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Aqueous extract of *Cola anomala* nuts protects against metabolic disorders in albino *Wistar* rats

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Abstract

The aim of this study was to evaluate the protective effect of *Cola anomala* nuts against metabolic disorders induced by a hypercaloric diet. For this purpose, the aqueous extract of *Cola anomala* nuts was prepared for a 28-day animal experimentation. Twenty male *wistar* rats were randomized according to body weight into four groups of five rats each and treated as follows : a negative control group fed with a normal calorie diet, a positive control group fed with a high-fat high-fructose diet (HFHFD) and two test groups fed with a HFHFD and also receiving either 200 or 400mg/kg body weight of the aqueous extract of *Cola anomala* nuts. At the end of the experiment, blood and organs were collected for the preparation of plasma, hemolysate and homogenates then used for the evaluation of lipid profile (total cholesterol, triglycerides), oxidative status (catalase, Malondialdehyde (MDA), Superoxide dismutase (SOD), Ferric reducing antyoxidant Power (FRAP)), hepatotoxicity (Alanine aminotransferase (ALT)) and nephrotoxicity (Creatinine). The extract at both doses significantly limited weight gain. Plasmatic triglyceride and cholesterol levels were lower in *Cola anomala*-treated groups when compared to the positive control. As for the oxidative stress status, plasmatic MDA levels, FRAP and catalase activities in liver and heart were lower in *Cola anomala*-treated groups when compared to the positive control group. Finally, the extract at both doses had a protective effect on renal and hepatic functions. The results obtained suggest that the aqueous extract of *Cola anomala* nuts has a protective effect against metabolic disorders induced by a hypercaloric diet.

Key words: *Cola anomala*, extract, metabolic disorders, hypercaloric diet, weight gain, glycemia

1. Introduction

Current dietary habits increase the risk of developing metabolic diseases, which may not only decrease life expectancy but also promotes the emergence of new pathologies **(Baraka, 2014).** Data from large cohort studies such as those conducted by **Benini *et al* (2017)** and **Schlienger (2019)** show an association between carbohydrate consumption and the risk of developing obesity, diabetes, or cardiovascular diseases **(Tappy 2020).** Indeed, a hypercaloric diet will generate hypertrophy of adipose tissue, followed by strong lipolysis, activation of macrophages, and low production of adiponectin, thus causing metabolic conditions like decreased glucose uptake, apoptosis of β-cells, overproduction of glucose which may result in hyperglycemia, hyperlipidemia and oxidative stress **(Chavanelle, 2018)**.

The management of these metabolic disorders is firstly based on dietetic measures, and the use of anti-hyperlipidemic and anti-hyperglycemic drugs **(Halimi 2020);** however, most of these drugs in addition to being expensive, present more or less complex side effects **(Guenzet 2019),** hence the need for an alternative approach that is less expensive and healthier, namely phytotherapy. Prior to searching for alternate less toxic treatments is the search for foods that could help prevent the development of metabolic disorders. Numerous studies have proven the efficacy of metabolites such as alkaloids, phenolic compounds, and terpenoids in the management of metabolic diseases (**Eddouks *et al.* 2019, Kassi *et al.* 2020).** Therefore, *Cola anomala*, whose content in these bioactive compounds has been demonstrated **(Mbong *et al*. 2020),** is our plant of interest. Indeed, *Cola anomala*, is a plant of the *Sterculiaceae* family and nuts of

plants of the *Cola* genus, are commonly consumed by most West and Central African populations by chewing to fight physical and intellectual fatigue as well as to improve cognitive performance **(Bureau 2013).** *Cola anomala* is abundant in Cameroon, and its nuts are more consumed by the patriarchs and most often during traditional ceremonies. These nuts have virtues that have been explored in some studies, such as their antidiabetic properties and their ability to protect against against toxicity induced by methotrexate **(Mbong *et al.* 2020, Mbong *et al.* 2022).** Based on this preexisting knowledge of its bioactive content and already proven biological activities, we found it judicious to evaluate its protective effect against metabolic disorders induced by a hypercaloric diet in rats.

