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## **Mustard Oil Based High Fat Diet is Associated with Decreased Body Weight Gain, Less Adiposity and Improved Glucose and Lipid Homeostasis in Wistar Rats**

Zafar Ahmad Malik, Amit Goyal and Pyare Lal Sharma  
Department of Pharmacology, ISF College of Pharmacy, Moga, India

*Corresponding Author: Zafar Ahmad Malik, Department of Pharmacology, ISF College of Pharmacy, Moga, India  
Tel: 01942455799*

### **ABSTRACT**

Mustard oil, traditional edible oil used in India and Bangladesh for centuries, has been associated with lower risk of metabolic disorders. The present study was performed to evaluate the potential antiobesity effect of mustard oil by analyzing the changes in body weight, visceral adipose mass and serum biochemicals in dietary obesity in wistar rats. Wistar rats were fed normal chow diet, lard based high fat diet, mustard oil based high fat diet or lard plus mustard oil based high fat diet for 10 weeks. Body weight and food intake were measured regularly during the experimental period. Total fat in fecal excretions were measured in the 8th week while as core body temperature (an index of thermogenesis) and oral glucose tolerance test were measured in 10th week of the experiment. At the end of experiment various visceral adipose tissues were weighed and serum glucose and lipids measured. Lard based high fat diet animals gained more body weight, had higher glucose and lipid levels compared to NC animals indicating induction of obesity. However, body weight gain and visceral adipose tissue mass of lard plus mustard oil based high fat diet animals were lower than that of lard based high fat diet animals and that of the mustard oil based high fat diet were the lowest. Furthermore, both mustard oil based high fat diet and lard plus mustard oil based high fat diet animals were associated with increased thermogenesis, decreased serum glucose and lipid levels and improved glucose tolerance compared to lard based high fat diet obese animals. These results suggest that mustard oil has potential antiobesity effect by regulating body weight gain, adipose tissue mass and lipid and glucose metabolism.

**Key words:** Mustard oil, obesity, adipose tissue, polyunsaturated fatty acid (PUFA), high fat diet

### **INTRODUCTION**

Obesity is a multifactorial, chronic disorder that has reached a pandemic proportion worldwide (Haslam and James, 2005). Nearly one third of the world's adult population (1.3 billion people) was overweight or obese in 2005 and if recent trends continue, by 2030 nearly two third of the world's adult population (3.3 billion people) could be either overweight or obese (Kelly *et al.*, 2008). Obese and overweight patients are at higher risk from coronary artery disease, hypertension, hyperlipidemia, diabetes mellitus, cancers, gall bladder disorders, cerebrovascular accidents, osteoarthritis, gastrointestinal diseases restrictive pulmonary disease and sleep apnoea (Hameed *et al.*, 2002; Afridi *et al.*, 2003; Ebrahimi-Mameghani *et al.*, 2008;

Afoakwah and Owusu, 2011). The rapid increase in the global prevalence of obesity suggests that common environmental and lifestyle factors are promoting and exacerbating the problem (Gluckman and Hanson, 2008; Mahajan *et al.*, 2010). Replacing traditional cooking oils with saturated fats may also contribute to high prevalence of obesity. Substantial evidence suggests that not only the amount of fat but also the type and composition of fat, can influence body weight, body composition and plasma comorbidity factors (Grundy, 1999). Furthermore, growing animal and human studies suggest that the association of fatty diet with weight gain and obesity may in part depend on the saturation of the fatty acids consumed (Storlien *et al.*, 1991; Delany *et al.*, 2000). Moreover, unsaturated particularly polyunsaturated fatty acids (PUFA) may protect against the development of obesity and associated biochemical disturbances and reduce body fat in established obese animals (Lichtenstein and Schwab, 2000; Buckley and Howe, 2009). These reports led to the speculation that PUFA rich mustard oil can be beneficial in high fat diet induced obesity and its biochemical complications.

