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# Anti-inflammatory, anthropometric and lipomodulatory effects Dyglomera® (aqueous extract of *Dichrostachys glomerata*) in obese patients with metabolic syndrome

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**Running Title:** Anti metabolic syndrome effects of Dyglomera®

## ABSTRACT

**Background:** Increased visceral fat, dyslipidemia and increased markers of inflammation and coagulation are cardiovascular risk factors commonly encountered in obese people with metabolic syndrome. Previous studies have shown that ground *Dichrostachys glomerata* (DG), a spice used in Western Cameroon, can have beneficial effects on inflammation and various other cardiovascular disease risk factors. The purpose of the present study was to evaluate the effects of Dyglomera®, an aqueous extract of DG (standardized to NLT 10% polyphenols) on certain anthropometric, biochemical (including pro-inflammatory and pro-thrombotic states) and hemodynamic parameters in obese patients with metabolic syndrome.

**Methods:** The study was an 8-week randomized, double-blind, placebo-controlled trial involving 116 males and 202 females aged between 24 and 58 years. Participants were randomly divided into two groups: treatment and placebo. Capsules containing the active treatment (200 mg Dyglomera®) or placebo (200 mg maize powder) were administered 30–60 minutes before lunch and dinner throughout the study period. Various biochemical (namely, blood glucose, lipid profile, pro-inflammatory and pro-thrombotic markers), anthropometric and hemodynamic parameters were measured at baseline and after 4 and 8 weeks of treatment.

**Results:** At the end of the study, the Dyglomera® group showed statistically significant differences in all 16 parameters compared to baseline values. Changes in BMI and waist circumference were accompanied by changes in biochemical parameters, with the exception of adiponectin levels which were not correlated to waist circumference and PAI-1 values. The results confirm the hypothesis that Dyglomera®, the aqueous extract of DG, has anti-inflammatory properties, and is effective in reducing cardiovascular disease risk factors associated with metabolic syndrome in obese human subjects.

**Key words:** *Dichrostachys glomerata* extract, inflammation, obesity, metabolic syndrome

## 1. INTRODUCTION

Obesity and its related complications are generally referred to as metabolic syndrome (MetS) and its prevalence is increasing worldwide, with some countries experiencing as much as a three-fold increase in the last three decades [1]. Due to the fact that MetS is a multifactorial disorder involving genetic and environmental factors, its management remains a challenge to scientists worldwide. Even though obesity-related complications appear to be secondary to the condition itself, some of the accompanying changes are life-threatening, requiring a combination of management strategies for appropriate control. This is the case in type 2 diabetes mellitus, dyslipidemia, atherosclerosis, hypertension, oxidative stress, and inflammation. MetS comprises a clustering of atherosclerotic factors, including visceral obesity, dyslipidemia, and disturbed carbohydrate metabolism [2]. It is associated with pro-inflammatory and pro-thrombotic states, in which the role of increased visceral fat deposits is thought to be central. Abdominal obesity leads to alteration of the normal physiological balance of adipokines, insulin resistance, endothelial dysfunction and a pro-atherogenic state [3,4]. In association with this, the presence of conventional cardiovascular risk factors such as hypertension results in a significantly elevated cardiometabolic risk. The pathway leading to MetS involves the abnormal production of hormones and cytokines from the adipose tissue [3], namely, excessive production of IL-6, TNF-α and the prothrombotic agent plasminogen activator inhibitor type 1 (PAI-1). This is accompanied by low secretion of the protective adipocytokine adiponectin, a molecule that exerts anti-inflammatory, anti-atherogenic, and anti-diabetic effects, and whose production is down-regulated in obese individuals with metabolic syndrome [3,5]. On the other hand, overproduction of IL-6 by the adipose tissue in obesity induces hepatic (C-reactive protein) CRP synthesis, which promotes the onset of cardiovascular complications [6].

