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ASSESSMENT OF THE EFFECTS OF THE AQUEOUS EXTRACT FROM TRUNK BARK OF *PARKIA BIGLOBOSA* (MIMOSACEAE) ON GLYCEMIA, RELEASE AND STORAGE OF HEPATIC GLUCOSE IN HEALTHY AND STREPTOZOTOCIN-INDUCED DIABETIC RATS

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ABSTRACT

Objective: *Parkia biglobosa* (Mimosaceae), is a plant commonly used in traditional African medicine to treat many diseases including hypertension, cardiac disorders and diabetes**.** This work aims to evaluate the effects of an aqueous extract from trunk bark of *Parkia biglobosa* (Mimosaceae) on blood sugar, the release and storage of hepatic glucose in healthy and diabetic rats. **Methods:** The measure of released glucose and glucose stored in the liver is performed using the reagent GOD-POD glucose in normoglycemic and diabetic rats. The blood sugar measure was performed on fasting rats using a glucometer. The animals were treated with glibenclamide at dose of 10 mg/Kg B.W and the aqueous extract of trunk bark of *Parkia biglobosa* at doses of 500 and 1000 mg/Kg B.W respectively. **Results:** The administration of EAqPB at doses of 500 and 1000 mg/Kg B.W reduced the release of liver glucose in normoglycemic rats after 60 minutes, with a significant percentage reduction ($p<0.0001$) of 13.83% for the group treated at a dose of 1000 mg/Kg B.W EAqPB. After 90 days of treatment, EAqPB at a dose of 1000 mg/Kg B.W, causes a significant decrease ($p<0.0001$) in blood glucose of diabetic rats by 58,79%. Moreover, after 90 days of treatment, EAqPB causes a significant increase ($p<0.0001$) of hepatic glucose stored in rats rendered diabetic by 142%. **Conclusion:** The study showed that the extract inhibits hepatic glucose release in normoglycemic rats and significantly reduces hyperglycemia in diabetic rats. In addition, EAqPB promotes the glucose storage in the liver of diabetic rats.

KEYWORDS: Diabetes, *Parkia biglobosa*, Streptozotocin, blood sugar, glycogenogenesis and glycogenolysis.

INTRODUCTION

Blood sugar balance is regulated by the combination of different organs and tissues. Among them we can mention: the intestine, the liver, the adipose tissue, the muscles, the brain and the pancreas.[1] This regulation is part of the process of maintaining homeostasis within the body. Therefore, any functional failure causing a disruption of metabolic functions in the body leads to the so-called metabolic diseases. One of the most dreadful metabolic affection is diabetes and represent also one of the most common diseases of civilization.[2]

Diabetes mellitus is a metabolic condition characterized by an abnormal rise in blood sugar level. It is a metabolic disorder of multiple etiology characterized by chronic hyperglycemia due to a defect in insulin's secretion or action or both.[3] According to the WHO[4], the number of people with diabetes continues to grow alarmingly (108 million in 1980 to 422 million in 2014). Ivory Coast is not immune to this pandemic with a prevalence that rose from 5.7% before 2000 to 9.6% in 2010 according to the Côte d'Ivoire Obesity and Diabetes Association.[5] The management of this condition is very expensive due to the high cost of treatment. These constraints lead people in developing countries to turn to traditional medicine.[6]

Several ethnobotanical surveys allowed Parkia biglobosa's classification among antidiabetic plants in the African pharmacopoeia.[7,8,9]

Parkia biglobosa (Mimosaceae), commonly known as *nere* in Ivory Coast Malinke language, is a plant used in the treatment of several infections. It is recommended in the treatment of amoebiasis, hookworm, roundworm, asthma, infertility, peptic ulcers and dental pain.[7,10] It is also used in the treatment of cardiac and renal disorders and hypertension as well.[11,12]

Our work has an overall objective to assess the pharmacological effects of an aqueous extract of the trunk bark of *Parkia biglobosa* (Mimosaceae) on blood

sugar, the release and storage of hepatic glucose in healthy rats and in diabetic rats to contribute to the enhancement of plants uses in traditional medicine and improve populations' health.

