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Research Article

Cuminum cyminum L. fruits distillate ameliorates the high fat diet-induced obesity

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ABSTRACT: **Introduction:** *Cuminum cyminum* L. (Fam. Apiaceae) is a widely consumed spice. It is a rich sources of essential oils and has been actively researched for its chemical composition and biological activities. **Aims:** The study aimed to investigate the antiobesity effects of *Cuminum cyminum* L. distillates (CD) in high-fat-diet-induced obese rats. **Methods:** Twenty four male rats were randomly assigned to one of four groups: normal diet (ND), high fat diet (HFD), HFD + CD and HFD + orlistat. The CD was administered orally 7.75 ml/kg/twice a day for 4 weeks. **Results:** The CD-treated group significantly ($p < 0.001$) decreased the body weight gain, fat-pad weights, serum and hepatic lipids levels, insulin, glucose, leptin and pancreatic lipase activity in HFD-induced obese rats. The histological analysis revealed that the CD-treated group showed a remarkable reduction of macro vesicular steatosis in hepatic tissues and a significantly decreased number of lipid droplets and size of adipocytes compared to the HFD group. **Conclusions:** These findings demonstrate that CD treatment has a protective effect against a high-fat-diet-induced obesity in rats through the decreased activity in lipogenesis, as well as the increase in fatty acid oxidation and reduced intestinal absorption of dietary fat.

KEYWORDS: Apiaceae, *Cuminum cyminum*, antiobesity, high-fat-diet, rats

INTRODUCTION

Obesity is a chronic metabolic disorder that results from the imbalance between energy intake and energy expenditure with storage of extra calories in the form of triglycerides in the adipose tissue. It is characterized by an excessive accumulation of fat in the body to a sufficient magnitude which adversely affects the health of an individual.[1,2] On a global scale, obesity has reached epidemic proportions and is a major contributor to the global burden of chronic disease and disability. Currently, more than one billion adults worldwide are overweight and at least 300 million of them are clinically obese.[3] Importantly, obesity is often associated with a variety of chronic diseases such as hyperlipidemia, diabetes mellitus, hypertension, coronary artery disease, and certain cancers.[4–7] Therefore, prevention and treatment of obesity are important for a healthy life.[8]

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In past years, many numbers of drugs have been approved for the treatment of obesity; however, most of them have been withdrawn from the market because of their adverse effects. Orlistat is presently the only available choice for the treatment of obesity because of its safety for cardiovascular events and positive effects on diabetic control.[9]

However, owing to the adverse effects associated with many antiobesity drugs, more recent trials have focused on screening natural sources that have been reported to reduce body weight with minimal side effects.[10] This may be an excellent alternative strategy for developing effective and safe antiobesity drugs in the future.[11–13] A variety of natural products, including crude extracts and isolated compounds from plants have been widely used traditionally to treat obesity.[14–16] A wealth of information indicates that numerous bioactive components from nature are potentially useful in obesity treatments. A good example of this is polyphenolic compounds showing strong antiobesity activity including genistein, apigenin, and the catechins.[17,18]

Cuminum cyminum L. (cumin) is widely consumed as spices and has been prominently considered carminative, eupeptic, antispasmodic, astringent and used in the treatment of mild digestive disorders, diarrhea dyspepsia and as a liver

tonic in India.[19] Distillate of *Cuminum cyminum* contains the essential oil an aqueous layer like oil aqueous type emulsion. Recently, it has been reported that *Cuminum cyminum* L. essential oil possesses various biological functions including anticonvulsant, antimicrobial, hypolipidemic and hypoglycaemic effect.[20–22] Essential oil from Indian cumin fruits contains cuminaldehyde, pinenes, and p-cymene as main components, whilst the essential oil from Iran cumin fruits contain terpinene (15.82%), 2-methyl-3-phenyl-propanal (32.27%) and myrtenal (11.64%).[23,24] In addition, these compounds from cumin oil fruits have shown to have antioxidant and antidiabetic activities.[25,26] However, little is known about the effects of *Cuminum cyminum* L. on obesity in animal model. Distillate of *Cuminum cyminum* is the major ingredient of Unani polyherbal formulation Arq Zeera which is thought to alter fat metabolism and increase the basal metabolic rate.[27] In the present study, we investigated the antiobesity effects of CD in high-fat diet-(HFD-) induced obese rats. Body weight gain, food intake, fat-pad weights, liver weight, serum and hepatic lipid profile, alanine aminotransferase (ALT), aspartate aminotransferase (AST), insulin, glucose, leptin and pancreatic lipase activity were measured.

