



Lipid Profile of Diabetic Rats Treated with a Combination of an Aqueous Extract of Roasted and Ground *Coffea canephora* Robusta Beans (Filter Coffee Beverage) and Glibenclamide (Hypoglycemic Sulphonamide)

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ABSTRACT

Introduction: Lipid disorders in diabetic lead to a decrease of HDL-cholesterol and an increase of triglycerides and total cholesterol regular and moderate consumption of filtered coffee would prevent the risk of developing type 2 diabetes. The aim of this study was to evaluate the impact of the combination of an aqueous extract of roasted and ground beans of *Coffea canephora* robusta (filtered coffee beverage) and glibenclamide on the lipid profile in diabetic rats. **Methodology:** 5 groups of 6 rats were formed. Group 1 composed of normal blood sugar level rats (205±4 g) and the other 4 groups of diabetic rats (177±3 g). The rats in group 1 each received 1mL of distilled water. The rats in group 2, 3, 4 and 5 received 1 ml of distilled water, 1 mL of glibenclamide (10 mg/kg bw), 1 mL filtered coffee drink at a dose of 20 mg/kg bw and 2 mL consisted of a mix of filtered coffee drink (1 mL) and glibenclamide (1 mL) taken separately. Triglycerides, total cholesterol and HDL-cholesterol were measured on day zero (D0) before the experiment and on day fourteen (D14) and day twenty-eight (D28) after administration of the various substances. **Results:** Triglycerides and total cholesterol decreased in rats from group 3, 4 and 5. HDL-cholesterol increase in group 3 and 5 but not in group 4 after treatment. In addition, triglycerides and total cholesterol decreased significantly ($p<0.01$) and less significantly ($p<0.05$) in group 5 compared with group 3. However, there was no significant variation ($p>0.05$) in HDL-cholesterol in group 5 compared with group 3. **Conclusion:** The aqueous extract of roasted and ground *Coffea canephora* robusta beans potentiates the effect of glibenclamide. This synergy of effect with glibenclamide is an interesting therapeutic way in the treatment of type 2 diabetes.

Keywords: Diabetes, Coffee, Lipid parameters, Management care, Glibenclamide

1. INTRODUCTION

Irregularities in the distribution of lipids and lipoproteins in the blood, diabetic dyslipidemias are often asymptomatic, but can lead to cardiovascular problems, increasing the mortality rate in type 1 or type 2 diabetics [1,2]. A national cross-sectional study by audit involved 2,473 people with type 2 diabetes. This study revealed that 55% of people diagnosed with diabetes within the last two years had dyslipidemia. This percentage increased to 66% in people who had had diabetes for 15 years or more [3]. Appropriate treatment of these lipid metabolic disorders is essential. This would help to improve the health of diabetics. In addition, this treatment has to be accompanied by the respect of dietary hygiene measures. For example, reducing consumption of foods rich in saturated fatty acids and increasing consumption of foods rich in dietary fibres and phytosterols would lead to a reduction in serum levels of total and LDL cholesterol [4]. Phytosterols are bioactive compounds with anticancer, hepatoprotective and cholesterol-lowering properties [5,6]. These phytosterols are also present in roasted and ground coffee beans. Research has been carried out to determine whether drinking filtered coffee is associated with a reduced risk of recurrence and death in people who have survived cardiovascular disease. The results of these studies suggest that drinking filtered coffee may be a protective factor against recurrence and mortality in individuals who have overcome cardiovascular disease [7,8,9]. Similarly, some experimental and clinical studies have also revealed that regular, moderate consumption of caffeine, and by extension filtered coffee, could play a role in preventing the risk of developing type 2 diabetes [10].

Given the positive effects of filtered coffee beverage consumption on human health and the role of diabetes treatment, which often requires drug combinations, it is surprising that few studies have assessed the impact of filtered coffee beverage on the treatment of diabetic patients, particularly on their lipid profile.



The main objective of this study was to assess the impact of the combined consumption of filtered coffee beverage and glibenclamide (an oral anti-hyper-glycemic substance) on the lipid profile in the case of experimental diabetes.

