

## Full Length Research Paper

## Impact of *Anthocleista vogelii* root bark ethanolic extract on weight reduction in high carbohydrate diet induced obesity in male wistar rats

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Obesity increases the risk of developing a number of diseases such as insulin resistance, type 2 diabetes, hypertension, hypercholesterolemia, stroke and heart attack. The aim of this study was to investigate the impact of ethanolic root bark extract of *Anthocleista vogelii* on weight reduction in high carbohydrate diet (HCD) induced obesity in male Wistar rats. Thirty male Wistar rats were housed in three steel cages containing 10 rats each. For the period of obesity induction, Group 1 was fed with normal pellet diet (NPD), while Groups 2 and 3 were fed with HCD for 14 weeks. During the 4 weeks treatment period, Group 1 was fed with NPD, Groups 2 and 3 were fed with HCD, and only Group 3 received 500 mg/kg b.w *A. vogelii* extract. The ethanolic root bark extract of *A. vogelii* significantly decreased ( $P<0.05$ ) food intake, body weight, total fat mass, adiposity index and low density lipoprotein cholesterol, but showed no significant difference ( $P<0.05$ ) in body mass index, total cholesterol, triglycerides, high density lipoprotein cholesterol, very low density lipoprotein cholesterol when compared with the HCD obese control. The results indicated that the ethanolic root bark extract of *Anthocleista vogelii* has potential to reduce weight in animals.

**Key words:** *Anthocleista vogelii*, obesity, body mass index, total fat mass, adiposity index, lipid profile, high carbohydrate diet.

### INTRODUCTION

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems (WHO, 2000). Obesity also increases the risk of developing a number of chronic

diseases including: insulin resistance, Type-2 diabetes, high blood pressure, hypercholesterolemia, stroke, heart attack, congestive heart failure, gallstones, gout and gouty arthritis, osteoarthritis, sleep apnea and mortality.

The change in the average weight of the population is

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**Abbreviations:** NPD, Normal pellet diet; HCD, high carbohydrate diet; BMI, body mass index; TC, total cholesterol; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; VDL-C, very low density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol.

occurring quickly, and within a few generations the bell-curve of human-weight distribution has shifted toward greater weight (Power and Schulkin, 2008). This suggests that aside genetic susceptibility, other factors like over-eating, high carbohydrate or fat diet, frequency of eating, slow metabolism, sedentary lifestyle and medications are playing important role in the increased prevalence of obesity.

Contemporary Western solid diets are frequently high in fats and sugars, and may be energy dense (Archer et al., 2007). High carbohydrate and fat diets are fast becoming the trend in developing countries like Nigeria. These solid dietary components are increasingly consumed in association with sweetened drinks, snacks and other liquid formulations. For example, evidence suggests that the consumption of sugar-sweetened soft drinks by children more than doubled between 1965 and 1996 (Cavadini et al., 2000). However, the role of carbohydrates in weight gain is not clear. For several decades obesity has been on the rise, although dietary fat has fallen and caloric intake for most pediatric groups, at least in the US, has been steady (Troiano et al., 2000). Simple carbo-hydrate intake may be to blame, particularly in children (Slyper, 2004), who regularly consume fruit juices and snacks like potato chips.

There are two main types of effective weight-loss medications: appetite suppressants (sibutramine) and lipase disruptors (Orlistat). As with almost all medications, weight-loss drugs have side effects, they are expensive, and are not safe for everyone. Traditional medicinal plants are often cheaper, locally available, and easily consumable (raw or as simple medicinal preparations) and have less adverse effect.

Some traditional healers have claimed that some medicinal plants in Nigeria like *Anthocleista vogelii* could be used to treat obesity. In Nigeria, *A. vogelii* is known by different names in different languages: 'Mpotó' (Igbo), apa oro, sapo (Yoruba) and kwari (Hausa). *A. vogelii* is a tree 6 to 20 m high, trunk 15–55 cm diameter, twigs with spines, leaves usually to 40 cm long, even to 150 cm, by 24 to 45 cm wide, inflorescence terminal with white sweet-scented flowers; of the closed-forest or mature regenerated jungle, usually in south west (Burkill, 1985).