2. Materials and methods

2.1. Plant material

Cola anomala nuts were obtained from a wholesale farmer Bandjoun (Western Region, Cameroon) in October 2019 in their mature state. Subsequently, these nuts were identified at the National Herbarium of Cameroon in comparison with *Cola anomala* K. Schum sample from collector B.A. Nkongmeneck No. 113 of Herbarium Collection specimen No. 48706/HNC (YA). After identification, the nuts were brought back to the Laboratory of Nutrition and Nutritional Biochemistry (LNNB) of the Department of Biochemistry, University of Yaounde I, Cameroon

2.2. Preparation of the aqueous extract of *Cola anomala* nuts

Once at the laboratoory, the *Cola anomala* nuts were washed and dried in the shade at room temperature until a constant weight was obtained. They were powdered with a Moulinex grinder and the powder obtained was macerated in distilled water at a ratio of 1:6 (w/v). The maceration lasted 24 hours, at the end of which the supernatant was collected and filtered using Nº3 Wattman paper. The filtrate obtained was dried at 40°C in a Binder oven for 72 hours to evaporate the solvent.

2.3 Animal experimentation

Twenty male *Wistar* rats weighing 183±10g were provided by the animal house of LNNB. They were placed in plastic cages containing wood pellets and maintained in a controlled environment under a 12-hour light/12-hour dark cycle with free access to water and food. The experimental protocol and the maintenance of the laboratory animals were carried out in accordance with the standard ethical guidelines for the use and care of laboratory animals as described by Joint Institutional Review Board for Animal and Human Bioethics, CRFD-SVSE of the University of Yaounde I.

The rats were divided into 4 groups of 5 each and treated for a period of 28 days as follows:

A Negative control (NC) group receiving a normal diet (ND) and distilled water

A Positive control (PC) group receiving a high-fat high-fructose diet (HFHFD) + distilled water

A Test 1 (AECa 200) group receiving a HFHFD + 200mg/kg bw of AECa

A Test 2 (AECa 400) receiving a HFHFD + 400mg/kg bw of AECa

The extract at corresponding concentration for the test group or distilled water for the control groups was administrated to rats through gastroesophageal intubation daily. Weight and fasting blood glucose levels were measured at the beginning, the middle, and the end of the experiment using an *electronic* scale and a glucometer, respectively. After 28 days of experimentation, the rats fasted for 12 hours and after anesthesia with ketamine were sacrificed by cervical dislocation. Blood was collected for the preparation of plasma and hemolysate, and organs (liver and kidneys) were collected for the preparation of a 10% (w/v) homogenates using NaCl (0.9%) solution. All biological preparations were stored at -20°C.

2.4. Biochemical analysis

Effect of experimentation on lipid markers

Total cholesterol and triglyceride levels were assessed using ChronoLab kits according to the protocols described by the manufacturer.

Effect of the extract on markers of oxidative stress

- **Catalase**

Catalase activity was assessed by the method described by **Sinha (1972).** The Lowry method was used to estimate the amount of plasmatic protein (**Lowry et *al.*, 1951)** and expressed in g/l. Catalase activity was expressed in mM hydrogen peroxide consumed per minute per mg of protein.

- **SOD**

The method described by **Misra and Fridovish (1972)** was used to estimate SOD activity expressed in units/mg of protein.

- **FRAP**

The reducing power of iron was evaluated as described by **Jayaprakash et *al.* (2001)** and expressed as a percentage of reduction.

- **MDA**

The MDA level was assessed using the protocol described by **Yagi (1976).** The intensity of the pink complex formed was proportional to the MDA level. Results were expressed in µmol/l.

- **Effect of the extract on a marker of hepatotoxicity (ALT)**

The method described by **Reitman and Frankel (1957)** was used to determine ALT activity expressed in IU/ml.

- **Effect of the extract on a marker of nephrotoxicity (Creatinine)**

Plasma creatinine was estimated using a Chronolab kit and the protocol was realized according to the manufacturers instructions; results were expressed in mg/ml.

