

Evaluation of Cafeteria Diet in Experimental Animal with Plant Extract of *Calotropis procera* for Obesity Parameter

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ABSTRACT

The function of obesity in an insulin-resistant syndrome associated with hyper insulinemia, hypertension, hyperlipidemia, atherosclerotic diseased illness is vital. Hunger, body weight and lipid profile investigations thus assess *Calotropis procera* extract in animal models.

Adult Wister rats (180-240g) 8 were used in each experimental group. The impact of Cactus Kalahari on hunger, body weight and profile of lipids. (A) Control in weight, (B) obesity and CP+ obesity. Control obesity caused by material of cafeteria cuisine (CD). (CP was induced at (100 mg/kg/day po. for 50 days). Every 10 days, the food give, animal body weight, blood glucose, serum lipids level examined—serum and term tests for Liver Function and Renal Function Tests were checked.

Our research has demonstrated that following obesity induction CP pretreatment and administration at 100mg/kg/day p<0.05) have resulted in substantial reductions in food consumption, increased body weight and improved lipid profile, liver enzyme and kidney function tests. Cafeteria food rats also showed considerable growth in body weight gain, famine, lipid profile, hepatic enzymes, and kidney function tests.

When administered with a protein-rich food at the same time, the Kalahari extract prevented and reduced body weight gain and profile of lipid alterations in experimental induced obesity(fats) in rats.

Keywords- obesity, Herbal medication, hunger, Kalhari cactus.

I. INTRODUCTION

Obesity is the most common in India, nutritional diseases are not even worldwide. In 2019 it was estimated that 38.2 million children under 5 years of age were overweight or obese. In country like Africa, since 2000, the number of children under the age of 5 has increased by more than 24 percent. In 2019, about half of overweight or obese children under 5 lived in

Asia. Over 340 million children and young people aged 5 to 19 years were overweight and obese in 2016. The frequency of overweight and obesity has grown significantly among children and adolescents aged 5-19 years, from barely 4 percent in 1975 to 18 percent in 2016. Both boys and girls saw a comparable increase: girls of 18% and 19% of boys were overweight in 2016. approx 1% of those aged 5-19yr had obesity in year of 1975, over 124 million school student and adolescents

(data of 6% of female and 8% of male) were obese in 2019. Overweight and obesity are more deadly than underweight globally. More people throughout the world are fat than flaky –everywhere save Sub-Saharan Africa and Asia. For all genders and adults, BMI is the most useful population-level assessment of obesity and overweight. But rough recommendations should be considered because different people may not correlate with the same fatness. Age must be taken into account for young people when defining obesity and overweight.

Boys / Girls under the age of 5:

Overweight must be more than an average of two from the WHO Children's Growth Standards; Obesity must exceed three percent from the WHO Children's Growth Standards.

Overweight and obesity, together with its accompanying non-communicable diseases, are typically preventable. Sustainable settings and communities are vital to influence the people's choices, making healthier food and regular physical activity the easiest decision to take, thereby lowering overweight and obesity.

Individual responsibility can operate only when people have access to a healthy way of life. Therefore, it is essential to encourage individuals in society to follow the aforementioned principles by adopting sustainable evidence-based policies that enable everyone, especially the poorest, to exercise physically regularly and offer healthier dietary alternatives. An example of this strategy is a tax on sugar-sweetened beverages.

Hoodia Gordonii is a succulent leafless prickly tree renowned for its therapeutic properties in the family of traditional medicine, popularly called Bushmans hat. It is endemic to Botswana, South Africa and Namibia. Collectors recognised and threatened the species internationally when a marketing push incorrectly claimed it was a suppressor of weight loss. The flowers instead smell like rotten flesh and are mostly pollinated by flies. This plant is called the Namibian San indigenous peoples of the Namib Desert and all *Hoodia* species are referred to by the African word "GAAP."

II. MATERIAL AND METHODS

We search from different scholar sources, like Pubmed, NCBI, Scopus data base.

Procedure:

The *Calotropis procera* extract (CP) was created. The bark parts of a plant extracted using alcohol for the manufacture of CP provide a lyophilized solution that contains 25% of embryonic glycosides.

Design Experimental

Rats divided into three groups (N=8): (a) control untreated animals; (b) coffee diet control and (c) coffee feed and treatment of CP. In the untreated control group, Rat was supplied with pellet chow. And then coffee diet in the CA and [CA+CP rat] treatment groups. At three doses, Gavage provided CP with (i) 25, (ii) 50, (iii) 100 mg/kg/day for regular day 50.

Obesity Induced Diet (DIO)

The obesity caused by the supply of modified versions of Harris' fatty café (1993). Three varieties existed: chocolate + cocoa + pellets cheese + tallow + flax+ potatoes boiled + chow pellets. (3:2:4:1), and chocolate + cacao + chow pellet (3: 2: 4: 1). (4: 3: 3: 2: 3). The different versions are displayed over alternating days during the therapy period.