MATERIAL AND METHODS

Vegetal material

The trunk bark of *Parkia biglibosa* (Jacq.) Benth. (Mimosaceae) was collected on August 25, 2017 in Zuénoula, 373 km away from Abidjan, Ivory Coast. The identification was made by Professor ZIRIHI Guédé Noel from the Botanical Laboratory using the herbariums number 10933 of 12 -22 -1969, 13329 of 02-08-1976 and 13336 of 02-09-1976 of the National Floristic Center (CNF) of Ivory Coast.

Animal material

Male rats of *Rattus norvegicus* specie, *Wistar* strain weighing between 160 and 180 g were used for this study. These animals came from the vivarium of the Superior Normal School, Abidjan. They had access to food and water *ad libitum*. The animals were acclimated to laboratory conditions before the experiment started.

Preparation of the aqueous extract of the trunk bark of *Parkia biglobosa*

The bark is cut into pieces, dried at 25 °C and then ground in a mechanical ball mill. Fifty grams (50 g) of ground material are mixed by slow magnetic stirring for 24 hours in 1 liter of distilled water. The macerate is filtered on hydrophilic cotton and "Wattman $n^0 2$" filter paper. The filtrate obtained was dried in an oven at 60°C. The fine brown powder obtained represents the crude aqueous extract of the trunk bark of *Parkia biglobosa* (EAqPB).

A stock solution is then prepared with a given amount of the powder and from which test solutions at different concentrations will be realized.

Mac-Ewen Solution preparation

One liter of Mac-Ewen solution is made of 130 mM NaCl; 5.63 mM KCl; 12.16 mM $CaCl_2$; 0.91 mM H_2PO_4Na; 11.90 mM HCO_3Na and 0.25 mM $MgCl_2$. Two (2) grams of glucose are added to this physiological solution before the experiments. The glucose Mac-Ewen is used to study the release of hepatic glucose.

Chemical and pharmacological substances

The chemical and pharmacological substances used for our work are: 95% ethanol, sulfuric acid, dinitrophenolphthalein, 2.5 N sodium hydroxide, the reagent GOD-POD (glucose oxidase-peroxidase), trichloroacetic acid, 0.9% NaCl, glucose Mac-Ewen, anhydrous glucose, glibenclamide (Daonil^R, Sanofi-Aventis, France), streptozotocin and nicotinamide (Sigma, Germany).

Glucose released measurement in normoglycemic rats' liver

Principle

In the presence of glucose oxidase (GOD), (+) D-glucose is oxidized into gluconic acid and hydrogen peroxide (H_2O_2). The hydrogen peroxide released during the reaction reacts with the action of peroxidase (POD), the chloro-4-phenol and 4-aminoantipyrine (AA) to form quinoneimine dye (a pink complex) and water. The intensity of the coloration is proportional to the concentration of glucose in the sample. The reactions involved are as follows:

$$\text{(+) D-glucose} + O_2 + H_2O \xrightarrow{\text{GOD}} \text{D-glucose} + H_2O_2$$

$$H_2O_2 + \text{Chloro-4-Phenol} + \text{AA} \xrightarrow{\text{POD}} \text{Quinoneimine (pink)} + H_2O$$

Experimental protocol

This study is carried out on 20 normoglycemic male rats of *Wistar* strain, with a body weight (B.W) ranging from 160 g to 180 g, divided into 4 groups of 5 rats. These rats fasted for an 18 hours time period. After determining their weight and basal blood glucose, the animals of group 1 (normal control) received distilled water by gavage for 28 days (duration of the experiment). Those from groups 2 and 3 were treated with the aqueous extract of trunk bark of *Parkia biglobosa* (EAqPB), at doses of 500 and 1000 mg/Kg B.W. Group 4's rats are treated with 10 mg/Kg B.W of glibenclamide. After 28 days of treatment, animals are sacrificed and liver fragment weighing 2 g are taken from each rat of each group. These liver fragments collected from the 4 lots are immersed individually in 4 ml of Mac-Ewen glucose, then brought to incubation for 60 minutes at 37°C. Before the pieces of liver are dissolved, the glucose concentration of each of the Mac Evan glucose solutions is 2 ± 0.02 g/l. The supernatant from each solution was collected for an assay measuring the amount of glucose in presence of GOD-POD glucose (reagent), The Quinoneimine's absorbance is proportional to the glucose concentration in the reaction. The absorbance was measured at 500 nm using a spectrophotometer (Biolabo, France), at specific times: 0 minutes (before the organs were dissolved), then 20 minutes, 40 minutes and 60 minutes after organs' immersion in the Mac Ewen glucose solution.