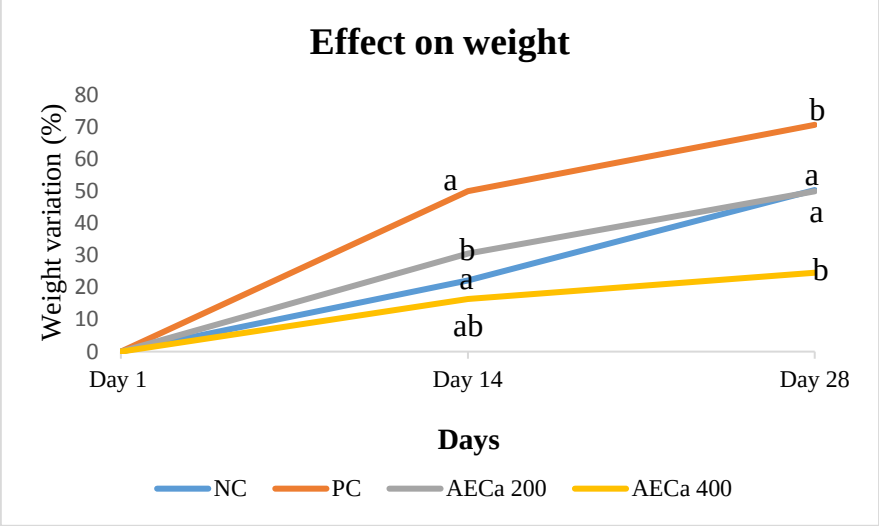
2.5. Data processing and statistical analysis

Excel spreadsheet was used for data processing. *Statistical Package for Social Science (SPSS)* version 25.0 for Windows was used for statistical analysis of the results. The one-way *Analysis Of Variance* (ANOVA) test followed by a LSD *post-hoc* test was used to compare the means between groups. Results were expressed as mean ± standard deviation and were considered significant at $p < 0.05$.

3. Results

3.1 Effect of the extract on the variation of the body weight

Figure 1 shows that the HFHFD induced a significant weight gain among PC group as compared to rats fed with normal diet (NC group). The administration of the extract at both doses considerably limited this weight gain throughout the experimentation to -56.73 % for 200 mg/kg.bw (P= 0.001) and -66 for 400mg/kg bw (p= 0.009) respectively compared to the PC group.



Values are expressed as mean ± standard deviation. NC: Negative control; PC: Positive control; AECa 200: Aqueous extract of Cola nuts anomala at the dose of 200mg/kg CP; AECa 400: Aqueous extract of Cola nuts anomala at the dose of 400mg/kg CP. Values with different letters are significantly different ($p < 0.05$).

Figure 1: Effect of aqueous extract of *Cola anomala* nut on weight change

3.3. Effect of the extract on blood lipid markers

The HFHFD caused a significant increase in triglycerides and total cholesterol levels. The administration of the extract at both doses significantly limited the increase in triglyceride levels by up to -62.11% and -38.59% for the 200 and 400mg/kg bw respectively compared to the PC group. For total cholesterol, the 200mg/kg bw rats were found to have an average level that was 20% lesser than that of the PC group (Table 1).

Table 1 : Effect of aqueous extract of Cola anomala nut on plasma cholesterol and triglyceride levels

Groups	Triglycerides in mg/ml (% of reduction as compared to PC group)	Total cholesterol in mg/ml (% of reduction as compared to PC group)
NC	59.15 ± 2.57[a]	48.02 ± 6.23[a]

PC	163.73 ± 0.52[b]	85.48 ± 11.12[b]
AECa 200	61.60 ± 2.61[a]	68.73 ± 9.55[a]
	-62.11%	-19.59%
AECa 400	100.54 ± 10.07	87.81 ±5.18 [b]
	-38.59%	2.72%

Values are expressed as mean ± standard deviation. NC: Negative control; PC: Positive control; AECa 200: Aqueous extract of Cola nuts anomala at the dose of 200mg/kg CP; AECa 400: Aqueous extract of Cola nuts anomala at the dose of 400mg/kg CP. Values with different letters are significantly different (p < 0.05).

