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Ameliorative Effects of an Aqueous Extract of *Garcinia kola* Heckel (Guittiferae) on Hyperlipidemia and Postprandial Hypertriglyceridemia Induced by High Fat and Sucrose Diet in the Wistar Rat

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ABSTRACT
Dyslipidemia is a major risk factor for cardiovascular diseases (CVD). In Ivorian traditional medicine, several preparations derived from natural product including *Garcinia kola* are used against metabolic disorders such as dyslipidemia. Therefore, aim of present study was to evaluate the ameliorative effect of an aqueous extract of *Garcinia kola* nuts (AE*Gk*) on dyslipidemia and postprandial hypertriglyceridemia induced high fat and sucrose diet (HFSD) in the rats. In the first step of experiments, to study antidyslipidemic effects of AE*Gk* twenty - five (25) Wistar rats randomly assigned five groups: normal control , group fed high-fat and sucrose diet untreated (HFSD), HFSD treated with Statin at 10 mg/kg (STAT) and HFSD those receiving EA*Gk* at 600 or 1000 mg/kg (EA*Gk* 600 or 1000). Normal control group rats were fed normal chow diet and the others groups were fed high-fat and sucrose diet. Next in order to evaluate the effect of AE*Gk* on postprandial hypertriglyceridemia thirty (30) rats including dyslipidemics and normal rats are selected, they received *per os* 5 mL/kg of olive oil. Then blood samples are taken every two hours for six hours to assess postprandial hypertriglyceridemia. For biochemical analysis, blood sample was taken from retro-orbital sinus, every 7 days during 28 days and each two hours during 6 hours focus, respectively in antihyperlipidemic group and postprandial hypertriglyceridemia tests. After 28 days, AE*Gk* treatments (600 or 1000 mg/kg) ameliorate dyslipidemia serum markers on HFSD rats. The effect of AE*Gk* at 1000 mg/kg seems to be strong versus the dose at 600 mg/kg, and also compare to STAT (10 mg/kg). For instance, at 28 days, AE*Gk* (1000 mg/kg), induce a significant reduction of hypertriglyceridemia (0.95 ± 0.04 versus 0.73 ± 0.02 g/L), hypercholesterolemia (1.01 ± 0.05 versus 0.66 ± 0.02 g/L) and LDL-C (0.58 ± 0.04 versus 0.18 ± 0.02 g/L), compared to untreated HFSD rats. Furthermore, AE*Gk* at 1000 mg/kg also increased significantly serum level of HDL-C (0.26 ± 0.008 versus 0.33 ± 0.02 g/L) and reduce significantly AUC on postprandial hypertriglyceridemia on six hours ($p < 0.01$). AE*Gk* induces an antihyperlipidemic and reduces postprandial effect in HFSD rats, which could partially justify its use in traditional medicine for treatment of metabolic disorder such as dyslipidemia. These ameliorative effects seems related to the presence of many microconstituents such as saponins and polyphenols in the *Garcinia kola* extract.

INTRODUCTION

Dietary habits of sub-Saharan Africa populations are undergoing considerable changes due to socio-economic development and especially to the strong industrialization of the food industries. Ivorian people are facing the nutritional transition and the consequence is the pathologies linked in part to nutrition including obesity, diabetes and cardiovascular dysfunction [29, 16]. Previews studies have shown, it is the leading cause of death for chronic diseases and dyslipidemia is a major risk factor [9, 16, 34, 18] . Synthetic lipid-lowering drugs, in particular statins, fibrates or ezitimibes, are in the first line of defense. Also, several preparations of herbal medicine are used for the medical care and/or diet and lifestyle in the management of this pathology, including nuts of *Garcinia kola* Heckel (Guttiferae). *Garcinia kola* is a medium sized evergreen tree, about 15-17m tall and 35m diameters [12, 14, 24]. It nuts are bitter, astringent and are consumed for these medicinals and nutritional properties. Commonly called « petit cola » in Côte d'Ivoire, they are also eaten as an aphrodisiac. They are usually used by west and central African people in folkloric medicine to treat many diseases such as cough, diabetes, high blood pressure and liver diseases [2 ; 24 ;]. Previews studies have shown that it contain many phenolic compounds such as biflavanones, GBl, GB2, GB1a, kolaflavanone and vitamin C. It possesses an important antioxidant property and can dose-dependently decrease lipid marker dysfunction in Wistar rats[7, 26, 36, 1]. Moreover, few work-related *Garcinia kola* nuts effects on nutritional dyslipidemia and postprandial hyperlipemia are developed. Even though previous investigations have also reported, postprandial hyperlipidemia is considered to be a substantial risk factor for atherosclerosis [3]. Accordingly, it is important to assess it in pharmacology and nutritionnal studies.

We hypothesized, *Garcinia kola* extracts decreasing lipid marker observed by Adejor et *al.* [1] on P407 induce dyslipidemia on Wistar rats might attributable yet to its possible effect on postprandial hyperlipemia. Furthermore, aim of this study is to assess *Garcinia kola* nuts aqueous extract effect on dyslipidemia and postprandial hypertriglyceridemia on Wistar rat.

