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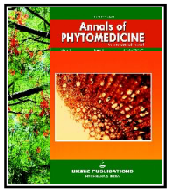

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Reckoning of antioxidant and antiobesity potential of *Anethum sowa* L. roots extract in experimental rodent model

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Abstract

Anethum sowa L. (Family: Umbelliferae) is a traditional herb, containing phenolic compounds and flavonoids having curative value. The total phenolics and flavonoid contents, antioxidant activity (*in vitro*), and antiobesity activity of the ethanolic extract of *Anethum sowa* roots (EASR) through high-fat diet induced antiobesity model in female SD rats were determined. The antioxidant activity was performed using standard procedure of 1,1-diphenyl-2picrylhydrazyl. Obesity was induced in female SD rats (*in vivo*) by feeding them with high-fat diet and ad *libitum*. The antiobesity results were estimated by oral administration of (EASR) at dosage levels of 100 and 200 mg/kg/ of body weight. During the study, physical parameters like body weight, food intake, food efficiency ratio, BMI, relative organ weight and lipid serum profile like total cholesterol (TC), total triglycerides (TG), low density lipoprotein (LDL), very low-density lipoprotein (VLDL), high-density lipoprotein (HDL), and atherogenic index (AI) were determined. Various oxidative stress markers such as superoxide dismutase (SOD), glutathione (GSH), glutathione peroxidase (GPx) and thiobarbituric acid reactive substances (TBARS) were also measured.

The present research exhibited that *A. sowa* roots exerted notable free radicals scavenging potentiality. Feeding high fat diet resulted in marked increase in the body weight, TC, TG, LDL and VLDL levels and a reduction in the HDL level. Treatment with EASR at dose 100 and 200 mg/kg/ body weight showed significant (P: *p<0.05 and **p<0.01) antiobesity effects. The present data exhibited that ethanolic extract of *A. sowa* roots exerted notable antiobesity potentiality.

1. Introduction

Obesity is one of the long-standing disorders with several diverse aetiology including genetics, environment, metabolism, lifestyle, and behavioural components. Factors such as proper nourishment, right eating habits and physical activity plays a significant role in controlling obesity (Wichtl, 1994). Obesity is abnormal accumulation of body fat which may have negative effect on health. Body Mass Index (BMI) is commonly used to determine a person's obesity. Not only the urban population of India is facing high rates of obesity due to their sedentary lifestyle, even the rural population is not resistant because of the use of modern technology in most of the farming occupations and less physical activity (Blumenthal *et al.*, 2000). WHO determined obesity as global epidemic. The latest study conducted in 2005 by WHO indicates that approximately 1.6 billion adults were overweight and about 400 million adults were obese (Giampiero *et al.*, 2002; Suresha, 2004). Several disorders are originated or made worse by obesity. These involve type-2 diabetes; hypertension; dyslipidaemia; ischemic heart disease; stroke; obstructive sleep apnoea; asthma; non-alcoholic steatohepatitis; gastroesophageal reflux disease; degenerative joint disease of the back, hips, knees, and feet; infertility and polycystic ovary syndrome; various malignancies; and depression (Cordatos *et al.*, 1992).

The Indian dill plant, *A. sowa* often known as dill is an annual odoriferous herb, belonging to family Umbelliferae (Singh and Panda, 2005). Dill plant has an extensive past of cultivation and utilized as culinary and therapeutic herb. The herb is cultivated throughout India, mainly in Punjab, Uttar Pradesh, Gujarat, Maharashtra, Assam and West Bengal (Randhawa *et al.*, 1995). *A. sowa* provides relief to digestive problems and stimulates milk to feeding mothers. It also possesses antimicrobial, antifungal, antioxidant, insecticidal, anti-inflammatory, antispasmodic, antidiabetic, anticancer and anti-hypercholesterolaemia, due to the existence of therapeutically active compounds (Chahal *et al.*, 2017). There are several chemically synthesized drugs are available in market for obese patients but they have significant adverse drug reactions and is not appropriate for old age patients. So, it is necessary to develop drugs which are safe for the treatment of obesity, and traditional systems of therapy provide the path for the same.