3.4. Effect of the extract on markers of oxidative stress status

As reported in Table 2, the catalase activity was significantly increased in erythrocytes, kidney, liver, and heart in the PC group as a consequence of the HFHFD. However, the extract prevented the installation of oxidation stress marked by a significant decrease in the activity of catalase in almost all explored compartments. The result was more significant with the AECa 400 group.

The HFHFD also induced a depletion of FRAP in most organs of PC group as compared to normal diet fed rats. Administration of the extract prevented the exhaustion of antioxidant potential by the hypercaloric diet. In the heart and erythrocytes, the administration of the extract ameliorated MDA levels that were found to be raised by the high calorie diet in the PC group.

Table 2: Effects of the extract on markers of oxidative stress status

Catalase activity (mmol/min/g protein)					
Groups	**Erythrocytes**	**Kidney**	**Liver**	**Pancreas**	**Heart**
NC	284.73 ± 18.44[a]	9.00 ± 0.19[a]	8.47 ± 0.44 [a]	2.93 ± 0.39[ab]	14.96 ± 1.31 [a]
PC	369.08 ± 0.00[b]	14.07 ± 1.02 [b]	15.73 ± 1.20 [b]	2.23 ± 0.24 [a]	155.48 ± 14.97 [b]
AECa 200	291.68 ± 20.05[c]	10.22 ± 0.66 [a]	12.36 ± 0.69 [c]	3.77 ± 0.21 [ab]	44.99 ± 0.80 [c]
AECa 400	200.38 ±22.49[d]	15.84 ± 1.55[bc]	8.95 ± 0.56 [a]	2.34 ± 0.36 [a]	17.24 ± 1.44 [a]
FRAP (%)					
NC	350.67 ± 8.17[abc]	168.34 ± 1.13 [a]	135.77 ± 4.61 [a]	122.62 ± 2.49 [a]	390.2± 9.65 [a]
PC	314.58 ± 11.26[a]	196.10 ± 22.37[ab]	101.21± 10.21[b]	86.90 ± 5.29 [b]	366.93 ± 1.65 [a]
AECa 200	361.51 ± 10.90[b]	209.39 ± 5.73 [b]	77.17 ± 4.55[cd]	80.81± 0.90[bc]	395.88 ± 12.80 [a]
AECa 400	374.46 ± 23.13[bc]	166.00 ± 12.08 [a]	92.32 ± 3.72[bd]	67.77 ± 0.83[d]	372.68 ± 13.14 [a]
MDA (µmol/l)					
NC	28.47 ± 11.30[a]	4.62 ±0.90 [a]	8.44 ± 0.33 [a]	5.44 ± 4.39 [a]	26.40 ± 1.01[ac]
PC	38.63 ± 3.09 [b]	2.92 ± 0.34 [b]	6.3 ± 0.32[b]	2.10 ± 1.42 [ab]	42.15 ± 0.34 [b]
AECa 200	31.44 ± 3.10 [b]	4.03 ± 0.60 [a]	7.51 ± 0.47[ab]	3.87 ± 11.96 [b]	51.71 ± 0.48 [ab]
AECa 400	22.07 ± 2.75 [a]	4.71 ± 0.60 [a]	15.58 ± 0.86[c]	6.21 ± 0.13 [ab]	35.1 ± 0.52 [c]

Values are expressed as mean ± standard deviation. NC: Negative control; PC: Positive control; AECa 200: Aqueous extract of Cola nuts anomala at the dose of 200mg/kg CP; AECa 400: Aqueous extract of Cola nuts anomala at the dose of 400mg/kg CP. Values with different letters are significantly different (p < 0.05).

3.6. Effect of the extract on ALT activity

The effect of the extract on ALT activity presented in Table 3 revealed that ALT activity was significantly elevated (41.6%) in the PC group compared to the NC group. On the other hand, the administration of the extract signficantly reduced it by -37.4% and -18.17% at the 200 and 400 mg/kg bw doses respectively compared to the PC group; the dose of 200mg/kg bw being the more effective.