MATERIAL AND METHODS

Plant material and extraction method

The nuts of *Garcinia kola* (Guittiferae) was collected from Elibou (Sikensi, Côted'Ivoire). This plant has been authenticated in July 10 1980 to south of Côte d'Ivoire, by an expert in Botany (Professor Ake-Assi Laurent). Voucher specimen was recorded under No. 10857 and

15189 in the *Centre National de Floristique* (UFR-Biosciences, Félix Houphouët-Boigny University, Abidjan, Côte d'Ivoire).

The fresh nuts of *Garcinia kola* Heckel (Guttiferae) are cut into small pieces, dried in ambient air, away from the sun. They are then milled in a micro mill (IKA LABORTECHNIK TYPE A 10). One hundred grams (100 g) of ground matter are mixed with slow magnetic stirring for 24 hours in one liter of distilled water. The solution obtained was carefully filtered on hydrophilic cotton and "Wattman" filter paper. The filtrate collected in a flask is then evaporated at 60 ° C., using a rotary evaporator of "Büchi" type and oven-dried at 50 ± 5 ° C [11, 39]. A water-soluble fine powder is obtained. An yield of 12.25 %. It represents an aqueous extract of *Garcinia kola* Heckel (AE*Gk*).

Animals

Rats (*Rattus norvegicus*, Muridae, L.1753) of Wistar strain were used to carry out this work. They are reproduced at the vivarium of the *Ecole Normale Supérieur* (ENS, Abidjan). The resulting litters are fed and watered ad libitum to reach a weight between 110 and 120 g under standard environmental conditions, temperature 25°C, with a light-dark cycle of 12 hours.

Assessment of anti-dyslipidemic effects of AEGk

Experimental diet for induction of dyslipidemia

Induction of dyslipidemia is similar to protocols previously described, followed by modifications [16, 4] . Experiments are carried out with ninety (90) rats, weighing between 110-120 g, without any pretreatment and adapted to metabolism cages. Eighty (80) of them are fed with high fat and sucrose diet (HFSD) and tap water added with saccharose (300 g of saccharose for one liter of water served every other day). HFSD content 18 % Proteins, 36 % Lipids and 40 % carbohydrate). And ten (10) others are subjected to the Standard Diet (Proteins18 %; Lipids 5 % carbohydrate 65 %) diet with tap water. The animals are monitored for 12 weeks, after which blood samples are taken for the determination biochemical markers of lipemia. Dyslipidemic rats only were selected for further work and the others excluded.

Effect of AEGk on dyslipidemia and postprandial hypertriglyceridemia

On the first series rats are randomly assigned into dyslipidemic four groups (six rats per group) and control for antidyslipidemic study. Dyslipidemics rats are subjected on high-fat and sucrose diet (HFSD). Whereas control groups fed on normal diet. Control group and dyslipidemic untreated HFSD rat receive either vehicle (distilled water). Next HFSD rats are treated respectively with statin (STAT) at 10 mg/kg or with AE*Gk* (600 or 1000 mg/kg).

In another series of experiment, Postprandial hypertriglyceridemia test of AE*Gk* is evaluate according to previous method describes by Toyoda-ono et *al.* [33], with slight modification. Briefly, thirty (30) rats weighing between 250 and 300 g are used, including twenty-four (24) dyslipidemic HFSD rats and six (6) healthy rats as control group and they receive *per os* 5 mL/kg of olive oil. Experimental group and different treatment are similar to those of obtained in antidyslipidemic study describe above. For biochemical analyses, after 12 hours of fasting the rats of each group are weighed, anesthetized with sodium thiopental blood sample is taken from rat orbital sinus, every 7 day during 28 days and each two hours during 6 hours focus, respectively in antihyperlipidemic group and postprandial hypertriglyceridemia tests.

Biochemical analysis

Blood samples taken in dry tubes are centrifuged at 3000 rpm for five minutes. The serum collected are used for the determination of triglycerides (TG), total cholesterol (CHOL-T), HDL-C levels using the Spectrophotometer (visible UV) HITACHI® 704R analyzer (JAPAN) at wavelengths adapted to dosing kit instructions. Determination of LDL cholesterol serum level derived concentration of TG, total cholesterol and HDL cholesterol previously determine [10].

Statistical analysis

The values are expressed as mean ± standard error of mean of six experiment (mean ± SEM). GraphPad Prism 7 software, (Microsoft, San Diego California, USA) was used for statistical analysis of data and graphical representations. The significance differences between treatments was determined using the variance analysis (ANOVA) of the Tukey-Kramer multiple comparison test. Difference was considered as statistically significant when $P < 0.05$.

RESULTS

Effects of EAGk on food intake, water intake and weight gain Feed and water intake

At day 28, there is a significant ($P < 0.001$) overall reduction in the amount of food consumed by HFSD untreated rat (11.13 ± 1.6 g/rat/day; Table I) compared to those of rat received control diet (30.59 ± 2.37 g/rat/day; Table I). However, treatment with STAT (10 mg/kg) and AE*Gk* (600 mg/kg) not affect this ($P > 0.05$). Furthermore, treatment with AE*Gk* at 1000 mg/kg increase significantly ($P < 0.001$) feed intake compared to those of HFSD untreated rat (31.33 ± 5.08 vs 11.13. ± 1.6 g/rat/day; Table I).