Although, *A. vogelii* have not been scientifically proven to have anti-obesity potential, they have been found to be medicinally useful in other ailments. The seed and bark are used as purgative and antidote for snake bite. The bark and root are used in the healing of dropsy, swellings, oedema, gout and venereal diseases. The leaf-bud serves as antidotes for venomous stings, bites (Burkill, 1985). Alaribe et al. (2012) demonstrated the antiplasmodial effects of petroleum ether extract of leaf of *A. vogelii*. The *in vivo* antimalarial activity of ethanolic leaf and stem bark extracts of *Anthocleista djalonsensis* (another species) was reported by Anita et al. (2009). In a different study, Ateufack et al. (2010) reported that the aqueous and methanol extracts of the stem bark of *A. vogelii* possesses

spasmogenic activity on both ileal and stomach smooth muscle fragments. The aim of this study was to investigate the impact of ethanolic root bark extract of *A. vogelii* on weight reduction in high carbohydrate diet (HCD) induced obesity in male Wistar rats.

## MATERIALS AND METHODS

### Plant material

The fresh root bark of *A. vogelii* Planch was collected from a farm land located in Umuekwune community, Imo State, Nigeria. The authentication of the plants was done by a taxonomist at the Forestry Research Institute of Nigeria (FRIN), Ibadan, Oyo State. A voucher specimen of the plant was deposited in the Herbarium, FRIN and the Herbarium, University of Benin, Nigeria.

### Preparation of plant extract

The fresh root bark of the plant was washed, chopped into pieces and air-dried at room temperature. The dried plant part was milled into powder and weighed. 250 g of the plant powder was soaked in 500 ml of absolute ethanol separately in a container for 72 h with intermittent shaking. Then, it was filtered through Whatman No. 1 filter paper. The resulting filtrate was evaporated under reduced pressure using a rotary evaporator and there after freeze dried to get powder. The yield was stored in a refrigerator (4°C) till when needed.

### Phytochemical screening

The phytochemical analysis of the root bark of *A. vogelii* plant was carried out using standard methods of analysis according to methods of Treatise and Evans (1989) and Sofowora (1993).

### Acute toxicity (LD<sub>50</sub>) of plants

The acute toxicity of the ethanolic extract of *A. vogelii* was carried out as described by Shah et al. (1997) and Burger et al. (2005). Thirty five albino rats were divided into seven groups of five (5) rats each weighing between (180-200 g). The rats were subjected to 24 h fasting (with only water) before the extract was administered. The extract was suspended in distilled water and administered in doses of 200, 400, 800, 1600, 3200 and 6400 mg/kg body weight orally. The seventh group served as control and received only distilled water. The rats were observed for signs of toxicity and mortality for the first critical 4 h and then for each hour for the next 12 h, followed by 6 hourly intervals for the next 56 h giving a total of 72 h observations, thereafter daily for 7 days.

### Experimental animals

In this study, thirty male Wistar rats weighing 85 ± 5 g was used. The rats were bought from Anatomy Department, University of Benin and moved to the animal house of the Department of Biochemistry, University of Benin, Nigeria. All animals were housed in steel cages and each cage contained 10 rats. Rats were maintained under controlled temperature (±23°C) and a 12:12 h light/dark cycle. During the two weeks acclimatization period, the rats had free access to tap water and normal pellet diet (NPD) until they were assigned to individual groups. This work was carried out

in accordance with the guidelines of Faculty of Life Sciences at University of Benin for animal use.

### Composition of experimental diet

The normal pellet diet had 5% fat, 60 % carbohydrates and 30% protein, while the HCD contained 5% fat, 80% carbohydrates and 10% protein, the fibre, mineral and vitamin content were of the same quantity for both diets. The food/supplement used to compose the diet based on the different classes of food include: carbohydrate (processed cassava locally called *garr*), protein (dried *bonga* fish), fat (butter), fibre, vitamins and mineral mixture were obtained from a retail goods manufactured by SuperMax Nig. Ltd.

### Induction of obesity in the rats

The thirty rats were randomly assigned into three groups of ten. The first group fed on NPD, while obesity was induced in the second and third group by feeding on HCD for 14 weeks.