2. Materials and methods

2.1. Plant material

The plant material used in this experiment is ripe cherries of *Coffea canephora* robusta. These cherries, harvested in the subprefecture of Agboville in Côte d'Ivoire, were shade-dried, peeled and then stored in a refrigerator at 8°C.

2.2. Animals

Albino white rats (*Rattus norvegicus*) of *Wistar* strain from the animal house of the Institute Pasteur in Adiopodoumé, Abidjan (Côte d'Ivoire) were used in the experiment. The animals weighed between 200-210 grams and were housed in ventilated metallic cages with a temperature of $24 \pm 3^\circ\text{C}$, with 12 hours light and 12 hours dark cycles and 50% hygrometry for 7 days. They were divided into several groups in these metal cages with free access to water and food.

2.3. Preparation of the aqueous extract of roasted and ground beans of *Coffea canephora* robusta

These peeled beans of *Coffea canephora* robusta were roasted at 220°C for 30 minutes and then ground to a medium particle size powder. Thirty grams (30 g) of this powder were infused for 10 minutes in 175 millilitres (mL) of hot distilled water using a filter coffee maker. The obtained filtrate was oven-dried at 60°C. The powder obtained, three grams (3 g), represented *Coffea canephora* robusta Aqueous Extract of Roasted and Ground beans (CcAERG). The CcAERG powder samples were stored in hermetically sealed green sterile glass jars and placed in a refrigerator at 8°C.

2.4. Chemicals and pharmaceutical products

Alloxane monohydrate 98%, a metabolic disorder which causes diabetes with intraperitoneal administration, destroys β -cells of the islets of Langerhans.

This drug was used to induce experimental diabetes. Glibenclamide, an oral anti-hyper-glycemic substance from the family of hypoglycemic sulphonamides, was used as an anti-hyper-glycemic substance.

2.5. Study of the effects of taking a combination of the aqueous extract of roasted and ground beans of *Coffea canephora* robusta (filtered coffee beverage) and glibenclamide on certain lipid parameters in diabetic rats.

2.5.1. Experimental protocol

This study was conducted with 30 *Wistar* rats, divided into 5 groups of 6 rats. This experiment took place during a period of 4 weeks. Group 1 is composed of normal blood sugar level rats and the other 4 groups of diabetic rats.

Experimental diabetes was induced intraperitoneally by injecting a single dose of 150 mg/kg bw alloxane monohydrate 98% dissolved in 9‰ NaCl.

Precisely thirty wistar rats were divided into 6 treatment groups as follows:

Group 1 (control group rats) : This group received distilled water at 10 mL/kg bw.

control (NCR);

Group 2: rats in this group were made diabetic by intraperitoneal injection of alloxane (150 mg/kg bw) and treated with 10 mL/kg bw distilled water (UDR);

Group 3: rats in this group were made diabetic by intraperitoneal injection of alloxane (150 mg/kg bw) and treated with 10 mg/kg bw of glibenclamide at a dose of 1 mL/rat;



Group 4: rats in this group were made diabetic by intraperitoneal injection of alloxan (150 mg/kg bw) and treated with 20 mg/kg bw CcAERG at 1 mL/rat.

Group 5: rats in this group were made diabetic by intraperitoneal injection of alloxane (150 mg/kg bw) and treated with CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) (DRTC+G).

2.5.2. Dosage of lipid parameters

Lipid parameters were dosed at the start of the experiment on day D0, before administration of the various substances, and then on day D14 and day D28 after administration of the same substances.

2.5.2.1. Measurement of Blood level of triglyceride

Quantitative determination of serum triglycerides was made by using a colorimetric enzymatic method according to the Spinreact data sheet [11].

2.5.2.1. Measurement of cholesterol levels and HDL cholesterol rates

Total cholesterol and HDL-cholesterol levels were measured using a colorimetric method based on the Spinreact data sheet [12].

2.6. Methods of statistical analysis and processing of results

Data analysis and graphing were carried out using Graph Pad Prism 5 software (San Diego, California, USA). The results were expressed in mean \pm SEM.

The difference between two values was determined by the student-Newman-Keuls test and considered as less significant for ($p < 0.05$); significant for ($p < 0.01$) and highly significant for ($p < 0.001$).