Mustard oil, traditional edible oil used in most parts of India and Bangladesh for centuries, is well known for its medicinal utilities (Rastogi *et al.*, 2004; Dasgupta and Bhattacharyya, 2007). It is extracted from seeds of *Brassica juncea* (and other members) of the Cruciferae family and is rich in  $\alpha$ -linolenic acid (well known omega 3 PUFA). The annual plant has much-branched stem with lobed, roughly lyre-shaped leaves. Bright yellow flowers are produced all summer, followed by small, erect, 4-angled pods with dark brown seeds. In herbal medicine, the seeds are used to aid digestion, to promote the appetite and for colds, chills, coughs, chilblains, rheumatism, arthritis, lumbago, and aches and pains (Duke, 2008). Long term mustard oil consumption has been associated with a number of beneficial effects such as prevention of dyslipidemia, coronary artery diseases, atherosclerosis and colon cancer (Singh *et al.*, 1997; Dwivedi *et al.*, 2003; Rastogi *et al.*, 2004; Risa *et al.*, 2008; Degirolamo *et al.*, 2010). However, the antiobesity effect of the mustard oil has not been evaluated. Therefore, the current study was designed to investigate the potential antiobesity effects of PUFA rich mustard oil by analyzing the changes in body weight, visceral adipose tissue mass and serum biochemicals in dietary obesity in male wistar rats.

## **MATERIALS AND METHODS**

**Preparation of High fat diets:** High Fat (HF) diets were made from 67% w/w Normal Chow (NC) and 33% w/w either mustard oil (HF-M), lard (HF-L) or equal mixture of both (HF-LM) (Bellush and Rowland, 1985). These isocaloric HF diets provide 43% energy as carbohydrate, 14% as protein and 36% as fat while as NC provides 65% of energy as carbohydrate, 21% as protein and 4% as fat.

The normal chow diet (Ashirwad Diets, Punjab, India), lard and mustard oil (P-Mark Grade 1 Mustard oil, Puri Oil Mills, Punjab, India) were procured from the commercial sources. Mustard oil was heated to smoking before adding to the diet as traditionally it is heated almost to smoking before used for cooking.

**Animal treatments:** Male wistar rats of 7-8 weeks of age were procured from the animal facility of the Institute. The animals were housed in standard polypropylene cages (two rats/cage) and maintained under controlled room temperature ( $25\pm 2^\circ\text{C}$ ) with 12:12 h light and dark cycle. The guidelines of committee for the purpose of control and supervision of experiments on animals (CPCSEA), Govt. of India were followed and prior permission was sought from the institutional animal ethics committee for conducting the study. The study was conducted between October 2009 and March 2010.

Animals were randomized on the basis of their body weight and divided into four groups. One group was assigned to NC diet and other three to different HF diet groups (n = 10) for 10 weeks. All the animals had free access to water and the animals were inspected daily. Food intake and body weight were measured twice weekly. Feces of rats were collected on three consecutive days in 8th week for the determination of total fat content. One week before the sacrifice, animals were subjected to Oral Glucose Tolerance Test (OGTT). Core body temperature, an index of thermogenesis (Harrold *et al.*, 2000) was recorded at the onset of the light phase via rectal route using digital thermometer two days before sacrifice. Animals were familiarized with the procedure 10 days before (except on the day of OGTT) the actual readings. At the end of the stipulated period, blood for various biochemical parameters was obtained retro-orbitally under light ether anesthesia and the animals were sacrificed by cervical dislocation. The blood was collected into tubes, serum separated and analyzed on the same day. The gastrocnemius muscle, epididymal, mesenteric and retroperitoneal white adipose tissue were dissected, weighed and stored in 10% buffered formalin solution. Lee index i.e. (Body wt. in g)<sup>1/3</sup>/(ano-nasal length in cm) (Bernardis and Patterson, 1968), an index of obesity, was calculated at the end of the experiment.