Table 3 : Influence of AECa on hepatic and renal toxicity marker

Groups	ALT (IU/ml)	Creatinine (mg/ml)
NC	25.18 ± 1.81[a]	2.5± 0.02 [a]
PC	43.15 ± 0.57[b]	4.44± 0.03[b]
AECa 200	27.04 ±0.89[a]	3.15± 0.04 [c]
AECa 400	35.31 ±1.43[c]	3.34±0.03[b]

Values are expressed as mean ± standard deviation. NC: Negative control; PC: Positive control; AECa 200: Aqueous extract of Cola nuts anomala at the dose of 200mg/kg CP; AECa 400: Aqueous extract of Cola nuts anomala at the dose of 400mg/kg CP. Values with different letters are significantly different (p < 0.05).

3.7. Effect of extract on creatinine levels

Creatinine which is a marker of kidney injuries was assessed and the results in Table 4 show that the HFHFD also increased the level of creatinine in the PC group (43.69%) compared to the NC group. While the extracts significantly decreased these levels as compared to PC (- 29.05% for AECa 200 and – 24.77% for AECa 400).

4. Discussion

An imbalance in energy consumption in favor of intake leads to an excess of energy that is stored as fat, thus favoring the occurrence of many metabolic disorders like hyperglycemia and dyslipidemia (**Chavanelle 2018).** In this study, the high fat, high carbohydrate and thus high energy content of the diet favored the accumulation of fat and the increase in weight of rats of the PC group. Moreover, a high energy density diet induces hyperphagia explained by an increase in palatability. Such a diet also induces a decrease in motor activity which decreases energy expenditure (**Rahoui Walid, 2018).** However, AECa at both doses prevented a significant increase in the weights of rats in the treated groups. This could be explained by its polyphenol content that restricted the accumulation of fat in adipose tissue and TG in the liver (**Bratoeva et *al.*, 2020).**

Significantly elevated levels of total cholesterol and triglycerides were observed in the group of rats receiving only the HFHFD; Indeed, a hypercaloric diet induces processes of adipose tissue hypertrophy and hyperplasia associated with intracellular abnormalities of adipocyte function that leads to increased plasma levels of total cholesterol and triglycerides associated with alterations in lipoprotein fractions, leading to elevated cholesterol and triglyceride content in the VLDL and LDL **(Rahoui Walid, 2018).** However, the groups of rats receiving AECa showed significantly low plasmatic triglyceride and total cholesterol levels which is in agreement with previous studies that demonstrated the ability of *Cola anomala* to prevent hyperlipidemia in rats fed with a normal diet (**Mbong et *al.* 2020)**. These hypotriglyceridemic and hypocholesterolemic abilities of extracts of *C. anomala* could be attributed to their polyphenols. They are able to stimulate lipoprotein lipase and inhibit HMG-CoA reductase both key enzymes involved in triglyceride metabolism and cholesterol synthesis respectively **(Abdulazeez 2011).**

The close relationship between metabolic disorders and oxidative stress led us to evaluate the markers of oxidative stress during this study. The main consequence of oxidative stress at the cellular level is lipoperoxidation characterized by the formation of primary markers such as hydroperoxides and secondary markers including MDA. The enzymes catalase serve as the first line of defense against pro-oxidant-induced cellular damage **(Bahadoran *et al.*, 2013, Ighodaro & Akinloye 2017)**. The assay of catalase activity revealed significantly elevated activity in the heart and kidney among PC group rats. Dietary excess of fat and carbohydrates leads to mitochondrial overproduction of ROS **(Matsuda & Shimomura, 2013)** leading to high catalase activity as a consequence. However, in the extract-treated groups the significant reduction of catalase activity noted could be explained by the presence of polyphenols, flavonoids and alkaloids that can readily donate hydrogen and consequently electrons to stop the