MATERIALS AND METHODS

Preparation and phytochemical investigation of CD

C. cyminum L. was purchased from Shamsi dawakhana (raw material drug supplier) Ballimaran, New Delhi, India, in December 2010 and identified from National Institute of Science Communication and Information Resources (NISCAIR), New Delhi. Voucher specimen and identification certificate reference number NISCAIR/RHMD/Consult/2011-12/1753/53 was obtained and a voucher specimen was deposited in the laboratory (faculty of pharmacy). Samples were rinsed with tap water to remove salt and dried in an air dryer at 37 °C for 40 h. A dried sample was ground and the coarse powder was stored at −20 °C until used.

The dried coarse powder of *Cuminum cyminum* L. (375 g) was soaked in purified water and transferred to the distillation plant along with purified water (12 liter). This was distillate at 100 °C for about five and half hrs and 7.5 liters of distillate was collected.[27] The distillate of *Cuminum cyminum* L was dissolved in diethyl ether and resulting in the formation of two layers, ether and aqueous layer. The ether layer was concentrated by evaporation and 0.2 µl of the solution was subjected to GC and GCMS (Shimadzu QP-2010 GC-MS system) for identification of chemical constituents. The chemical constituents were identified by using NIST and Wiley libraries. GC and GCMS chromatogram are shown in Figures 1 and 2.

Animals and experimental diet

The study was approved by the Institutional Animal Ethics Committee (IAEC) of Hamdard University (Letter number – 607, 13/6/11), New Delhi, which is registered with the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India. Wister Albino male rats, weighing 150–200 g, were procured from the Central Animal House Facility, Hamdard University, New Delhi, and acclimatized

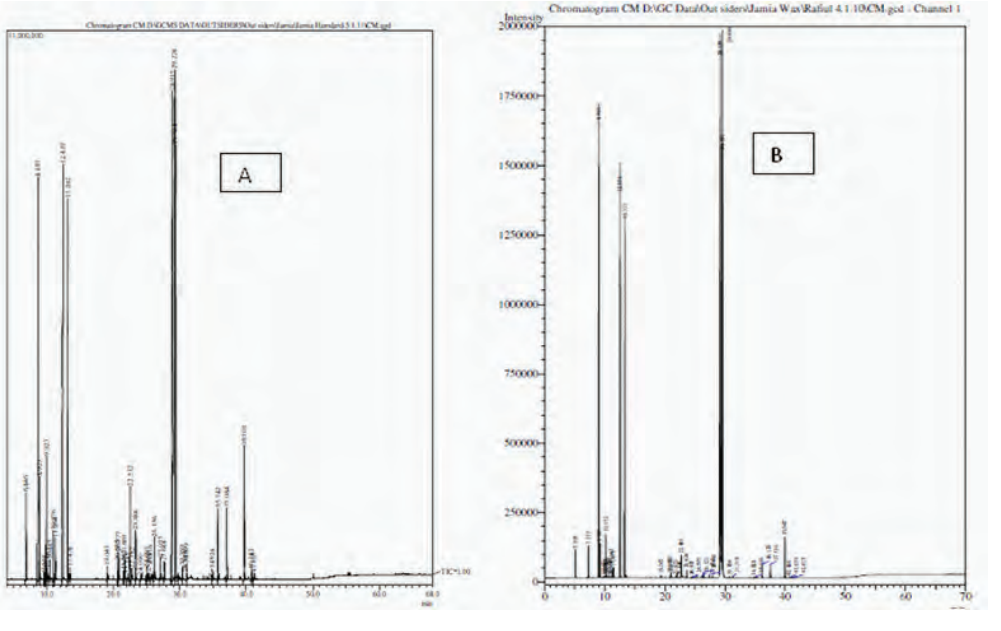

Figure 1. GC-MS (A) and GC (B) chromatogram of ether fraction of CD.

under standard laboratory conditions at 25 ± 2 °C, and relative humidity (50% ± 15%) and normal. The animals were kept in polypropylene cages under standard laboratory conditions (12 hour light and 12 hour dark: day: night cycle) and had a free access to tap water ad libitum.

