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Original article

Anti-obesity potential of almond (Prunus dulcis) in experimental animals under cafeteria and atherogenic diets

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ABSTRACT

Background & objectives: Natural dietary supplements are progressively getting famous to supplant synthetic substances particularly in chronic morbidities. The aim of this study was to evaluate the anti-obesity potential of almond on the normal, Cafeteria, and Atherogenic diets.

Materials and methods: Parameters such as change in body weight, body temperature, lipid profile, organ weights, and fat pad weights were assessed. Central Nervous System related studies (Despair Swim test and Elevated Plus maze test) were also performed to comprehend the effect of the diets, and almond on the brain. All of the experimental animals were randomly assigned to one of three diet categoriesregular, cafeteria, or atherogenic, and fed those diets for 40 days. Each diet had the control group, standard drug group and three almond groups (low dose: 50; medium dose: 100 and high dose: 200 mg/kg body weight). Body weight was recorded every alternate day. On 40th day, body temperature was measured. On day 41, lipid parameters, organ weights, fat pad weights and the CNS parameters were evaluated. ANOVA followed by Duncans Multiple Range Test were used for statistical analysis.

Results: Treatment of animals with either a low or high dose of almond as well as a standard herb prevented a rise in body weight significantly ($p = 0.01$) in all three diet groups. When a regular diet was replaced with a cafeteria and atherogenic diet, the serum levels of triglycerides and LDL increased significantly, while HDL levels decreased significantly. Overall, almond preparation reduced lipid parameters, organ weights, fat-pad weights, and stabilized CNS parameters substantially.

Interpretation & conclusion: The almond high dose was the most effective of all the almond preparations. Our study suggests that chronic administration of almond independently reduces the body weight in experimental animals.

1. Introduction

Obesity implies overabundance fat in the body. It isn't equivalent to being overweight, which means weighing excessively. An individual might be overweight from additional muscle, bone, or water, just as from having an excessive amount of fat. The two terms imply that an individual's weight is higher than what is thought to be healthy for his or her height (Garb et al., 2009; Spiegelman and Flier, 2001). Obesity is a spreading epidemic, and effective strategies are required to curb this condition. The rate and duration of treatment depend from patient to patient. Despite

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the fact that a decent measure of treatments exist, quiet consistence to them diminishes with time. This can prompt a backslide of the condition (MacLean et al., 2004). Nonstop family uphold, self-checking systems, and mental counselling is fundamental for these patients.

The present scenario of obesity is much direr than what was expected. Statistics from WHO show that over a million people have been affected with obesity. It has become the 5th leading risk for global deaths. A research done on Saudi Arabian population reported for 38.3% and 27.6% prevalence of overweight and obesity, respectively (Al-Qahtani, 2019). Recent advances in research have come out with therapies and drugs that effectively cope with the fast growing condition of obesity. Current treatment includes utilizing non-pharmacological, pharmacological, and surgery (Caterson, 2009). At times, combinational therapies are used to combat obesity. Despite the best efforts of the healthcare professionals, obesity seems to worsen with time. Mental resistance takes its toll with time in patients. Secondly, treatments are tedious and torrid. Hence with time, the patient compliance decreases. In most cases, relapse of the condition can occur (Heymsfield et al., 2007).