**Measurements:** Serum glucose, triglyceride, total cholesterol and HDL cholesterol, SGOT and SGPT concentrations were measured by using commercially available kits (Tulip Diagnostic (P) Ltd, Goa, India). During OGTT glucose levels were quantified at the start (t = 0), 30, 60, 90 and 120 min after the administration of the glucose load (2 g kg<sup>-1</sup>). The total fat content in feces was determined gravimetrically. The samples were dried (105°C for 12 h) and then extracted with petroleum ether under reflux (Chen and Chan, 2003).

**Histological analysis and morphometry:** Epididymal adipose tissue was fixed in 10% formalin and then embedded with paraffin. Tissue sections (10 µm) were cut and mounted on microscope slides. After being air-dried, they were stained with hematoxylin and eosin and photographed at 100X magnification. At least two fields per slice and six slices per fat mass were analyzed for the purpose of quantifying adipocyte size.

**Statistics:** All values are expressed as Mean±SD. The significance of the differences between the means of various groups was established by one way ANOVA with a Tukey's post hoc test using the Graphpad Prism 4 software. The p value<0.05 was considered to be statistically significant.

## RESULTS

**Effect on body weight and adipose tissue weight:** All four groups of rats gained body weight during the 10 weeks of the diet regimens (Table 1). Rats on the HF-L diet gained body weight significantly more than the NC (HF-L 345.4±25.7 g vs. NC 285±40.3 g, p<0.05) indicating induction of obesity. However, the final body weight of HF-M and HF-LM animals were approximately 16% (HF-M 297.3±35.4 g vs. HF-L 345.4±25.7 g, p<0.05) and 11% (HF-LM 315.6±31.3 g vs. HF-L 345.4±25.7 g, p>0.05), respectively, lesser than the HF-L group.

Parallel to the body weight change, the weights of visceral adipose pads (including epididymal, retroperitoneal and mesenteric adipose pads) were significantly greater in HF-L (p<0.05) and HF-LM (p<0.05) groups but not in HF-M than NC rats (Table 1). However, in the HF-M and HF-LM groups the total weight of visceral fat deposits was nearly 31% (p<0.05) and 28% (p<0.05), respectively, lesser than the HF-L group.

Table 1: Body weight gain, tissue mass, and serum and hepatic biochemistry of male wistar rats fed normal chow (NC), lard based high fat diet (HF-L), mustard oil based high fat diet (HF-M) or lard plus mustard oil based high fat diet (HF-LM) for 10 weeks

