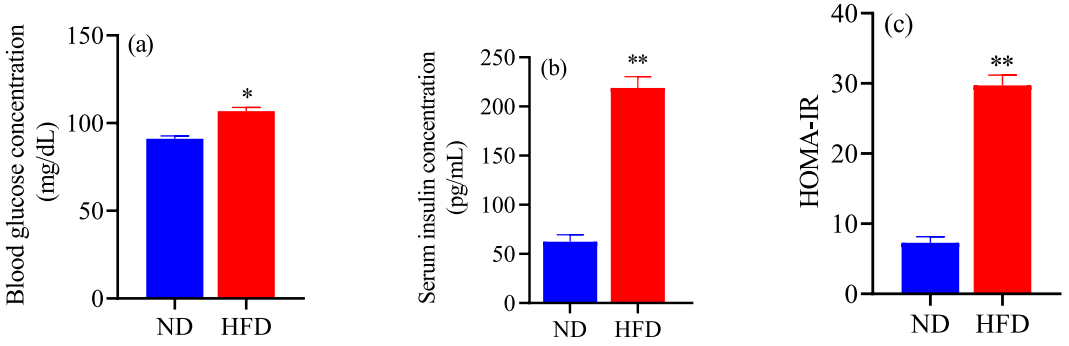

Fig. 4. Fasting level of glucose (a), insulin (b), and HOMA-IR index (c) of rats after four months of diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each column represents the Mean ± SEM. Significant difference: *P < 0.05, **P < 0.01 compared to ND.

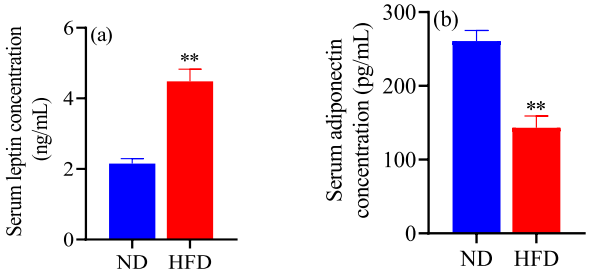

Fig. 5. Serum levels of leptin (a) and adiponectin (b) of rats after four months of diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each column represents the Mean ± SEM.Significant difference: **P < 0.01 compared to ND.

control [30,31]. Food intake in the HFD rats decreased compared to the control group. This result is in disagreement with the findings of Dìaz-Urbina and collaborators who found that HFD led to excessive food consumption and increased body weight [32]. However, in our study, the high energy content of the high fat diet could explain why MACAPOS 2-fed rats ate less but significantly gained weight and accumulated fat compared to those fed with the standard food. Because of the high energy content (fat) of the diet, animals required less of the HFD meal to be satisfied. Visceral obesity is characterized by the accumulation of abdominal fat. MACAPOS 2 increased visceral fat mass (mesenteric, peri-renal, and peri-testicular fats) in rats. This could be the consequence of the high energy value of macronutrients (palm oil and sucrose) present in the diet. Some investigations have revealed that excess energy from the diet can be accumulated over time in adipose tissue by the process of lipogenesis leading to adipocyte hypertrophy. In fact, energy is stored in cells in the form of fatty acids. Lipogenesis is the mechanism by which, cells convert carbohydrates into fatty acids which are often converted into neutral lipids for storage in the form of lipid droplets [33]. The cumulate action of palm oil and sucrose could enhance the accumulation of fat in adipose tissue [33]. Sucrose (simple carbohydrate) contained in the MACAPOS 2 diet could provide energy immediately available for the animal's needs while lipids are stored. On the other hand, complex sugars contained in maize, and cassava could be fermented and metabolized by the gut microbiome producing substrates (short-chain fatty acids such as acetate,