Study of the effects of the aqueous extract of *Parkia biglobosa* trunk bark on diabetic rats' blood glucose

Blood sugar measurement

Rats' blood glucose level is measured using Accu-Chek test strips (Roche, Germany). In this study, the rats fasted for 18 hours before the experiment. The test substances are administered to them orally.

Experimental protocol

For this study, a total of 25 Wistar rats weighing between 160 and 180 g were used. The rats were divided into 5 groups of 5 rats in metal cages lined with wood chips.

After the determination of their basic blood sugar, these animals underwent a treatment by oral administration of glibenclamide (10 mg / Kg B.W) and two (02) doses of our aqueous extract from trunk bark of *Parkia biglobosa* for 90 days (duration of experiment). The treatment with the plant's aqueous extract or the reference product glibenclamide or distilled water was initiated 24 hours after the confirmation of experimental diabetes.

The blood sugar measurement was carried out on fasting rats, every week (D0, D7, D14, D21 and D28), then every 4 weeks (D56 and D90) after 28 days of treatment until the experiment's end.

Healthy animals and diabetic ones were treated as follows: the rats of Lot 1 (healthy control lot) received 2 ml of distilled water. The rats of lot 2 (diabetic control lot) received 2 ml of distilled water. The rats of lots 3, 4, and 5 (test lots) received respectively 10 mg/Kg B.W of glibenclamide, 500 and 1000 mg/Kg B.W of EAqPB.

Measurement of the glucose stored in diabetic Rats' liver

Principle

The dosage of glucose stored in the liver is done using the GOD-POD glucose reagent.

Experimental protocol

This study was carried out on 25 Wistar rats divided into 5 lots. Their weight varied between 160 and 180g. Group 1 is the normoglycemic control which receives distilled water. Lot 2 constitutes the diabetic control receiving distilled water. Lots 3, 4 and 5 are lots of diabetic rats treated with respectively 500, 1000 mg/Kg of B.W of EAqPB and glibenclamide at a dose of 10 mg/Kg B.W. After 90 days of treatment, the animals are sacrificed and 5 g of liver are taken from each of the rats of each group, cut into small pieces and then ground in 30 ml of trichloroacetic acid 4%. The ground product obtained is placed in a test tube and centrifuged at 4500 rpm during 10 min and the supernatant collected. Ethanol 95% is then added to the supernatant (ethanol/supernatant, 2 v/v); the mixture is stirred and heated in a slow-boiling water bath to the boiling point. The glycogen precipitates and the suspension obtained is cooled and centrifuged at 4500 rpm for 10 min. 2 ml of 2.5 N sulfuric acid (H_2SO_4) is added to the pellet (precipitated glycogen) and the tube is heated for 30 minutes. This step allows the glycogen's hydrolysis into glucose. After the hydrolysis, the tube is cooled and 1 drop of dinitrophenolphthalein is added, followed by sodium hydroxide 2.5N until a red-pink coloration appears. This step allows the neutralization of the hydrolyzate's acidity. For each sample, the glucose thus formed is measured by the Beer colorimetric method[13] in the presence of the GOD-POD reagent Beer.[14] The formed glucose level is dosed using a spectrophotometer (Biolabo, France), at 500 nm.

Statistical analysis methods and treatment of results

The statistical analysis of the values and the graphical representation of the data were carried out using the software and Graph PadPrism 7 (San Diego, California, USA). The statistical difference between the results was obtained through the analysis of variances (ANOVA), followed by the Tukey-Kramer multiple comparison test, with a significance level $P < 0.05$. All values are presented as mean ± SEM (Standard Error of the Mean).