### Experimental design and animal grouping

After the period of obesity induction, Group 1 was fed the NPD, Groups 2 and 3 were fed with HCD, and only Group 3 received 500 mg/kg b.w *A. vogelii* as treatment for 4 weeks. The 500 mg/kg b.w dose of *A. vogelii* extract was reached from careful study of few works done by other researchers (Mbiantcha et al., 2013; Ogbonnia et al., 2011) and a pilot dose dependent study done in our laboratory. The *A. vogelii* extract was suspended in normal saline and then it was administered orally to the rats in Group 3, whereas the control groups (Groups 1 and 2) received normal saline for 4 weeks using a gavage tube. During the 4 weeks of treatment, all the rats had free access to their diets and water.

### Food intake and body weight measurement

The daily food intake of the rats was measured in the morning using a weighing balance. Food intake was calculated by subtracting the amount of food left over in each cage (that is, the refusal and spillage for the individual solid diets) from the measured amount of food provided at the previous day (gm/day/cage). The mean of food intake was represented in gm/day/group. The body weight of the rat was measured weekly in grams (g).

### Anthropometrical determinations

The body weight of the rat was measured weekly in grams (g). The body length (nose-to-anus length) was determined weekly in centimeter (cm) in all rats. The body weight and body length was used to determine the body mass index (BMI) as described by Noveli et al. (2007):

$$\text{Body mass index (BMI)} = \text{body weight (g)} / \text{length}^2 \text{ (cm}^2\text{)}$$

### Blood sample preparation

At the end of the experiment, rats were fasted for 12 to 14 h. Blood was collected by cardiac puncture from the rats at fasting state after being anesthetized with chloroform. The blood samples were collected in plain tubes, allowed to coagulate at room temperature and centrifuged at 3500 rpm for 15 min at room temperature for separation of serum. The clear, non-haemolysed supernatant was

separated using clean dry Pasteur pipette and stored at -20°C. Serum was used to assay for the lipid profile levels of the rats.

### Adipose tissue dissection and fat mass determination

After abdominal incision, five different white adipose depots (two subcutaneous and three intra-abdominal) and interscapular BAT were harvested from each rat. The WAT and BAT were dried on separate filter papers and weighed in grams (g). The total fat mass was the combined weight of the WAT and BAT.

### Adiposity index

Adipose tissue was isolated from the epididymal, visceral and retroperitoneal pad. These were dried on filter paper and weighed (g.) Adiposity index was determined by the sum of epididymal, visceral and retroperitoneal fat weights divided by body weight  $\times$  100, and expressed as adiposity percentage (Taylor and Phillips, 1996).

### Lipid profile assay

Serum was used to assay for the following parameters: total cholesterol (Trinder, 1969), triglycerides (Tietz, 1990), high density lipoprotein cholesterol (Tietz, 1976), very low density lipoprotein cholesterol (triglycerides/5) and low density lipoprotein cholesterol (Friedewald et al., 1972).

### Serum glucose

Serum was used to assay for glucose as described by Trinder (1969).

### Insulin ELISA assay

The quantitative measurement of insulin in serum was performed using a solid phase enzyme-linked immunosorbent assay (ELISA) based on the sandwich principle according to the manufacturer's instructions (DRG Insulin ELISA kit, DRG International, Inc. USA). The microtitre wells were coated with a monoclonal antibody directed towards a unique antigenic site on the insulin molecule. An aliquot of serum sample containing endogenous insulin was incubated in the coated well with enzyme conjugate, which is an anti-insulin antibody conjugated with biotin. After incubation, the unbound conjugate was washed off. During the second incubation step Streptavidin Peroxidase Enzyme Complex binds to the biotin-anti-insulin antibody. The amount of bound HRP complex was proportional to the concentration of insulin in the sample.