Water intake recorded during the experiment showed significant ($P < 0.001$) difference in the comparison between HFSD untreated rat and to those receive control diet (38.67 ± 5.89 vs 20.28 ± 2.69 mL/rat/day; Table I). Treatment with AE*Gk* at 1000 mg/kg not affect significantly ($P > 0.05$; Table I) water intake compared to those of HFSD untreated rat (24.54 ± 4.84 vs 20.28 ± 2.69 mL/rat/day; Table I). However, treatment with STAT (10 mg/kg) and AE*Gk* at 600 mg/kg increase significantly water intake in comparison to those of HFSD untreated rat ($P < 0.05$; Table I). Means recording are 20.28 ± 2.69; 33.06 ± 7.43; 30.94 ± 7.18 mL/rat/day; respectively for HFSD untreated rat; STAT (10 mg/kg) and AE*Gk* at 600 mg/kg (Table I).

Table I: Effects of AE*Gk* on food and water intake in Wistar rats after 28 days

Groups	Control	HFSD	HFSD		
			STAT (10 mg/kg)	AE*Gk* (600 mg/kg)	AE*Gk* (1000 mg/kg)
Food intake (g/rat/day)	30,59 ± 2,37	11,13 ± 1,6$^{\#\#\#}$	13,87 ± 1,42 $^{\#\#\#}$	17,91 ±2,16$^{\#\#\#}$	31,33 ± 5,08***
Water intake (mL/rat/day)	38,67 ± 5,89	20,28 ± 2,69$^{\#}$	33,06 ± 7,43*	30,94 ± 7,18*	24,54 ± 4,84

HFSD: high fat and sucrose diet; STAT: statin; AE*Gk*: Aqueous extract of *Garcinia kola*; Results are show as means (m) ± SEM of 6 different rats. $^{\#}p < 0.05$; $^{\#\#}P < 0.01$; $^{\#\#\#}P < 0.001$ $^{\#\#\#\#}P < 0.0001$ versus control group and *p < 0.05; **P < 0.01; ***P < 0.001; ****P < 0.0001 versus HFSD group.

Evolution of Body Weight

At 16 weeks of dyslipidemia induction, the weight is similar to rat fed with HFSD compare to those received control diet ($P > 0.05$; Table II). After 28 days of treatment, the weight of the control rats subjected to control diet, increased to about 30.7 ± 0.11 g, whereas those of untreated rat decreased to about 17 ± 0.69 g. This variation represents about 55 % of the loss weight compared to those of control group rats ($P < 0.0001$). STAT (10 mg/kg) or AE*Gk* (1000 mg/kg) treatments reduce significantly the weight loss, respectively to 4.7 ± 0.6g and 6.7 ± 0.71 g in HFSD dyslipidemic of untreated rat. This variation represents respectively about 72.35 % and 60.06 % of inhibition in weight loss in HFSD dyslipidemic of untreated rat (Table II). Moreover, AE*Gk* treatment at 600 mg/k abolish the weight loss and promote significantly the weight gain to about 12.3 ± 0.81 g in comparison with the HFSD group ($P < 0.0001$; Table II).

Table II: Effects of AE*Gk* on weight gain in Wistar rats after 28 days

Groups	Control	HFSD	HFSD		
			STAT (10 mg/kg)	AE*Gk* (600 mg/kg)	AE*Gk* (1000 mg/kg)
Weight (g)					
Day 0		303 ± 4,74	299 ± 17,85	302,7 ± 14,83	300 ± 6,64
Day 28	302,30 ± 0,10	286 ± 11,82 $^{\#}$	294,3 ± 23,19	315 ± 15	293,3 ± 11,58
Variation (g)	333 ± 10,60	-17 ± 0,69 $^{\#\#\#/****}$	-4,7 ± 0,6 $^{\#\#\#\#/****}$	12,3 ± 0,81 $^{\#\#\#\#/****}$	-6,7 ± 0,71 $^{\#\#\#\#/****}$
% of weight loss	30,7 ± 0,11	55	15,30	_	21,67
% of weight loss inhibition	_	_	72,35	_	60,6

HFSD: high fat and sucrose diet; STAT: statin; AE*Gk*: Aqueous extract of *Garcinia kola*; Results are show as means (m) ± SEM of 6 different rats. $^{\#}p < 0.05$; $^{\#\#}P < 0.01$; $^{\#\#\#}P < 0.001$ $^{\#\#\#\#}P < 0.0001$ versus control group and $*p < 0.05$; $**P < 0.01$; $***P < 0.001$; $****P < 0.0001$ versus HFSD group.

Ameliorative effect of AEGk on HFSD increase triglyceridemia, total cholesterolemia, LDL-Cholesterolemia and decrease HDL-cholesterolemia of serum levels in rat.

After 16 weeks, induction of dyslipidemia increases significantly serum level of triglyceridemia in HFSD to about 61 % ($P < 0.0001$; Fig. 1 A). Different treatments with STAT (10 mg/kg) and AE*Gk* (600 or 1000 mg/kg) modulate serum triglyceridemia level from at day 7 to day 28. But the significant effect was obtained at AE*Gk* with 1000 mg/kg.

For instance, at day 28 AE*Gk* (1000 mg/kg) decrease triglyceridemia serum level to 38 % compared to those of untreated dyslipidemic HFSD rat ($P < 0.05$; Fig. 1 A).