The management of disorders clustered in this syndrome and the elucidation of the mechanisms involved in its development are of great interest for the benefit of health. While lifestyle modification through dietary intervention and exercise have had limited success in treating these disorders, they have not stopped the increasing prevalence of MetS and may have low long-term sustainability. Pharmacological therapy has therefore been proposed as an adjunct to diet and lifestyle changes to improve long-term weight loss [7]. The numerous synthetic drugs developed to combat obesity and metabolic syndrome have had only marginal success, due in

part to their accompanying adverse effects [8,9]. This has turned the focus of some researchers towards nutraceuticals, compounds derived from foods or other natural products that are considered to be safer. Medicinal foods and herbal drugs are therefore widely prescribed, even when their biologically active compounds are unknown [10,11].

*Dichrostachys glomerata* (DG) (Forssk.). Chiov. is a deciduous tree found in Cameroon and other tropical countries. DG produces edible fruits and seeds, and the dried fruits are commonly used as a spice in a traditional soup of the Western provinces of Cameroon called "*Nah poh*"[12]. The hypotensive property of the plant was first reported more than four decades ago [13]. This plant has been shown to have antiviral, anti-infectious, [14] anti-inflammatory, and analgesic effects in rats [15] and cicatrizing effects [16]. A study from our laboratory showed that DG fruits also exhibit *in vitro* and *in vivo* antioxidant activity and can inhibit oxidation of low-density lipoproteins (LDL) [17]. A study of diabetic rats showed the ability of DG to reduce fasting blood glucose and glycosylated hemoglobin levels [18]. A recent study indicated that whole, ground DG had a positive effect on cardiovascular risk factors associated with obesity and type 2 diabetes [19]. While these reports suggest that DG can have beneficial effects on health, the bioactive components of DG have not been well characterized. Toward this end the current study was undertaken to evaluate the effects of an aqueous extract of standardized DG on MetS markers in obese humans.

## 2. METHODS AND MATERIALS

*2.1*. Test material: Dyglomera®, an aqueous extract of DG (standardized to NLT 10% polyphenols), was supplied by Gateway Health Alliances, Fairfield, California, USA. They were supplied as 200 mg capsules. Identical-looking placebo capsules were also manufactured containing 200 mg of maize-based powder.

### *2.2. Study population and intervention*

The study was a double-blind, placebo-controlled trial lasting 8 weeks. A total of 318 participants (202 females and 116 males) were randomly selected from a pool of 1,360 obese subjects previously recruited from the city of Yaounde and its environs. The participants included males and non-pregnant/non-lactating females aged 24–58 years, with a BMI between 30-40 $kg/m^2$. The NCEP ATP III criteria were used for the diagnosis of MetS, such that if any three of the following conditions were present in the same patient they were considered to have MetS: waist circumference > 102 cm in men and > 88 cm in women; triglycerides (TAG) ≥ 150 mg/dL; high-density lipoprotein (HDL) < 40 mg/dL in men and < 50 mg/dL in women; fasting glucose ≥ 110 mg/dL and blood pressure ≥ 130/85 mm Hg (or use of antihypertensive agents) [20]. A physician examined participants to ascertain their eligibility for inclusion in the study. Participants were randomly divided into two groups, and were instructed to take 200 mg of either Dyglomera® or placebo 30-60 min before lunch or dinner throughout the study period. They were asked to report any lapses in taking the pills. Since the capsules were identical in size, shape, and appearance, neither the researchers nor participants knew which treatment was given. Participants were encouraged to maintain their prior lifestyle and dietary habits throughout the study.

*2.3. Exclusion criteria*

Exclusion criteria included impaired kidney function, cardiac problems, serious hypertension (systolic and diastolic blood pressure above 180 mm Hg, and 110 mm Hg, respectively), need for daily insulin management, and enrollment in another clinical study within the past 6 months. Also excluded were volunteers with a history of drug or alcohol abuse, those on cholesterol-lowering, inflammation-reducing and/or other medications (e.g., steroids) that interfere with blood clotting and wound repair, as well as participants with infections including HIV/AIDS or cancer.

*2.4. Approval and informed consent*

The study was approved by the local ethical committee (Approval No. 006/CNE/MP/07). The purpose, nature, and potential risks of the study were explained to all participants, who gave their written informed consent before participation. The study was done in full accordance with the ethical provisions of the World Medical Association Declaration of Helsinki (as amended by the 52nd General Assembly, Edinburgh, Scotland, October 2000).