| Parameters                      | NC           | HF-L                      | HF-M                      | HF-LM                    |
|---------------------------------|--------------|---------------------------|---------------------------|--------------------------|
| Initial body weight (g)         | 180.60±11.3  | 182.10±10.7               | 183.40±10.2               | 179.80±12.7              |
| Final body weight (g)           | 285.00±40.3  | 345.40 ±25.7 <sup>a</sup> | 297.30±35.4 <sup>b</sup>  | 315.60±31.3              |
| Avg. food intake (kcal/day/rat) | 68.00±4.9    | 77.60±7.9 <sup>a</sup>    | 78.70±9.7 <sup>a</sup>    | 77.20±6.8 <sup>a</sup>   |
| Avg. fecal fat (g/day/rat)      | 0.052± 0.014 | 0.317±0.034 <sup>a</sup>  | 0.302±0.039 <sup>a</sup>  | 0.303±0.030 <sup>a</sup> |
| Core body temperature (°f)      | 98.40±0.5    | 98.70±0.7                 | 99.60±0.6 <sup>a,b</sup>  | 99.30±0.5 <sup>a</sup>   |
| Lee index                       | 0.284±0 .01  | 0.298±0 .01 <sup>a</sup>  | 0.286±0 .01               | 0.292±0 .01              |
| Fat pad weight (g)              |              |                           |                           |                          |
| Epididymal                      | 2.32±0.31    | 4.90±0.92 <sup>a</sup>    | 3.78±1.29 <sup>a</sup>    | 3.57±1.16 <sup>a,b</sup> |
| Retroperitoneal                 | 2.55±0.42    | 5.00±1.41 <sup>a</sup>    | 3.00±1.1 <sup>b</sup>     | 3.47±1.21                |
| Mesenteric                      | 2.58±0.45    | 5.60±1.55 <sup>a</sup>    | 3.82±0.79 <sup>b</sup>    | 4.08±1.69 <sup>a,b</sup> |
| Total visceral fat              | 7.45±0.80    | 15.50±3.69 <sup>a</sup>   | 10.60±2.76 <sup>b</sup>   | 11.12±3.06 <sup>a</sup>  |
| Gastrocnemius mass              | 2.10± 0.31   | 2.20±0.24                 | 2.10±0.19                 | 2.20±0.22                |
| Liver weight (g)                | 8.58±1.62    | 10.31±2.67                | 9.42±1.87                 | 9.59±2.12                |
| Heart weight (g)                | 0.877±0.21   | 0.921±0.23                | 0.898± 0.24               | 0.855±0.19               |
| Glucose (mg dL <sup>-1</sup> )  | 93.60±13.4   | 123.40±18.2 <sup>a</sup>  | 96.90±14.2 <sup>b</sup>   | 103.60±17.5 <sup>b</sup> |
| TG (mg dL <sup>-1</sup> )       | 106.30±19.2  | 98.60±22.3                | 102.70±25.4               | 104.20±20.1              |
| TC (mg dL <sup>-1</sup> )       | 48.20±4.1    | 65.60±7.4 <sup>a</sup>    | 39.00±5.7 <sup>a,b</sup>  | 51.20±6.6                |
| HDL (mg dL <sup>-1</sup> )      | 23.10±4.3    | 28.40±5.7                 | 27.10±5.2                 | 27.40±5.4                |
| TC/HDL ratio                    | 2.18±0.26    | 2.31 ±0.21                | 1.44 ±0.31 <sup>a,b</sup> | 1.87 ±0.28 <sup>b</sup>  |
| SGOT (IU L <sup>-1</sup> )      | 51.4 0±13.4  | 90.60±15.4 <sup>a</sup>   | 64.20±18.8 <sup>b</sup>   | 60.10±16.5 <sup>b</sup>  |
| SGPT (IU L <sup>-1</sup> )      | 47.60±11.6   | 73.70±13.4 <sup>a</sup>   | 52.10±14.7 <sup>b</sup>   | 49.30±10.9 <sup>b</sup>  |

Mean±S.D. <sup>a</sup> p<0.05 vs. NC ; <sup>b</sup> p<0.05 vs. HF-L

The Lee's index was significantly increased in HF-L (p<0.05) but not in HF-M and HF-LM groups compared to NC animals (Table 1). However, the Lee's index did not differ significantly among the HF diet groups. Moreover, dietary intervention did not produce any significant change in gastrocnemius muscle mass, an index of lean tissue mass, indicating no effect on lean tissue mass (Table 1).

**Energy intake and core body temperature:** During the 10 weeks of the diet regimens, average daily energy intake and fecal fat content of rats among the three HF diet groups were not significantly different indicating no difference in apparent fat absorption (Table 1). However, all the HF diets were calorically denser than the NC diet. Therefore, average daily energy intake and fecal fat content differed significantly (p<0.05) among the HF and NC animals.

Core body temperature, an index of thermogenesis, was significantly higher in HF-M (p<0.05) and HF-LM (p<0.05) rats compared to NC rats and among HF diet groups core body temperature of HF-M (p<0.05) was significantly higher than the HF-L group (Table 1) suggesting energy expenditure was higher in mustard based HF diet groups.