3. Results

Study of the effects of taking a combination of the aqueous extract of roasted and ground beans of *Coffea canephora robusta* (filtered coffee beverage) and glibenclamide on certain lipid parameters in diabetic rats.

3.1. Blood level of Triglyceride

Injection of alloxane (150 mg/kg bw) induced a significant increase in triglyceride concentration in diabetic rats compared with control rats (1.39 ± 0.06 g/L versus 0.82 ± 0.05 g/L) ($p < 0.001$) (Table 1).

At day 14, triglyceride levels remained higher in untreated diabetic rats compared with control rats (1.40 ± 0.04 g/L versus 0.80 ± 0.05 g/L) ($p < 0.001$) (Table 1). The different treatments with glibenclamide (10 mg/kg bw), CcAERG (20 mg/kg bw) and the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) reduced the blood level of triglyceride in diabetic rat ($p < 0.001$, $p < 0.05$, $p < 0.001$) (Table 1).

This drop was greater when diabetic rats were treated with CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw (1.40 ± 0.04 g/L versus 1.08 ± 0.03 g/L). Blood level of triglyceride was also higher in diabetic rats treated with CcAERG (20 mg/kg bw) compared with the triglyceride concentration of diabetic rats treated with glibenclamide at 10 mg/kg bw (1.35 ± 0.04 g/L versus 1.13 ± 0.05 g/L) ($p < 0.001$) (Table 1).

However, treatment with the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) lowered triglyceride concentrations in diabetic rats compared with treatment with glibenclamide alone at 10 mg/kg bw (1.08 ± 0.04 g/L versus 1.13 ± 0.05 g/L) ($p < 0.05$) (Table 1).

After 28 days, triglyceride concentration increased from 0.83 ± 0.03 g/L in control rats to 1.38 ± 0.05 g/L in untreated diabetic rats ($p < 0.001$) (Table 1). At this stage, treatment with glibenclamide (10 mg/kg bw), CcAERG (20 mg/kg bw) and CcAERG (20 mg/kg



bw) + glibenclamide (10 mg/kg bw) induced a significant decrease in triglyceride concentration in diabetic rats ($p < 0.001$, $p < 0.01$, $p < 0.001$) (Table 1). In addition, treatment with the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) resulted in a significant decrease in triglyceride concentration compared with the other treatments.

This decrease was 34.78% compared with the triglyceride concentration of untreated diabetic rats (0.90 ± 0.04 g/L versus 1.38 ± 0.05 g/L, $p < 0.001$) (Table 1).

Table 1: Triglyceride concentrations at different periods Day 0, Day 14 and Day 28 in different groups of experimental animals.

Parameter	Rat groups	Period		
		D0	D14	D28
Triglyceride concentrations (g/L)	NCR	0,82±0,05	0,80±0,05	0,83±0,03
	UDR	1,39±0,06***	1,40±0,04***	1,38±0,05***
	DRTG	1,36±0,05***	1,13±0,05 ^{###}	0,97±0,05 ^{###}
	DRTC	1,37±0,03***	1,35±0,04 ^{#,φφφ}	1,33±0,06 ^{#,φφφ}
	RDTC+G	1,38±0,04***	1,08±0,03 ^{###,φ}	0,90±0,04 ^{###,φφ}

NCR: Non-glycemic Control Rats; UDR: Untreated Diabetic Rats; DRTG: Diabetic Rats Treated with Glibenclamide; DRTC: Diabetic Rats Treated with CcAERG; RDTC + G: Diabetic Rats Treated with CcAERG + Glibenclamide.

Triglyceride concentrations are expressed as mean followed by standard error on the mean ($m \pm SEM$), $n = 6$; ANOVA, Newman-Keuls Test; *** $p < 0.001$ vs control rats and same time period; [#] $p < 0.05$, ^{##} $p < 0.01$, ^{###} $p < 0.001$ compared with diabetic rats untreated and the same period; ^φ $p < 0.05$, ^{φφ} $p < 0.01$, ^{φφφ} $p < 0.001$ compared with diabetic rats treated with glibenclamide and the same period.