After 16 weeks, HFSD induce dyslipidemia in rat and increasing significantly serum total cholesterol level (TC) to about 78 % compared to control group fed normal diet ($P < 0.0001$; Fig. 1 B).

From days 7 to days 14 different traetments with STAT (10 mg/kg), and AE*Gk* (600 or 1000 mg/kg) seem not affect hypercholesterolemia in dyslipidemic HFSD rats. Futhermore at day 21 only AE*Gk* at 1000 mg/kg decrease significantly TC serum level in HFSD rats ($P < 0.0001$; Fig. 1 B). This effect was more pronounced at day 28 with a reduction of hypercholesterolemia in dyslipidemic HFSD rats compared to those of control group ($P < 0.0001$; Fig. 1 B). Otherwise, at this stage treatment with STAT (10 mg/kg) also reduce serum TC in HFSD rats to about 48 % ($P < 0.05$; Fig. 1 B).

Dyslipidemia induction, also increase serum LDL cholesterol (LDL-C) levels in HFSD group compare to those of control group fed normal diet ($P < 0.0001$; Fig. 1 C). After 7 days, different treatments with STAT (10 mg/kg) and AE*Gk* (600 or 1000 mg/kg) failled to reduce the increase of LDL-C serum level in HFSD rat ($P > 0.05$; Fig. 1 C). The LDL-C serum levels remain unaffected with AE*Gk* treatment (600 or 1000 mg/kg) after 14 days, but at this stage STAT (10 mg/kg) reduce slightly, but significantly the serum LDL-hypercholesterolemia in HFSD dyslipidemic rat ($P < 0.05$; Fig. 1 C). At day 21, STAT at 10 mg/kg and AE*Gk* at 1000 mg/kg reduce significantly serum LDL-hypercholesterolemia respectively to about 43 % and 46 % ($P < 0.05$; $P < 0.01$) compared to untreated HFSD rats (Fig 1 C). Both treatments with STAT (10 mg/kg) and AE*Gk* (600 or 1000 mg/kg) down-regulate serum LDL-hypercholesterolemia at day 28 (Fig. 1 C). For instance, at this stage STAT (10 mg/kg) decrease LDL-C serum level from 0.57 ± 0.02 mg/L to 0.32 ± 0.02 mg/L ($P < 0.01$; Fig. 1 C).

The strong effect AE*Gk* on serum LDL-hypercholesterolemia HFSD dyslipidemic rat was obtained at the dose of 1000 mg/kg (0.57± 0.02 mg/Lin HFSD untreated rat versus 0.18 ± 0.01 mg/L in rat treated with AE*Gk* at 1000 mg/kg ; Fig. 1 C).

At day 0 after dyslipidemia induction during 16 weeks, serum HDL cholesterol (HDL-C) level decreased significantly about to 34 % in rats subjected to HFSD diet, compared to those of control group, fed normal diet ($P < 0.001$; Fig. 1 D). AE*Gk* at 1000 mg/kg increased

significantly HDL-C serum level compared to those of HFSD group (0.27 ± 0.02 untreated rat versus 0.37. ± 0. 01. mg/L in rat treated with AE*Gk* at 1000 mg/kg) at day 7 (P <.05; Fig. 1 D). At day 14 AE*Gk* treatment (600 or 1000 mg/kg) seem not affect HDL-C serum level, whereas STAT (10 mg/kg) increase significantly HDL-C serum level in rats to about 102 % compared to those of HFSD group rats (P < 0.01; Fig. 1 D). At day 21, serum HDL-C level significantly increased to 34 % in rats subjected to HFSD after STAT (10 mg/kg) treatment (P < 0.05; Fig. 1 D). AE*Gk* (1000 mg/kg), also up regulate serum HDL-C level to about 59 % in comparison to those of HFSD group of (P < 0.05; Fig. 1 D). Similar effects are obtained at 28 days after STAT (10 mg/kg) or AE*Gk* (1000 mg/kg) treatment (P < 0.05; Fig. 1 D).

