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Growth Performance, Fasting Plasma Glucose and Lipid Profile of Sprague-Dawley Rats Fed Different Levels of Omani Halwa

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Abstract: Omani halwa is a traditional sweet delicacy. The present study evaluated the effects of feeding different levels of Omani halwa on the growth performance, fasting plasma glucose, glycated hemoglobin (HbA1c) and plasma lipid profile of Sprague-Dawley rats. Forty two, 4 weeks old male SD-rats were randomly divided into 7 groups containing 6 rats in each. Six experimental diets, (in which the normal rat chow was replaced with either white or black Omani halwa at 10, 15 and 20% level), were prepared and fed for 10 weeks. The group fed on rat chow acted as control. No significant ($p<0.05$) differences were observed in the feed consumption and growth performance of rats fed diets containing different levels and types of halwa. Significant ($p<0.05$) differences were observed in fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) values in rats fed different experimental diets. Diets containing 20% of halwa showed significantly ($p<0.05$) higher FPG and HbA1c values. Similarly the rats fed diets containing 20% of halwa showed significantly ($p<0.05$) higher lipid profile i.e., TC, TG, HDL-C and LDL-C values as compared to control. However, the TC/HDL-C ratio did not differ significantly ($p>0.05$). The plasma creatinine levels differed significantly ($p<0.05$) whereas the plasma albumin levels did not differ ($p>0.05$) in rats fed various experimental diets. Feeding Omani halwa at 15% level in diets did not affect ($p>0.05$) the growth, fasting plasma glucose, glycated hemoglobin (HbA1c) and lipid profile of rats.

Key words: Omani halwa, feeding rats, blood glucose, HbA1c, lipid profile

INTRODUCTION

Diet is one of the many lifestyle factors that influences the risk of developing non-communicable diseases (NCDs), the leading cause of death and disabilities worldwide (Wagner and Brath, 2012; Siegel *et al.*, 2014; Vineis *et al.*, 2014). In the Arab-Gulf countries, including Oman, the recent improvements in the socio-economic status of the people, as well as an increased availability of a wide variety of foods has led to drastic changes in their lifestyle and food habits. The consumption of plant based natural diets has reduced, whereas the use of high fat, energy-dense, highly processed ready-made foods has increased (Ali *et al.*, 2013a). The prevalence of obesity, impaired glucose tolerance, type 2 diabetes mellitus, hypertension, hyperlipidemia and the rate of cancer in Omani population are on the increase (Asfour *et al.*, 1995; Al-Riyami *et al.*, 2012). Although infant mortality rates have decreased significantly in Oman, the adult death rate due to NCDs, such as diabetes, cardio-vascular diseases and cancer is on the increase (Al-Lawati *et al.*, 2008; Al-Riyami *et al.*, 2012). The prevalence of metabolic syndrome (MetS) in the Gulf Cooperative Council (GCC) states, including Oman, is also on the increase and is generally higher in women than in men (Al-Lawati *et al.*, 2003; Sliem *et al.*, 2012). The available evidence indicates that the most cases of MetS may be attributed to inappropriate dietary patterns and sedentary life style (Aballay *et al.*, 2013; Ahluwalia *et al.*, 2013; Calton *et al.*, 2014).

Considerable epidemiological evidence exits concerning the dietary lipids and sugar intake as the etiological factors in obesity. High-fat diets and high intake of dietary sugars lead to excessive energy intakes and are strongly linked to the pandemic of obesity that is directly associated in the causation of non-communicable diseases such as diabetes, hypertension, cardiovascular diseases and cancers (Al-Khudairy *et al.*, 2013; Bolsinger *et al.*, 2013; Schwab *et al.*, 2014). Humans are known for their passion of eating sweets and every nation has its own delights that are synonymous with their ethnic, cultural, regional and religious practices. The frequent consumption of sweets, in particular by children can lead to excessive intake of calories and is the risk factor for dental caries, weight gain, obesity and cardiovascular risk factor in adults (Schneider *et al.*, 2013; Nicklas and O'Neil, 2014).