After 7 days of acclimation, animals were randomly divided into four groups (n = 6): one normal control group, one HFD control and remaining 3–4 as treatment groups. Animals in normal control group were fed with normal diet (ND) consisting of 12.5% lipids, 62.3% carbohydrate and 24.3% protein (Amrut rat feed, Mfd by: Pranav Agro Industries Ltd, Maharashtra, India) while the other groups were fed with HFD (Research Diets Inc., NIN, Hyderabad, India) consisting of 60% fat, 20% protein, and 20% carbohydrate ad libitum, respectively, throughout the experiment. Treatments were started from 15th day and continued for four weeks. The treatment groups HFD + CD and HFD + orlistat were given CD and orlistat respectively at 7.75 ml/kg, twice per day and, 30 mg/kg of the body weight once per day of body weight by oral root. During the course of treatment the treatment groups were continued to feed with HFD. Food intake was recorded daily, and body weights were monitored every week during the feeding period. At the end of the treatment period, rats were subjected to fasting for 12 hours. After anesthetization with diethyl ether, the epididymal, abdominal, visceral fat-pads and livers were removed from the rats, rinsed with phosphate-buffered saline, and then weighed. The liver samples were stored at −70 °C until biochemical analysis.

Biochemical analysis

Blood was collected from the retro-orbital plexus of the all groups of overnight fasted rats using microcapillary tubes containing heparin on day 43. Serum was separated by centrifugation (4000 rpm, 10 min) and transferred to Eppendorf tubes. The concentrations of glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), free fatty acid (FFA), blood urea nitrogen (BUN), total cholesterol (TC), triglyceride (TG; all the three from Span Diagnostics Ltd, Surat, Gujarat, India) and high-density lipoprotein-cholesterol (HDL-C; Reckon Diagnostics Pvt Ltd, Baroda, Gujarat, India) in serum were measured with commercial kits. The concentrations of insulin, leptin and pancreatic lipase in the serum were measured, respectively, with rat insulin ELISA kit (Alpco Diagnostics, Salem, USA), rat leptin ELISA kit (BioVendor, Czech Republic) and Quanti ChromTM Lipase Assay Kit (DLPS-100), (Corporate Place, Hayward, CA 94545, USA). All other chemicals used were of analytical grade. Double distilled water was used for all biochemical assays. Estimations of serum low-density lipoprotein-cholesterol (LDL-C) cholesterol were measured by the using of Friedewalds equation.[28]

Hepatic lipid profiles

Hepatic lipids were extracted using the method developed by Folch et al.[29] and the dried lipid residues were dissolved in 1 mL ethanol. High density lipoprotein cholesterol (HDL-C), TC, TG, and FFA concentrations in the hepatic lipid extracts were measured using the same enzymatic kits that were used for the plasma analysis.

Hematoxylin and eosin staining

Tissue samples of liver and epididymal fat pads were fixed with 10% buffered formalin and embedded in paraffin. Standard 5 μm thick sections were stained with hematoxylin and eosin, viewed with an optical microscope (Olympus Optical, Tokyo, Japan), and photographed at a final magnification of 400 x for adipocytes and 100 x, 10 x for liver tissue. A total of 32 planes (8 planes per group) were used to determine the size of adipocytes. The average size of adipocytes was measured by using Image J software (National Institute of Mental Health, Bethesda, USA).

Statistical analysis

Statistical analysis was carried out using Graphpad Prism 3.0 (Graphpad software, San Diego, California, USA). All of the data were expressed as mean ± SEM. Statistical analysis was performed by Dennett's t- test and $p < 0.05$ were considered to be significant.

RESULTS

GC and GCMS analysis of CD

The chemical composition of the CD was examined by GC and GC–MS; 36 components, were identified. Cuminaldehyde (22.58%), 4-ethyl-3-nonen-5-yne (19.21%), phenyl propanol (15.18%), γ-terpinene (13.12%), α-pinene (11.76%), O-cymene (10.12%) were the major components. It is shown in Table 1.

Effect of CD treatment on body weight, food intake and fat-pad weights

The body weight and food intakes are shown in Table 2. During the 4 weeks experimental period, the body weight changes were measured every week. The body weight gain of the HFD group was significantly elevated than those of the ND group. The CD and orlistat administration significantly reduced body weight gain compared to no treatment (HFD group). The food intake of the obese rats given CD was significantly lower than the values for the rats fed HFD only, suggesting that CD regulates food intake and energy metabolism. In addition, epididymal, abdominal and visceral fat-pad weights were significantly higher in the HFD group than the value for the ND group, and these increases was lower by CD treatment (Table 2).