RESULTS

Effects of the aqueous extract from trunk bark of *Parkia biglobosa* on the hepatic glucose released by the liver in normoglycemic rats

Figure 1 shows the effects of EAqPB and glibenclamide on the rats' hepatic glucose release. The glucose concentration of the control solution (S1) containing the liver of the control rats having received distilled water increases from 2 ± 0.02 g/l to 3.76 ± 0.10 g/l at the end of the experience, an 88% increase in the glucose levels. The glucose concentrations in S2 and S3 solutions, containing the livers of rats treated with EAqPB at the doses of 500 and 1000 mg/Kg B.W, increased to 3.63 ± 0.07 g/l and 3.24 ± 0.05 g/l respectively after sixty (60) minutes; representing an 81.5% and 62% increase respective in the glucose level in these solutions compared to their initial glucose concentration. In the solution (S4) containing the livers of rats treated with glibenclamide at a dose of 10 mg/Kg B.W, the glucose concentration after one hour is 2.90 ± 0.08 g/l, a 45% increase. The glucose concentration in solution S2 (3.63 ± 0.07 g/l) is substantially equal to solution S1 (3.76 ± 0.10 g/l). On the other hand, the glucose levels in the S3 and S4 solutions are significantly reduced ($p<0.0001$) compared to the glucose level in the control solution S1. At the end of the experiment, this reduction is 13.83% for S3 and 22.87% for S4, compared to the control solution S1.

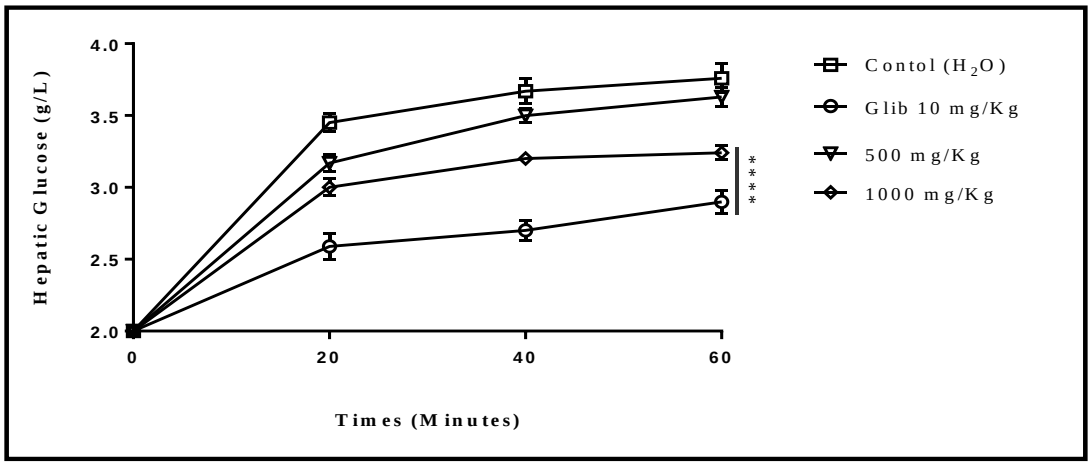

Figure 1: **Effects of the aqueous extract of *Parkia biglobosa* trunk bark (EAqPB) and glibenclamide on the release of hepatic glucose in normoglycemic rats.**
*The hepatic glucose concentrations are expressed as the mean followed by the Standard Error of the Mean (m ± SEM), n = 5, **** p < 0.0001 compared to the control.*

Effects of aqueous extract of trunk bark of *Parkia biglobosa* on blood glucose levels in diabetic rats

Figure 2 shows blood glucose changes of animals from different groups tests during the experiment. After the induction of experimental diabetes by streptozotocin (D0), a significant increase ($p<0.0001$) in blood glucose is observed in all the groups with different peaks compared to the non-diabetic controls. The peak for the diabetic control group increased from 81.75 mg/dl ± 2.180 to 254.8 mg/dl ± 3.683, an increase of 211.68 %; the value for the group treated with glibenclamide increased from 81.75 mg/dl ± 2.180 to 251.30 mg/dl ± 4.270, an increase of 207.4 %. The peaks observed in the groups treated with EAqPB at the doses of 500 and 1000 mg/Kg B.W go respectively from 81.75 mg/dl ± 2.180 to 241.5 mg/dl ± 4.9 and from 81.75 mg/dl ± 2,180 to 257.5 mg/dl ± 4.9; a 195.41% and 214.98% increase respectively. However, the glycemia in non-diabetic controls rats shows no significant variation ($P>0.05$). It is around 83 mg/dl ± 3.51 during experimentation.