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The Omani halwa is a traditional Omani sweet delicacy that mainly consists of butter oil, starch and sugar. It is not only served on special occasions but is also offered and consumed together with Omani coffee as a gesture of hospitality in everyday life. The details about its ingredient composition, methods of preparation, chemical composition and glycemic index value have already been reported by us in a previous study (Ali *et al.*, 2013b). Although the glycemic index values of different types of Omani halwa were found to be within the low GI category (<55), its total fat and carbohydrate contents were found to be about 14 and 73%, respectively (Ali *et al.*, 2013b).

Being an energy-dense sweet dish (403-421 kcal/100 g) containing high amounts of sugar and fat (mainly saturated fatty acids), the increased daily intake of Omani halwa may lead to over consumption of daily discretionary calories allowances and ultimately to overweight and obesity. The American Heart Association recommends reductions in the intake of added sugars to half of the discretionary calorie allowance (Johnson *et al.*, 2009). No data is available on the effect of eating Omani halwa, in particular its effects on weight gain, obesity and other physiological parameters in humans or animals. Experimental dietary models in rats have been developed to mimic the features of over consumption and to study the weight gain, obesity, hyperlipidemia, insulin resistance and other markers of cardiovascular diseases (la Fleur *et al.*, 2013; Munshi *et al.*, 2014). The present study was therefore conducted to determine the effects of feeding different levels of traditional white and black Omani halwa on the growth performance, glycated hemoglobin (HbA1c), fasting plasma glucose and lipid profile of Sprague-Dawley rats.

MATERIALS AND METHODS

Preparation of experimental diets and animal experiment: Representative samples of two different types of Omani halwa (white and black) were collected from the local market. The details about the preparation process and chemical composition of Omani halwa were given in our previous paper (Ali *et al.*, 2013b). The percentage moisture, crude protein, total fat, ash, crude fiber and nitrogen free extract (NFE) in white and black halwa was 11.81, 12.13, 0.28, 0.44, 13.84, 12.94, 0.68, 0.57, 0.15, 0.27, 73.24 and 73.65, respectively. The energy value of white and black halwa was 418.6 and 403.8 kcal/100 g, respectively. The normal rat chow was obtained from the local Oman Flour Mills Company (S.A.O.G.), Muscat, Oman. The proximate composition of normal rat chow indicated 11.7% moisture; 19.5% crude protein; 4.9% crude fiber; 2.7% total fat; 6.4% ash and 54.8% nitrogen free extract (NFE). The gross energy value of normal rat chow was 322 kcal/100 g. Six experimental diets, in which the rat chow was replaced with either white or black Omani halwa at 10, 15 and 20% level respectively, were prepared. The substitution of rat chow with either white or black Omani halwa at 10, 15 and 20% level in experimental diets increased their gross energy values by 6.20, 9.34 and 12.52 or 4.78, 7.17 and 9.56 kcal/100 g, respectively. The diets were randomly allocated to different experimental groups. Forty-two (4 weeks old) male Sprague-Dawley rats with an average weight of (72.9±3.6) were used for this study. They were randomly divided into 7 groups containing 6 rats in each group. The rats were kept in individual cages with wire-meshed floors equipped with feeders to facilitate the assessment of food intake and food spillage. The rats were housed in a temperature controlled room (24±1°C) under the standard housing conditions with a 12 h light-dark cycle. The rats were given a weighed mount of diet twice daily to ensure *ad libitum* feeding. The leftover food was collected daily and the daily food intake was recorded. The rats had 24 h continuous access to water. The animals were weighed weekly and their body weight gain was recorded. The experiment lasted for 10 weeks. The group fed on normal rat chow acted as control. The study was approved by the Animal Research Ethics Committee of Sultan Qaboos University, Muscat, Oman.