*2.5. Anthropometric measurements*

Various anthropometric parameters were measured at baseline and at biweekly follow-up visits for the 8 weeks of treatment. Height was measured with a Harpenden™ stadiometer (Cranlea & Company, Birmingham, UK), which measures height to the nearest 0.5 cm. Body weight and percentage body fat were assessed using a Tanita™ BC-418 Segmental Body Composition Analyzer/Scale (Arlington Heights, Illinois, USA) that uses bioelectrical impedance analysis to compute body composition. BMI was calculated as the ratio of weight (kg) to height squared ($m^2$). Waist (average of narrowest and the widest parts of the trunk) and hip (widest point) circumferences were measured to the nearest 0.1 cm. The participants were asked to fast for 12 hours and to wear light clothing for visits when measurements were taken. The participants were measured at approximately the same time of day and by the same technician across visits.

*2.6. Blood pressure*

As with the anthropometric measurements, blood pressure was recorded at baseline and at biweekly follow-up visits. Blood pressure was measured on the left arm after a 10-minute rest. Triplicate readings were taken over 5-minute intervals and the average was recorded.

*2.7. Sample collection*

At Week 4 and Week 8, blood samples (5 ml of blood) were collected after a 12-hour overnight fast. The plasma or serum obtained from each blood sample were split into multiple 500 µl aliquots and stored at -20 °C until needed.

*2.8. Analytical methods*

This study used the Trinder glucose activity test [21], which determines glucose in the blood using glucose oxidase with an alternative oxygen receptor. Plasma total cholesterol was assayed

by the cholesterol oxidase method [22], while triglycerides were assayed following the method described by Bucolo and David [23]. HDL cholesterol was determined using a heparin manganese precipitation of Apo B-containing lipoproteins [24], and LDL cholesterol was calculated using the Friedewald formula [25]. C-reactive protein was measured using an ELISA method (BioCheck™ hsC Reactive Protein ELISA kit, Foster City, CA USA). . Fasting insulin (mIU/l) was determined using the Medgenic immunoenzymetric assay by Biosource-Europe SA (Nivelles, Belgium). Insulin sensitivity was assessed by the homeostasis assessment model (HOMA-IR [mmol/L × mU/L] = (fasting glucose [mmol/L] × fasting insulin [mU/L]/22.5). Fasting serum adiponectin was measured using the Linco RIA, and PAI-1 concentrations were assessed by using an enzyme-linked immunosorbent assay (Diagnostica Stago, Asnières-sur-Seine, France).

*2.9. Statistical analysis*

The data were summarized (mean and standard error) for Week 0 (baseline), Week 4, and Week 8 (final), and the intra-group variation and the data were analyzed using SPSS 16.0 for Windows (SPSS Inc., Chicago, IL, USA). Because the repeated measurements on each participant were correlated in nature (covariance), a mixedmodel approach was used to characterize variation between patients and within patients because it is a flexible tool for analyzing repeated, longitudinal treatments. The interaction between time and intervention was tested at the 0.05 level of significance. If the interaction was significant, comparisons were made between the treatment and placebo groups for each month. Between-treatment changes were tested using one-way analysis of covariance (ANCOVA), adjusted for baseline values, with the initial value as a covariate. Bilateral correlations between variables were examined using Pearson's correlation coefficients. P values less than 0.05 were considered to be statistically significant.

## 3. RESULTS

*3.1. Baseline characteristics* A total of 297 individuals (154 in the Dyglomera® treatment group and 143 in the placebo group) out of 318 initial participants completed the study (21 participants dropped out of the study). The number of premature withdrawals was higher in the placebo group than in the Dyglomera® group (16 *vs* 5). The baseline (T0) anthropometric, hemodynamic, and metabolic characteristics of the two study groups are listed in Tables 1–4. Plasma TAG, total cholesterol, plasma glucose, insulin resistance (HOMA-IR), CRP, and circulating PAI-1 were slightly higher at T0, whereas HDL cholesterol and adiponectin were lower at T0 than normal values, which is reflective of the obese status of the patients in the study. The baseline characteristics across the two experimental groups were not significantly different.