2. Materials and Methods

2.1 Collection and certification of plant

The roots of *A. sowa* were collected from our kitchen garden Paikramau, Lucknow in November 2018. The plant specimen was authenticated from "CSIR-National Botanical Research Institute" Lucknow with Voucher Specimen ref. No: NBRI/CIF/668/2018.

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2.2 Chemicals and drugs

Triglycerides (TG) kit, total cholesterol (TC) kit low-density lipoprotein (LDL) kit and high-density lipoprotein (HDL) kit were purchased from Agappe Diagnostics Ltd., Kerela, India. All the other chemicals used including the solvents, were of analytical grade procured from SD Fine Chemicals Limited and Fischer Scientific Ltd. The standard drug Orlistat has been approved by the US Food and Drug Administration (FDA) precisely for obesity (Kaur and Kulkarni, 2000). This drug was procured from Quantum In. 64 C, Khasra 133 Narayan Nagar, Virat Khand Rd, Kamta, Lucknow, Uttar Pradesh 226028.

2.3 Extract preparation

Roots were collected from our kitchen garden, thoroughly wash with tap water and shade dried at room temperature for 4 weeks to get a consistent weight and grinded to obtain coarse powder. 50 g of powdered root was extracted with 250 ml of ethanol using Soxhlet apparatus. The extraction was carried out until the solvent became clear. The filtrate was concentrated to dryness under reduced pressure at 40°C through rotary evaporator (Sachan *et al.*, 2011). The yield of ethanol extract was 3.7% w/w. To check the primary and secondary metabolites in the ethanolic extract of *A. sowa* roots (EASR) were performed by the qualitative preliminary phytochemical screening (Arif and Fareed, 2010; Modi *et al.*, 2018).

2.4 Animals

Thirty female Sprague Dawley (SD) rats (142-158 g) were obtained from CDRI (Lucknow, Uttar Pradesh) and maintained in standard laboratory conditions (temperature 20-25°C, humidity 50 ± 15% and 12 h each of dark and light periods). Rats were provided with commercial pellet diet and water *ad libitum*. Acclimatisation of rats with the laboratory environment was done for one week before the initiation of the experiment. Experimental procedures were carried out in accordance with the guidelines of Institutional Animal Ethics Committee (Approval Number: IU/IAEC/19/13).

2.5 Experimental schedule

After one week of acclimatisation, obesity was induced by randomly allotting the animals into normal and obese groups and fed with normal pellet diet (NPD) and high-fat diet (HFD) and *ad libitum*, respectively for one week (Srinivasan *et al.*, 2005). The composition of HFD (Harlan Teklad TD93075) in % kcal is contains carbohydrates (24.0%), protein (21.2%) and fat (54.8%) and component of each mentioned in Table 1.

The rats were divided in to 5 groups of 6 rats in each (n = 6) for study. In the diet induced antiobesity model, normal control group (I) was fed with normal pellet diet and received normal saline, group (II) was fed with high-fat diet, groups (III and IV) received the 100 and 200 mg/kg/day of ethanolic extract of *A. sowa* roots (EASR), and (group V) was treated with standard drug Orlistat (5 mg/kg). All groups except normal control group (I) was fed with high fat-diet for 10 days, then groups (III, IV and V) were treated test drugs for 28 days along with HFD, respectively. On 29th day, the rats were measured, weighed and sacrificed with diethyl ether for the evaluation of physical parameters, lipid serum profile and oxidative stress (Vijaya *et al.*, 2011).