Anti Appetite Indirect Assay

Group (iii) Day One coffee shop and CP Diet. The weighting of feed clearance affected food consumption on a daily basis. By monitoring (a) a 50-day intake of food, (b) baseline, weekly and term of body weight of the animals, and (c) fat pad of liver on time, the CP suppressed the appetite.

Indicator Serum Obesity Analysis

Semi-automatic analyzer commercial kit for obesity serum indicators comprising cholesterol high-density lipoprotein (HDL) Triglyceride (TG). The suggested formula is computed with lipoprotein low density and lipoprotein extremely low density.

Serum Leptin Analysis

In the longer term, mild pentothal anaesthesia with heart puncture is used to draw the blood; the serum has been separated and stored until analysis with –20 livres. Leptin was also detected in the Commercial Rats using the Enzyme-Linked Immunosorbent Assay (ELISA) kit.

Analyze Data

All findings were presented as medium \pm SEM and evaluated using the statistic from the Brown System and post hoc comparisons from Games-Howell to assess the differences between groups using a one-way variance analysis (ANOVA). These options been chosen since the variance assumptions need not be homogeneous.

III. RESULTS

Behaviour Feeding:

Food habit occurs in examine animals for 2 month (50 days) (Fig 1). As, diet in CA-fed animals was substantially greater than in the supplied pellet-chew group ($p < 0.5$). Compared with both CA and pellet chow ($p < 0.5$) groups, concurrent treatment with CA of CP of 25, 50, and 100 mg/kg reduced considerably the intake of food. The grade and timing of the reduction in food intake varied on the dose. week 7 the end of examination ($p < 0.05$) of the 50 mg/kg/day, the lower dose (25 mg/day) anorexigenic effects were noticeable in 4th week and impact of the highest dose level (100 mg/kg/day) was apparent from the start of the third week.

Weight Of Body:

Animals in all groups gained body weight (Table 1). However, the weight increases in the CP-treated group significantly lower ($p < 0.001$) than in the obese group after 50 days.

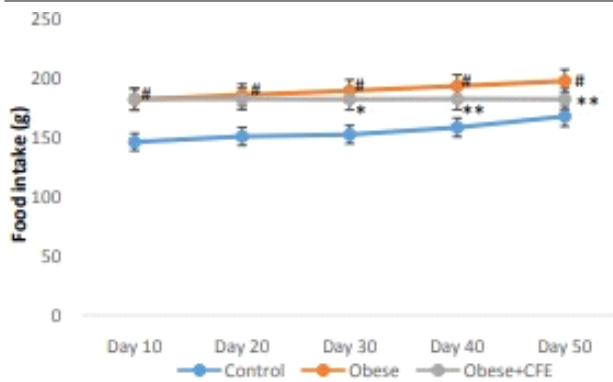


Figure 1: Feed Intake Duration Over 50

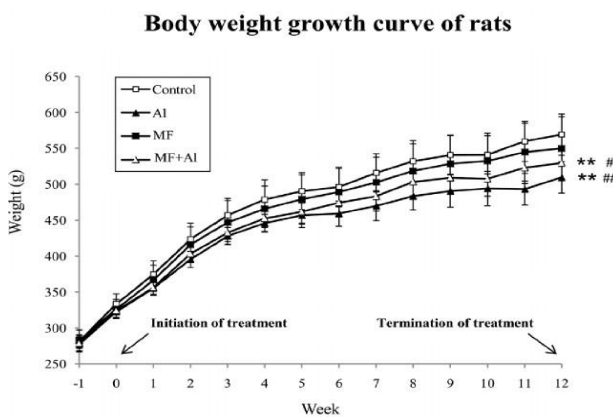


Figure 2: CP effect on body weight of rats in gram over 12 weeks

Table 1 indicates that administering a CA diet substantially increases the weight, compared with rats fed pellet chow, of ($P = .0001$) here weight growth of the animals given CA + CP was significantly less than in the CA group in all fat places, with the dosage of 50 & 100 mg/kg/day resulting near group of untreated animals. Fatty diet liver led to a 132.66% increase in the mass of the liver comparing to untreated rats ($P < .0001$). In the rats administered CP + CA, the dose-related increase in liver weight decreased. Dose at 25 mg/kg/day CP, the importance of liver was successfully reduced to 105.14%, the untreated control value. CP dose of 50 mg/kg/day, weight of the liver was lower than that of untreated control, at percentage of 78.57%, while the weight of CP of 100 mg/kg/day decreased to percentage 75.14%.