Similarly, the triglyceride concentration of diabetic rats treated with glibenclamide at 10 mg/kg bw was lower compared with that of diabetic rats treated with CcAERG at 20 mg/kg bw (0.97 ± 0.05 g/L versus 1.33 ± 0.06 g/L, $p < 0.001$) (Table 1).

However, this concentration is elevated compared with that of diabetic rats treated with CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw (0.97 ± 0.05 g/L versus 0.90 ± 0.04 g/L) ($p < 0, 01$) (Table 1).

3.2. Cholesterol levels

A significant increase in cholesterol levels was observed in diabetic rats ($p < 0.001$). It was 70.45% higher than the control group (1.50 ± 0.08 g/L versus 0.88 ± 0.04 g/L) (Table 2).

At day 14, the untreated diabetic rats increased significantly cholesterol level compared with the control group (1.49 ± 0.10 g/L versus 0.86 ± 0.1 g/L) ($p < 0.001$) (Table 2). At this stage, the different treatments with glibenclamide (10 mg/kg bw) and the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) showed a reduction of cholesterol levels in diabetic rats ($p < 0.01$).

Table 2: Total cholesterol concentrations at Day 0, Day 14 and Day 28 in the different experimental different batches of experimental animals

Parameter	Rat groups	Period		
		Day 0	Day 14	Day 28
Total Cholesterol concentration (g/L)	NCR	0,88±0,04	0,86±0,1	0,87±0,04
	UDR	1,50±0,08***	1,49±0,1***	1,53±0,08***
	DRTG	1,51±0,05***	1,40±0,03 ^{##}	1,03±0,05 ^{###}
	DRTC	1,48±0,06***	1,47±0,06 ^φ	1,44±0,04 ^{#,φφφ}
	RDTC+G	1,52±0,07***	1,38±0,05 ^{##}	0,97±0,03 ^{###,φ}

NCR: Non-glycemic Control Rats; UDR: Untreated Diabetic Rats; DRTG: Diabetic Rats Treated with Glibenclamide; DRTC: Diabetic Rats Treated with CcAERG; RDTC + G: Diabetic Rats Treated with CcAERG + Glibenclamide.



Total cholesterol concentrations are expressed as mean followed by standard error on the mean ($m \pm SEM$), $n = 6$; ANOVA, Newman-Keuls Test; *** $p < 0.001$ vs control rats and same time period; $^{\#}p < 0.05$, $^{\#\#}p < 0.01$, $^{\#\#\#}p < 0.001$ compared with diabetic rats untreated and the same period; $^{\phi}p < 0.05$, $^{\phi\phi\phi}p < 0.001$ compared with diabetic rats treated with glibenclamide and the same period.

This cholesterol reduction was more significant in diabetic rats treated with CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw (1.49 ± 0.10 g/L versus 1.38 ± 0.05 g/L) (Table 2). However, cholesterol levels in diabetic rats treated with CcAERG (20 mg/kg bw) were similar to those in untreated diabetic rats (1.49 ± 0.10 g/L versus 1.47 ± 0.06 g/L) ($p > 0.05$) (Table 2).

In addition, treatment with glibenclamide at a dose of 10 mg/kg bw induced a decrease in total cholesterol concentration compared with treatment with CcAERG at a dose of 20 mg/kg bw (1.40 ± 0.03 g/L versus 1.47 ± 0.06 g/L) ($p < 0.05$) (Table 2).

A similar concentration of total cholesterol was observed in diabetic rats treated with glibenclamide (10 mg/kg bw) and in diabetic rats treated with CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw (1.40 ± 0.03 g/L versus 1.38 ± 0.05 g/L) ($p > 0.05$) (Table 2).

After 28 days, cholesterol levels increased from 0.87 ± 0.04 g/L in control rats to 1.53 ± 0.08 g/L in untreated diabetic rats ($p < 0.001$).