Table 1: High fat diet (HFD) composition

Formula	g/kg	Representative components
Casein	289.0	Ground corn
DL-methionine	3.33	Dehulled soybean meal
Corn starch	207.3	Ground oats
Sucrose	90.5	Wheat middlings
Vegetable shortening, hydrogenated	274.1	Dehydrated alfalfa meal
Corn oil	16.0	Soybean oil
Cellulose	53.12	Corn gluten meal
Vitamin mix, teklad (40060)	13.33	Iodized salt
Mineral mix, AIN-76 (170915)	46.66	Brewers dried yeast
Calcium carbonate	6.66	Calcium carbonate

2.5.1 Evaluation of physical parameters

The body weight (g), food intake (g) and food efficiency ratio were documented once in a week for 28 days of each group (Ordonez *et al.*, 2006). Body Mass Index (BMI) of animals was calculated by measuring the weighed and length (Nasal to anal length) every week using electronic balance and ruler. To calculate BMI of rats following formula was used:

$$\frac{\text{Body weight (gm)}}{\text{Length}^2(\text{cm}^2)}$$

The relative organ weight of liver, kidneys, spleen, heart, lungs and brain to body was determined. The organs were deliberately dissected out and weighed. These organs were stored in 10% neutral buffered formalin. The relative organ to body weight was calculated using the following formula (Vani and Reddy, 2000).

Relative organ to body weight:

$$= \frac{\text{Actual weight of the organ (g)}}{\text{Body weight of an individual rat on day of sacrifice (g)}}$$

2.5.2 Assessment of lipid serum profile

On 29th day, the blood was collected by retro orbital puncture method using diethyl ether and serum was separated using centrifugation. Then, the biochemical parameters such as cholesterol, TG, HDL, LDL, VLDL and atherogenic index were determined.

$$\text{VLDL} - \text{C levels} \left(\frac{\text{mg}}{\text{dl}}\right) = \text{Triglyceride level}/5$$

$$\text{LDL} - \text{C level} \left(\frac{\text{mg}}{\text{dl}}\right) = \text{Total cholesterol} - (\text{HDL} - \text{C} + \text{VLDL} - \text{C})$$

$$\text{Atherogenic index} = (\text{TC} - \text{HDL} - \text{C}) / \text{HDL} - \text{C}$$

The levels of serum Very Low-Density Lipoprotein (VLDL) were calculated in accordance with a formula as described by Friedewald and Fredrickson (Friedewald *et al.*, 1972: Nwagha *et al.*, 2010).

2.5.3 Parameters for oxidative stress

2.5.3.1 Effects of EASR on superoxide dismutase (SOD)

The superoxide dismutase (SOD) activity was spectrophoto metrically determined by method developed by Marklund and Marklund (Kumar *et al.*, 2009). SOD activity was revealed based on its ability to impede superoxide-mediated non-acceptance. One unit of SOD activity was defined as the amount of enzyme that inhibited the oxidation of pyrogallol by 50% and was expressed as unit/g Hb and that from the tissue as unit/mg protein.

2.5.3.2 Effects of EASR on reduced glutathione (GSH)

The procedure is based on the depletion of 5, 5 di-thiobis (2-nitrobenzoic acid) (DTNB) with reduced glutathione (GSH) to give rise a yellow compound. The reduced chromogen is proportionate to GSH concentration and its absorbance can be estimated at 405 nm (Jaishree *et al.*, 2010).

2.5.3.3 Effects of EASR on glutathione peroxidase (GPx)

NADPH, *i.e.*, oxidation substrate was estimated at 340 nm. The activity of GPx was measured as quantity of NADPH oxidised per min per mg protein. The estimation of glutathione peroxidise activity was done by Mohandas *et al.* method (Janardanan *et al.*, 1984).

2.5.3.4 Effects of EASR on thiobarbituric acid reactive substances (TBARS)

The results were measured as nM of TBARS formed per min per mg. Absorbance was taken at 535 nm and the determination of lipid peroxidation was done according to the method of Iqbal *et al.* (2017).

2.5.4 *In vitro* antioxidant activity

2.5.4.1 Estimation of total phenolic content in EASR

Modified folin ciocalteu method was used to estimate the total phenolic content of the ethanolic extract of *A. sowa* roots (EASR) (Gezici *et al.*, 2020). In a 25 ml volumetric flask (1 ml) of extract (50, 100 and 200 mg/ml) was diluted in distilled water (9 ml) and added 1 ml of Folin Ciocalteu reagent with stirring. After 5 min, 3 ml sodium carbonate was added and made up the volume 25 ml. A reagent blank using distilled water was prepared and all samples were allowed to stand in dark for 1 h. Absorbance was measured at 765 nm using UV spectrophotometer. The equation based on the calibration curve: $y = 0.002\ x$, $R^2 = 0.994$, was used to calculate the total phenolic content, where x was the absorbance and y was the gallic acid equivalent (µg/g).