Natural dietary supplements have been considered promising contenders as weight loss and anti-obesity agents. One of the most potential and promising herbal remedy are dry fruits. Cashews, Fig, Almonds are showing positive influence on weight loss (Kris-Etherton et al., 1999). Almonds (*Prunus dulcis*, also known as *Prunus amygdalus*) have been linked to a variety of health benefits (de Souza et al., 2020). Its antioxidant activity is typically attributed to the presence of alpha-tocopherols and polyphenols, the latter of which has been linked to a lower risk of metabolic syndrome (via regulation of postprandial hyperglycemia and a reduction in the occurrence of diabetes mellitus type II) and a potential antimicrobial effect (Kamil and Chen, 2012). Almond polyphenols are primarily present at the lipid interface and, owing to their antioxidant and antimicrobial properties, help to extend the shelf life of whole almonds (Bolling et al. 2010). Almond is a rich source of vitamin E, calcium, phosphorous, iron, and magnesium. It likewise contains zinc, selenium, copper, and niacin. Almond contains some significant phytochemicals, for example, sphingolipid, beta-sitosterol (Barreca et al., 2020). Almond likewise contains a protein called Amandine. Researchers have studied the flavanol content of 16 different varieties, and 4 flavanol glycosides were obtained in almond seedcoats. As mentioned above, almonds in particular are especially rich in many tocopherols, including α-tocopherol, the most active form of vitamin E, which has also shown potent anti-atherogenic effects (Jenkins et al., 2002). Alpha-Tocopherol is known to diminish the measure of LDL in the body (Jenkins et al., 2002). This thus causes other lipid substances like TGL, and TC to diminish. The decrease of LDL is ascribed to the expansion in HDL levels. It is referred to go about as an insulin sensitizer (Jenkins et al., 2006). An overall beneficial effect is achieved while having almonds every day.

Since there is an immediate connection between the bringing down of lipid level and an abatement in weight. Despite the fact that there are reports of the hypolipidemic impact of almonds, the likely part of almonds on high-fat diet regimens is yet to be investigated. Consequently, this examination is an endeavor to investigate the part of almond as an anti-obesity regimen under Cafeteria and Atherogenic diets in animals.

2. Methods

2.1. Procurement of drug, chemicals and estimation kits

Almond was bought from the neighborhood food store. *Triglycerides*, Cholesterol kits, and Glucose pack was secured from Span Diagnostics, Bangalore. Chocolates, Biscuits, Condensed Milk, Bread, Potatoes, Coconut, and Cheese were bought from the nearby food stores.

2.2. Selection of animals

Laboratory bred albino Wistar rats of either sex weighing between 150 and 200 g were chosen for this investigation. They were housed six for each cage and under standard research facility conditions at room temperature 22 °C + 5 °C with 12 h day night cycle. The ethical board of the Krupanidhi College of Pharmacy endorsed experimental protocol (KCP/IAEC-45/2011-12).

2.3. Selection of dose

The dose for Almond was discovered via doing the Acute Toxicity studies according to the OPPTS rules 67. Test portion of 2 g/kg and 5 g/kg was given to albino mice (20–30 g). 1/tenth, 1/25th and 1/50th of the most extreme safe dose was chosen as low, medium and high dosages (50, 100 and 200 mg/kg body weight) respectively. For the standard medication (AyurSlim Capsules), no pre-clinical information was available. Henceforth the dose was changed over from recommended human dose to the suitable animal dose by means of the FDA dose calculator available on the US FDA website. Almond preparation was suspended in distilled water and administered orally twice daily for 40 days at a steady volume of 0.5 ml/100 g of body weight. A standard solution of 3 mg/ml of AyurSlim was prepared by dissolving the appropriate quantity in distilled water, followed by series of dilutions. The dose were injected per oral, twice a day.

2.4. Description of diet

As indicated by a report published by Rolls BJ et al, the accessibility of an assortment of food sources is a significant factor in the amount eaten in the meal and in the etiology of obesity (Rolls et al., 1983). At the point when animals are presented to various diets, their energy intake is expanded, which prompts animals in lipid profile, and consequently advancement of obesity. In this way, our study contains 3 separate diets.

2.4.1. Normal pellet chow

This is the staple eating regimen for rats. It is supplemented with water, and is allowed for the duration of the day. The first model comprises of this diet only.

2.4.2. Cafeteria diet

The cafeteria diet comprised of 3 eating regimens (consolidated milk, 40 g + bread, 40 g), (chocolate, 15 g + rolls, 30 g + dried coconut, 30 g), (cheddar, 40 g + bubbled potatoes, 50 g). The three regimens were introduced to group of 6 rats on day 1, 2 and 3 separately and repeated in same succession. This diet were provided in addition to normal pellet chow (Harris, 1993).

2.4.3. Atherogenic diet

The atherogenic diet comprises of cholesterol 1%, cholic acid 0.5%, and lard oil 5%. This eating regimen was blended altogether with the typical pellet chow diet given together (Jiao et al., 1991).