Serum Indicators Obesity

The cafeteria diet generated predicted changes in all the blood obesity indicators, generally favourably changed by CP, as seen in Table 2. shows a 130.80% increase in total cholesterol in the CA group compared to untreated controls ($P < .0001$) (74.76 ± 0.48 mg/dL). A

relatively low effect of co-administering CP by 25 mg/kg/day (115.28 percent; 87.15 ± 1.61 ; $P = 0.001$); an increase of 110.76 percent (82.88 ± 1.73 ; $P \geq 0.025$) at 50 mg/kg/day, not statistically different between group and control ($P = 0.797$), and an overall standardisation of cholesterol at 101.01 percent. (11.15 ± 76.35) In the CA group, 207.78 percent (150.9 ± 1.65) of serum triglycerides were greater than untreated checks ($P < 0.0001$). This was lowered to 161.59% (1161.25 ± 0.92 ; $P = 0.0001$), 149.25% (108.35 ± 1.51 ; $P = 1.0001$) and 132.06% (95.5 ± 0.75 ; $P = 0.0001$). Coadministered at 25, 50 and 100 mg/kg/day CP correspondingly. The LDL levels of the CA group were significantly higher in comparison with untreated controls ($P = 0.0001$). While CP was not changed statistically substantially in 25 mg/kg/day co-administration; CP was effectively averted in 50 ($P = 0.0001$) and CP 100 mg/kg/day ($P = 0.0001$). Compared to untreated controls, serum HDL levels in the CA group dropped ($P = .0001$). The HDL decline ($P = 0.001$) was reduced progressively by CP 50 mg/kg/day, whereas CP 100 mg/kg/day effectively prevented an HDL reduction ($P = 0.0001$). In contrast with untreated testing, VLDL levels were significantly elevated in CA-fed animals ($P = .0001$). This dosage dependent increase was considerably decreased by CP with coadministration of 25 ($P = 0.001$), 50 ($P = 0.0001$) and 100 mg/kg/day ($P = 0.0001$).

Serum Concentration

Table 2 further shows that blood leptin concentrations in the CA group have been significantly greater than in untreated rats ($P = .0001$). However, this increase ($P = 0.0001$), with CP simultaneous administration, was effectively prevented at all doses.

Level of Blood Glucose

($p < 0.05$) rise in blood sugar levels in both obese groups compared with untreated controls. In compared with an untreated obese group, CP therapy dramatically reduced blood glucose levels over the next 50 days ($P < 0.01$) (Fig. 4).

Liver Function Test

Concentrations of SGOT, GPT and ALP elevated in obese and CP treatment groups (< 0.001). The induce of CP ($p < 0.001$) is significantly reversed at the SGOT and ALP levels. However, there no significant difference in the quantity of SGPT in all groups (Table 3).

Renal Function Test

Serum creatinine and uric acid were dramatically raised for both treated and untreated CP obese rats, with blood urea levels decreasing markedly ($P < 0.05$) on day 50. CP treatment has reversed these changes (Table 4).

Table 1: Gain in Body Mass (Gram) in Different Examine Group Over 50 Day

GROUP N= 8	DAY 0 (G)	DAY 10 (G)	DAY20 (G)	DAY 30 (G)	DAY 40 (G)	DAY 50 (G)
CONTROL	208.25 ±4.56	212.13±5.86	220.85± 8.06	226.90±5.98	235.27±5.58	248.47±5.96
OBESE	318± 2.26*	325.98 ± 2.17	335.98±1.98	341.41± 1.98#	342.96±1.59#	351±1.61#
OBESE + CP	315± 1.70	321± 1.74	327±1.89	335.02± 1.85	331.98±1.82##	329±2.00##

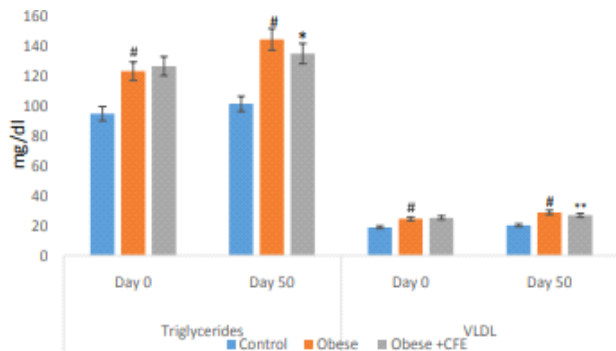


Fig. 2a: Total Cholesterol & LDL Level at Day 50.