**Effect on biochemical and hepatic parameters:** The serum glucose concentrations in HF-L, HF-M and HF-LM were approximately 32% (HF-L 123.4±18.2 mg dL<sup>-1</sup>, p<0.05), 4% (HF-M 96.9±14.2 mg dL<sup>-1</sup>, p>0.05) and 11% (HF-LM 103.6±17.5 mg dL<sup>-1</sup>, p>0.05), respectively, greater than the NC animals (NC 93.6±13.4 mg dL<sup>-1</sup>) (Table 1). However, the glucose concentrations in HF-M and HF-LM were 21% (HF-M 96.9±14.2 mg dL<sup>-1</sup>, p<0.05) and 16%

(HF-LM  $103.6 \pm 17.5$  mg dL<sup>-1</sup>,  $p < 0.05$ ), respectively, lesser than the HF-L group (HF-L  $123.4 \pm 18.2$  mg dL<sup>-1</sup>). The total cholesterol concentrations in HF-L and HF-LM were approximately 36% (HF-L  $65.6 \pm 7.4$  mg dL<sup>-1</sup>,  $p < 0.05$ ) and 6% (HF-LM  $51.2 \pm 6.6$  mg dL<sup>-1</sup>,  $p < 0.05$ ) respectively, greater but 19% (HF-M  $39.0 \pm 5.7$  mg dL<sup>-1</sup>,  $p < 0.05$ ) lower in HF-M rats than the normal animals (NC  $48.2 \pm 4.1$  mg dL<sup>-1</sup>) (Table 1). However, compared to NC animals, TG and HDL-C concentrations did not alter significantly in HF diet groups (Table 1).

The relative weights of the liver as well as SGOT and SGPT levels were not statistically significant among groups indicating liver function was not affected with the dietary regimens (Table 1).

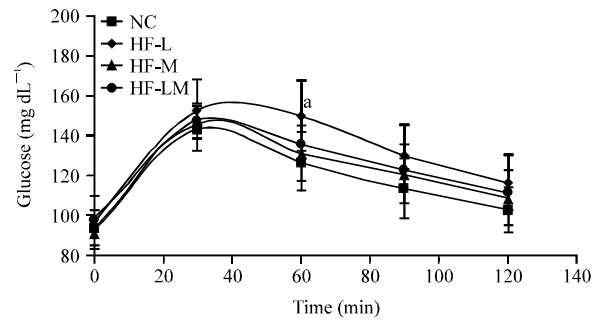


Fig. 1: OGTT in the last week of the 10 weeks experimental period. Values represent the Mean $\pm$ SD (n = 10). a =  $p < 0.05$  vs. NC. NC: Normal chow, HF-L: Lard based high fat diet, HF-M: Mustard oil based high fat diet. HF-LM: Lardplus mustard oil based high fat diet

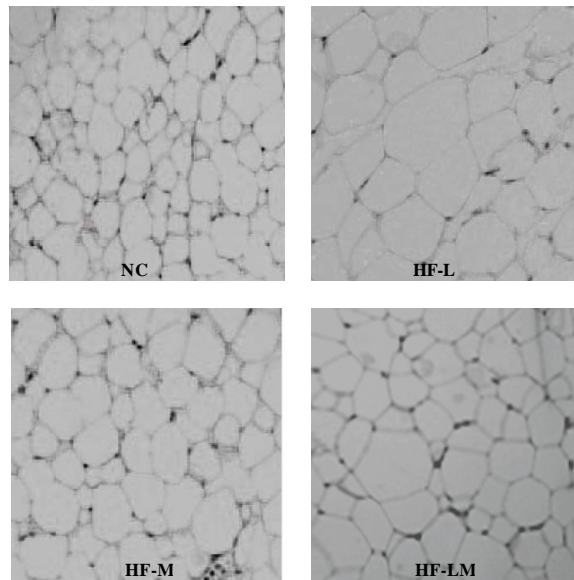


Fig. 2: Histological changes in epididymal adipose tissue. Representative sections of hematoxylin-eosin stained epididymal adipose tissue from normal chow (NC), lard based high fat diet (HFD-M), mustard oil based high fat diet (HFD-M), lard + mustard oil based high fat diet (HFD-LM) fed animals (magnification, x100)

**Effect on oral glucose tolerance test (OGTT):** The serum glucose concentrations at various time points after the oral glucose administration are shown in Fig. 1. The postprandial blood glucose levels in the HF-L group was significantly ( $p < 0.05$ ) greater than NC group 60 min after the glucose administration. However, no significant difference in serum glucose response was observed at any time point after the glucose administration to the HF-LM and HF-M groups when compared with the NC group.