One week after the treatment (D07), the glycemia of the diabetic controls remained significantly high ($p<0.0001$) compared to the one in non-diabetic controls until the end of the experiment, with a value of 339.8 mg/dl ± 10.05, which represent a 315% increase. On the other hand, when the diabetic rats are treated with glibenclamide and EAqPB at respective doses of 500 and 1000 mg/Kg B.W, a progressive and significant decrease ($p<0.0001$) in the glycemia is observed until the end of 28, 56 and 90 days of treatment compared to diabetic controls.

Thus, at the end of 28 days of treatment Figure 2 A, the blood glucose of the group treated with glibenclamide at a dose of 10 mg/Kg B.W went from 251.30 mg/dl ± 4.270 to 106.30 mg/dl ± 3.10, an 57.70% decrease. Regarding the groups treated with EAqPB at the doses of 500 and 1000 mg/Kg B.W, it went respectively from 241.5 mg/dl ± 4.9 to 120 mg/dl ± 3.46 and from 257.5 mg/dl ± 4.9 to 108 mg/dl ± 2.64, a 48.11% and 58.06% decrease respectively.

After 56 days of treatment Figure 2 B, the glycemia of the group treated with glibenclamide (10 mg/Kg B.W) decreased from 251.30 mg/dl ± 4.270 to 104.4 mg/dl ± 5.22, i.e. a 59.02% of reduction. In the groups treated with EAqPB at doses of 500 and 1000 mg/Kg B.W, the values went respectively from 241.5 mg/dl ± 4.9 to 121 mg/dl ± 4.52 and from 257.5 mg/dl ± 4.9 to 109.3 mg/dl ± 7.1, i.e. a 52.51% and 57.10% decrease respectively.

Finally, at the end of the 90 days of treatment Figure 2 C, the glycemia of the group treated with glibenclamide at a dose of 10 mg/Kg B.W went from 251.30 mg/dl ± 4.270 to 100.8 mg/dl ± 3, 89, representing a 60.43% reduction. Moreover, in the groups treated with EAqPB at doses of 500 and 1000 mg/Kg B.W, the glycemia decreased respectively from 241.5 mg/dl ± 4.9 to 118.7 mg/dl ± 3.45 and from 257.5 mg/dl ± 4.9 to 105 mg/dl ± 5.21, i.e. a 53.41% and 58.79% reduction respectively.

In addition, the daily administration of glibenclamide and EAqPB at doses of 500 and 1000 mg/Kg B.W, after 90 days of treatment resulted in a non-significant variation ($p> 0.05$) in the blood glucose levels of the rats treated in comparison to healthy controls.