The different treatments with glibenclamide (10 mg/kg bw), CcAERG (20 mg/kg bw) and the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) induced a decrease in cholesterol concentration in diabetic rats ($p < 0.001$, $p < 0.05$, $p < 0.001$) (Table 2). However, a significant reduction in cholesterol levels was observed with the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) compared with the other treatments. This reduction in cholesterol levels was 36.60% compared with the cholesterol concentration in untreated diabetic rats (0.97 ± 0.03 g/L versus 1.53 ± 0.08 g/L). In addition, the cholesterol concentration of diabetic rats treated with glibenclamide at a dose of 10 mg/kg bw was lower compared with that of diabetic rats treated with CcAERG at a dose of 20 mg/kg bw (1.03 ± 0.05 g/L versus 1.44 ± 0.04 g/L) ($p < 0.001$) (Table 2).

However, this concentration is increased compared with that of diabetic rats treated with CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw (1.03 ± 0.05 versus 0.97 ± 0.04) ($p < 0.05$) (Table 2).

3.3. HDL cholesterol

HDL cholesterol concentration was significantly reduced in rats after induction of diabetes ($p < 0.001$). This reduction was 33.33% compared with HDL cholesterol levels in control rats (0.22 ± 0.02 g/L versus 0.33 ± 0.03 g/L) (Table 3).

After 14 days, the reduction in HDL-Cholesterol concentration remained significant in the untreated diabetic rats compared with the HDL-Cholesterol level in the control group (0.21 ± 0.04 g/L versus 0.33 ± 0.05 g/L) ($p < 0.001$). Treatment with glibenclamide (10 mg/kg bw) and the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) induced a significant increase in HDL cholesterol concentration in diabetic rats ($p < 0.01$) (Table 3). This increase was higher when diabetic rats were treated with glibenclamide (0.21 ± 0.04 g/L versus 0.27 ± 0.03 g/L). However, treatment with CcAERG (20 mg/kg bw) had no effect on the HDL cholesterol concentration of untreated diabetic rats (0.21 ± 0.03 g/L versus 0.22 ± 0.02 g/L) ($p > 0.05$) (Table 3). In addition, treatment with glibenclamide (10 mg/kg bw) resulted in an increase in HDL cholesterol concentration compared with treatment with EAGTCC at a dose of 20 mg/kg bw (0.27 ± 0.03 g/L versus 0.22 ± 0.02 g/L) ($p < 0.01$) (Table 3). Furthermore, a non-significant decrease in HDL cholesterol concentration was observed with the combination of CcAERG (20 mg/kg bw) and glibenclamide (10 mg/kg bw) compared with treatment with glibenclamide at a dose of 10 mg/kg bw (0.26 ± 0.04 g/L versus 0.27 ± 0.03 g/L) ($p > 0.05$) (Table 3).

After 28 days, HDL cholesterol concentration increased from 0.34 ± 0.02 g/L in control rats to 0.20 ± 0.03 g/L in untreated diabetic rats ($p < 0.001$) (Table 3).

**Table 3: HDL-cholesterol concentrations at different periods Day 0, Day 14 and Day 28 in different groups of experimental animals**

Parameter	Rats groups	Period		
		D0	D14	D28
HDL cholesterol concentration (g/L)	NCR	0,33±0,03	0,33±0,05	0,34±0,02
	UDR	0,22±0,02***	0,21±0,04***	0,20±0,03***
	DRTG	0,24±0,04***	0,27±0,03 [#]	0,31±0,02 [#]
	DRTC	0,23±0,02***	0,22±0,02 ^{φφ}	0,23±0,03 ^{φφφ}
	DRTC+G	0,21±0,03***	0,26±0,04 [#]	0,31±0,04 [#]

NCR: Non-glycemic Control Rats; UDR: Untreated Diabetic Rats; DRTG: Diabetic Rats Treated with Glibenclamide; DRTC: Diabetic Rats Treated with CcAERG; DRTC + G: Diabetic Rats Treated with CcAERG + Glibenclamide.

HDL cholesterol concentrations are expressed as mean followed by standard error on the mean ($m \pm SEM$), $n = 6$; ANOVA, Newman-Keuls Test; *** $p < 0.001$ vs control rats and same time period; [#] $p < 0.01$, ^{##} $p < 0.001$ compared with diabetic rats untreated and the same period; ^{φφ} $p < 0.01$, ^{φφφ} $p < 0.001$ compared with diabetic rats treated with glibenclamide and the same period.