Effect of CD treatment on lipid parameters

Rats in the HFD group exhibited significantly higher TG, TC, LDL- C and FFA levels and lower HDL-C compared to rats in the ND group (Table 3). However, CD or orlistat treatment led to a reversal of the aforementioned parameters to the levels similar to those of the ND group. CD treatment significantly decreases TG, TC, LDL- C, and FFA and an increase in HDL-C compared with no treatment (HFD group). These results indicate that oral administration of CD suppresses the accumulation of body fat, resulting in improved lipid profiles in serum.

Effect of CD treatment on liver weight and hepatic lipid

Liver weights and hepatic lipid levels are shown in Table 4. Liver weight was about 2.5 times greater in the HFD group than that in the ND group. In addition, hepatic TG, TC, and FFA levels in the HFD group were significantly higher than ND group. Conversely, HDL-C

Table 1: Chemical composition of CD

S. no	Retention time	Compounds	Area %
1	8.697	**α-pinene**	**11.7657**
2	8.927	β-phellandrene	0.6031
3	9.617	Delta- 3 carene	0.6031
4	9.801	4-ethyl Bicyclo-[3.3.0]oct-2-ene	0.0335
5	9.927	Myrcene	0.8506
6	10.055	1-phellandrene	0.0432
7	10.445	α-terpinene	0.0738
8	10.976	Limonene	0.2477
9	11.264	Sabinene	0.3152
10	12.439	**γ-Terpinene**	**13.9454**
11	13.202	**O-Cymene**	**10.1289**
12	13.478	Carene	0.0435
13	20.675	Limonene epoxide	0.1185
14	20.777	1-(3-isopropenyl-2,2-Dimethyl Cyclopropyl)-2-Methyl-Propan-1-one	0.1185
15	21.561	Linalool	0.0206
16	21.669	4-thujanol	0.0300
17	22.145	4-isopropyl-1-methyl-2-cyclohexen-1-ol	0.0300
18	22.512	Phellandral	0.6445
19	22.782	Borneol acetate	0.1935
20	23.386	4-terpineol	0.1935
21	24.090	4-isopropyl-1-methyl-2-cyclohexen-1-ol	0.0167
22	24.997	Pinocarveol	0.0200
23	25.259	β-farnesene	0.0653
24	25.616	O-menth-8-ene	0.0653
25	26.136	β-menchol	0.1299
26	27.075	Phellandral	0.0984
27	27.666	Azulene	0.0593
38	28.915	**Cuminaldehyde**	**22.582**
39	29.226	**4-ethyl-3-nonen-5-yne**	**19.215**
30	29.364	**1-phenylpropanol**	**15.181**
31	29.749	α-bisabolol	0.0415
32	30.369	4-isopropylphenyl Methnol	0.0476
33	30.999	4-isopropyl-2-cyclohexen-1-yl) methanol	0.0154
34	35.742	(4-isopropyl-1,4-Cyclohexadien-1-yl) Methanol	0.3653
35	37.084	Cuminol	0.4017
36	39.701	Thymol	1.2681

levels in liver were significantly lower than those in the ND group. The CD treated significantly decreases hepatic TG, TC, and FFA and an increase in HDL-C compared with the HFD group.

Effect of CD treatment on serum glucose, insulin, leptin, and pancreatic lipase activity

Serum glucose, insulin, leptin, and pancreatic lipase activity were increased by the high-fat-diet (Figure 2). However, CD or orlistat administration showed decreased pancreatic lipase activity, leptin, glucose, and insulin levels in serum compared to the HFD group.

Effect of CD treatment on serum AST, ALT and BUN level

To evaluate the effect of CD on hepatic and renal function, we determined the plasma AST, ALT and BUN level in CD-treated obese rat (Table 5). Although serum levels of AST, ALT and BUN of the HFD group were significantly higher than those of the ND group, and orlistat or CD treatment reduced the serum AST, ALT and BUN levels to values of the ND groups, they all were within normal range. These results indicate that CD administration has no liver and kidney toxicity.

Histopathological studies

Our histological examinations revealed that the sizes of the adipocytes were significantly increased in HFD induced obese rats. Conversely, the size of adipocytes from the epididymal white adipose tissue of the obese rats fed CD was remarkable smaller than those for the HFD group (Figure 3a and b). Our histological examination showed macro vesicular steatosis in liver tissues of the HFD group (Figure 4). However, CD administration noticeably attenuated the extent of steatosis. These lipid droplets were strikingly reduced both in size and number in the liver of CD or orlistat-treated rats, suggesting that CD treatment effectively inhibits lipid accumulation in liver.