2.5. Grouping of animals

All animals used in the study are grouped into three major categories depending on the kind of diet they received, they were normal diet, Cafeteria Diet and Atherogenic Diet. Group I of each diet class was considered as control, group II were treated as standard group (3 ml/kg body weight, Ayurslim), while group III, IV and V,

respectively were given low (50 mg/kg body weight), medium (100 mg/kg body weight) and high dosages of almond (200 mg/kg body weight).

2.6. Experimental procedure

The body weight (g) was recorded on day 1 and afterward on alternate days for 40 days in each group. The body temperature was recorded on day 40 using rectal tele-thermometer before and after drug administration at 30, 60, 90, 120 and 180 min with a contact time of 1 min. Locomotor activity was recorded on day 40 utilizing open field behaviour test device and 30 min after almond administration to treated groups. The device comprised of a circular wooden field of 75 cm distance across wall with a height of 25 cm. Open field test was performed by setting the rats in the middle circle and recording the walking activity, the recurrence of raising and prepping for a 5 min trial.

On day 41 changes in glucose, total cholesterol, HDL and triglyceride levels, were measured from serum samples using the biochemical kits (glucose, total cholesterol and triglycerides) (Asdaq and Inamdar, 2010). The animals were sacrificed by cervical dislocation and afterward various organs (kidney, liver, heart, and spleen) and fat cushions (mesenteric, left and right perirenal and uterine fat cushions) were eliminated and weighed.

2.7. Statistical analysis:

The results were expressed as mean ± SEM. Comparisons between the treatment groups and control were performed by analysis of variance (ANOVA) followed by Duncan's multiple range test. In all tests the criterion for statistical significance was $P < 0.05$.

3. Results

3.1. Change in body weight

In all three diet categories, treatment of animals with either medium or high dose of almond as well as standard herb resulted significantly ($p < 0.01$) in preventing an increase in the body weight when compared to control (Table 1). The activity of high dose of almond is similar to standard as it puts a check on the increase in the body as effectively as standard therapy.

3.2. Biochemical parameters

3.2.1. HDL and triglycerides

Both cafeteria diet and atherogenic diet has caused a fall in the HDL level compared to normal diet. Significant ($p < 0.01$) increase in the HDL level was noticed in all treated groups, wherein, high dose of almond and standard group showed more elevation in HDL level in all three diet categories (Table 2). Overall, no significant difference between high dose of almond and standard groups was noticed in any of the diet categories. Replacement of normal diet with Cafeteria and atherogenic diet demonstrated remarkable increase in the serum triglycerides of experimental animals. However, when animals were treated with various doses of almond and standard herb during these high fat diets, significant decrease was noticed in serum TG level (Table 2).

Table 1
Percentage increase in body weight.

Groups	Normal Diet	Cafeteria Diet	Atherogenic Diet
Control	50 ± 1.22	79.83 ± 0.49	88 ± 0.52
SAH	37 ± 1.07*	62.66 ± 0.60*	73.16 ± 0.68*
ALD	46 ± 0.94^{O}	73.05 ± 0.80*^{O}	82.82 ± 0.54*^{O}
AMD	42.5 ± 0.51*^{O}	70.5 ± 0.45*^{O}	79.16 ± 0.86*^{O}
AHD	39.5 ± 1.14*	67.83 ± 0.89*^{O}	76.16 ± 1.03*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); All values are Mean ± SEM. *$p < 0.01$ when compared to control group. $^{O}p < 0.01$ when compared to standard anti-obesity herb group.

3.2.2. Total cholesterol and VLDL

In normal diet category, low dose of almond failed to produce significant ($p < 0.01$) alteration in the TC (total cholesterol) and VLDL level, whereas, medium and high dose of almond caused a significant decrease in TC level. Also, no significant effect of medium dose of almond was observed on serum VLDL level (Table 3). Both Cafeteria and atherogenic diets produced an increase in the serum TC and VLDL levels of experimental rats. Twice daily administration of almond doses produced significant ($p < 0.01$) dose dependent decrease in serum TC levels. However, serum VLDL levels were significantly decreased only with medium and high doses of almond in both these diet categories (Table 3).