2.5.4.2 Estimation of total flavonoid content in EASR

Aluminium-chloride colourimetric assay method was used to determine the total flavonoidal content (Muhammad and Sheeba, 2011). In a 10 ml volumetric flask (1 ml) of extract (50, 100 and 200 mg/ml) was diluted in distilled water (4 ml) and mixed with 0.3 ml of 5% sodium nitrite and after 5 min, add 0.3 ml of 10% aluminium chloride added. At the 6th min add 2 ml 1 M-NaOH and made up the volume 10 ml with distilled water. Then, the mixtures were allowed to stand at room temperature for 30 min at room temperature and the absorbance was measured at 520 nm. Total flavonoid content was calculated using the equation based on the calibration curve: $y = 0.001\ xs$, $R^2 = 0.991$, where x was the absorbance and y was the rutin equivalent (µg/g).

2.5.4.3 Free radical scavenging activity of EASR by DPPH method

The DPPH assay is established on the quantification of the scavenging potentiality of an antioxidant using the stable 1,1-diphenyl-2-picrylhydrazyl (DPPH) free radical (Wali *et al.*, 2019). The colour of free radical DPPH is purple in ethyl alcohol and is reduced to the equivalent hydrazine, as it reacts with a hydrogen donor, which is yellow in colour. It is a discoloration assay, which is determined by the addition of the antioxidant to a DPPH solution in ethyl alcohol and the decrease in absorbance is established at 490 nm and calculated by using the following formula (Qadir *et al.*, 2020).

$$\% \text{ Inhibition} = [(Ax - Ay) / Ax \times 100]$$

where,

Ax - absorbance of the control (blank, without extract) and

Ay - absorbance in the presence of the extract.

3. Statistical analysis

The data was represented as mean ± SEM and ± S.D for six rats. One-way analysis of variance (ANOVA) followed by Dunnett's test was performed by using Graph Pad Prism 2.01 (Graph Pad Software Inc.). The data were expressed as the mean ± standard error of the means (SEM) and a value of $p<0.05$ was considered as statistically significant.

4. Results

4.1 Body weight and food intake

The body weight (g) of each group once were taken before starting of feeding with high-fat diet, then after 10 days of high-fat diet feed and final body weight after dosing of test drugs at 28 days (Table 2). Food intake (g), food efficiency ratio and BMI were calculated at 28 days and documented in (Table 4).

Table 2: Effect of *A. sowa* on body weight

Treatment groups (n = 6)	Dose (mg/kg p.o)	Initial body weight (g) (0 Day)	Body weight (g) (10 Day)	Final body weight (g) (28 Day)
Normal control-I	-ve Control	143.66 ± 3.39	145.46 ± 2.32	144.33 ± 3.65
High fat diet (HFD)-II	+ve Control	155.16 ± 4.86	215.13 ± 3.56##	284.50 ± 2.86
HFD + Orlistat-III	05	150.66 ± 8.13	208.34 ± 4.10##	162.50 ± 7.54**
HFD + EASR-IV	100	146.83 ± 2.38	211.33 ± 3.35##	186.66 ± 3.71*
HFD + EASR-V	200	157.33 ± 4.55	217.65 ± 2.74##	166.48 ± 4.83**

Each value is expressed in Mean ± SEM One-way ANOVA followed by Dunnett's test.
P: #$p<0.05$ and ##$p<0.01$compare to normal control group.
P: *$p<0.05$ and **$p<0.01$compare to (HFD) positive control group.