Table 2: Effects of the CD on body weight, food intake and fat-pad weights (value are Mean ± SEM from six rats in each group)

Parameter	ND	HFD	HFD + CD	HFD + orlistat
Initial body weight (g)	155.2 ± 2.16	158.32 ± 1.26	179.09 ± 3.18	182.1 ± 5.43
Weight (g) on 14th day	180.87 ± 4.34	212.56 ± 3.56	243.76 ± 3.79	248.75 ± 3.67
Weight (g) on 43rd day	251.08 ± 3.12	341.45 ± 10.2	326.79 ± 3.12	308.97 ± 4.31
Body weight gain (g)	70.21 ± 5.12	128.89 ± 6.02$^{\$\$}$	83.03 ± 3.12**	60.22 ± 5.90**
Food intake (g/150 g rat/day)	14.13 ± 0.26	20.20 ± 0.23$^{\$\$}$	19.05 ± 0.375*	19.34 ± 0.248
Visceral fat pad weights (g/150 g rat)	8.5 ± 1.18	22.42 ± 4.2$^{\$\$}$	14.32 ± 2.36**	8.69 ±1.26**
Epididymal fat weights (g/150 g rat)	10.03 ± 0.18	18.32 ± 1.02$^{\$\$}$	14.09 ± 0.75**	15.90 ± 0.05**
Abdominal fat weights (g/150 g rat)	13.43 ± 0.08	38.32 ± 6.02$^{\$\$}$	18.32 ± 0.75**	18.09 ± 0.35**

$^{\$\$}p < 0.001$ as compared to control groups. *p < 0.05 as compared to HFD groups, **p < 0.001 as compared to HFD groups

Table 3: Effect of CD on serum lipid profiles levels (Mean ± SEM) (value are Mean ± SEM from six rats in each group)

Parameters	ND	HFD	HFD + CD	HFD + orlistat
Total triglycerides level (Mg/dL/150 g rat)	48.67 ± 2.51	108.105 ± 4.37$^{\$\$}$	68.55 ± 1.93**	42.23 ± 3.36**
Total cholesterol level (mg/dL/150 g rat)	62.87 ± 2.18	99.59 ± 2.88$^{\$\$}$	76.9 ± 1.34**	74.098 ± 1.17**
HDL-cholesterol level (Mg/dL/150 g rat)	34.18 ± 0.89	24.07 ± 2.59$^{\$\$}$	36.78 ± 1.67**	29.656 ± 1.09**
LDL-cholesterol level (Mg/dL/150 g rat)	12.25 ± 1.82	21.71 ± 3.82$^{\$\$}$	15.75 ± 2.43**	13.7 ± 1.81**
FFA(mEq/L)	0.45 ± 0.08	0.78 ± 0.02$^{\$\$}$	0.46 ± 0.01**	0.48 ± 0.02**

$^{\$\$}p < 0.001$ as compared to control group, **p < 0.001 as compared to HFD group

Table 4: Effect of CD on liver weight and hepatic lipid (value are mean ± SEM from six rats in each group)

Parameters	ND	HFD	HFD + CD	HFD + orlistat
Total triglycerides level (Mg/g liver)	1.67 ± 0.05	4.105 ± 0.07$^{\$\$}$	2.55 ± 0.07**	2.23 ± 0.061**
Total cholesterol level (Mg/g liver)	0.47 ± 0.01	0.89 ± 0.08$^{\$\$}$	0.5 ± 0.03**	0.598 ± 0.04$^{\$\$}$
HDL-cholesterol level (Mg/g liver)	0.03 ± 0.89	0.011 ± 0.59$^{\$\$}$	0.027 ± 0.07**	0.026 ± 0.09**
FFA (mEq/L)	1.45 ± 0.08	2.78 ± 0.021$^{\$\$}$	1.48 ± 0.021**	1.48 ± 0.029**
Liver weight (g/150 g rat)	4.96 ± 0.13	11.47 ± 0.02$^{\$\$}$	7.156 ± 0.09**	5.23 ± 0.084**

$^{\$\$}p < 0.001$ as compared to control group. **p < 0.001 as compared to HFD group

DISCUSSION

In the current study, CD exhibited a promising antiobesity effect on the obesity rate induced by high fat diet. A model of diet-induced obesity in rats is well controlled and shares many features with human obesity. Dietary fat is one of the most important environmental factors associated with the incidence of obesity; high triglycerides, cholesterol and saturated fat diets have been shown to promote obesity.[30] A rodent