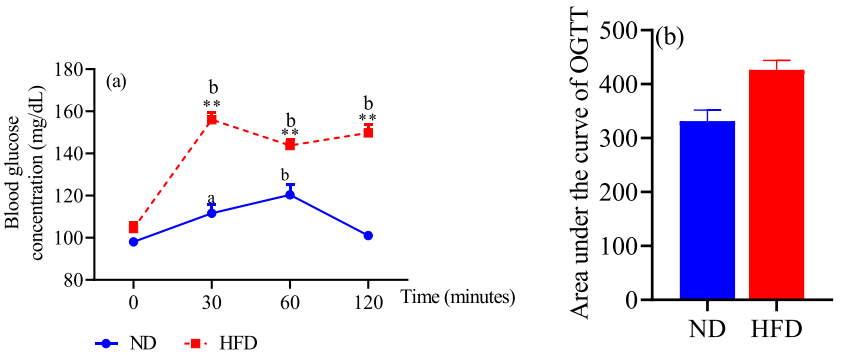

Fig. 6. Glucose level (a) and area under the curve (AUC) (b) after the OGTT in rats after four months of diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each point or column represents the Mean ± SEM. Significant difference: **P < 0.01 compared to the ND, and [a]P < 0.05, [b]P < 0.01 as compared to the initial value.

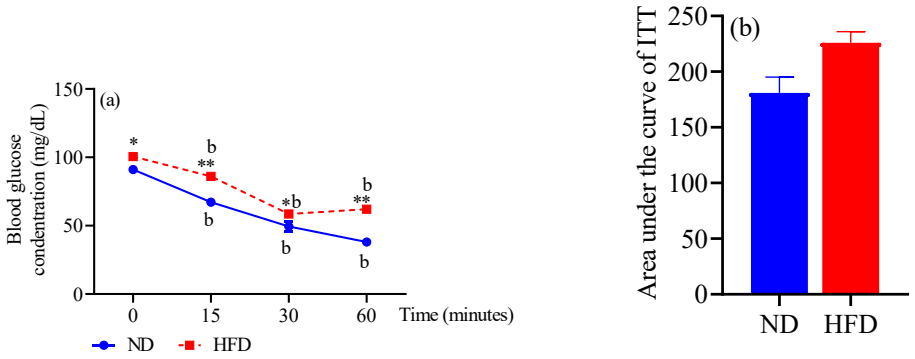

Fig. 7. Glucose level (a) and area under the curve (AUC) (b) after the ITT in rats after four months of diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each point or column represents the Mean ± SEM. Significant difference: [b]P < 0.01 compared to the initial value and *P < 0.05; **P < 0.01 compared to the ND.

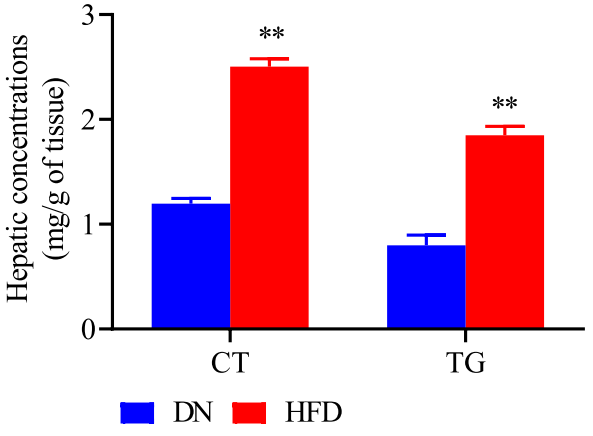

Fig. 8. Hepatic Total cholesterol (TC) and Triglycerides (TG) of rats after four months diet. ND: Normal diet, HFD: High-fat diet. n = 5. Each column represents the Mean ± SEM. Significant difference: **P < 0.01 compared to ND.

propionate and butyrate, glycerol, ATP) necessary for the biosynthesis of triglycerides and lipogenesis, promoting fat storage [34]. The main source of fat "Palm oil" in MACAPOS 2 is mostly composed of saturated fatty acids (44 % of palmitic acid) [35]. Saturated fatty acids are known to enhance the accumulation of fat in white adipose tissue and are considered more deleterious for human health than unsaturated fats [36,37].