*3.2. Anthropomorphic characteristics and blood pressure.* Table 1 shows the changes in the various anthropometric variables (body weight, BMI, waist and hip circumference, and percent body fat) over the 8-week trial period.

**Table 1.** Anthropometric effects of DG in obese subjects with metabolic syndrome

| Variable | Group | T0 | T4 | T8 | Change from baseline (T8 - T0) (%) |
|---|---|---|---|---|---|
| Weight (kg) | DG | 99.26± 1.12 | 93.73± 1.07[a] | 88.12± 1.05[b] | -11.15± 0.18 (-11.33)[†] |
| | Placebo | 98.69± 1.15 | 98.72± 1.12 | 98.17± 1.11 | -0.53± 0.11 (-0.49) |
| BMI (kg/m$^2$) | DG | 36.63± 0.25 | 34.59± 0.25[a] | 32.51±0.26[b] | -4.13± 0.06 (-11.33)[†] |
| | Placebo | 36.02± 0.26 | 36.04± 0.26 | 35.84± 0.26 | -0.18± 0.04 (-0.49) |
| Waist (cm) | DG | 106.20± 0.88 | 99.79± 0.98[b] | 95.48± 0.99[b] | -10.72± 0.28[†] (-10.23)[†] |
| | Placebo | 105.43± 1.00 | 105.49± 0.97 | 104.65± 0.97 | -0.77± 0.08 (-0.70) |
| Hip (cm) | DG | 127.37± 0.73 | 122.92± 0.75[a] | 118.34± 0.73[b] | -9.03± 0.22 (-7.10)[†] |
| | Placebo | 126.52± 0.82 | 126.34± 0.85 | 125.88± 0.84 | -0.64± 0.21 (-0.51) |
| Body fat (%) | DG | 44.88± 0.58 | 42.32± 0.56[a] | 40.15± 0.55[b] | -4.73± 0.11 (-10.68)[†] |
| | Placebo | 44.28± 0.61 | 44.37± 0.60 | 44.08± 0.59 | -0.20± 0.10 (-0.37) |

[a]$p < 0.05$ compared with placebo, adjusted for baseline
[b]$p < 0.001$ compared with placebo, adjusted for baseline
[†]$p < 0.05$ compared with baseline, intra-group analysis

Compared to the placebo group, the Dyglomera® treatment group showed a significant average weight reduction of 11.15 kg (-11.33% of total body weight) ($p < 0.001$) after 8 weeks of treatment with Dyglomera®. The amount of weight loss achieved in the current study using Dyglomera® was higher than that obtained with whole, ground DG in a previous study[19]. This reduction in weight was accompanied by a loss of visceral fat as measured by waist circumference (-10.23%). In general, changes to BMI, waist and hip circumferences, and body fat paralleled the loss in weight. Similarly, blood pressure also decreased significantly in the Dyglomera® treatment group compared with the placebo group ($p < 0.001$) (Table 2).

**Table 2.** Effects of DG on blood pressure in obese subjects with metabolic syndrome

| Variable | Group | T0 | T4 | T8 | Change from baseline (T8 - T0) (%) |
|---|---|---|---|---|---|
| SBP (mm Hg) | DG | 143.93± 0.48 | 122.51± 1.07[b] | 119.75± 0.92[b] | -24.18± 1.00 (-16.69)[†] |
| | Placebo | 142.60± 0.54 | 142.92± 0.83 | 144.29± 0.95 | 1.69±0.83 (+1.22) |
| DBP (mm Hg) | *DG* | 93.43± 0.52 | 85.62± 0.6[b] | 82.25± 0.69[b] | -11.18± 0.66 (-11.81)[†] |
| | Placebo | 93.29± 0.54 | 94.77± 0.61 | 95.27± 0.68 | 1.99± 0.47 (2.19) |

[a]$p < 0.05$ compared with placebo, adjusted for baseline
[b]$p < 0.001$ compared with placebo, adjusted for baseline
[†]$p < 0.05$ compared with baseline, intra-group analysis

*3.3. Blood parameters:* As shown in Table 3, there were significant variations in the lipid profile from baseline to Week 8 of Dyglomera® treatment in TAG, TC, HDL and LDL concentrations.