Fig. 1

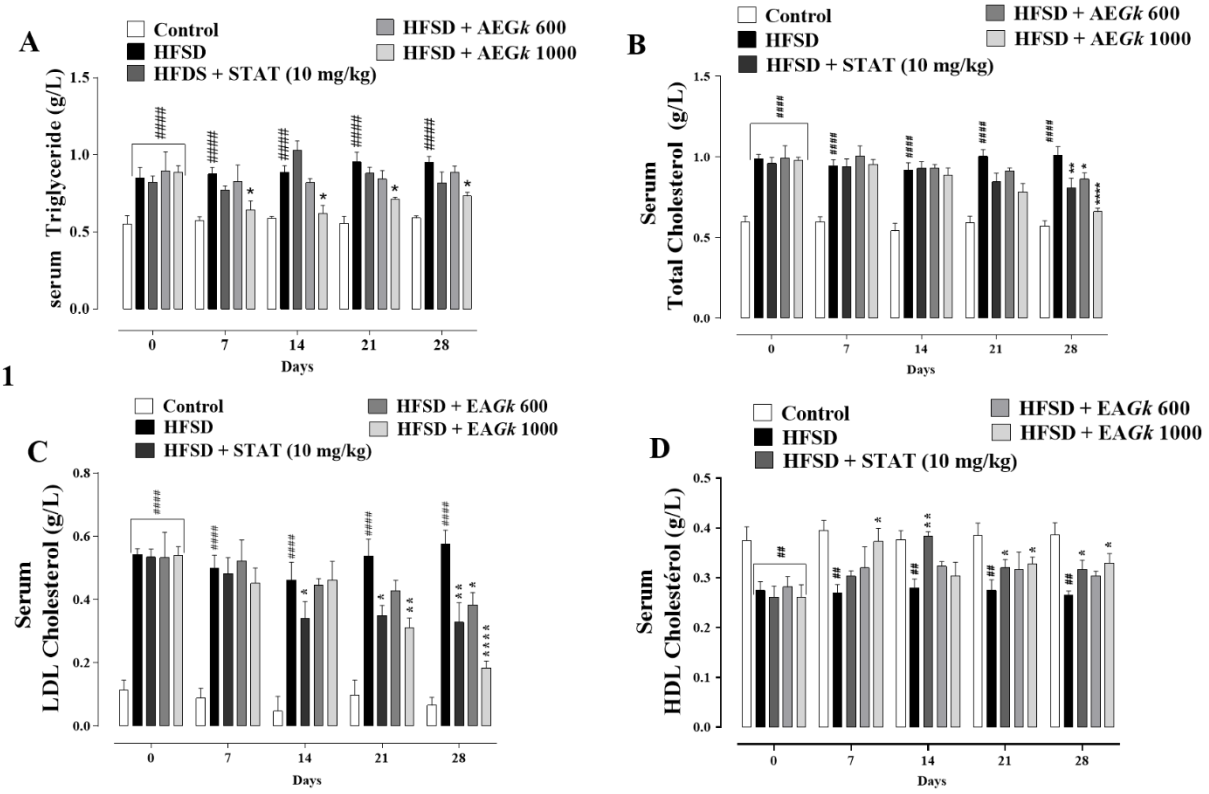

Figure 1: Effects of aqueous extract of *Garcinia kola* and the Statin on the triglyceridemia (A), total cholesterolemia (B), HDL-cholesterolemia (C) and LDL- cholesterolemia in rats after 28 days. Results are show as means (m) ± SEM of 6 different experiments. $^{\#}P < 0.05$; $^{\#\#}P < 0.01$; $^{\#\#\#}P < 0.001$ and $^{\#\#\#\#}P < 0.0001$ versus control group and *p < 0.05; **P < 0.01; ***P < 0.001 and ****P < 0.0001 versus HFSD group.

Ameliorative effect of AEGk on the olive oil overload-induced postprandial hypertriglyceridemia in HFSD rats Kinetics and areas under the curve (AUC) of postprandial triglycerides.

Basal serum triglyceride level in post-absorptive stage (0 hour) remain unaffected after different treatments ($P > 0.05$; Fig 2 A). Two hours, after olive oil administration, serum postprandial triglyceridemia increased significantly in HFSD untreated rat, compared to those of control group (0.82 ± 0.02 mg/L in control versus 2.07 ± 0.08 mg/L in HFSD rat ;Fig 2 A). At this stage, only AE*Gk* treatment at 1000 mg/kg promote slightly, but significant reduction of postprandial triglyceride concentration in HFSD rat which amount to about 82 % ($P < 0.05$; Fig 2 A). After 4 hours, the effect of AE*Gk* (1000 mg/kg) on postprandial triglyceride serum level will sustained, without statistical difference compared to HFSD untreated rat (1.33 ± 0.04 mg/L in rat treated with AE*Gk* at 1000 mg/kg, versus HFSD untreated rat; Fig 2 A).

After 6 hours, differents treatments with STAT (10mg/kg) or AE*Gk* (600 or 1000 mg/kg) decreased postprandial triglyceride serum level near to their respective basal value obtained in post-absorptive stage, but not significant difference are observed compared to HFSD dyslipidemic rat ($P < 0.05$; Fig 2 A).

After six hours, postprandial triglyceridemia AUC increased significantly to about 20 % in untreated rats subjected to HFSD, compared to those of control group fed normal diet ($P < 0.05$; Fig. 2B). No significant changes are observed in postprandial triglyceridemia AUC in rats treated with STAT (10 mg/kg) or AE*Gk* at 600 mg/kg ($P > 0.05$; Fig. 2 A). However, significant reduction of the postprandial triglyceridemia AUC to about 161 % was obtained after AE*Gk* treatment at 1000 mg/kg, compared to those of untreated rats subjected to HFSD diet ($P < 0.01$; Fig. 2 B).