production of free radicals responsible for oxidative stress**.** The plasma MDA level of the PC group was significantly higher than that of the negative control group, which is consistent with the works of **Murase et *al*. (2002); Alberti et *al.* (2005); Nguele et *al*. (2012)** who stated that elevated plasma MDA level is the result of lipid peroxidation and increased protein oxidation at the tissue level. Administration of *C. anomala* at doses of 200 and 400 mg/kg bw to rats resulted in a significant decrease in MDA concentration in all treated groups in some compartements. The results of **Xydakis et *al*. (2004)** and **Raneva (2005)** showed that flavonoids could reduce the concentration of metabolites involved in lipid peroxidation, DNA and protein damage, thus reducing the risk of CVD. The reducing power of Iron in the plasma, liver and pancreas was significantly lower in the group receiving only HFHFD compared to the negative control group. This could be explained by an oxidation of compounds responsible for free radical scavenging by the HFHFD that would induce a state of oxidative stress **(Nguele et *al.* 2012)**. Nevertheless, the administration of *C. anomala* extract at the dose of 200mg/kg bw resulted in a significant increase in iron reducing power that could be attributed to flavoids present in the extract which reduce Iron an prevents its pro-oxidant effect in the body **(Pandey et *al.* 2012).**

ALAT is an indicator of the structural integrity of the liver **(Pradeep 2010)**, The results obtained reveal a significant increase in ALAT activity in the plasma of rats in the PC group compared to the NC group confirming liver injury caused by the HFHFD. Similar results were obtained by **Rahoui (2018).** *Cola anomala* aqueous extract, however, significantly reduced plasma ALAT activity in the 200mg/kg bw dose treated group compared to the PC group. This hepato-protective property could be due to the decrease of hepatic oxidative stress and hepatic steatosis by suppression of the expression of lipogenesis genes, with a reduction of the process of gluconeogenesis which requires the intervention of transaminases **(Chandan et *al*., 2007; Saito et *al*., 2012)**.

The determination of plasma creatinine levels revealed a significantly higher level of creatinine in the PC group compared in the NC group, which could be explained by the fact that oxidative stress caused by HFHFD leads to renal damage charecterized by a high expression of the caspase-3 marker, which triggers apoptosis in the kidney cells **(Gehad et *al.* 2021).** However, the extract led to a significant decrease in creatinine levels; in fact, this could be due to the presence of phenolic compounds which, through their role as antioxidants, limit the effects of pro-oxidants at the renal level, maintaining thus an adequate glomerular filtration in the extract treated groups (**Li *et al.* 2014).**

5. Conclusion

Based on the reduction in body weight, lowering of triglycerides and total cholesterol levels in treated groups, it could be suggested that the aqueous extract of *Cola. anomala* nuts presented a protective effect against some metabolic disorders. *Cola anomala* due to its numerous bioactive compounds also contributed in the improvement of antioxidant status; thus preventing the occurrence of liver and kidney damage in an obesogenic environment.

Authors Contributions: MAM-A and OJE designed the study, NFR analysed the data, NFR, ABK, MAM-A and YJA wrote the manuscript, DPG, DHT, MI and FTH carried-out the experimentations and biochemical analyses.

Competing Interest: The authors in this paper have no conflict of interest

References

Abdulazeez, M. (2011). Effect of Peristrophe bicalyculata on lipid profile of P-407-induced hyperlipidemic Wistar rats. *Journal of Medicinal Plants Research*. 5: 490-494.

Alberti KG, Zimmet P, Shaw J. (2005). The metabolic syndrome-a new Worldwide definition. *Lancet.* 366: 1059-62.

Bahadoran Z, Mirmiran P, Azizi. **(2013)**. Dietary polyphenols as potential nutraceuticals in management of diabetes: a review. *Journal of Diabetes & Metabolic Disorders*. 12: 1-9.

Baraka-Vidot J. **(2014).** Oxidative stress and diabetic pathology in Reunion Island-Identification and characterization of structural and functional properties of glycated albumin. Human medicine and pathology. University of Reunion, French. NNT: 2014LARE0013. tel-01155664.