Table 5: Effect of CD on the liver and renal functions (value are mean ± SEM from six rats in each group)

Parameters	ND	HFD	HFD + CD	HFD + orlistat
AST level (IU/L/150 g rat)	62.44 ± 1.6	87.75 ± 1.77$^{\$\$}$	72.29 ± 2.74**	64.68 ± 1.39**
ALT level (IU/L/150 g rat)	42.46 ± 2.92	67.09 ± 3.39$^{\$\$}$	53.55 ± 2.98**	44.22 ± 2.38**
BUN (mg/dL)/150 g rat)	16.43 ± 0.23	19.71 ± 0.93$^{\$\$}$	15.98 ± 0.73**	15.42 ± 0.36**

$^{\$\$}$p < 0.001 as compared to control groups, **p < 0.001 as compared to HFD group

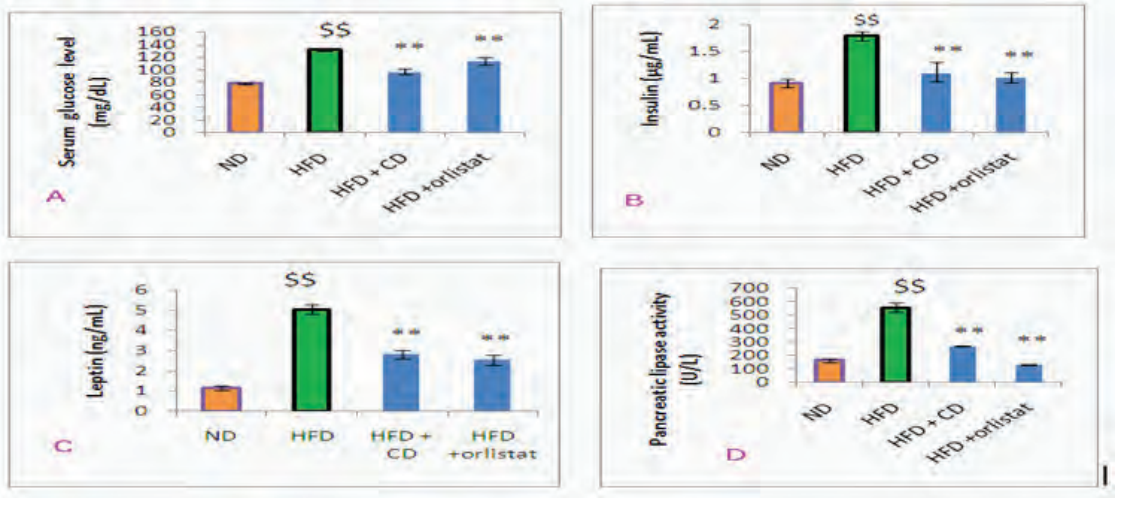

Figure 2. Effect of CD on the level of (A) glucose (B) insulin, (C) leptin, and (D) pancreatic lipase activity. All values were expressed as mean ± SEM for six rats in each group. $^{\$\$}$p < 0.001 as compared to control group. **p < 0.001 as compared to HFD groups.