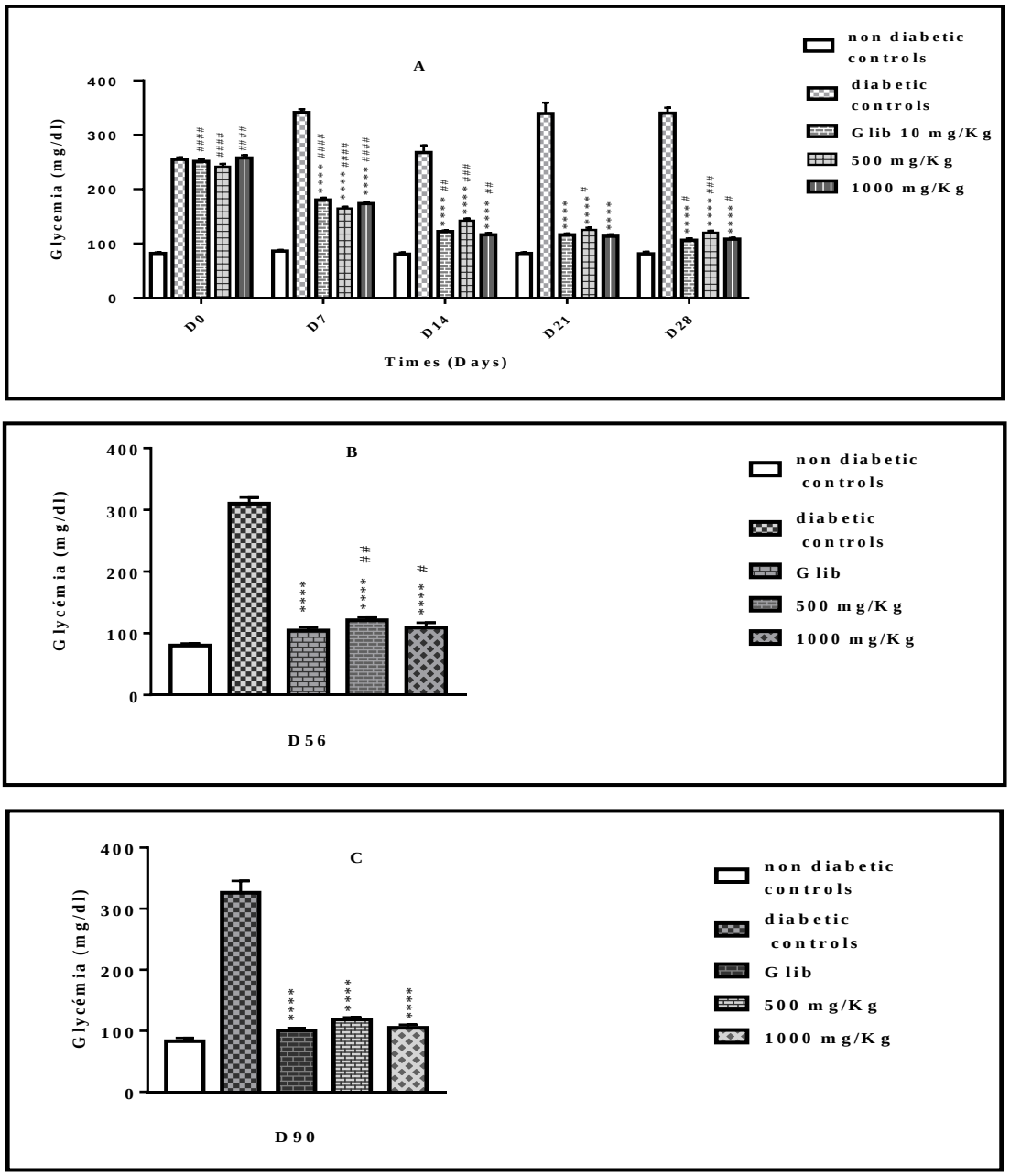

Figure 2: **Effects of aqueous extract of *Parkia biglobosa* trunk bark and glibenclamide on blood sugar levels in diabetic rats after 90 days of treatment.**

D28 (A): After four weeks of treatment of the rats
D56 (B): After eight weeks of treatment of the rats
*D90 (C): After thirteen weeks of treatment of the rats. Blood glucose values are expressed as the mean followed by the standard error of the mean (m ± SEM), n = 5, **** p < 0.0001, *** p < 0.001 compared to diabetic controls and # # # #p < 0.0001, # # # p < 0.001, # # p < 0.01, # p < 0.05 compared to non-diabetic controls.*

Effects of the aqueous extract of *Parkia biglobosa*'s trunk bark on hepatic glucose stored by the liver of diabetic rats