**Effect on adipocyte size:** Histological examination of epididymal adipose tissue revealed that lard based HFD rats had markedly increased adipocyte size (Fig. 2) than normal chow-fed rats. However, HFD-M fed rats had adipocyte size almost similar to NC fed rats. HFD-LM had markedly suppressed epididymal adipocyte size compared to HFD-L fed rats (Fig. 2).

## DISCUSSION

The present study was undertaken to determine whether chronic administration of mustard oil can prevent the development of obesity and associated biochemical anomalies in wistar rats. We observed that rats fed a mustard oil based high fat diet for 10 weeks were associated with decreased body weight gain, less adiposity and improved glucose and lipid homeostasis. To our knowledge, this is the first study describing the antiobesity effect of mustard oil in wistar rats.

Obesity is an increasing health problem, with the overeating of fat-rich diets implicated in the rise of obesity (Dreon *et al.*, 1988; West *et al.*, 1992; Boozer *et al.*, 1995; Levin *et al.*, 1997). Together with elevated body fat, dietary obesity induced by exposure to long term high-fat diet consumption causes disturbance in glucose and lipid homeostasis (Yaquooob *et al.*, 1995; Briaud *et al.*, 2002; Sivabalan and Menon, 2008; Norazmir *et al.*, 2010). In the present study, the lard based HF diet animals gained more weight, had higher glucose and lipid levels and showed less efficient glucose tolerance than normal animals. These results are in agreement with a large number of earlier studies of HF diets based on this fat type (Buettner *et al.*, 2000; Yaspelkis *et al.*, 2001; Gustafson *et al.*, 2002). Dietary fat being calorically dense and extremely palatable is easily overconsumed because it causes less satiety than other macronutrients (Rolls and Hammer, 1995). Moreover, diet induced thermogenesis following high fat diets (particularly saturated fats) is much lower than following high protein and high carbohydrate diets which triggers positive energy balance and promote fat accumulation (Schutz *et al.*, 1989; Flatt, 1995). Thus, the observed obesity and subsequent biochemical disturbances in HF-L animals may be due to high caloric intake and inefficient diet induced thermogenesis. This contention is supported by the results obtained in the present study that compared to normal animals HF-L animals had higher average daily energy intake without significant increased in core body temperature, an index of thermogenesis.

In the present study, animals on mustard oil based HF diets continued to gain body weight at a slower rate and had decreased visceral fat accumulation compared to lard based HF diet rats. The observed lesser adiposity in HF-M animals than other HF diet groups cannot be explained by difference in caloric intake or fat absorption as significant differences between these parameters in three HF diet types were not detected. However, the isocaloric HF diets employed in the present study were containing distinct types of dietary fat with different levels of saturated and polyunsaturated fatty acids. The HF-L, HF-M and HF-LM diets contained 14, 4.3 and 9.2% of energy as saturated fat and 4, 8 and 6% of energy as polyunsaturated fat, respectively. Clinical and animal studies have shown that polyunsaturated fat is oxidized more rapidly than saturated fat