Table 3: Effect of *A. sowa* on food intake, food efficiency ratio and BMI

Treatment groups (n = 6)	Dose (mg/kg p.o)	Final weight gain (gm)	Final food intake (gm)	FER (weight gain/food intake)	BMI
Normal control-I	-ve Control	15.67 ± 2.45	9.51 ± 0.12	1.647739	0.69 ± 0.01
High fat diet (HFD)-II	+ve Control	129.34 ± 3.78##	17.21 ± 0.18	7.515398##	0.9 ± 0.016##
HFD + Orlistat-III	05	17.84 ± 2.76**	10.21 ± 0.15	1.747307**	0.67 ± 0.028**
HFD + EASR-IV	100	63.83 ± 2.58*	14.38 ± 0.11	4.438804*	0.77 ± 0.007*
HFD + EASR-V	200	29.15 ± 2.1**	11.23 ± 0.09	2.595726**	0.72 ± 0.023**

Each value is expressed in Mean ± SEM One-way ANOVA followed by Dunnett's test.
P: #$p<0.05$ and ##$p< 0.01$compare to normal control group.
P: *$p<0.05$ and **$p< 0.01$compare to (HFD) positive control group.

Table 4: Effect of *A. sowa* on relative organ weight

Treatment groups(n = 6)	Dose(mg/kg p.o)	Heart (g)	Liver (g)	Spleen (g)
Normal control -I	-ve Control	1.1 ± 0.034	7.96 ± 0.036	1.26 ± 0.025
High fat diet (HFD)-II	+ve Control	2.49 ± 0.117##	10.72 ± 0.056##	2.53 ± 0.078##
HFD + Orlistat-III	05	1.14 ± 0.015**	8.03 ± 0.069**	1.32 ± 0.025**
HFD + EASR-IV	100	2.18 ± 0.128*	10.19 ± 0.08*	2.04 ± 0.047*
HFD + EASR-V	200	1.84 ± 0.1 **	8.75 ± 0.141**	1.59 ± 0.03**

Each value is expressed in Mean ± SEM One-way ANOVA followed by Dunnett's test.
P: #$p<0.05$ and ##$p<0.01$ compare to normal control group.
P: *$p<0.05$ and **$p<0.01$ compare to (HFD) positive control group.

Table 5: Effect of *A. sowa* on lipid serum profile

Treatment groups (n = 6)	Dose (mg/kg p.o)	VLDL (mg/dl)	HDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	LDL (mg/dl)	AI
Normal control-I	–ve Control	19 ± 0.37	58.7 ± 0.87	127.97 ± 1.01	95.05 ± 1.9	50.26 ± 1.76	0.209 ± 0.009
High fat diet (HFD)-II	+ve Control	37.12 ± 0.25##	34.33 ± 0.49##	172.6 ± 0.73##	185.64 ± 1.29##	101.14 ± 0.63##	0.732 ± 0.004##
HFD + Orlistat-III	05	19.13 ± 0.4**	54.52 ± 0.61**	130.39 ± 1.09**	95.68 ± 2.03**	56.73 ± 0.34**	0.243 ± 0.009**
HFD + EASR-IV	100	25.37 ± 0.3*	41.96 ± 0.45*	153.92 ± 0.63*	126.87 ± 1.51*	86.61 ± 0.57*	0.480 ± 0.004*
HFD + EASR-V	200	19.78 ± 0.26**	42.48 ± 0.39**	136.13 ± 1.08**	98.95 ± 1.32**	67.86 ± 0.92**	0.309 ± 0.006**

Each value is expressed in Mean ± SEM One-way ANOVA followed by Dunnett's test.
P: #$p<0.05$ and ##$p< 0.01$ compare to normal control group.
P: *$p<0.05$ and **$p< 0.01$ compare to (HFD) positive control group.

4.2 Determination of relative organ weight

The weight of the liver, spleen and heart were increased when animals of group II, III, IV and V feed with high-fat diet for 10 days. The weight of liver, spleen and heart were normalized significantly ($p<0.05$ and $p< 0.01$) by the treatment with standard drugs Orlistat and both doses of test drug (EASR) as compared to (HFD) positive control group after 28 days (Table 4).