At this stage, an increase in HDL cholesterol concentration was observed respectively in diabetic rats treated with glibenclamide (10 mg/kg bw), CcAERG (20 mg/kg bw) and CcAERG (20 mg/kg bw) combined with glibenclamide at a dose of 10 mg/kg bw ($p < 0.001$, $p < 0.05$, $p < 0.001$) (Table 3).

HDL cholesterol concentrations in diabetic rats treated with glibenclamide (10 mg/kg bw) and diabetic rats treated with the combination of CcAERG (20 mg/kg bw) + glibenclamide (10 mg/kg bw) were similar (0.31 ± 0.02 g/L versus 0.31 ± 0.04 g/L). In addition, the HDL cholesterol concentration of diabetic rats treated with glibenclamide (10 mg/kg bw) was higher than that of diabetic rats treated with CcAERG at a dose of 20 mg/kg bw (0.31 ± 0.02 g/L versus 0.23 ± 0.03 g/L) (Table 3).

4- Discussion

The results obtained in his study show serum concentration increase of lipid parameters in diabetic rats. The hyperlipidemia observed, could be generated by alloxane and would result from the intense degradation of lipid compounds in adipose tissue [13,14].

Indeed, insulin has an inhibitory action on 3- hydroxy-3-methyl-glutaryl coenzyme A reductase (HMG-COA reductase), a key enzyme in cholesterol biosynthesis [15].

Furthermore, the partial improvement in lipid disorder observed in diabetic rats treated with CcAERG could be due to the effects of the extract's constituents. These constituents could act either by storing fatty acids as triglycerides in adipose tissue, or by inhibiting HMG-COA reductase, or by increasing the activity of lipoprotein lipases or by reducing hepatic triglyceride synthesis [15,16].

The chlorogenic (CAG) and caffeic (CA) acids contained in coffee could play an important role in preventing atherosclerosis, a complication of hyperlipidemia in diabetics. Their action could inhibit the oxidation of low-density lipoproteins (LDL) in plasma [17].

An anti-hyperlipidemic effect is also attributed to ACG and depends on its ability to increase the activities of 3-hydroxy-3-methyl-glutaryl coenzyme A reductase in the liver and kidney and to increase the activities of lipoprotein lipase and lecithin cholesterol acyltransferase in plasma [18]. In addition, coffee consumption is correlated with a reduction in LDL oxidability [19]. ACG briefly reduces the sensitivity of LDL oxidase and lowers LDL cholesterol and malondialdehyde levels [19]. The trigonelline in coffee also has lipid-lowering effects [20]. Treatment with trigonelline reduces serum cholesterol and triglyceride levels in type 2 diabetic rats [21,22].

The hyperlipidemia observed in this study has been reported in several studies. these authors reported an increase in serum lipids in diabetic rats [23,24,25]. This increase in serum lipids could play an important role in the pathology of diabetes [25]. On the other hand, experimental studies have demonstrated an effect of ACG on serum lipid metabolism [26]. ACG could reduce lipid



concentrations (total cholesterol, triglycerides, free fatty acids, phospholipids and LDL) and increase HDL-cholesterol levels in plasma and in certain tissues such as the liver and kidney [18]. The polyphenols contained in coffee are could also have an effect to sensitize insulin, improve the lipid profile and improve inflammation and endothelial function [27].

However, consumption of boiled coffee is associated with high serum concentrations of LDL-cholesterol, whereas consumption of filtered coffee leads to a slight increase in cholesterol in human serum [28]. The low presence of diterpenes (cafestol and kahweol) in filtered coffee could be responsible for this increase in cholesterol [29].

Conclusion

This study shows that the aqueous extract of roasted and ground beans of *Coffea canephora* robusta has an anti-hyperlipidemic effect. This extract potentiates the effect of glibenclamide for most of the lipid parameters studied.

This synergy of effect with glibenclamide could be an interesting therapeutic way in the treatment of type 2 diabetes. In addition, experimental tests could be carried out with the various components of the aqueous extract of roasted and ground beans of *Coffea canephora* robusta in order to identify the compound(s) responsible for this anti-hyperlipidemic effect.

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