(Delany *et al.*, 2000). Hence, a diet high in saturated fat induces a relatively low resting metabolic rate and a reduced diet-induced thermogenesis that would exacerbate weight gain (Clarke, 2000). Activation of the thermogenesis has been reported to prevent the development of HF diet induced obesity (Levin *et al.*, 1986). Moreover, increased consumption of PUFA rich diet prevents the development of obesity both in animals as well in humans when exposed to high fat diet (Sahin *et al.*, 2008; Buckley and Howe, 2009). Therefore, it is possible to speculate that decreased body weight gain observed in HF-M animals may be due to increased ratio of PUFA in the diet. This may have consequently lead to enhanced diet induced thermogenesis and suppression of fat accumulation in adipose tissue. This contention is supported by the observation that core body temperature, an index of thermogenesis, was directly proportional to the amount of PUFA in the diets. A decrease in the size of the adipocyte further support the argument that PUFA present in HF-M suppresses fat accumulation in adipose tissue. Moreover, in the present study HF-L (saturated fat diet) animals exhibited higher glucose and lipid levels and less efficient glucose tolerance while as normoglycemia, reduction in lipid levels and better glucose tolerance were observed in HF-M (polyunsaturated fat diet) animals. It is well reported that diets high in saturated fat induce insulin resistance and dyslipidemia (Storlien *et al.*, 1991) while as diets rich in PUFA have shown beneficial effects on glucose (Alsaif, 2004) and lipid metabolism (Howard *et al.*, 1995; Riserus *et al.*, 2009). Polyunsaturated fats (e.g., fish oil) are also reported to lower triglycerides and cholesterol levels (Howard *et al.*, 1995). Thus, it may be suggested that PUFA present in the mustard oil had improved glucose and lipid homeostasis in mustard oil based HF diet groups. Better glucose tolerance and decreased serum lipids in mustard oil based HF diet groups are supportive of this contention. However, in present study, the hypotriglyceridemic effect was not observed. This discrepancy may be due to the reason that previous authors report fasting triglycerides while as we have measured fed triglycerides level. Furthermore, mustard oil consumption significantly decreased the TC/HDL-C ratio which is considered the best single lipid predictor of coronary artery disease (CHD) risk (Lewington *et al.*, 2007). This observation is supported by the clinical reports that traditional mustard oil consumption is associated with lower risk of heart diseases (Singh *et al.*, 1997; Rastogi *et al.*, 2004). On the other hand, the results demonstrated that there were no general toxicities associated with consumption of mustard oil. Based on earlier report that erucic acid (mustard oil contains 48% erucic acid) may cause cardiac lipidosis in rats by accumulation of fat in the cardiac muscle (Kramer *et al.*, 1992), mustard oil was considered unsuitable for human consumption. Subsequent studies have, however, shown that it is a species specific effect and to the contrary mustard oil has been found to afford protection against cardiovascular diseases in regions where it is consumed traditionally (Charlton *et al.*, 1975; Singh *et al.*, 1997; Rastogi *et al.*, 2004).

One more important observation in the present study was that partial replacement of saturated with polyunsaturated fat attenuated the deleterious effects of saturated fat. This is based on the observation that HF-LM animals were to a large extent prevented from obesity and its associated biochemical anomalies compared to HF-L animals. Replacing saturated fat with either polyunsaturated or monounsaturated fat has been found beneficial in metabolic syndrome (Riserus *et al.*, 2009). To our knowledge this is the first study to report that replacing saturated fat with polyunsaturated fat can prevent obesity and its associated biochemical complications. This is significant with respect to Indian scenario where the wave of obesity is on rise (Wang *et al.*, 2009) and saturated fat in the form of vanaspati ghee is consumed in large quantity than in the US (Singh *et al.*, 1996). Consequently most nutritional guidelines issued by government and health



organizations recommend increased consumption of foods rich in PUFA, especially omega 3 PUFA (Lichtenstein *et al.*, 2006). Fish oil is the richest dietary source of omega 3 PUFA (Lichtenstein *et al.*, 2006) and its beneficial effects are attributed to omega 3 PUFA. Vegetable oils such as mustard oil containing  $\alpha$  linolenic acid can serve as alternative sources of omega 3 PUFA in areas where fish intake is low. Thus mustard oil can be a feasible and cost-effective alternative for preventing the obesity, particularly in areas where health care resources are limited.

## CONCLUSION

In conclusion, the results from the present study suggest the preventive role of mustard oil in obesity. Consumption of mustard oil is associated with low body weight gain, less visceral fat accumulation and improved glucose and lipid homeostasis. The potential for mustard oil to counteract the obesity and its negative effects appears substantial and deserves further investigation.

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