MACAPOS 2 increased the concentration of serum insulin in rats at the end of the experiment. Hyperinsulinemia of obese rats

MACAPOS 2 could result from carbohydrates of diet and insulin resistance. HOMA-IR, a marker of insulin resistance was increased in obese rats MACAPOS 2, indicating the resistance to the action of insulin in these rats. In visceral obesity, a high blood glucose level reduces insulin sensitivity and increases insulin secretion leading to insulin resistance. Insulin resistance could be the result of excess free fatty acid from visceral fat since, the high lipolytic activity of visceral adipose tissue in obese subjects constantly releases free fatty acid leading to the ectopic accumulation of free fatty acid from pancreatic cells, inhibiting the insulin release and lead to the insulin resistance. This result confirmed those of Ngakou et al. [38] who found that obese rats MACAPOS 2 were insulin resistant after four months of diet. This can also explain the high blood glucose of MACAPOS 2 rats [39].

MACAPOS 2 increased total cholesterol, LDL-cholesterol, and triglycerides and decreased HDL-cholesterol levels, indicating dyslipidemia. This result is in agreement with the study of Kamgang and collaborators [16]. Similar results were also found in studies that demonstrated the implication of a HFD rich in saturated fat in dyslipidemia [40,41]. MACAPOS 2 saturated fat content is most likely responsible for the observed lipid rise.

Triglyceride and total cholesterol had increased in the liver of MACAPOS 2 rats. This ectopic accumulation of fat may have resulted from the increased uptake of free fatty acids from the MACAPOS 2 diet, from neoglucogenesis following insulin resistance, or from the decrease of β-oxidation of these fatty acids in the liver. These actions could explain the implication of the MACAPOS 2 diet in hepatic-induced steatosis in rats [42].

Adipose tissue secretes hormones such as leptin and adiponectin which contribute to the obesity related disorders such as cardiovascular diseases and type 2 diabetes. Plasma leptin increases proportionally to fat accumulation [39]. Leptin reduces food intake and increases energy expenditure [39]. Leptin levels rise in obesity [39]. We found in the current study an increase in blood leptin levels in the diet-induced obesity group compared to the control. This might be regarded as an attempt of the animal organism to overcome the leptin resistance. Hyperleptinemia could be the result of hyperinsulinemia and hypertriglyceridemia in MACAPOS 2 rats. Adiponectin is known to enhance insulin sensitivity, reduce inflammation, and regulate cholesterol metabolism. Adiponectin concentrations are low in obese subjects [43]. The level of adiponectin decreased in obese rats MACAPOS 2. Hypoadiponectinemia is correlated to the reduction of the stimulation of fatty acid oxidation and glucose uptake in skeletal muscles, worsening body energy homeostasis [39].

In comparison to other HFD, most of MACAPOS 2 components were purchased at their natural state in local markets, without any chemical modifications. They are made for human consumption. In the MACAPOS 2 diet, palm oil was the main source of lipids, compared to other HFD which rather use some manufactured, and therefore, expensive oils. Palm oil is the cheapest basic oil, it is entirely produced without any chemical modification. All these characteristics make the MACAPOS 2 diet less expensive, and available to researchers from developing or developed countries for obesity studies.

In the current investigation, the MACAPOS 2 diet successfully induced visceral obesity in rats after 16 weeks, which is a little too long. Our main challenge now is to reduce the induction time in future projects. Also, our results would have sounded better and more accurate if investigations had been carried out *in vitro* on isolated cultured cells.

This study showed that MACAPOS 2 could generate visceral obesity related to insulin resistance, glucose intolerance, hyperinsulinemia, hypoadiponectinemia, hepatic steatosis, and hyperleptinemia with possible leptin resistance in the *Wistar* rats. MACAPOS 2 could therefore be used as a model of diet-induced obesity in scientific studies.