**Table 3.** Effects of DG on blood glucose and lipid parameters in obese subjects with metabolic syndrome

| Variable | Group | T0 | T4 | T8 | Change from baseline (T8 - T0) (%) |
|---|---|---|---|---|---|
| Glucose (mg/dL) | DG | 110.25± 0.48 | 85.82± 0.82[a] | 79.55± 0.51[b] | -30.70± 0.66 (-27.67) [†] |
| | Placebo | 108.99± 0.74 | 110.54± 0.68 | 111.75± 0.77 | 2.76± 0.59 (+2.72) |
| TAG (mg/dL) | DG | 151.92± 3.01 | 46.80± 2.48[a] | 44.01± 1.86[b] | -105.12± 3.54(-69.49) [†] |
| | Placebo | 150.79± 1.78 | 156.11± 3.04 | 151.77± 2.39 | 0.98± 2.91 (+4.33) |
| TC (mg/dL) | DG | 219.57± 1.82 | 137.84± 2.73[b] | 122.76± 3.36[b] | -96.82± 3.65 (-43.67) [†] |
| | Placebo | 216.47± 3.26 | 217.91± 2.98 | 216.35± 3.16 | -0.12± 1.88 (+0.63) |
| HDL (mg/dL) | DG | 37.68± 0.54 | 50.39± 1.58[a] | 58.23± 1.07[b] | 20.55± 0.84 (+55.52) [†] |
| | Placebo | 27.24± 0.99 | 19.87± 1.09 | 24.92± 1.13 | -2.32± 1.05 (+0.41) |
| LDL (mg/dL) | DG | 151.51± 1.96 | 78.09± 3.02[a] | 76.27± 3.44[b] | -75.23± 3.60 (-49.18) [†] |
| | Placebo | 159.07± 3.26 | 166.82± 3.12 | 161.07±3.39 | 2.00± 2.26 (+1.61) |

[a] $p < 0.05$ compared with placebo, adjusted for baseline
[b] $p < 0.001$ compared with placebo, adjusted for baseline
[†] $p < 0.05$ compared with baseline, intra-group analysis

These variations were significant compared with the placebo group ($p < 0.001$). There was also a significant reduction of blood glucose by 27.67% in the Dyglomera® group from baseline to Week 8 ($p < 0.001$) (Table 3). As shown in Table 4, the Dyglomera® group demonstrated significant reductions in insulin and insulin resistance (HOMA-IR) respectively by 10.38% and 35.11%, CRP by 17.16%, PAI-1 by 39.61 % in response to the weight loss during the Dyglomera® supplementation from baseline to Week 8 ($p < 0.001$). Inverse to the change in body weight, adiponectin levels increased by 21.86% in the Dyglomera® group from baseline to Week 8.

**Table 4.** Effects of DG on insulin resistance and circulating markers of inflammation and blood coagulation in obese subjects with metabolic syndrome

| Variable | Group | T0 | T4 | T8 | Change from baseline (T8 - T0) (%) |
|---|---|---|---|---|---|
| Insulin (mIU/L) | DG | 13.01± 0.15 | 12.27± 0.13[a] | 11.67± 0.15[a] | -1.34± 0.10 (-10.38) [†] |
| | Placebo | 13.80± 0.74 | 13.71± 0.17 | 13.66± 0.19 | -0.15± 0.11 (-1.05) |
| HOMA-IR (mmol/L×mU/L) | DG | 3.55± 0.04 | 2.60± 0.04[a] | 2.30± 0.03[a] | -1.25± 0.03 (-35.11) [†] |
| | Placebo | 3.71± 0.05 | 3.74± 0.05 | 3.77± 0.06 | 0.06± 0.04 (+1.65) |
| Adiponectin (µg/mL) | DG | 5.80± 0.11 | 6.51± 0.14 [a] | 7.08± 0.17[b] | 1.28± 0.10 (21.86) [†] |
| | Placebo | 5.48± 0.15 | 5.51± 0.19 | 5.54± 0.19 | 0.05± 0.11 (+0.40) |
| CRP (mg/L) | DG | 6.43± 0.14 | 5.80± 0.07[a] | 5.10± 0.11[b] | -1.32± 0.14 (-17.16) [†] |
| | Placebo | 6.12± 0.99 | 6.25± 0.21 | 6.07± 0.21 | -0.05± 0.14 (-1.73) |
| PAI-1 (ng/L) | DG | 32.58± 0.22 | 26.25± 0.34[a] | 19.81± 0.37[b] | -12.77± 0.26 (-39.61) [†] |
| | Placebo | 33.14± 0.26 | 32.39± 0.28 | 32.31±0.28 | -0.83± 0.09 (-2.52) |