3.2.3. LDL and glucose

As shown in table 4, serum LDL level was significantly ($p < 0.01$) decreased in normal diet category by long term (40 days) administration of either almond (dose dependently) or standard herb. Significant ($p < 0.01$) hypoglycemic effect of almond was also noticed in the normal diet groups. The administration of almond during high fats diets (Cafeteria and atherogenic) significantly ($p < 0.01$) prevented an incline in the serum LDL and glucose levels compared to non-treated group (control). The protection offered by high dose of almond was almost similar to standard group (Table 4).

3.3. CNS related parameters

3.3.1. Despair swim test

As evident from table 5, the immobility time was high in cafeteria diet groups that further get enhanced in animals kept on atherogenic diet. However, almond administration dose dependently decreased significantly ($p < 0.01$) the immobility period compared to the respective controls.

3.3.2. Elevated plus maze test

The time spend in the open arm is significantly ($p < 0.01$) increased by all doses of almond preparation in normal, cafeteria and atherogenic diet categories. On the contrary the spend by animals in the closed arms are decreased significantly ($p < 0.01$) by all treated groups. It is interesting to note that in addition to high dose, even medium dose almond administration produced almost similar level of potency when compared to standard anti-obesity herb (Table 5).

3.4. Fat weights

3.4.1. Kidney and uterine fat

Both kidney and uterine fat was significantly ($p < 0.01$) reduced by chronic therapy of almond and standard anti-obesity regimen in normal diet category of animals. In fact, medium and high doses of almond caused a significant ($p < 0.01$) decrease in the uterine fat compared to even standard therapy (Table 6).

Animals who received cafeteria and atherogenic fat had a higher contents of fat in both kidneys and uterine. The administration of almond during exposure to the high fat diets prevented an increase in the fat to a significant ($p < 0.01$) level compared to their respective controls (Table 6).

Table 2
Effect of almond and standard drug on HDL and triglyceride levels.

Groups	Normal Diet		Cafeteria Diet		Atherogenic Diet	
	HDL	TG	HDL	TG	HDL	TG
Control	55.2 ± 0.97	87.4 ± 1.65	37.76 ± 0.42	135.46 ± 1.30	35.96 ± 1.09	154.91 ± 1.08
SAH	77.53 ± 1.76*	74.6 ± 0.69*	69.56 ± 0.45*	113.63 ± 1.10*	61.31 ± 1.53*	123.16 ± 0.87*
ALD	66.15 ± 0.97*O	83.08 ± 0.26 O	53.03 ± 0.99* O	131.85 ± 1.46 O	48.21 ± 1.61* O	138.65 ± 0.76* O
AMD	69.05 ± 0.62*	80.48 ± 0.55 O	60.01 ± 1.43* O	125.2 ± 0.92* O	52.55 ± 1.04* O	134.18 ± 0.56* O
AHD	72.9 ± 0.97*	77.75 ± 1.65*	64.80 ± 0.86* O	119.05 ± 1.21*	59.01 ± 1.34*	129.1 ± 1.23*

ALDAlmond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); TG (triglycerides, mg/dl); HDL (high density lipoprotein, mg/dl); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

Table 3
Effect of almond and standard drug on total cholesterol and VLDL levels.

Groups	Normal Diet		Cafeteria Diet		Atherogenic Diet	
	TC	VLDL	TC	VLDL	TC	VLDL
Control	144.03 ± 0.85	17.03 ± 0.29	156.41 ± 0.64	27.09 ± 0.26	164.81 ± 0.68	30.98 ± 0.21
SAH	127.76 ± 1.91*	15.41 ± 0.17*	133.95 ± 1.26*	22.72 ± 0.22*	147.3 ± 1.67*	24.65 ± 0.16*
ALD	139.06 ± 0.50O	16.47 ± 0.10O	145.16 ± 0.85* O	26.37 ± 0.29 O	156.4 ± 0.76* O	28.72 ± 0.14 O
AMD	134.70 ± 1.77* O	16.07 ± 0.12	142.5 ± 0.59* O	25.04 ± 0.18* O	154.36 ± 1.32* O	26.83 ± 0.11* O
AHD	130.95 ± 0.66*	15.5 ± 0.33*	137.33 ± 1.06*	23.67 ± 0.26*	151.41 ± 0.78*	25.76 ± 0.28*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); TC (Total cholesterol, mg/dl); VLDL (very low density lipoprotein, mg/dl); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

Table 4
Effect of almond and standard drug on blood glucose and LDL levels.