Bratoeva K., Bekyarova, G., & Kiselova, Y. (2010). Effect of bulgarian herb extracts of polyphenols on metabolic disorders - induced by high-fructose diet. *Trakia Journal of Sciences*. 8 :56-60.

Benyaich, A. (2017). Effects of the Mediterranean diet on chronic diseases: Cardiovascular disease, oxidative stress, dyslipidemia, diabetes mellitus, blood pressure, cancer, neurodegenerative diseases and obesity. *Nutrition Research Reviews*. 1-37.

Benini A., Nezzal L., Mekhancha D-E., Dahel-MekanchaC-C. (2017). Cohort study of diabetic shift workers in an Algerian company (1995-2014). *Medicine of metabolic diseases*. 11: 300-306.

Bureau, L. (2013). Kola. *Phytotherapy*. 11: 126-129.

Chandan BK, Saxena AK,Shukla S, Sharma N,Gupta DK, Suri KA, Suri J, Bhadauria M, Singh B. **(2007).** Hepatoprotective potential of Aloe barbadensis Mill. Against carbon tetrachloride induced hepatotoxicity. *Journal of Ethnopharmacology*. 111: 560-566.

Chavanelle V. (2018). Effects of two training modalities and plant extract supplementation on the development of type 2 diabetes: Physiology. Education. University of Clermont Auvergne. French. NNT: 2018CLFAS021. tel-02885485.

Eddouks M., Amssayef A., Ajebli M., Hebi M. (2019). Ethnopharmacological study on the use of medicinal plants in the treatment of tuberculosis in southeast Morocco. *Lavoisier SAS* 18:340-348.

Gornall A, Bardawill C, David M. (1949). Determination of serum proteins by means of the biuret reaction. J. Biol. Chem. 1949; 177: 751-767.

Gehad E. E. and Sara T. E. (2021). Cinnamon aqueous extract attenuates diclofenac sodium and oxytetracycline mediated hepato-renal toxicity and modulates oxidative stress, cell apoptosis, and inflammation in male albino rats. *Veterinary sciences*.8,9.

Guenzet A. (2019). Effect of lyophilized extract of clove (Sywygium aramaticum) on lipid profile, redox and inflammatory status, in rats with diabetes or hypetcholesterolemia. Faculty of Natural and Life Sciences. University of Oran 1.

Halimi S. (2020). Blood glucose-lowering sulfonamides still have a place in the treatment of type 2 diabetes in 2021. Highlights of the case for hypoglycemic sulfonamides. *Metabolic Disease Medicine* 15: 53-61.

Ighodaro OM, Akinloye OA. **(2017).** First line defense antioxidants-superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX): Their fundamental role in the entire antioxidant defense grid. *Alexandria Journal of Medicine*. 54: 287-293.

Jayaprakash G, Singh P and Sakariah K. (2001). Antioxidant activity of grape seed extracts on peroxidation models in-vitro. *Journal of Agricultural Food Chemistry*.55. 1018-1022.

Kassi A. B. B., Ballo D., Kabran A. F., Sissouma D. and Adjou A. (2020). Evaluation of the antioxidant power and total polyphenol content of six medicinal plants used in the treatment of cardiovascular diseases. *Journal of Applied Biosciences* 153: 15788 -15797.

Lavau M., Gradnauer-Griglio S., & Lowry R. (2017). In-vitro metabolism of epididymal adipose tissue in the wistar rat (CF) subjected to a hyperlipidic diet. *Enzymologia Biologica et Clinica*. 8:298-310.

Li Y, Zhang Y, Zhang L, Li X, Yu J, Zhang H, Tan B, Jiang L, Wang Y, Liang Y. **(2014).** Protective effect of tea polyphenols on renal ischemia/reperfusion injury via suppressing the activation of TLR4/NF-kappaB p65 signal pathway. *Gene*. 542: 46-51.

Lowry O, Rosebrough N, Farr A and Randall R. (1951). Protein measurement with the Folin phenol reagent. *Journal of Biological Chemistry*. 193: 265-275.