Blood sampling and analysis: At the end of the 10 weeks experimental period after an overnight fast (12 h), the rats were euthanized by cervical dislocation and the blood was collected in heparinized tubes. The blood samples were centrifuged to collect the plasma, which was then analyzed for determining the glucose, total cholesterol, triglycerides, HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), TC/HDL-C ratio, creatinine and albumin by enzymatic methods using an Alcyon 300 automated biochemical analyzer (Abbott Laboratories, USA). The instrument was calibrated and validated using the standard solutions before the actual samples were analyzed. Fresh blood was used to determine the glycated hemoglobin (HbA1c). The glycated hemoglobin (HbA1c) was measured with the help of NycoCard HbA1c method. NycoCard HbA1c is a Boronate Affinity Assay. The kit contains test devices, which include a porous membrane filter, test tube pre-filled with reagent and a washing solution. The reagent contains agents that lyse the erythrocytes and specifically precipitate the hemoglobin. The reagent also contains a blue boronic acid conjugate that binds cis-diols of glycated hemoglobin. When blood is added to the reagent mixture in the test device, the erythrocytes immediately lyse and all the hemoglobin is precipitated. The conjugate-bound and unbound glycated hemoglobin remains on top of the filter in the test device. Any excess of the coloured conjugate is removed with the help of washing solution. The precipitate is then evaluated by measuring the blue (glycated hemoglobin) and the red (total hemoglobin) colour intensity with the help of NycoCard Reader II. The ratio between the glycated and total hemoglobin is proportional to the percentage of HbA1c in the sample (Axis-Shield PoC AS, 2003).

Statistical analysis: The results obtained were analyzed statistically using descriptive statistics and one way analysis of Variance (ANOVA). The means were compared by least significant difference (LSD) using Duncan's Multiple Range Test (DMRT) as described by Snedecor and Cochran (1989). The data was analyzed with the help of statistical software package, SPSS v. 16. The data is presented as mean±SD and the values p<0.05 were considered as significant.

RESULTS AND DISCUSSION

Dietary intake and growth performance of rats: The data on the average weekly feed intake and average weekly body weight gain in rats is shown in Fig. 1 and 2. No significant (p<0.05) differences were observed in the average weekly consumption of control and experimental diets. The rats fed diets containing different levels of Omani halwa showed slightly higher intakes as compared to control (normal rat chow) in the beginning of experiment. However, as the experiment progressed, the dietary intake was almost similar in rats fed diets containing different levels of Omani halwa as compared to control (Fig. 1). This may be attributed to improved taste and flavour of the experimental diets because of Omani halwa that is mainly composed of butter oil, starch and sugar. The substitution of rat chow at 10, 15 and 20% levels with Omani halwa increased the energy value of experimental diets from 4.78 to 12.52 kcal/100 g. There was a linear trend in the body weight gain of rats in all the experimental groups (Fig. 2). No significant (p<0.05) differences were observed in body weight gain and the average growth rate of rats fed various experimental diets as compared to control group. The weight gain in rats was also well correlated with their feed intake. As the animals were offered diets *ad-libitum*, they might have compensated their energy intake by adjusting their dietary intake and ultimately didn't show any significant differences in weight gain. The average growth rate of rats decreased significantly (p<0.05) with the progress in the age of the animals. Our results are in line with those reported by Wilkes *et al.* (1998), who observed that the body weights of rats, fed either high fat or high carbohydrate diets, did not differ at any time during a 3 week study period and the rats in all the dietary groups consumed similar amounts of food throughout the study. Our results are also in line with the previous findings reported by Leonardi *et al.* (2010), Caton *et al.* (2012) and Jurgonski *et al.* (2014).