Groups	Normal Diet		Cafeteria Diet		Atherogenic Diet	
	LDL	Glucose	LDL	Glucose	LDL	Glucose
Control	71.08 ± 1.33	94.1 ± 0.79	91.55 ± 0.43	121.83 ± 0.72	97.85 ± 1.17	109.83 ± 0.43
SAH	35.31 ± 2.79*	82.1 ± 1.32*	41.65 ± 1.18*	104.50 ± 1.12*	61.34 ± 2.27*	96.16 ± 0.43*
ALD	56.29 ± 1.19* O	89.0 ± 0.88* O	65.76 ± 1.50* O	114.16 ± 0.54* O	85.45 ± 1.18* O	105.83 ± 0.95* O
AMD	49.54 ± 2.28* O	87.0 ± 0.66* O	57.43 ± 1.82* O	111.33 ± 1.30* O	74.97 ± 0.98* O	102.33 ± 0.50* O
AHD	42.49 ± 1.60*	85.1 ± 1.23*	48.59 ± 1.55*	107.66 ± 0.99*	65.89 ± 1.88*	99.33 ± 0.60*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); LDL (low density lipoprotein, mg/dl); Glucose (mg/dl); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

Table 5
Effect of almond and standard drug on immobility time.

Groups	Normal Diet	Cafeteria Diet	Atherogenic Diet
Control	119.66 ± 1.46	125.16 ± 0.79	133.00 ± 0.97
SAH	89.33 ± 1.40*	98.5 ± 0.56*	112.50 ± 1.14*
ALD	100.6 ± 1.40* O	110 ± 1.14* O	122.83 ± 1.14* O
AMD	91.5 ± 0.99* O	103 ± 1.45* O	118.33 ± 0.90* O
AHD	87.00 ± 1.33*	97.5 ± 1.26*	110.83 ± 1.44*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); Immobility time (secs); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

3.4.2. Mesenteric fat

The mesentric fat was significantly ($p < 0.01$) decreased by almond treatment compared to both control and standard therapy (Table 7) in normal diet category of animals. Additionally, the pattern of response was similar in cafeteria diet category as well, whereas, in atherogenic diet category only high dose of almond was significantly ($p < 0.01$) more effective than standard anti-obesity herb (see Table 8).

3.5. Body temperature

The body temperature recordings of animals in normal, cafeteria and atherogenic diet categories were done (data not included). No significant change in the body temperature was noticed in any treated group compared to their respective controls. This shows that neither the standard drug nor almond chronic therapy alter body temperature of animals.

4. Discussion

It is a well-known fact that obesity does not develop overnight. So, it develops gradually over a period of time and considered as chronic condition. Additionally, herbal medications set aside a more extended effort to act when contrasted with the synthetic medicines. Subsequently we chose 40 days as a period limit for both the eating regimen and the medication treatment. Food prompts satiety. This thus causes increasingly more liking towards the food we love (Rolls et al., 1983). As we consume food, we are also consuming calories. Variety in diet is responsible for causing people to become obese.

Table 6
Effect of almond and standard drug in elevated plus maze test.