Matsuda M, Shimomura I (2013). Increased oxidative stress in obesity: Implications for metabolic syndrome, diabetes, hypertension, dyslipidemia, atherosclerosis, and cancer. *Obesity Research & Clinical Practice*. 7 :330-341.

Mbong, M.-A. A., Manga Ngandi, L. C., Ebouel Edoun F. L., Fotso Youvop J. A., Orang Orang R., Fotso Tienoue H. M., Ngalla Nwang F., Ngondi J. L., & Oben J. (2020). Comparative Study of the Protective Effect of Cola anomala and Coffea arabica Against Induced Toxicity in Rats. *Journal of Food Research*. 9: 1927-0887.

Mbong Angie Mary-Ann, Ntentie Françoise Raïssa, Bakam Viviane Merveille' Oben Julius Enyong Effects of Aqueous Extract of Cola anomala Nuts on Hyperglycemia and Associated Complications. Am. J. Pharm Health Res 2022;10(02) 2321–3647

Misra HP, Fridowich I. (1972). The generation of superoxide radical during the autoxidation of hemoglobin. *Journal of Biological Chemistry*. 247: 6960-6962.

Murase T, Nagasawa A, Suzuki J, Hase T, Tokimitsu I. 2002. Beneficial effect of tea catechins on diet induced obesity: stimulation of lipid catabolism in the liver. *International Journal of. Obesity* 26: 1459-1464.

Nguele L. R., Fokunang C. N. , Etoundie C. , Chakokam R. M. , Ngondi J. L. , Tembe E. A. , Kechia F. , Ngameni B. , Gatsing D. & Oben J. (2016). Use of Aframomum species (*Aframomum aulacocorpus, A. citratum, A. daniellii*) for weight control, lipid profile and antioxidant status in *Wistar* rats fed an atherogenic diet. *International Journal of Biological and Chemical Sciences*. 10 : 2575-2586.

ORS. (2012). Diabetes in Reunion Island. In: Tableau de bord de l'ORS de la Réunion.

Pradeep K. (2010). Renal function in diabetic nephropathy. *World Journal of Diabetes*. 1: 48-56.

Pandey A, Mishra K & Mishra A. (2012). Antifungal and antioxidative potential of oil and extracts derived from leaves of Indian spice plant cinnamomum tamala. *Cellular and Molecular Biology*.58 :142-147.

Rahoui Walid (2018). Benficial effects of Aloe vera gel on lipid profile, lipase activities and oxidant/antioxidant status in obese rats. *Journal of Functional Foods*. 48 :525-532.

Raneva VG, Shimasaki H. (2005). Green tea catechins decrease lipid peroxidation in plasma and organs of C57BL/6J mice fed atherogenic diet. *Journal of Oleo Science*. 54: 641-648.

Reitman S. and Frankel S. (1957). Serum transaminase assay. *American Journal of Clinical Pathology*. 28: 56.

Saito M, Tanaka M, Misawa E, Yamada M, Yamauchi K, Iwatsuki K (2012). Aloe vera gel extract attenuates ethanol-induced hepatic lipid accumulation by suppressing the expression of lipogenic genes in mice. *Bioscience Biotechnology and Biochemistry*. 76: 2049-2054.

Schlienger J. L. (2019). From sugar delight to sugar crime. About a public health controversy . *Medicine of metabolic diseases* 13: 156-163.

Sinha, K. (1972). Colorimetric Assay of Catalase. *Analytical Biochemistry*. 47 :389-394.

Tappy L. (2020). Fructose, sugars and metabolic diseases. *Cahier de nutrition et de diététique*. 55:233-239

Xydakis AM, Case CC, Jones PH Adams UP, Brian CA. (2004). Adiponectin, inflammation, and the expression of the metabolic syndrome in obese individuals: the impact of rapid weight loss through calorie restriction. *Journal of Clinical Endocrinology & Metabolism*. 89: 2697-2703.

Yagi, K. (1976). Simple Fluorometric Assay for lipoperoxide in blood plasma. *Biochemistry Medicine*. 15: 212-216

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