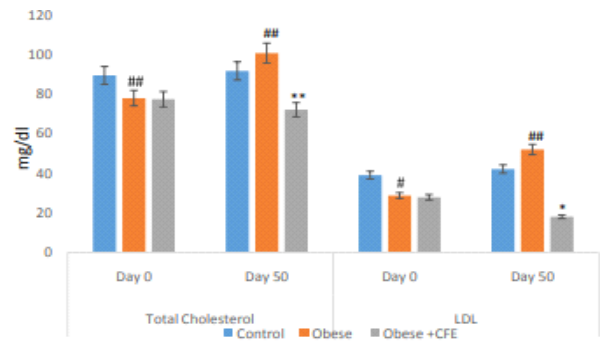


Fig. 2b: VLDL Levels and Triglycerides in Given Group to Day 0 to 50

Table 2: HDL Level at Day 50 Different Group

GROUP N=8 RATS	HDL (mg/dl)		
	Control	Obese	Obese + CP
DAY 0 of 50	29.4±0.29	23.5±0.60#	24.2±0.66
DAY 50 of 50	28± 0.35	18.9±0.58#	27±0.67

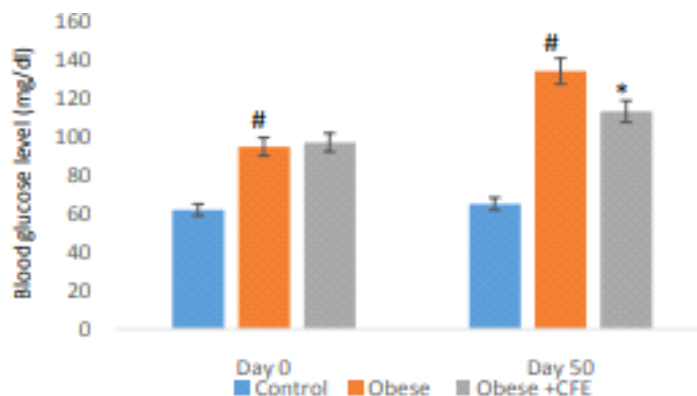


Fig. 3: Blood Glucose Level at Day 50 Different Group

Table 3: Liver Test to Experimental Groups

SHOT			SGPT			ALP	
GROUP N= RATS	8	DAY 0 OF 50	DAY 50 of 50	DAY050 of	DAY 50 of 50	DAY 0 of 50	DAY 50 of 50
CONTROL		22.3 ± 0.69	53.8 ±1.42	18.4 ± 0.50	51.4 ± 1.34	219± 5.58	306.7 ± 3.9
OBESE		51.96± 2.96*	94.6 ± 3.52	43.8 ±2.84	57.4± 1.91#	287± 8.56#	388± 1.6.96
OBESE CP	+	50.12± 3.70	75.3 ±3.74	44.2 ± 2.80	53.8 ± 2.85	278.8 ± 11.45##	349± 10.79#

Table 4: Experimental Groups Rena Test

SERUM CREATININE			BLOOD UREA TEST		URIC ACID TEST		
GROUP	N=	DAY 0 (SZ)	DAY 50(SZ)	DAY 0 (SZ)	DAY 50 (SZ)	DAY 0(SZ)	DAY (SZ)50
CONTROL	8	0.78± 0.02	0.73 ±0.03	49.70 ± 1.50	53.4 ± 1.00	3.09 ± 0.03	2.79 ± 0.06
OBESE		0.86± 0.02*	1.02 ± 0.04	43.8 ±2.84	35.4± 1.91#	3.39 ± 0.11#	3.96± 0.10
OBESE	+	0.88± 0.04	0.89 ± 0.02	44.2 ± 2.80	40.8 ± 2.20	3.50 ±	3.56 ± 0.10#
CP						0.11##	

IV. DISCUSSION

Cafeteria dietary obesity (DIO) is a common paradigm of obesity as high fat foods constantly encourage hyperphagia and raise body weight. The simulation of clinical obesity. Our findings show that CP has appetite suppression and anti-obesogenic effects in this scenario. These effects were seen after consuming the meal, body weight and serum lipid treatment with CP. It indicates that co-treating CP with the cafeteria diet reduces obesity in rats. Previous study has shown that co-determining CP with a coffee diet protects mice from getting obese. Two clinical studies have shown CP's anti-obesity efficacy. The precise mechanism of action of this impact is not completely known. CP-pregnancy glycosides can work in several ways. The decrease of food consumption can suggest a direct role in the control of hypothalamic appetite. In which glycosides are known to be active in pregnancy. Calotropis procera contains pregnancy glycosides that inhibit lyase citrate action. By inhibiting this enzyme, Calotropis procera may decrease fat synthesis. Calotropis procera also inhibits the malonyl coenzyme A enzyme. By blocking this enzyme, fat synthesis is further inhibited, and the body forces itself to spend its fat reserves. It can accelerate the pace of fat removal of the body. CP can otherwise lower the synthesis of ghrelin in the abdomen and subsequent neuropeptide Y in the brain by reducing hunger.

V. CONCLUSION

Calotropis procera reduced the development of body weight and lipid profile in experimentally induced obesity of the mouse. As demonstrated in preliminary clinical trials, obesity treatment can therefore benefit. However, additional study is confirm and its effects on obesity and to discover its unique mode of action.

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