Groups	Normal Diet		Cafeteria Diet		Atherogenic Diet	
	Open arm	Closed Arm	Open arm	Closed Arm	Open arm	Closed Arm
Control	46.50 ± 1.61	253.5 ± 1.61	40.33 ± 0.76	259.66 ± 0.76	46.5 ± 0.50	253.50 ± 0.50
SAH	115.33 ± 1.44*	184.66 ± 1.44*	108.83 ± 0.98*	191.16 ± 0.98*	86.5 ± 1.00*	213.50 ± 1.00*
ALD	99.00 ± 1.39* O	202.00 ± 1.94* O	90.66 ± 0.80* O	209.33 ± 0.80* O	60.83 ± 1.22* O	239.16 ± 1.22* O
AMD	118.16 ± 0.86*	181.83 ± 0.86*	114.66 ± 0.80*	209.33 ± 0.80*	65.00 ± 0.88*	235.00 ± 0.88*
AHD	130.16 ± 1.21*	169.83 ± 1.21*	107.66 ± 0.80*	192.33 ± 0.80*	84.66 ± 2.11*	223.33 ± 2.11*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group. Open arm: Open Field behavior test-time spent in open arm in seconds; Closed arm: Elevated Plus Maze test- time spent in closed arm in seconds

Table 7
Effect of almond and standard drug on kidney and uterine fat.

Groups	Normal Diet		Cafeteria Diet		Atherogenic Diet	
	Kidney fat	Uterine fat	Kidney fat	Uterine fat	Kidney fat	Uterine fat
Control	1.58 ± 0.01	0.28 ± 0.01	2.48 ± 0.2	1.16 ± 0.3	3.01 ± 0.14	2.06 ± 0.08
SAH	1.25 ± 0.06*	0.24 ± 0.01*	1.13 ± 0.1*	0.51 ± 0.1*	2.4 ± 0.10*	1.38 ± 0.14*
ALD	1.32 ± 0.01* O	0.22 ± 0.01*	1.25 ± 0.06*O	1.15 ± 0.08 O	2.5 ± 0.09*	1.81 ± 0.14
AMD	1.24 ± 0.05*	0.20 ± 0.01* O	1.23 ± 0.06*O	0.95 ± 0.1 O	2.16 ± 0.13*	1.36 ± 0.09*
AHD	1.21 ± 0.01*	0.19 ± 0.01* O	1.14 ± 0.2*	0.46 ± 0.01*	2.03 ± 0.08*	1.25 ± 0.06*

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); Fat weight (g); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

Table 8
Effect of almond and standard drug on mesenteric fat.

Groups	Normal Diet	Cafeteria Diet	Atherogenic Diet
Control	0.83 ± 0.01	1.61 ± 0.3	3.05 ± 0.13
SAH	0.49 ± 0.01*	0.91 ± 0.1*	2.21 ± 0.07*
ALD	0.47 ± 0.01*	0.73 ± 0.04*	2.66 ± 0.06*
AMD	0.43 ± 0.01* O	0.53 ± 0.04* O	2.31 ± 0.09*
AHD	0.38 ± 0.01* O	0.47 ± 0.05* O	1.15 ± 0.14* O

ALD: Almond low dose (50 mg/kg body weight); AMD: Almond medium dose (100 mg/kg body weight); AHD: Almond high dose (200 mg/kg body weight); SAH: Standard anti-obesity herb (3 ml/kg body weight, Ayurslim); Fat weight (g); All values are Mean ± SEM. *p < 0.01 when compared to control group. O p < 0.01 when compared to standard anti-obesity herb group.

Cafeteria diet (Harris, 1993) includes a mixture of Cheese, condensed milk, biscuits, chocolate, and potatoes. Once consumed, the excess calories turn to fat. Although there is a considerable debate whether conversion of glucose to fat is a major factor responsible for increase in body weight, research suggests that it does contribute to the development of obesity. The cafeteria diet is basically a carbohydrate rich diet. When excess calories are consumed, two things happen. There is marked increase in glucose metabolism, and there is conversion of remaining glucose to glycogen. Glucose breakdown produces pyruvate and acetyl CoA. Acetyl CoA is used via 2 pathways. The first one is the citric acid cycle pathway, which involves the conversion of Acetyl CoA to produce ATP. This pathway is required for energy output. If a person is involved in strenuous activities, this pathway dominates. The second pathway is the lipogenesis pathway. In this pathway, the Acetyl CoA gets converted to fat. This pathway predominates when the need for energy output is low. For example, if a person sleeps for few hours after having lunch, the carbohydrates in the food are broken down, and the resulting Acetyl CoA is converted to fat. On chronic consumption of cafeteria diet, the fat formed is deposited in various parts of the body, leading to obesity. Atherogenic diet (Jiao et al., 1991) consists of fatty foods. Thus any fat food devoured is either assimilated and separated, or retained and kept in the body.