Fasting plasma glucose (FPG) and blood glycated hemoglobin (HbA1c): The results on fasting plasma glucose (FPG) levels and blood glycated hemoglobin (HbA1c) values of rats fed control and experimental diets are shown in Table 1. Diets containing different concentrations of Omani halwa significantly (p<0.05) affected both the FPG and HbA1c concentration in rats. The blood glucose levels in rats fed various experimental diets ranged between 114.2 to 135.7 mg/dL. The highest value was observed in rats fed diet containing 20% of white halwa. No significant (p<0.05) differences were however observed in the FPG values of rats fed various levels of black Omani halwa. The glycated hemoglobin values in rats fed various experimental diets ranged from 3.02 to 3.48% (Table 1) and differed significantly (p<0.05). The rats fed diet containing 20% of black halwa showed the highest HbA1c values (3.48%).

Table 1: Fasting plasma glucose (mg/dL)* and blood glycated hemoglobin (%) values in rats fed various levels of Omani halwa.

Group	Fasting plasma glucose	HbA1c (%)
Control	119.83±12.21[b]	3.02±0.27[c]
BH10%	114.17±8.450[b]	3.20±0.14[bc]
BH15%	131.67±13.32[ab]	3.17±0.27[bc]
BH20%	123.33±18.29[b]	3.48±0.10[a]
WH10%	128.67±12.56[ab]	3.33±0.22[ab]
WH15%	117.50±16.59[b]	3.23±0.26[bc]
WH20%	135.67±16.06[a]	3.30±0.13[ab]

*Values are means±SD; a, b, c: Different alphabets in the same column means significantly different at 5% (p<0.05)

The relationship between the daily dietary nutrient composition, in particular the intake of fats and carbohydrates, with glucose homeostasis has been recognized since long (Heer and Egert, 2014; Kawamura *et al.*, 2014). High fat diets have been shown to produce quantitative effects on glucose homeostasis in experimental animals and result in impaired glucose tolerance (Lichtenstein and Schwab, 2000). Type of fat e.g., saturated fats, relative to monounsaturated and polyunsaturated fats appear to be more deleterious with respect to fat induced insulin sensitivity, inflammation and other risk factors for non-communicable diseases (Chisholm and O'Dea, 1987; Schwab *et al.*, 2014). Metabolic studies suggest that high fat diets containing higher proportions of unsaturated fats result in better measures of glucose metabolism than high carbohydrate diets (Pedersen *et al.*, 1991). Fasting plasma glucose is commonly used as a marker for the evaluation of glycemic control in diabetic patients. However, it is only a short-term indicator of diabetic control. The glycated hemoglobin (HbA1c) is considered as a more useful biomarker in assessing the long-term glycemic control as it reflects the retrospective time averaged glycemic status in individuals (Davidson *et al.*, 2008). HbA1c has also been shown to be associated with increased risk of CVD and cancer incidence (Kim *et al.*, 2010; Haring *et al.*, 2014). Our results are in agreement with these findings.

Table 2: Fasting plasma lipid profile (mg/dL)*in rats fed various levels of Omani halwa

Group	TC	TG	HDL-C	LDL-C	TC/HDL-C
Control	68.00±4.24[d]	35.67±4.68[a]	28.83±5.56[c]	32.00±5.57[b]	2.36±0.43[NS]
BH10%	84.50±4.55[b]	32.50±5.58[ab]	36.02±7.14[b]	41.98±5.13[ab]	2.34±0.62[NS]
BH15%	79.50±7.87[c]	28.17±6.31[b]	35.83±7.11[b]	38.07±7.45[b]	2.22±0.67[NS]
BH20%	100.31±5.57[a]	33.00±5.53[ab]	39.53±9.53[ab]	54.18±7.32[a]	1.85±1.09[NS]
WH10%	75.03±9.00 [c]	27.00±6.61[b]	33.00±4.73[bc]	36.60±8.55[b]	2.27±0.58[NS]
WH15%	85.67±9.7[b]	26.67±7.91[b]	42.83±5.19[a]	37.51±7.19[b]	2.00±0.41[NS]
WH20%	85.33±8.5[b]	36.17±6.33[a]	38.5±9.80[ab]	39.61±7.31[ab]	2.40±1.05[NS]

*Values are means±SD. a, b, c: Different alphabets in the same column means significantly different at 5% ($p<0.05$)