Figure 3 shows the effects of EAqPB and glibenclamide on rats' hepatic glucose storage. After 90 days of experimentation, the level of hepatic glucose stored in diabetic rats decreased significantly ($p<0.0001$) compared to the non-diabetic rats. This value is 0.19 ± 0.022 g/l for diabetic rats versus 0.51 ± 0.023 g/l for the non-diabetic ones, a 62% reduction. The various treatments with glibenclamide (10 mg/Kg B.W) and EAqPB (500 and 1000 mg/Kg B.W) resulted in a significant increase of the stored hepatic glucose levels in the diabetic rats treated compared to the untreated diabetic rats. The stored hepatic glucose level of diabetic rats treated with glibenclamide is 0.60 ± 0.024 g/l, a significant increase ($p<0.0001$) of 215%. Those values for diabetic rats treated with EAqPB at doses of 500 and 1000 mg/Kg B.W, are respectively 0.33 ± 0.027 g/l and 0.46 ± 0.027 g/l, a significant increase of 73.68% ($p<0.001$) and 142% ($p<0.0001$), respectively.

In addition, compared to healthy control rats, the stored hepatic glucose level of diabetic rats treated with glibenclamide (10 mg/Kg B.W) increased significantly ($p<0.05$) with a value of 0.60 ± 0 0.02 g/l versus 0.51 ± 0.01g/l for non-diabetic controls rats. On the other hand, this increase is not significant ($p>0.05$) when diabetic rats are treated with EAqPB at a dose of 1000 mg/Kg B.W with a value of 0.46 ± 0.01 g/L versus 0.51 ± 0.01 g/L for non-diabetic control rats. Furthermore, a significant decrease ($p<0.0001$) in this rate is observed when diabetic rats are treated with EAqPB at a dose of 500 mg/Kg B.W with a value of 0.33 ± 0.01 g/L against 0.51 ± 0.01 g/L for non-diabetic control rats.

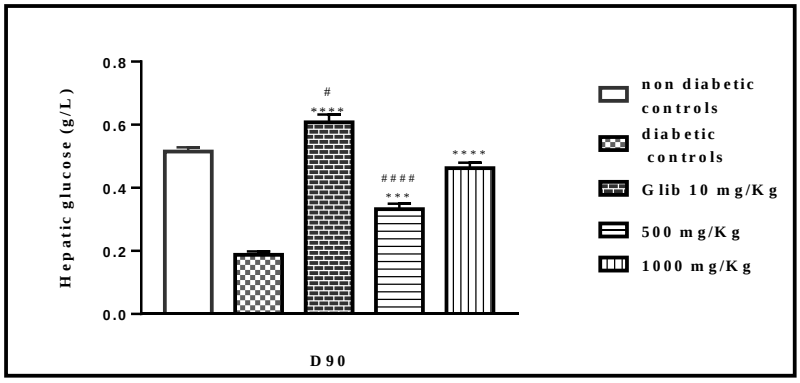

Figure 3: **Effects of the aqueous extract of *Parkia biglobosa* trunk bark (EAqPB) and glibenclamide on hepatic glucose storage in diabetic rats after 90 days.**
*The hepatic glucose concentrations are expressed as the mean followed by the standard error on the mean (m ± SEM), n = 5, **** p < 0.0001, *** p < 0.001 compared to the non-diabetic controls and # # # #p < 0.0001, # p < 0.05 compared to diabetic controls.*

DISCUSSION

Our results revealed that the glucose level released by the liver of normoglycemic control rats and normoglycemic rats treated with EAqPB or glibenclamide increases progressively, as a function of time. Treatments with EAqPB at doses of 500 and 1000 mg/Kg B.W lead to a decrease in the release of glucose by the liver compared to the control rats. This glucose release from the liver of rats treated with EAqPB at a dose of 1000 mg/Kg B.W is greater than the one observed with rats treated with EAqPB at the dose of 500 mg/Kg B.W and is similar to the one recorded for rats treated with glibenclamide at a dose of 10 mg/Kg B.W. These results are consistent with those from Takin *et al*.[15], Kahou Bi *et al*.[16] and Aka *et al*.[17] who respectively showed that aqueous extracts of *Khaya senegalensis* (Meliaceae), *Pseudarthria hookeri* (Fabaceae) and *Coffea canephora* (Rubiaceae) reduce the release of hepatic glucose in rats.