[a] $p < 0.05$ compared with placebo, adjusted for baseline
[b] $p < 0.001$ compared with placebo, adjusted for baseline
[†] $p < 0.05$ compared with baseline, intra-group analysis

With regard to lipid profiles, this study confirms the strong hypolipidemic effects of Dyglomera® and suggests it could protect against MetS through reduced TC (mean = 96.82 mg/dL (-43.67%)), reduced LDL (mean = 75.23 mg/dL (-49.18%)), reduced TAG (mean = 105.12 mg/dL (-69.49%)) and increased HDL (mean = 20.55 mg/dL (+55.52%)).

*3.4. Correlation of select anthropometric changes with metabolic parameters and adipocytokines before and after weight loss.*

Pearson correlations between outcome variables are presented in Table 5 and they reveal that the change in BMI was significantly associated with changes in insulin, HDL, LDL, CRP, adiponectin, insulin and PAI-1 levels ($p < 0.01$). Similarly, waist circumference reduction was significantly associated with the all the above mentioned parameters except adiponectin. While there was no significant correlation between the adipocytokines adiponectin and PAI-1, increased HDL was positively correlated with adiponectin levels and was negatively correlated with other anthropometric and biochemical parameters.

**Table 5.** Correlation between BMI, waist circumference, cholesterol and circulating markers of inflammation and coagulation in obese subjects with metabolic syndrome

| | | **Correlations** | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Parameters | | BMI | Waist | HDL | LDL | CRP | Adiponectin | Insulin | PAI-1 |
| BMI | Pearson Correlation | 1 | 0.474** | -0.260** | 0.351** | 0.221** | -0.180** | 0.160** | 0.365** |
| | Sig. (2-tailed) | | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.001 | 0.000 |
| Waist | Pearson Correlation | 0.474** | 1 | -0.128** | 0.092* | 0.176** | -0.041 | 0.159** | 0.307** |
| | Sig. (2-tailed) | 0.000 | | 0.006 | 0.048 | 0.000 | 0.378 | 0.001 | 0.000 |
| HDL-C | Pearson Correlation | -0.260** | -0.128** | 1 | -0.475** | -0.188** | 0.184** | -0.135** | -0.402** |
| | Sig. (2-tailed) | 0.000 | 0.006 | | 0.000 | 0.000 | 0.000 | 0.004 | 0.000 |
| LDL-C | Pearson Correlation | 0.351** | 0.092* | -0.475** | 1 | 0.246** | -0.225** | 0.161** | 0.501** |
| | Sig. (2-tailed) | 0.000 | 0.048 | 0.000 | | 0.000 | 0.000 | 0.000 | 0.000 |
| CRP | Pearson Correlation | 0.221** | 0.176** | -0.188** | 0.246** | 1 | 0.308** | 0.552** | 0.495** |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.000 | 0.000 |
| Adiponectin | Pearson Correlation | -0.180** | -0.041 | 0.184** | -0.225** | 0.308** | 1 | 0.557** | 0.001 |
| | Sig. (2-tailed) | 0.000 | 0.378 | 0.000 | 0.000 | 0.000 | | 0.000 | 0.988 |
| Insulin | Pearson Correlation | 0.160** | 0.159** | -0.135** | 0.161** | 0.552** | 0.557** | 1 | 0.471** |
| | Sig. (2-tailed) | 0.001 | 0.001 | 0.004 | 0.000 | 0.000 | 0.000 | | 0.000 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| PAI-1 | Pearson Correlation | 0.365** | 0.307** | -0.402** | 0.501** | 0.495** | 0.001 | 0.471** | 1 |
| | Sig. (2-tailed) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.988 | 0.000 | |