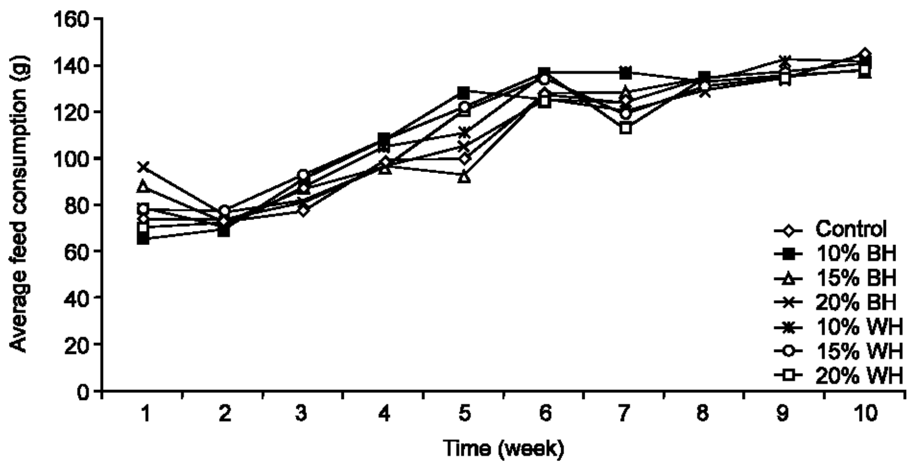

Fig. 1: Average weekly feed consumption (g) of rats fed various experimental diets

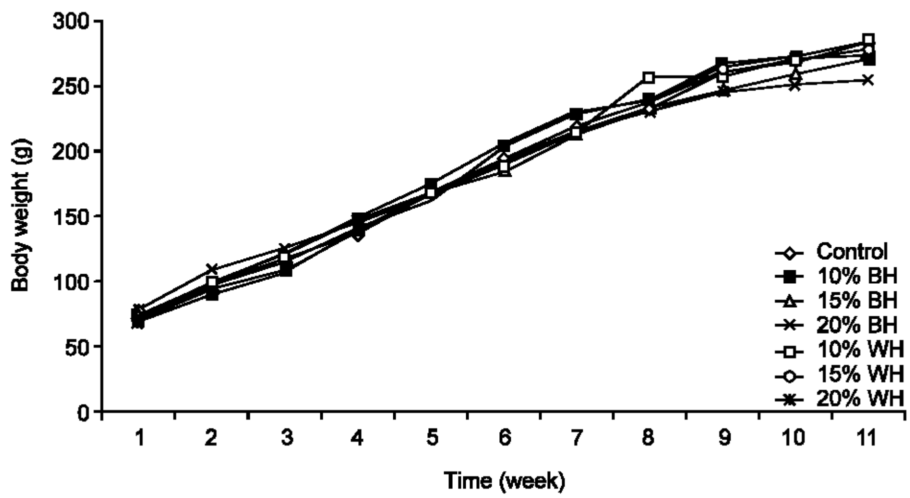

Fig. 2: Average weekly body weight gain (g) in rats fed various experimental diets

The fasting plasma glucose concentrations in rats fed different concentration of halwa correlated well with the HbA1c values and indicated the differences in the glucose homeostasis of the rats. Glycation of hemoglobin is a non-enzymatic process and the degree of glycation reflects the time-averaged concentration of glucose over the previous 3 to 10 weeks period. The results of this study could be attributed to prolonged exposure of rats to higher dietary fat and carbohydrate concentrations contributed from halwa that might have affected the pancreatic sensitivity causing a reduction in insulin level in the blood stream and consequently the elevated HbA1c levels. It has been shown that rats fed higher levels of fat developed hyperglycemia that was accompanied by impaired glucose tolerance (Zaragoza-Hermans, 1974). Higher intake of dietary fat can induce a reduction in glucose uptake and oxidation, or its conversion to fatty acids that may cause a decrease in