Indeed, Claude Bernard in[18], showed that under normal physiological conditions, the liver releases glucose to meet organism's physiological needs. The hepatic glucose production and release into the bloodstream are thought to be due to the hydrolysis of glycogen into glucose by the enzyme glycogen phosphorylase.[9]

The reduction of the hepatic glucose released in the presence of EAqPB may be due to the inhibition of the enzyme glycogen phosphorylase by the chemical compounds it contains.

The injection of streptozotocin to rats leads to an increase in blood sugar which goes from 81.75 mg/dl ± 2.180 to 254.8 mg/dl ± 3.683, a 211.68% increase and maintained, reflecting the onset of experimental diabetes due to the beta cells of the pancreas' necrosis. Indeed streptozotocin is an antibiotic antitumor isolated from the fermentation of a fungus, Streptomyces achromogenes. Administered parenterally (intravenously, intraperitoneally, subcutaneously), STZ specifically penetrates β cells *via* the GLUT 2 transporters (glucose transporter in the cell) and induces cell death by production of free radicals by DNA alkylation.[20]

During the 90 days of testing, the hyperglycemia persisted in untreated diabetic rats. Other hand, when diabetic rats are treated with EAqPB or glibenclamide, the hyperglycemia decreases significantly and the glycemia tends to return to normal. EAqPB's effects have already been observed by Kahou Bi *et al*.[16], Onsiyor *et al*.[21] and Nanti *et al*.[22] respectively with *Pseudarthria hookeri* (Fabaceae), *Ageratum conyzoïdes* (Asteraceae) and *Annona senegalensis* (Annonaceae) and *Hallea ledermannii* (Rubiaceae). Indeed, these authors have shown that extracts of these plants significantly reduced hyperglycemia in diabetic rats treated respectively after 28 days and 90 days.

Moreover, EAqPB effects on blood sugar are similar to glibenclamide. We can infer that EAqPB, just like the glibenclamide, has antidiabetic properties and could act by the same mechanism as this reference substance. Indeed, the glibenclamide administered on an empty stomach, stimulates insulin secretion, decreases glucagon secretion, inhibits hepatic glucose release and potentiates the effects of insulin in the liver.[23]

The stored hepatic glucose level is reduced by 62% in diabetic rats, compared to healthy control rats. The decrease in hepatic glucose storage observed in diabetic rats may be explained by an impaired insulin secretion after the injection of streptozotocin. Indeed, Basha and Sankaranarayanan[24] showed that STZ alters the insulin's secretion, which causes an inhibition of the glucokinase activity. After 90 days, the stored hepatic glucose level increases significantly in diabetic rats treated with EAqPB and with glibenclamide (10 mg/Kg B.W). However, compared to normal control rats, the stored hepatic glucose level in diabetic rats treated with at a dose of 1000 mg/Kg B.W is substantially identical, whereas it is higher with diabetic rats treated with glibenclamide (10 mg/Kg B.W).

Our results show that EAqPB promotes glucose's storage in the liver. They are similar to those obtained by Kahou Bi *et al*.[16] and Aka *et al*.[17] These authors have respectively shown that aqueous extracts of *Pseudarthria hookeri* (Fabaceae) and *Coffea canephora* (Rubiaceae) promote the storage of glucose in the liver. The effects of EAqPB on hepatic glucose storage are similar to the glibenclamide. Thereby, like the glibenclamide, EAqPB's mechanism of action relies on its direct action on Glut 2 receptors (glucose transporter in the cell) or stimulation of residual β cells of the pancreas to allow storage of the glucose.[25,26]

CONCLUSION

The study of the pharmacological effects of the aqueous extract of *Parkia biglobosa*'s trunk bark on the glycemia of rats has shown that this extract inhibits the release of hepatic glucose in normoglycemic rats and significantly reduces hyperglycemia in diabetic rats. In addition, EAqPB promotes the glucose' storage in the liver of diabetic rats. It emerges from this study that EAqPB has anti-diabetic properties which are similar to certain insulin secretors.

These results are therefore favorable to the use of this plant for the treatment of diabetes in traditional medicine and provide a scientific basis for its use.

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