4. Material and methods

4.1. Ethics statement

Animal handling and experiments were performed according to the European Union directives on the ethical evaluation of animal experiments adopted by the Cameroon Institutional National Ethic Committee of the Ministry of Scientific Research and Innovation guidelines and regulations [20] and were reviewed and approved by the University of Buea Institutional Animal Care and Use Committee (UB-IACUC), authorization number 14/2022 of June 2022.

4.2. Experimental animals

The animals used in this study were male albino *Wistar* rats (6–8 weeks), weighing 100 ± 10 g. They were purchased from the Laboratory of Human Metabolism and Non-Communicable Diseases of the Institute of Medical Research and Medicinal Plants Studies (IMPM), Yaounde, Cameroon, raised in a pathogen-free environment under normal environmental conditions in the animal house and kept in polypropylene cages with metal mesh cover, at the ambient temperature. Animal handling and experiments were performed according to the European Union directives on the ethical evaluation of animal experiments adopted by the Cameroon Institutional National Ethic Committee of the Ministry of Scientific Research and Innovation guidelines and regulations [20] and were approved by the University of Buea Institutional Animal Care and Use Committee (UB-IACUC), authorization number 14/2022.

4.3. Obesity induction

After two weeks of acclimatization period, male albino *Wistar* rats (6–8 weeks) were divided into two groups receiving food *ad libitum* for four months: the control group (ND) was fed with a normal diet (50 % carbohydrate, 20 % fat and 30 % proteins), and experimental group (HFD) fed with high-fat diet (35 % carbohydrate, 55 % fat and 10 % proteins) (Table 1) [18]. It should be noted that all the food components were obtained from local markets where they are sold for people's consumption. Except for the palm oil,

they were further ground and autoclaved for 30 min at 121 °C and kept in sterile closed containers. Foods were then prepared daily, and according to the regimen by mixing all its components with fresh palm oil. Also, diets were prepared under aseptic conditions in sterilized bowls.

The body weight was taken weekly during the four-month study period using an electronic weighing balance (Electronic compact scale SF-400 A).

The net food consumption of each group was calculated every two days as the difference between the quantity of food given and that left after two days. To determine obese rats, the Lee index (Li) was calculated using the body weight (bw) and naso-anal length (Lna) as follows:

$\mathrm{Li} = \frac{\sqrt[3]{\mathrm{bw}}}{\mathrm{Lna}}$ [21]

4.4. Serum collection and organ homogenate preparation

After four month-diet, the animals were fasted for 12 h and sacrificed. Blood was collected in dry tubes and then, centrifuged at 2500 rpm for 15 min. The serum was then collected for biochemical analysis. After dissection, adipose tissues (mesenteric, subcutaneous, renal, and testicular fats) and the liver were collected and weighed. The liver was immediately washed with an ice-cold saline solution (NaCl 0.9 %) and 400 mg were homogenized in 2 mL of Tris-HCl (0.2 M, pH 7.4) buffer solution. The homogenates were centrifuged at 2500 rpm for 25 min. The supernatants were collected and stored at −20 °C for biochemical analysis.

4.5. Oral glucose tolerance test (OGTT)

In the 15th week of experimentation, rats were subjected to OGTT. Then, after a 12-h overnight fasting period, glucose (2.5 g/kg body weight) was orally administered to each rat. Glycemia was taken at 0 h (just before the administration of glucose), 30 min, 1, or 2 h(s) using test strips (CERA-CHEK, Green Cross Medis Corp, Korea) [22].

4.6. Insulin tolerance test (ITT)

Two days after OGTT, rats were subjected to ITT. Then, after a 12-h overnight fasting period, 0.75 UI/kg of insulin (Novo Nordisk Laboratory, ACTRAPID, Human insulin, Denmark) was administered by subcutaneous injection, glycemia was evaluated just before insulin injection, then at 15, 30 and 60 min after insulin administration using test strips (CERA-CHEK) [19]. The homeostasis model assessment of insulin resistance (HOMA-IR) (a marker of IR) was calculated as follows: HOMA-IR = [Fasting blood glucose (mg/dL) × insulin (μUI/mL)/405] [23].