### Leptin (sandwich) ELISA assay

The quantitative measurement of leptin in serum was performed using a solid phase enzyme-linked immunosorbent assay (ELISA) based on the sandwich principle according to the manufacturer's instructions (DRG Leptin ELISA kit, DRG International, Inc. USA). The microtitre wells were coated with a monoclonal antibody directed towards a unique antigenic site on a leptin molecule. An aliquot of sample containing endogenous leptin was incubated in the coated well with a specific biotinylated monoclonal anti leptin antibody. A sandwich complex was formed. After incubation the unbound material was washed off and a Streptavidin Peroxidase Enzyme Complex was added for detection of the bound leptin.

**Table 1.** Food intake of rats during obesity treatment with *A. vogelii*.

Group	Food intake (g)				
	Week 14	Week 15	Week 16	Week 17	Week 18
Normal control	64.84 ± 2.55 <sup>a</sup>	60.54 ± 1.76 <sup>a</sup>	63.60 ± 1.71 <sup>b</sup>	68.19 ± 2.28 <sup>a</sup>	63.13 ± 2.11 <sup>b</sup>
HCD obese control	54.16 ± 2.99 <sup>b</sup>	50.67 ± 2.43 <sup>c</sup>	69.76 ± 3.07 <sup>a</sup>	70.54 ± 2.35 <sup>a</sup>	76.72 ± 1.64 <sup>a</sup>
HCD + <i>A. vogelii</i>	61.92 ± 3.24 <sup>a</sup>	55.33 ± 1.24 <sup>ab</sup>	63.37 ± 0.72 <sup>b</sup>	46.02 ± 1.31 <sup>b</sup>	40.21 ± 1.22 <sup>c</sup>

Values are expressed as means ± SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ).

**Table 2.** Body weight of rats during obesity treatment with *A. vogelii*.

Group	Body weight (g)				
	Week 14	Week 15	Week 16	Week 17	Week 18
Normal control	270.33 ± 6.39 <sup>b</sup>	274.00 ± 4.93 <sup>c</sup>	275.67 ± 8.09 <sup>b</sup>	290.33 ± 8.41 <sup>b</sup>	293.67 ± 9.17 <sup>e</sup>
HCD obese control	352.67 ± 9.21 <sup>a</sup>	368.33 ± 9.94 <sup>a</sup>	365.00 ± 11.02 <sup>a</sup>	389.00 ± 6.35 <sup>a</sup>	399.33 ± 7.06 <sup>a</sup>
HCD + <i>A. vogelii</i>	350.00 ± 3.51 <sup>a</sup>	360.33 ± 5.36 <sup>ab</sup>	359.33 ± 0.88 <sup>a</sup>	368.33 ± 4.37 <sup>a</sup>	368.33 ± 6.12 <sup>b</sup>

Values are expressed as means ± SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ).

**Table 3.** BMI of rats during obesity treatment with *A. vogelii*.

Group	BMI (g/cm <sup>2</sup> )				
	Week 14	Week 15	Week 16	Week 17	Week 18
Normal control	0.74 ± 0.05 <sup>b</sup>	0.71 ± 0.02 <sup>b</sup>	0.66 ± 0.01 <sup>b</sup>	0.66 ± 0.01 <sup>b</sup>	0.64 ± 0.00 <sup>c</sup>
HCD obese control	0.99 ± 0.03 <sup>a</sup>	0.96 ± 0.04 <sup>ab</sup>	0.87 ± 0.01 <sup>a</sup>	0.89 ± 0.01 <sup>a</sup>	0.86 ± 0.01 <sup>ab</sup>
HCD + <i>A. vogelii</i>	1.04 ± 0.03 <sup>a</sup>	0.99 ± 0.04 <sup>a</sup>	0.83 ± 0.01 <sup>a</sup>	0.84 ± 0.01 <sup>ab</sup>	0.84 ± 0.02 <sup>b</sup>

Values are expressed as means ± SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ).

Having added the substrate solution, the intensity of colour developed was proportional to the concentration of leptin in the sample.

### Statistical analysis

The experimental results were expressed as the mean ± S.E.M. Statistical significance of difference in parameters amongst groups was determined by One way ANOVA followed by Duncan's multiple range test.  $P < 0.05$  was considered to be significant.