Fig. 2

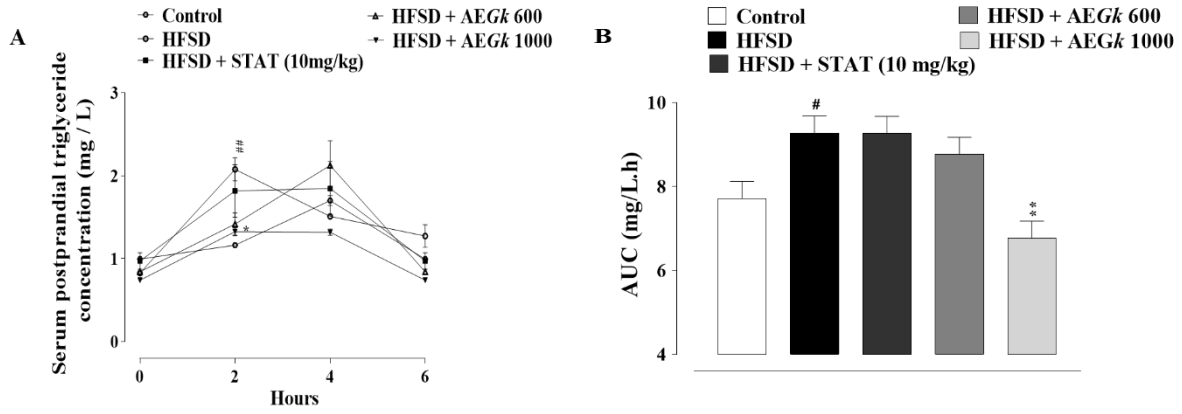

Figure 2: Effects of aqueous extract of *Garcinia kola* and the Statin on postprandial triglyceridemia (A) and its area under the curve (B) in the rat after 28 days following an administration of 5 ml / kg of P.C. olive oil in rats. Results are show as means (m) ± SEM of 6 different experiments. $^{\#}P < 0.05$; $^{\#\#}P < 0.01$; $^{\#\#\#}P < 0.001$ and $^{\#\#\#\#}P < 0.0001$ versus control group and *p < 0.05; **P < 0.01; ***P < 0.001 and ****P < 0.0001 versus HFSD group.

DISCUSSION

Pharmacological effects of an aqueous extract of *Garcinia kola* (AE*Gk*) on dyslipidemia and postprandial hypertriglyceridemia induced by HFSD diet was performed in the Wistar rat.

Previous studies have reported deleterious effects of HFSD on lipemia and CVD in humans and rodents such as mice or rat [13, 16, 25]. HFSD causes obesity, dysfunctions of lipid metabolism and cardiovascular pathologies, therefore it is good model to study dyslipidemia [38, 35, 19].

The mechanism causing these dysfunctions observed involve a mechanistic and genetic transformation of the intestine, an increasing of the absorption surface of the anterocytes and an increase amount of lipid absorbed [28, 8]. It therefore causes disorders of HDL metabolism by excess exogenous LDL which inhibits the activity of Lecithin cholesterol acyltransferase (LCAT). In addition, an increase in hepatic production of VLDL lipoproteins resulting in hypertriglyceridemia is observed [32].

During this study, the 16-week HFSD diet induced hypertriglyceridemia, hypercholesterolemia (total), hyper-LDL-cholesterolemia, hypo-HDL-cholesterolemia, and postprandial hypertriglyceridemia in HFSD rats compared to those of normal control group. These results are similar to those obtained by Mandukhail et *al.*[20] in Wistar rat with high-protein diet by adding cholesterol (2 %), cholic acid (0.5 %) and 2 % butter for seven weeks. Such diet led to a gain in weight in contrast to the present finding which weight losses are recorded. This difference could be explained by the type of fat used for diet and the duration of induction [5].

Moreover, hyperlipidemia and hepatic steatosis has been observed in rats after, 12 weeks of dyslipidemia induction with non-significant weight loss [15]. The weight loss observed in the present study could also be explained by fatty liver disease which would induce a decrease in hepatic protein synthesis caused by a decrease in intake or absorption and abnormal metabolism or an increase in protein requirements [23, 21].

AE*Gk* corrects weight loss, dyslipidemia and postprandial hypertriglyceridemia induced by HFSD diet. In addition, it causes a decrease at 1000 mg/kg of hypertriglyceridemia, total hypercholesterolemia and LDL, and induces an increase in HDL hypocholesterolemia on the 21st and 28th day. At the same dose, postprandial hypertriglyceridemia observed in HFSD diet animals was significantly reduced, thereby decreasing the area under the curve obtained and thereby reducing the cardiometabolic risk in the animals in this group.

These effect are similar to those of obtain with several plant extract from pharmacopoeias and deemed antidyslipidemic effects [37, 20, 33, 1].

For instance, Mandukhail et *al.* [20] showed that hydroethanolic extracts of leaves, fruits and roots of *Morinda citrifolia* at 1000 mg/kg significantly reduce dyslipidemia induced in Sprague-Dawley rats, with a hyperlipidemic diet. It causes a decrease in this dose of hypertriglyceridemia, total hypercholesterolemia and LDL, and induces an increase in HDL hypocholesterolemia. Moreover, biflavonoid fractions of nuts and bark, trunk and *Garcinia kola* roots also decrease in lipid marker dysfunction in Wistar rats on dyslipidemia induced with poloxamer 407 [1].

Moreover, commercial preparation of citrus fruit and olive leaves in Wistar rats induces a significant reduction in postprandial triglyceride levels and pancreatic lipases following the administration of an emulsion, olive oil, saline serum and lecithin respectively to about 20 %,

50 % and 3 %) [22]. AE*Gk*(1000 mg/kg) provides a significant reduction in AUC to about 160 % of postprandial hypertriglyceridemia induced in Wistar rats. These results are similar to those obtained in normal mice and rat by Toyoda-ono et *al.* and Kurihara et *al.* [17, 33]. According to these authors, AUC of the plasma triglycerides of mice treated with the polymerized polyphenol extract of *Camellia sinensis* L., (oolong tea) significantly decreased by 53 % and 76 %, after five hours respectively for doses of 500 and 1000 mg/kg compared to those of the control. Preview studies have showed many phytoconstituents of *Garcinia kola in* aqueous extract [7, 26, 36, 1]. Phytoconstituents such as saponins and polyphenols contained in this extract could be responsible of these ameliorative effects [27].