4.7. Lipid parameters

Total cholesterol, Triglycerides, HDL-C (High-density lipoprotein-cholesterol) and LDL-C (Low-density lipoprotein-cholesterol) were assayed in the serum and liver homogenates by enzymatic methods using kits and according to the manufacturer's instructions: CHOD– PAP method for TC (Biolabo SAS, France) [24], liquid triglycerides GPO–PAP method for TG (Biolabo SAS, France) [25] and using HDL Cholesterol Precipitant method for HDL-C (Biolabo SAS, France) [26]. LDL was calculated using the Friedwald formula as follows:

$\mathrm{LDL-C\ (mg/dL)} = \mathrm{CT} - \left(\mathrm{HDL-C} + \frac{\mathrm{TG}}{5}\right)$ [27]

The atherogenic index (AI) was calculated as follows:

$\mathrm{AI} = \frac{\mathrm{TC}}{\mathrm{HDL\text{-}C}}$ [28]

4.8. Serum adipokines: leptin and adiponectin

Serum leptin and adiponectin were assayed by enzymatic methods using ELISA-specific kits for leptin (Rat LEP ELISA Kit, Elabscience/USA Catalog N°: E-EL-R0582) and for adiponectin (Rat ADP/Acrp30 ELISA Kit, Elabscience/USA Catalog N°: E-EL-R0329) according to the manufacturer's instructions.

4.9. Serum insulin

Serum insulin was assayed by enzymatic method using a specific kit (Rat INS ELISA Kit, Elabscience/USA Catalog N°: E-EL-R3034) according to the manufacturer's instructions.

4.10. Statistical analysis

The results were expressed as Mean ± Standard Error of Means and analyzed using Graph Pad Prism 8.01. The comparison was performed by analysis of variance (ANOVA) and *t*-test. To compare body weight, food consumption, OGTT, ITT over time between the ND group and HFD group, two-way ANOVA was used followed by Bonferroni's multiple comparisons test. For non-repeated measures

Table 1
Diet compositions.

Groups	Maize	Wheat	Stepped cassava	Sucrose	Soya bean	Fish flour	Cabbage palm cake	Palm oil	Bones flour	Vitamins complex	Energy (kcal/kg)
ND	250	400	–	–	150	100	80	–	10	10	3400
HFD	80	110	220	50	280	30	–	200	20	10	4730

Components obtained from Yaoundé local market (Cameroon) expressed in g/kg of diet. ND: normal diet, HFD: hypercaloric fat diet. Each value represents the Mean ± SEM.

(fat mass, lipid profile, atherogenic index, blood glucose concentration, Lee index, HOMA-IR, serum insulin, leptin, and adiponectin concentrations, hepatic CT and TG) as well as AUC of OGTT and ITT, *t*-test was used. $P < 0.05$ was considered statistically significant.

CRediT authorship contribution statement

Sandrine Nkoubat Tchoundjwen: Writing – original draft, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Armel Georges Kamgang Tchawou:** Methodology, Investigation, Formal analysis. **Clémence Mvongo:** Methodology, Investigation, Formal analysis, Conceptualization. **Adamou Mfopa:** Writing – original draft, Software, Investigation. **Joseph Ngakou Mukam:** Writing – original draft, Software, Methodology, Investigation, Conceptualization. **Paul Aimé Noubissi:** Writing – review & editing, Validation, Software, Project administration, Investigation, Formal analysis, Conceptualization. **Gaetan Olivier Fankem:** Methodology, Investigation, Formal analysis, Data curation. **René Kamgang:** Writing – review & editing, Supervision, Project administration. **Jean Louis Essame Oyono:** Writing – review & editing, Validation, Supervision, Project administration.

Ethics approval

This study was reviewed and approved by the University of Buea Institutional Animal Care and Use Committee (UB-IACUC), authorization number 14/2022 of June 2022.

Data availability

The data supporting the findings of this study are available within the article. Any additional data or materials can be requested from the corresponding author.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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