4.3 Assessment of lipid serum profile

Table 5 showed that the concentration of TC, TG, LDL and VLDL levels significantly increase and a reduction in the HDL by HFD fed in rats compared to the normal control group I. Treatment with standard drug 5 mg/kg Orlistat of group III and EASR 100 and 200 mg/kg of groups IV and V showed significant decrease ($p< 0.05$ and $**p< 0.01$) in TC, TG, LDL and VLDL levels and increases the concentration of HDL. The results also showed the serum AI significantly increased ($p<0.01$) in HFD induced obese animals when compare to normal control group and tested drugs showed significantly decrease the AI level.

4.4 Parameters for oxidative stress

Table 6 showed the various oxidative stress markers such as SOD, GSH, GPx decreases and TBARS were significantly increased in high-fat diet group II when compare with normal control group I. Treatment with standard drug 5 mg/kg Orlistat of group III and EASR 100 and 200 mg/kg of groups IV and V showed significant increase ($p< 0.05$ and $**p< 0.01$) level of SOD, GSH, GPx decreases and TBARS.

Table 6: Effect of *A. sowa* on lipid serum profile

Treatment groups (n = 6)	Dose (mg/kg p.o)	SOD (unit/mg tissue)	GSH (mg/g tissue)	GPx (nmol/min/mg protein)	TBARS (nmol of MDA formed/g tissue)
Normal control-I	-ve Control	40.11 ± 0.144	37.84 ± 0.485	13.25 ± 0.411	25.73 ± 0.398
High fat diet (HFD)-II	+ve Control	16.04 ± 0.221$^{\#\#}$	17.17 ± 0.499$^{\#\#}$	6.45 ± 0.247$^{\#\#}$	82.54 ± 0.396$^{\#\#}$
HFD + Orlistat-III	05	37.14 ± 0.095**	33.27 ± 0.661**	11.97 ± 0.359**	31.57 ± 0.551**
HFD + EASR-IV	100	23.12 ± 0.167*	21.02 ± 0.364*	7.96 ± 0.353*	63.55 ± 0.405*
HFD + EASR-V	200	31.92 ± 0.113*	26 ± 0.381**	11.18 ± 0.321**	37.14 ± 0.576**

Each value is expressed in Mean ± SEM One-way ANOVA followed by Dunnett's test.
P: $^{\#}p< 0.05$ and $^{\#\#}p< 0.01$compare to normal control group.
P: $*p< 0.05$ and $**p< 0.01$compare to (HFD) positive control group.

4.5 Total phenolic and flavonoidal content

The results of phenolic and flavonoids contents of the ethanol extract of *A. sowa* roots (EASR) are summarized in Table 7.

Table 7: Phenolic and flavonoidal content present in *A.sowa* roots

EASR	Phenolic content*	Flavonoids contents$^{\#}$
50	114.32 ± 1.053	270.23 ± 1.137
100	342.25 ± 1.062	435.53 ± 1.121
200	532.43 ± 1.026	609.43 ± 1.124

All values are expressed as mean ± SD (n = 3).

*µg of gallic acid equivalent per 50, 100 and 200 mg of extract.

$^{\#}$µg of rutin equivalent per 50, 100 and 200 mg of extract.

4.6 DPPH scavenging activity

The ethanol extract of *A. sowa* roots was compared with ascorbic acid for DPPH radical scavenging activity. The DPPH % inhibition activity of *A. sowa* roots extract was 43.94% at 100 µg/ml, respectively (Figure 1). It was observed that the % inhibition (at 15 min) with the IC_{50} values of the ethanol extract of *A. sowa* roots (EASR) was found to be 97.5 µg/ml compared to standard ascorbic acids 68.74 µg/ml.