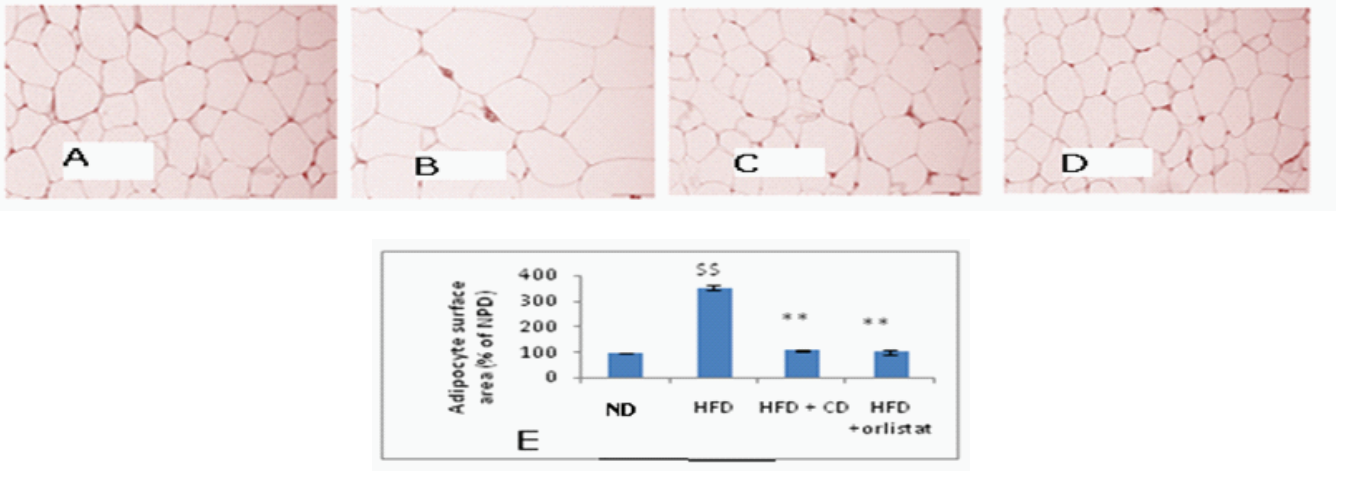

Figure 3. (a) Effect of CD or orlistat on epididymal adipose tissue and (b) adipocyte surface area (E) of HFD induced obese rat. All sections were stained with hematoxylin and eosin; magnification, × 400. Magnification bar = 100 µm; (A) ND groups section showing normal sizes of the adipocytes; (B) HFD group section showing bigger sizes of the adipocytes; (C) HFD + CD treated group section (Almost near normal); (D) HFD + orlistat treated group section showed (Almost near normal). Mean surface area for epididymal white adipocytes was measured using Image J software. All values were expressed as mean ± SEM for six rats in each group. $^{\$\$}$p < 0.001 as compared to HFD group. **p < 0.001 as compared to control groups.