** Correlation is significant at $p < 0.01$ (2-tailed).

* Correlation is significant at $p < 0.05$ (2-tailed)

## 4. DISCUSSION

The purpose of this study was to evaluate the effects of an extract of DG (Dyglomera®) on anthropometric parameters, blood lipids, and other variables in obese patients with MetS. Because obesity results in increased visceral fat deposits that promote insulin resistance and inflammation through alteration of adipokine secretion, even modest reductions to weight and waist circumference are associated with favorable changes in serum adipocytokines [6].

Compared to whole, ground powder [19], the extract (Dyglomera®) induced a greater reduction in weight (11.15 kg vs. 7.67kg), BMI (4.13 kg/m$^2$ vs. 3.00 kg/m$^2$), abdominal circumference (10.72 cm vs. 7.17 cm), body fat (4.43 % vs. 3.20 %), systolic blood pressure (24.8 mm Hg vs. 13.09 mm Hg), and fasting blood glucose (30.7 mg/dL vs. 28.91 mg/dL). Weight loss in overweight and obese individuals reduces mortality and morbidity, and is important in the treatment of obese patients. The current data suggest that weight loss *per se* leads to significant improvement in numerous cardiometabolic risk factors. In particular the Dyglomera®-treated subjects exhibited significant decreases in PAI-1, CRP, and fasting insulin levels. In addition, they showed a significant increase in adiponectin concentrations, an adipokine whose plasma concentrations correlate positively with insulin sensitivity, and levels of which are lower both in obese and type 2 diabetic patients than in lean, healthy individuals [26]. Hypoadiponectinemia is independently associated with the development of obesity-related MetS, as well as insulin-resistant diabetes and atherosclerosis [27], and this relationship is stronger than that of any other inflammatory marker [28]. Mechanisms of action of Dyglomera® may include antioxidant activity and inhibition of LDL oxidation [17] as well as modulation of blood glucose and HbA1c levels [19], all of which may have contributed to the effects conferred by Dyglomera® treatment in the present study.

Dyslipidemia is a component of MetS, which has an underlying cause of altered metabolism of triglyceride-rich lipoproteins such as VLDL and IDL remnants [29,30]. Elevated TAG levels are also very common in both MetS and the general population [31]. The use of Dyglomera® by the general population could therefore have a preventive effect on dyslipidemia and MetS. Reducing LDL levels is generally considered a key factor in the management of cardiovascular risk because LDL particles are the main carriers of circulating cholesterol and play a key role in cholesterol transfer and metabolism [32]. The guidelines in the Third Report of the National Cholesterol Education Program (Adult Treatment Panel III) (NCEP ATP III) focus on LDL levels as the primary target of cholesterol-lowering therapy [20]. Supplementation with Dyglomera® could therefore be a useful strategy to lower LDL levels.

In summary, Dyglomera® reduces the weight and improves the atherogenic risk factors associated with MetS after 8 weeks of treatment. The effects in the current study, which focused on the extract of DG (Dyglomera®), appears to be stronger than those observed in previous

studies using whole, ground DG, thus conferring a superior anti-atherogenic capacity on Dyglomera®.

**Competing interests**

The authors declare that they have no competing interests.

**Authors' Contributions**

Julius Oben conceived and coordinated the study, and prepared the manuscript. Ngondi Judith Laure co-designed and worked on the initial draft of the manuscript. Dieudonne Kuate carried out anthropometric measurements, analytical work, statistical analyses and prepared the draft of the manuscript. Blanche CO Etoundi carried out anthropometric measurements, analytical work and processed results. All authors read and approved the final manuscript.

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