## RESULTS

### Phytochemical screening and acute toxicity (LD<sub>50</sub>) of plant

The ethanolic root bark extract of *A. vogelii* contained alkaloid, saponin, tannin, steroid and cardiac glycosides. Acute toxicity study revealed general weakness and sluggishness as the major behavioral changes observed in the rats at 6400 mg/kg b. wt. oral dose. These behavioral changes disappeared after 1 h of observation.

No death was recorded at any of the doses administered. Oral LD<sub>50</sub> was therefore not determined because mortality was not observed.

### Obesity treatment period of the rats

The food intake of the HCD obese control was increased significantly as compared to the normal control at the 18<sup>th</sup> week. The food intake of the HCD treated group was significantly decreased as compared to the HCD obese control throughout the treatment period (Table 1).

The body weight of HCD obese control group maintained significantly increased body weight as compared to the normal control through out the treatment period. Again, the body weight of group treated with *A. vogelii* was significantly decreased as compared to the HCD obese control at the 18<sup>th</sup> week (Table 2).

The BMI of the HCD obese control group remained significantly increased as compared to the normal control at the end of the treatment. But there was no significant difference within the HCD obese and that treated with

**Table 4.** Fat mass of rats during obesity treatment with *A. vogelii*.

Group	BAT (g)	WAT (g)	Total fat mass (g)	Adiposity index
Normal control	0.93 ± 0.02 <sup>ab</sup>	19.65 ± 0.88 <sup>b</sup>	20.58 ± 0.50 <sup>c</sup>	3.29 ± 0.18 <sup>c</sup>
HCD obese control	0.93 ± 0.00 <sup>ab</sup>	28.50 ± 0.68 <sup>a</sup>	29.43 ± 0.68 <sup>a</sup>	4.63 ± 0.11 <sup>a</sup>
HCD + <i>A. vogelii</i>	1.03 ± 0.08 <sup>a</sup>	20.27 ± 0.74 <sup>b</sup>	21.30 ± 0.68 <sup>bc</sup>	2.78 ± 0.16 <sup>b</sup>

Values are expressed as means ± SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ). BAT= brown adipose tissue; WAT= white adipose tissue.

**Table 5.** Lipid profile of rats during obesity treatment with *A. vogelii*.

Group	TG (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	VLDL-C (mg/dl)	LDL-C (mg/dl)
Normal control	45.58 ± 0.91 <sup>c</sup>	43.78 ± 1.45 <sup>b</sup>	29.97 ± 1.70 <sup>ab</sup>	9.12 ± 0.11 <sup>c</sup>	4.68 ± 0.50 <sup>b</sup>
HCD obese control	83.57 ± 5.00 <sup>ab</sup>	54.50 ± 2.09 <sup>a</sup>	23.98 ± 1.51 <sup>e</sup>	16.71 ± 1.00 <sup>ab</sup>	13.82 ± 1.39 <sup>a</sup>
HCD + <i>A. vogelii</i>	91.47 ± 5.27 <sup>ab</sup>	51.60 ± 1.61 <sup>ab</sup>	25.78 ± 0.69 <sup>bc</sup>	18.29 ± 1.05 <sup>a</sup>	7.53 ± 1.0 <sup>b</sup>

Values are expressed as means ± SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ). TG = triglycerides, TC = total cholesterol, HDL-C = high density lipoprotein cholesterol, VLDL-C = very low density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol.

#### *A. vogelii* (Table 3).

The HCD obese control group had significantly greater total fat mass and adiposity index as compared to the normal control. The HCD treated group had significantly decreased total fat mass and adiposity index as compared to the HCD obese control. The HCD group treated with *A. vogelii* had the least total fat mass and adiposity index throughout the treatment period (Table 4).

As shown in Table 5, the HCD obese rats had significantly increased serum triglycerides, total cholesterol, VLDL cholesterol and LDL cholesterol, with a decrease in HDL cholesterol which was significant as compared to the normal control. Only the LDL cholesterol level of the HCD treated with *A. vogelii* was significantly decreased when compared with the HCD obese control.

The glucose, insulin and leptin level of the HCD obese control was significantly increased as compared to the normal control. With exception of insulin, the glucose and leptin levels of the HCD obese rats treated with *A. vogelii* showed significant decrease when compared with the HCD obese control.