Body weight is a significant parameter of identifying obesity. Before the administration of diet and the treatment, the weight of the animals was recorded. On day 40, the weight was recorded again and the adjustment in body weight was estimated. The change in body weight is directly proportional to weight gain due to the diet. The animals spend most of their time in the cage. High intake of calories, coupled with less exercise causes the conversion of glucose to fat. There is an imbalance between energy input and energy output. The deposition of fat maybe crucial because it increases the circulating levels of lipid parameters such as Triglycerides and LDL. Such circulating levels of fats can lead to formation of atherosclerotic plaques, and block the blood supply to the major organs like brain and heart. The outcome could be unfortunate causing stroke and respiratory failure respectively. Almond reduced the rate of body weight gain. The mechanism which is responsible for the LDL-cholesterol reduction which is observed with almond consumption is likely to be associated with the nutrients which are provided by the almonds, i.e., decreased absorption of cholesterol and bile acid, increased bile acid and cholesterol excretion and an increased LDL-cholesterol receptor activity. The nutrients which are present in almonds regulate the enzymes which are involved in cholesterol synthesis and bile acid production (Berryman et al., 2011). Jenkins et al shown that almonds reduced the biomarkers of lipid per oxidation in hyper lipidaemic patients (Jenkins et al., 2008). The aqueous preparation of almond preserve most of the active phytochemicals responsible for cholesterol lowering properties and hence it is used in this study.

Estimation of Lipid parameters included LDL, VLDL, HDL, TGL, and Total Cholesterol (TC). Cholesterol was estimated utilizing the CHOD-PAP (Kaplan et al., 2003) end point technique. Serum cholesterol has been a pointer of inclination towards Coronary Heart diseases (Asdaq et al., 2009), Liver function, Biliary function, Intestinal absorption, Thyroid function and Adrenal disease. Cholesterol is transported through either the HDL, or the LDL. Almond high dose was powerful in diminishing the TC levels in animals in all diets. Almond medium dose was effective as well, however not as compelling as the Almond high portion. AyurSlim preparation especially decreased the TC levels in the rats. Almond

may owe its activity to tocopherol, which helps in decrease of LDL. LDL decrease causes indirect decrease of different other parameters such as TC.

Triglycerides are group of lipids delivered endogenously from carbohydrates, absorbed from the diet and are found in all plasma lipoproteins. Triglyceride estimation is a significant tool for the recognition of hyperlipidemia and consequently obesity. Elevated concentration is typically found in hypertriglyceridemia, ischemic heart illnesses, hyperlipoproteinimia type 1 and type 5, Diabetes Mellitus, acute pancreatitis, glycogen storage disease and Tangier sickness. Almond preparations decreased triglyceride levels in all diets. When compared to control, only almond high dose and standard anti-obesity herb showed significant reduction in triglyceride levels in the normal diet. Almond owes its activity due to a probable mechanism that it prevents lipid absorption from the GI tract. Almond also causes increase in transportation of TGL from the tissues to the liver for metabolism. This causes increase in the HDL levels, and decreases LDL and TGL levels in the body.

The exchange of cholesterol from liver to cells and the other way around is completed by lipoproteins. LDL does the transportation of cholesterol from liver to different parts of the body for deposition. Despite what might be expected, HDL completes the transportation of cholesterol from tissues to liver for its catabolism. In this manner, assessment of HDL and LDL is a significant parameter for assurance of transport of lipids. There was a reduction in LDL levels in all groups, and Almond high dose was very effective in ameliorating the LDL levels. A possible mechanism is that it could enhance the breakdown of fat in the liver. A study performed on Almonds suggests that almond has high content on monounsaturated fatty acids (MUFA). Almond tends to replace the carbohydrate with the MUFA (Jiao et al., 1991). This has been attributed to a reduction in LDL cholesterol levels in the animals. The standard drug however markedly reduced the LDL levels. Our values of LDL are akin to that of the research carried out by Jenkins et al (2006).