CONCLUSION

Aqueous extract of *Garcinia kola* nuts induces an antihyperlipidemic and reduces postprandial hypertriglyceridemia induced in rats, which could partially justify its use in traditional medicine for treatment of metabolic disorder. These ameliorative effects seem related to the presence of many microconstituents in the *Garcinia kola* extract.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

REFERENCES:

1. Adejor EB, Ameh DA, James DB, Owolabi OA, Ndi U.S. Effects of *Garcinia kola* biflavonoid fractions on serum lipid profile and kidney function parameters in hyperlipidemic rats. *Clinical Phytoscience.* 2016; 2:19
2. Adesina SK, Gbile ZO, Odukoya OA. Survey of indigenous plants of West Africa with special emphasis on medicinal plants and issues associated with management. *The United Nations Programme on Natural Resources in Africa*; 2nd edition. 1995; 84-5.
3. Alipour A, Elte JWF, van Zaanen HCT, Rietveld AP, Castro Cabezas M. Novel aspects of postprandial lipemia in relation to atherosclerosis. *Atherosclerosis Supplements.* 2008**;** 9: 39-44
4. Amin KA, Kamel HH, Abd Eltawab MA. Protective effect of Garcinia against renal oxidative stress and biomarkers induced by high fat and sucrose diet. *Lipids in Health and Disease*. 2011 ; 10 (6):13p

5. Buettner R, Parhofer KG, Woenckhaus MW, Kunz-Schughart CE, Schölmerich LAJ, Bollheimer LC. Defining high-fat-diet rat models: metabolic and molecular effects of different fat types. *journal of Molecular Endocrinology*. 2006;.36: 485-501
6. Correia J, Pataky Z, Golay A. Comprendre l'obésité en Afrique : poids du développement et des représentations perspectives. *Revue Médicale Suisse.* 2014 ; 10 : 712-6
7. Cotterhill, P.J., Scheinmann, F. and Stenhouse, I.A. Extractives from Guttiferae. Part 34: Kolaflavanone, a new biflavanone from the nuts of *Garcinia kokz* Heckel. Applications of W nuclear magnetic resonance in elucidation of the structures of flavonoids. *Journal of the Chemical Society.* 1978 ; 532-538.
8. De Wit WNJ, Bosch-Vermeulen H, De Groot PJ, Hooiveld JEJG, Bromhaar M MG, Jansen J, Müller M, Van der Meer R. The role of the small intestine in the development of dietary fat-induced obesity and insulin resistance in C57BL/6J mice. *BMC Medical Genomics*. 2008; 1 :14
9. Durrington P. Dyslipidaemia. *Lancet.* 2003;.362: 717-731.
10. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, Without Use of the Preparative Ultracentrifuge. *Clinical Chemistry.* 1972; 18(6):499-502 ·
11. Guédé-Guina F, Vangah-Manda M, Harouna D, Bahi C. Potencies of misca, a plant source concentrate against fungi. *Journal of Ethnopharmacology*. 1993; 14:45-53.
12. Heckel E, Schlagdenhauffen F. Some African kolas, in their botanical, chemical and therapeutical aspects. *The American Journal of Pharmacy*. 1884; 56: 81-177.
13. Hegsted DM, McGrancy RB, Myers ML Stare FM. Quantitative effects of dietary fat on serum cholesterol in man. *American Journal of Clinical Nutrition.*1965; 17.: 281-295
14. Iwu M. Handbook of African medicinal plants.CRC Press, Boca Raton, FL. 1993; 12 : 32-38.
15. Ji G, Zhao X, Leng L, Liu P, Jiang Z. Comparison of dietary control and atorvastatin on high fat diet induced hepatic steatosis and hyperlipidemia in rats. *Lipids Health Diseases*. 2011; 26 (10) :23
16. Kawasaki T., Kashiwabara A., Sakai T., Igarashi K., Ogata N., Watanabe H., Ichiyanagi K, Yamanouchi T . Long-term sucrose-drinking causes increased body weight and glucose intolerance in normal male rats *British Journal of Nutrition***.** 2005; 93 (5): 613-8.
17. Kurihara H, Shibata H, Fukui Y, Kiso Y, Xu J, Yao X, Fukami H. Evaluation of the Hypolipemic Property of *Camellia sinensis* Var. ptilophylla on Postprandial Hypertriglyceridemia. *Journal of. Agriculture and. Food Chemistry***.** 2006; 54: 4977-4981
18. Lei L, Zeng B. "Risk Factor Differences in Acute Myocardial Infarction between Young and Older People: A Systematic Review and Meta-Analysis. *International Journal of Cardiovascular Sciences.* 2019; 32 (2): 163-176
19. Li L, Zhao Z, Xia J, Xin L, Chen Y, Yang S, Li K. A Long-Term High-Fat/High-Sucrose Diet Promotes Kidney Lipid Deposition and Causes Apoptosis and Glomerular Hypertrophy in Bama Minipigs. *Plos one*. 2015 ; 10 (11): e0142884
20. Mandukhail SR, Aziz N, Gilani A-H. Studies on antidyslipidemic effects of *Morindacitrifolia* (Noni) fruit, leaves and root extracts. *Lipids in Health and Disease*. 2010; 9: 88
21. Mc cullough AJ, Tavill AS. Disordered energy and protein metabolism in liver disease. *Seminars in Liver Disease*. 1991; 11(4):265-77
22. Merola N, Castillo J, Benavente-García O, Ros G, Nieto G. The effect of consumption of *Citrus* fruit and olive leaf extract on lipid metabolism. *Nutrients*. 2017; 9: 1062
23. Mezey E. Liver disease and protein needs. *Annual Review of Nutrition.* 1982; 2: 21-50.
24. N'guessan KA, Koffi E, Gnahoua GM, Coulibaly B, Tahouo O. Produire des plants pour sauver l'arbre du «petit cola» en Côte d'Ivoire. *Le C.N.R.A.* 2012; 52:7p
25. Nwibo DD, Eze MI, Okonkwo TM. Effects of *Hibiscus rosa-sinensis* leaf products on haematological indices, lipid profile and hepatic parameters of hyperlipidemic *rat. African. Journal of Pharmacy and Pharmacology*. 2016; 10 (12): 223-229
26. Nyamien Y, Adje F, Niamké F, Chatigre O, Adima A, Biego GH. Caffeine and Phenolic Compounds in *Cola nitida* (Vent.) Schott and Endl and *Garcinia kola* Heckel Grown in Côte d'Ivoire. *British Journal of Applied Science & Technology.* 2014 ; 4(35): 4846-4859