## DISCUSSION

The ethanolic root bark extract of *A. vogelii* contained alkaloid, saponin, tannin, steroid and cardiac glycosides. The phytochemistry of all the parts of *A. vogelii* has been shown to have alkaloids; glycosides, saponins and steroids (Burkill, 1985). A synergistic relationship amongst phytochemicals has been adduced to be responsible for the overall beneficial effect derivable from plants (Liu, 2004). Saponins are capable of neutralizing some enzymes in the intestine that can become harmful,

building the immune system and promoting wound healing (Akinmoladun et al., 2007). Also, saponins also promote wound healing (Okwu and Okwu, 2004).

Acute toxicity study of the ethanolic root bark extract of *A. vogelii* revealed that the extract was not toxic to the rats at 6400 mg/kg b. wt. oral dose. Although, the rats exhibited some behavioral changes as stated above, these changes disappeared after 1 h of observation. In a different toxicity study on *A. vogelii*, no lethality was observed at 2000 mg/kg body weight i.p. in mice (Alaribe et al., 2012).

Obesity is a chronic metabolic disorder that occurs from the imbalance between energy intake and energy expenditure which is followed by increased body weight, enlarged fat mass and elevated lipid concentration in blood. Food intake and body weight are direct measures of obesity (Haslam and James, 2005). The food intake and body weight of the HCD obese rats increased significantly as compared to the normal control. Thus, it showed that the rats in the HCD obese group were obese. However, *A. vogelii* caused significant decrease in food intake and body weight in HCD induced obese rats during the treatment period. The reduction in the food intake in the HCD obese group treated with *A. vogelii* may be due to its ability to suppress the animals' appetite indicating action of bioactive components like saponin, and this was similar to the results reported by Chidrawar (2011).

The BMI of the HCD obese control remained significantly increased as compared to the normal control at the end of the treatment; this corroborates the work done by Novelli et al. (2007), which showed that high caloric diet increased significantly the BMI of rats as compared to the control (standard diet). Although there

**Table 6.** Blood glucose, insulin and leptin of rats treated with *A. vogelii* extracts.

Group	Glucose (mg/dl)	Insulin ( $\mu$ U/ml)	Leptin (ng/ml)
Normal control	65.00 $\pm$ 2.89 <sup>c</sup>	2.33 $\pm$ 0.03 <sup>b</sup>	2.63 $\pm$ 0.15 <sup>b</sup>
HCD obese control	89.67 $\pm$ 3.18 <sup>a</sup>	2.50 $\pm$ 0.06 <sup>a</sup>	2.86 $\pm$ 0.09 <sup>a</sup>
HCD + <i>A. vogelii</i>	79.33 $\pm$ 0.33 <sup>b</sup>	2.50 $\pm$ 0.06 <sup>a</sup>	2.30 $\pm$ 0.12 <sup>b</sup>

Values are expressed as means  $\pm$  SEM. Means in the same column not sharing common letter(s) are significantly different ( $p < 0.05$ ).

was a slight decrease in the BMI of the rats treated with *A. vogelii*, there was no significant difference between the HCD obese control and HCD obese rats treated with *A. vogelii* (Table 3).

Obesity is a condition of abnormal or excessive fat accumulation in adipose tissue. So, by measuring fat mass in the rats, we can directly correlate fat mass with obesity. In this study, total fat mass included: WAT and interscapular BAT of each rat. It was discovered that the HCD obese control group had significantly greater total fat mass as compared to the normal control. The *A. vogelii* extract significantly decreased the WAT and total fat mass of HCD obese rats.

Adiposity index was determined by the sum of epididymal, visceral and retroperitoneal fat weights divided by body weight  $\times$  100, and expressed as adiposity percentage (Taylor and Phillips, 1996). The HCD obese control group had significantly higher adiposity index as compared to the normal control rats, which was similar to the results found in other studies (Jeyakumar et al., 2009). The *A. vogelii* extract produced a significant decrease in the total fat mass and adiposity index as compared to the obese controls.