In the present study, almond preparations increased the concentration of HDL in serum. In all diets, almond preparation and the standard anti-obesity herb significantly increased the level of HDL when compared to control group. This shows that a large quantity of cholesterol is being transported to the liver for catabolism. When compared to the standard group, almond low dose, medium dose and high dose showed significant activity. This states that the standard drug markedly increased the HDL levels in the animals.

Glucose that isn't utilized is either put away as glycogen, or is changed over to fat. Subsequently it is changed over to fatty substance. Albeit this is certifiably not a critical factor contributing to obesity, it is regardless significant for controlling the circulating triglycerides levels, and thus diminishing degree of fats in the serum. Additionally, in obese patients, glucose is a contributing component for diabetes. Subsequently it is basic that ideal degrees of glucose be kept up in the body. Estimation of glucose was performed using glucometer (Accucheck). Blood from the rats tail was collected on the strips of glucometer and blood glucose values were obtained. Almond preparation decreased blood glucose in animals. This could either be due to decrease systemic absorption of glucose, or increase uptake of glucose by cells. Research shows that Almond causes insulin sensitization in cells (Rolls et al., 1983). This enhances the glucose uptake in the cells, and decrease blood sugar level is observed.

It is scientifically proved that obese patients are more prone to depression than people who are not obese (Brett and Pratt, 1990; Rolls et al., 1983). Depression maybe caused due to decrease secretion of serotonin and Nor-epinephrine in the brain. Therefore it is important to assess the effect of obesity on the central nervous system and to understand the parameters that cause depression. In the present study, two models were used to determine CNS activity in brain. Despair Swim test (DST) and the Open Field Behavior Test (OFBT). Despair Swim test checks the depression state of the rats or mice. Immobility time is the period in which the limbs of the animal are in a state of immobility (Stunkard et al., 2003). When the animal was first placed in the water, it vigorously moving legs. As the time goes on, the animal attained a state of immobility. It has given up swimming and now assumes that it is going to die. Animals that are obese had greater immobility time compared to the non-obese ones. In our present study, Almond high dose had significant effect in reducing the immobility time. This is mostly attributed to the weight loss, and lowering of lipid parameters in the body. However, we don't know whether the action is central or peripheral. Elevated plus maze test is a test for the anxiolytic activity of a drug. Obese patients are less anxious compared to the non-obese patients. Elevated plus maze test reveals the extent to which anxiety affects the rats/mice. In our present study, all the control groups had higher grooming time. That is they spent most of their time in closed arm (Naitoh et al., 1992). This was observed in all diets, the highest time being the cafeteria diet. Almond preparations and the standard anti-obesity herb decreased the grooming time, and increased the ambulatory time significantly. In normal diet, ambulatory time was increased significantly by all almond preparations. However, in cafeteria and atherogenic diet, only the almond medium and high dose significantly increased the ambulatory time.

Organ weights provide an in-vitro assessment of the deposition of fat present in the body. Left and Right kidney, Heart, Liver, Spleen, Mesenteric, Uterine, and Kidney fat was measured. There was a significant change between the liver and heart weights of control groups in all diets. Almond high dose significantly reduced the fat pads. This shows that there is sufficient fat mobilization for its breakdown.

5. Conclusion

It can be concluded that almond high dose (200 mg/kg) is effective in reducing body weight, lipid parameters, and immobility time. High dose of almond also increased the ambulation time in the open field behavior test. Hence almond may have some direct or indirect effect on the Central Nervous System. Additionally high dose of almond was successful in reducing the kidney fat, mesenteric fat, and uterine fat. Therefore almond high dose can be potential candidate to be used as anti-obesity agent. Further research must be carried out to accurately figure out the mechanism of action of almond.

Declaration of Competing Interest

The authors declare that they have no conflicts of interest.

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