Table 3: Fasting plasma Creatinine and Albumin levels (mg/dL)* in rats fed various levels of Omani halwa

Group	Creatinine	Albumin
Control	0.58±0.14[a]	4.15±0.99[NS]
BH10%	0.50±0.12[b]	4.00±0.47[NS]
BH15%	0.54±0.05[ab]	3.67±1.65[NS]
BH20%	0.40±0.15[c]	3.70±1.73[NS]
WH10%	0.42±0.04[c]	4.12±0.34[NS]
WH15%	0.49±0.11[b]	4.15±0.79[NS]
WH20%	0.36±0.03[c]	4.07±0.86[NS]

Values are means±SD; NS: Non-significant. a,b,c: Different alphabets in the same column means significantly different at 5% (p<0.05)

insulin sensitivity (O'Dea *et al*., 1989). Shimakawa *et al*. (1993) reported that the percentage of energy consumed as fat was significantly correlated with HbA1c levels in insulin-dependent male diabetic patients. Our results are in agreement with these findings.

Plasma lipid profile: The results on the fasting plasma lipid profile of rats fed various experimental diets are shown in Table 2. The components of lipid profile included total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and total cholesterol to high density lipoprotein cholesterol ratio (TC/HDL-C). Significant (p<0.05) differences were observed in the TC, TG, HDL-C and LDL-C values of rats fed various levels of Omani halwa as compared to control group. The TC values in rats fed various experimental diets ranged between 68.0 to 100.3 mg/dL. The highest TC values were observed in rats fed diet containing 20% of black Omani halwa, followed by diets containing 15 and 20% of white Omani halwa (Table 2). The TG values in rats fed various experimental diets ranged between 26.7 to 36.2 mg/dL. The diet containing 20% of white halwa showed the highest TG values in rats. However, the TG values in rats fed diets containing 20% of black and white Omani halwa didn't differ significantly (Table 2). The HDL-C values in rats fed various experimental diets ranged between 28.8 to 42.83 mg/dL. The highest HDL-C value (42.83 mg/dL) was observed in rats fed 15% of white halwa followed by 20% of black and white halwa (Table 2). The LDL-C values ranged between 32.0 to 54.18 mg/dL. The highest LDL-C value was observed in rats fed diet containing 20% of black halwa (Table 2). No significant (p<0.05) differences were observed in TC/HDL-C values of rats fed various experimental diets as compared to control as well as among each other (Table 2).

Plasma lipid profile identifies a panel of risk factors that are considered to be associated with the risk of cardiovascular diseases (CVD). Out of these risk factors, total cholesterol has traditionally been estimated to study the effects of dietary lipids on developing the risk of CVD (Munshi *et al*., 2014). Relying on cholesterol alone, as a marker of CVD risk is however not enough, as the disease picture may change if the HDL-C and other associated risk factors are not taken into account (Mensink *et al*., 2003). Measuring the ratio of TC to HDL-C is now considered a more reliable marker than the TC alone in assessing the risk of coronary artery diseases. High serum triglyceride levels have also been recognized as an independent risk factor and biomarker for cardiovascular diseases (Sarwar *et al*., 2007; Schwab *et al*., 2014). The rats in the control group showed the lowest TC value whereas the highest TC values were observed in rats fed diet containing 20% of black halwa (BH20% group). Similarly the rats fed diet containing 20% of white and black halwa showed higher TG values. The differences in the TC and TG values of rats fed various levels of Omani halwa could be attributed to the higher amount of dietary fat consumed by the animals. Wang *et al*. (2003) demonstrated that long term feeding with a high fat/low carbohydrate diet might produce adverse effects on the plasma lipid profile, with elevated levels of triglycerides, NEFA, TC and reduced levels of HDL cholesterol. High-fat/low-carbohydrate diets have been shown to raise the plasma cholesterol levels associated with the formation of large low-density lipoprotein (LDL) particles (Guay *et al*., 2012), which may adversely increase the cardiovascular risk profile (Gerber and Berneis, 2012). The short term exposure to high fat diets upregulates the key genes involved in lipid and lipoprotein metabolism at the enterocyte level (Tremblay *et al*., 2013). Low carbohydrate and high fat diets have also been shown to promote the hepatic steatosis in rats (Leonardi *et al*., 2010). On the other hand, the high-carbohydrate/low-fat diets may dictate the development of type 2 diabetes and may also induce dyslipidemia and metabolic syndrome (Bolsinger *et al*., 2013; Rajaie *et al*., 2014). The type of fat rather than the total amount of fat in the diet also plays an important role in determining the risk of coronary heart diseases (CHD) (Stradling *et al*., 2013, Jurgonski *et al*., 2014). Cintra *et al*. (2006) observed that rats fed high fat diets, in particular containing saturated fatty acids showed significantly (p<0.05) higher levels of total serum cholesterol, larger lipid molecules and increased cholesterol deposition in their livers as compared to those fed the normal diet. Consumption of high amounts of carbohydrates at the expense of polyunsaturated fatty acids increased the total and LDL-cholesterol, reduced the HDL-C and promoted the triglycerides concentration (Hauner *et al*., 2012). The quantity of ingested carbohydrates also affects the quality of LDL particles by decreasing their size and increasing their density, which may increase the risk of cardiovascular profile (Gerber and Berneis, 2012). Our results on the lipid profile of SD-rats are in agreement with these results reported in the literature.