Some of the chemical constituents, such as saponins, flavonoids, and some triterpenoids, have been reported for their antiobesity effect in various plants (Yun, 2010). A wide variety of plants possess pancreatic lipase inhibitory effects, including *Panax japonicus* (Han et al., 2005), *Platy-codi radix* (Han et al., 2000), *Salacia reticulata* (Kishino et al., 2006), *Nelumbo nucifera* (Ono et al., 2006). These pancreatic lipase inhibitory phytochemicals include mainly saponins, polyphenols, flavonoids and caffeine (Kim and Kang, 2005; Moreno et al., 2006; Shimoda et al., 2006). Saponins are known bioactive substances that can reduce the uptake of cholesterol and glucose at the gut through intra-luminal physiochemical interaction (Price et al., 1987). Saponins as a class of natural products are also involved in complexation with cholesterol to form pores in cell membrane bilayers (Francis et al., 2002) as such may be used as anticholesterol agents or cholesterol lowering agent. Thus, it is suggestive that the *A. vogelii* root bark extract used in this present study, which contains saponins reduced the fat accumulation in the treated obese rats by inhibiting the activity of pancreatic lipase.

In the present study, there was a significant increase in

serum triglycerides, total cholesterol, VLDL cholesterol and LDL cholesterol, with a decrease in HDL cholesterol (which was not significant) of the HCD obese rats. Also, it is well known that excess sugar in the human diet can be converted both into glycerol and fatty acids and, thus, into lipids such as triglycerides (Murray et al., 2006). This explain why the triglyceride level and VLDL cholesterol in the HCD obese rats is significantly higher than the normal control. The *A. vogelii* extract had no significant difference in the TG, TC and VLDL as compared to the HCD obese rats.

The increased level of LDL cholesterol is a common feature of Obesity. LDL cholesterol is found to cause endothelial damage, oxidative stress and inflammation, which further aggravates obesity (Mistry et al., 2011). This necessitates the elevated LDL level in the HCD obese control. It has been observed that increase in lipid level particularly LDL-cholesterol is predictive for coronary events such as atherosclerosis and coronary heart disease (Blake et al., 2002). So, it is necessary to reduce LDL cholesterol level in obesity to treat as well as protect the disease from expansion (Shibano et al., 1992). The *A. vogelii* root bark extract had significantly decreased LDL cholesterol in the HCD obese treated group.

Serum glucose, insulin and leptin concentrations were significantly higher in HCD obese group as compared to the normal control. Leptin is a hormone produced mainly by adipocytes, and is involved in controlling body weight by increasing both satiety and energy expenditure (Tentolouris et al., 2008; Viguera-Villaseñor et al., 2011). Leptin levels are excessively high in obese people as a result of leptin resistance, which is associated with weight gain. In this present study, the HCD obese rats showed significant increase in leptin concentration. This result corroborates other studies that show high leptin levels in models of rodent DIO obesity (Ghanayem et al., 2010; Tentolouris et al., 2008; Fam et al., 2007).

The extract of *A. vogelii* significantly decreased glucose and leptin concentration in the treated rats, but there was no significant difference insulin concentration between the treated and HCD obese rats. Leptin concentration is related to the amount and distribution of body fat such that the higher the body weights, the higher the leptin concentration in human and rodents (Aizawa-Abe et al., 2000). This implies that decrease in body weight or body fat will bring about a corresponding decrease in leptin

concentration in the rats as seen in Group 3 of this study (Table 6). Also, this suggests that *A. vogelii* extract might have bioactive component(s) that decreased the accumulation of fats and as such reduce the body weight of HCD obese rats. *A. vogelii* extract caused decreased glucose level in the treated rats, this could be attributed to its ability to facilitate glucose entrance into cells and increase insulin sensitivity. *A. vogelii* might as well contain bioactive components that possess hypoglycemic effects.

## Conclusion

A number of medicinal plants, including crude extracts of these plants can induce body weight reduction and prevent diet-induced obesity. The ethanolic root bark extract of *A. vogelii* significantly decreased food intake, body weight, total fat mass, adiposity index and low density lipoprotein cholesterol. Thus the ethanolic root bark extract of *A. vogelii* possess some potential to reduce weight and therefore, might be explored in the treatment of obesity. However, there is need for further research on extensive identification of the active ingredients of the plant and the role they play in weight reduction.

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