27. Pandey KB, Rizvi SI. Plant polyphenols as dietary antioxidants in human health and disease *Oxidative Medicine and Cellular Longevity.* 2009 ; 2 (5) :270-278
28. Petit V, Arnould L, Martin P, Monnot MC, Pineau T, Besnard P, Note I. Chronic high-fat diet affects intestinal fat absorption and postprandial triglyceride levels in the mouse. *Journal of Lipids Research.* 2007; 48(2) :278-87.
29. Popkin B M. The nutrition transition and obesity in the developing world. *Journal of. Nutrition*. 2001; 131(3):871S-873S.
30. Shi Y, Guo R, Wang X, Yuan D, Zhang S. The regulation of Alfalfa saponin extract on key genes involved in hepatic cholesterol metabolism in hyperlipidemic rats. *Plos one* 2014 ; 9 (2): e88282.
31. Schonewille M, Brufau G, Shiri-Sverdlov R, Groen K A, Plat J. Serum TG- lowering properties of plant sterols and stanols are associated with decreased hepatic VLDL secretion. *Journal of Lipids Research*. 2014; 55(12): 2554-2561
32. Taskinen MR. Diabetic dyslipidemia from basic research to clinical practice. *Diabetologia*. 2003; 46(6):733-49.
33. Toyoda-ono Y, Yoshimura M, Nakai M, Fukui Y, Asami S, Shibata H, Kiso Y. Ikeda Suppression of Postprandial Hypertriglyceridemia in Rats and Mice by Oolong Tea Polymerized Polyphenols. *Bioscience Biotechnology and Biochemistry*. 2007; 71:4, 971-976,
34. World Health Organization (WHO). Prevention of Cardiovascular Disease. Guidelines for assessment and management of total cardiovascular risk. 2007; Genève, 20p
35. Yang ZH, Miyahara H, Takeo J, Katayama M. Diet high in fat and sucrose induces rapid onset of obesity-related metabolic syndrome partly through rapid response of genes involved in lipogenesis, insulin signalling and inflammation in mice. *Diabetol. Metab. Syndr*. 2012; 4 (32) : 10 p
36. Yété P, Togbé A, Yaya K, Agbangnan P, Ndahischimiye V, Djènontin TS, Wotto D, Azandégbé EC, Sohounhloue D. Etude comparative des Composés phénoliques et activité antiradicalaire des extraits des Graines de *Garcinia kola* (Guttifféraea) et de *Cucumeropsis* edulis (cucurbitacéae) du Bénin. *International Journal of Innovation and Scientific Research.* 2015 ; 15 (1) : 217-227
37. Zhao SP, Liu L, Cheng YC, Li YL. Effect of xuezhikang, a cholestin extract, on reflecting postprandial triglyceridemia after a high-fat meal in patients with coronary heart disease. *Atherosclerosis.* 2003 ; 168: 375-380
38. Zhao S; Chu Y; Zhang C; Lin Y; Xu K; Yang P; Fan J; Liu E. Diet-Induced Central Obesity and Insulin Resistance in Rabbits. *Journal of Animal Physiology and Animal Nutrition*. 2007 ;92 :105-111
39. Zirihi GN, Kra AM, Guédé-Guina F. Evaluation de l'activité antifongique de *Microglossa pyrifolia* (Larmarck) O. kuntze (Asteraceae) " pymi " sur la croissance in vitro de *Candida albicans*. *Revue de Médecine et de Pharmacopées Africaines*. 2003; 17:11- 19.