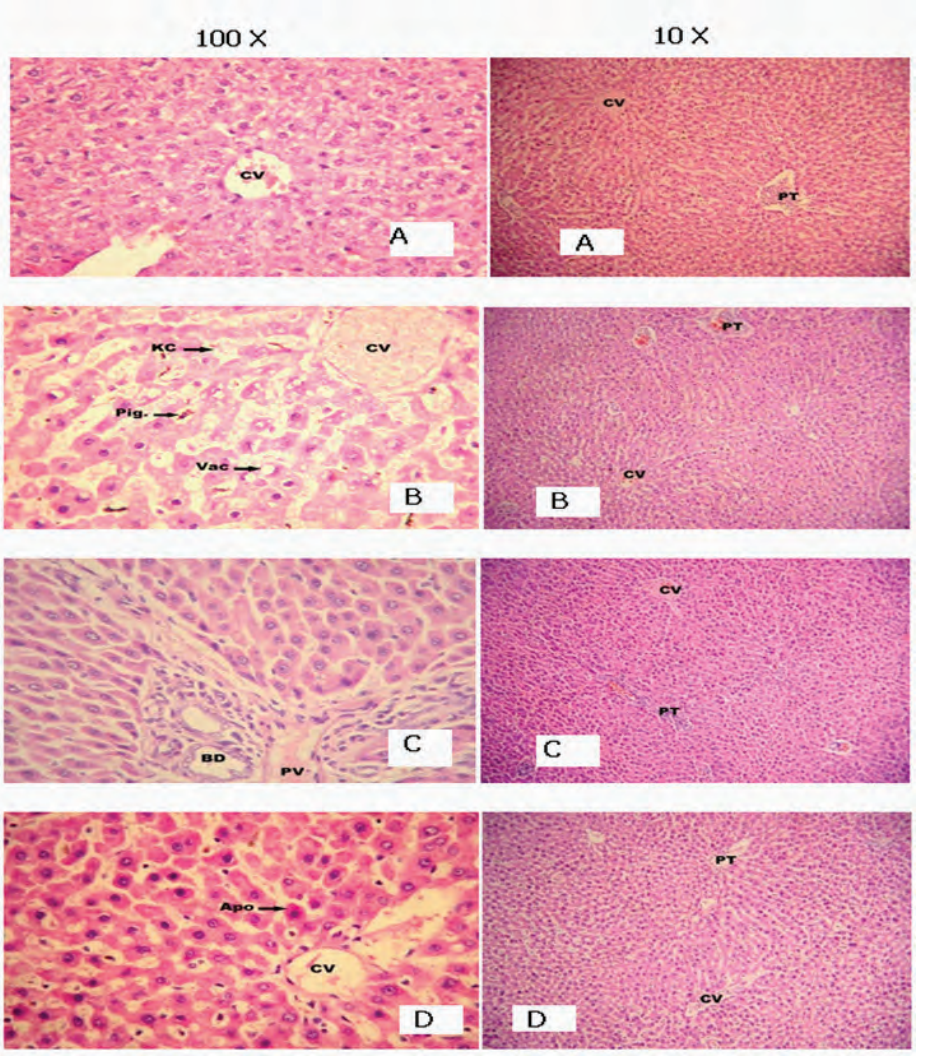

Figure 4. Effect of CD or orlistat on liver tissue of HFD induced obese rat. The liver was removed, fixed in 10% formalin, paraffinized, and stained using hematoxylin and eosin, magnification: 100 X and 10 X; (A) ND group section showing normal liver architecture; (B) HFD control section showing patches of liver cell necrosis with inflammatory collections; (C) HFD + CD treated group section (Almost near normal); (D) HFD + orlistat treated group section shows lesser damage of hepatocytes and low index of necrosis. (KC = Kuffer cell, PT = portal Triad, hepatocytes vacuolation, sinusoidal dilation, Pig = lipofuchsin pigment and apo = apoptotic heptocytes.

model of obesity based on the intake of HFD is advantageous in studying obesity-related factors.[31] In this study, antiobesity activity of CD was investigated by measuring body weight gain, food intake, and lipid profiles in CD -treated obese rats. High-energy diets are widely used in nutritional experiments as a strategy to induce overweight conditions and fat deposition in animals.[32] We also observed that body-weight gain, and total fat-pad weight in the HFD group were greater than of ND group. Administration of CD or orlistat for 4 weeks remarkably decreased the body weight gain compared with that of the HFD group (Table 2). The CD significantly decreased the food intake. In general, a high-fat diet significantly increases the TC and TG levels in serum and liver.[33] Our data also showed that rats in the HFD group exhibited significantly higher TG, TC, LDL-C and FFA levels, and lower HDL-C. However, the administration of CD or orlistat reduced these parameters to near normal levels in serum and liver (Table 3 and 4). These results indicate that oral administration of CD suppresses the accumulation of body fat, resulting in improved lipid profiles in serum and liver, and decrease of insulin level. Notably, CD treatment did not show any renal or hepatic toxicity (Table 5). Blood levels of leptin, which is a key fat-derived regulator of appetite and energy expenditure,

normally correlate positively with the extent of the TG stores in adipocytes.[34–36] In our study, the plasma leptin level was decreased by CD or orlistat treatment (Figure 2). Our histological examinations revealed that the sizes of the adipocytes were significantly reduced in CD -treated rats (Figure 3). These results suggest that the decreased plasma leptin levels after CD administration might be attributable to decreased lipid accumulation in white adipose tissue. In addition, HFD is known to increase the synthesis of fatty acids in the liver and the delivery of free fatty acids to the liver and decrease β-oxidation of free fatty acids, resulting in fat accumulation in the liver.[37,38] Our histological examination also showed macro vesicular steatosis in liver tissues of the HFD group (Figure 4). However, CD administration noticeably attenuated the extent of steatosis, suggesting that CD may regulate lipid storage and mobilization in adipocytes by modulation of the leptin level.

It is noticeable that CD treatment significantly reduced the serum glucose, and insulin levels in obese rats. It is well known that CD reduces the intestinal absorption of glucose. Considering that the increase in blood glucose and insulin in animals on the high-fat diet is a strong indicator of obesity-induced insulin resistance and progression to type-2 diabetes, these results suggest that CD has protective effect against the development of obesity-induced insulin resistance.

Han et al. have suggested that long term high fat diet may result in increased pancreatic lipase activity.[39] Our data also showed that rats in the HFD group exhibited significantly higher pancreatic lipase activity. Interestingly, CD treatment significantly reduced the serum pancreatic lipase activity levels in obese rats. Pancreatic lipase inhibitors which help to limit intestinal fat absorption at the initial stage, have been proven as useful medications for the treatment of hyperlipidemia and have promise as anti-obesity agents.[40]

CONCLUSION

This study conclusively demonstrated that CD has beneficial effects for the suppression of high-fat-diet induced obesity in rats. It provided evidence that CD administration decreases body weight gain, food intake, lipid levels in serum and liver, size and number of adipocytes, pancreatic lipase activity and improvement in insulin and leptin sensitivity in HFD-induced obese rats. CD appears to show such activities by modulating the lipid metabolism through the decreased activity in lipogenesis as well as the increase in fatty acid oxidation and reduction in fat absorption through inhibiting of pancreatic lipase, suggesting that CD has excellent potential as an effective antiobesity agent with no obvious toxicity.

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