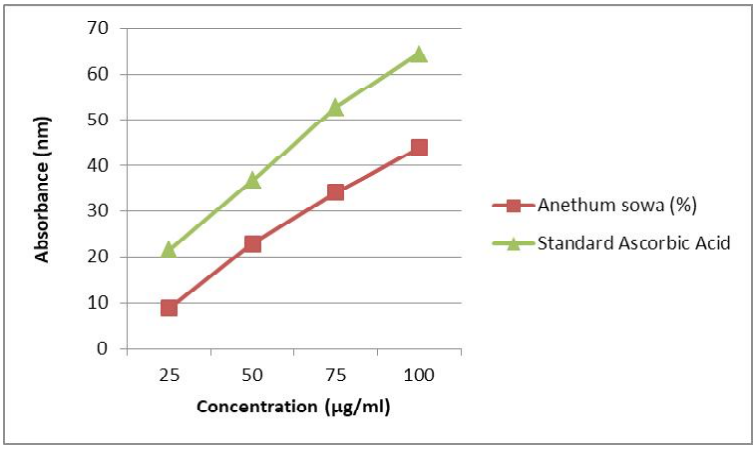

Figure 1: DPPH scavenging activity.

5. Discussion

Preliminary phytochemical screening of the ethanol extract of *A. sowa* roots (EASR) revealed the presence of reducing sugar, phenolics, flavonoids, terpenoids and steroids as major phytoconstituents. Hence, the presence of phenolics, flavonoids and terpenoids in EASR were responsible for the antioxidant and antiobesity potential of *A. sowa* roots. The HFD induced model comprises of high calorie value and high fat content, which was fed to rats to induce obesity (Kumar *et al.*, 2011). As per the various studies conducted rats fed with high-fat diet are exceptional model of obesity where dietary conditions can be compared with the dietary obesity that occurs in humans (Duca *et al.*, 2012; Bull, 1988). In the HFD model, the physical parameters such as body weight, food intake, food efficiency ratio, BMI and the biochemical parameters such as AI, TC, TG, HDL, LDL and VLDL levels were determined (Kwon *et al.*, 2003). On feeding, the rats with HFD diet the body weight escalated to a great extent due to the deposition of fat in the body and the drug, *A. sowa* showed its protective nature in rats by averting their weight gain. The weight of organs like liver was remarkably increased due to the deposition of fat all over the organ and the drug, *A. sowa* averted the increase in weights of the organs in rats. One of the predominant changes that occur in obese people is their biochemical parameters which are lipid serum profile. During obesity, the levels of TC, TG, LDL and VLDL elevates whereas HDL also known as good cholesterol decreases. Increase in the levels of TC, TG and LDL are crucial risk factors which are responsible the evolution of cardiovascular disorders such as hypertension, coronary heart disease, hyper lipidaemia, atherosclerosis, *etc*.

The increased level of TC and TG due to the feeding of HFD diet is transported to the tissues through blood. The rats getting the drug *A. sowa* did not have increased TC, TG, LDL and VLDL levels as compared to HFD fed rats. Also, the levels of oxidative stress markers such as SOD, GSH, GPx and TBARS in rats receiving the drug, *A. sowa* were significantly increased. The drug *A. sowa* protected the rats by intercepting the changes in physical parameters, lipid profile and oxidative stress markers. The free radical scavenging capacity of EASR was investigated by DPPH. DPPH activity of antioxidants is due to the donating capability of hydrogen. The results from the DPPH assay revealed that the EASR showed an efficient quenching of DPPH radicals, and thus contain free radical quenching compounds, which act as primary radical and, scavengers that react with DPPH (Kamal *et al.*, 2019).

6. Conclusion

The research concentrated on the estimation of *in vivo* antiobesity effects of ethanol extract of *A. sowa* roots (EASR) in high-fat diet induced obese female SD rats. The outcome stipulated that *A. sowa* revealed antiobesity effects through the depletion in body weight, food intake, food efficiency ratio, BMI and relative organ weights. Apart from that, a reduction in levels of atherogenic index (AI), serum triglycerides, total cholesterol, LDL-C, VLDL and an increase in HDL-C alongwith oxidative stress markers are evocative that *A. sowa* roots extracts have antiobesity potential.

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Author contribution

All the authors have accepted responsibility for the entire content of manuscript of this submitted manuscript and contributed equally.

Conflict of interest

The authors declare no conflicts of interest relevant to this article.

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