Plasma creatinine and albumin: The results of the plasma creatinine and albumin values in rats fed various experimental diets are shown in Table 3. The creatinine levels in rats fed various experimental diets ranged between 0.36 to 0.58 mg/dL. The control group showed the highest creatinine level whereas the rats fed diets containing 20% white halwa showed the lowest creatinine level. Although significant ($p<0.05$) differences were observed in creatinine values of the rats fed various experimental diets, the values were within the normal range. Jayapalan *et al.* (2000) reported that serum creatinine was 49% higher in male rats fed the high fat diet. Creatinine is a catabolic product of creatinine phosphate, which is important in the skeletal muscle contraction. Creatinine is excreted by the kidneys and is therefore directly proportional to the renal function. Creatinine test is therefore used to diagnose the impaired renal functions. The albumin levels of the rats fed various experimental diets ranged from 3.67 to 4.15 mg/dL. No significant ($p<0.05$) differences were observed in albumin values of rats fed various experimental diets (Table 3). Serum albumin is the main protein synthesized by the liver and plays an important role in maintaining the colloid osmotic pressure and transporting fatty acids, toxins, drugs and bilirubin. Chronic renal and liver diseases may lead to long-term changes in the plasma concentrations of albumin (Baumgartner *et al.*, 1996). The results of this study indicated that the albumin levels in rats fed different levels of Omani halwa were within the normal range.

Conclusion: Omani halwa is a traditional sweet delicacy that is served in Omani homes together with Omani coffee as a gesture of cultural hospitality. The present study evaluated the effects of feeding different levels of Omani halwa on the growth performance, fasting plasma glucose, glycated hemoglobin (HbA1c) and plasma lipid profile of male Sprague-Dawley rats. Feeding Omani halwa at 15% level in diets did not affect ($p>0.05$) the growth, fasting plasma glucose, glycated hemoglobin (HbA1c) and lipid profile of rats. Being an energy-dense traditional sweet dish containing high amounts of sugar and fat, mainly saturated fatty acids, the habitual increased intake of Omani halwa may lead to over consumption of daily discretionary calorie allowances and ultimately to weight gain and obesity. Based on the results of this study it is suggested that only moderate amounts of Omani halwa should be consumed daily to avoid weight gain and the risk of developing non communicable chronic diseases (NCDs).

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REFERENCES

Aballay, L.R., A.R. Eynard, P. Diaz Mdel, A. Navarro and S.E. Munoz, 2013. Overweight and obesity: a review of their relationship to metabolic syndrome, cardiovascular disease and cancer in South America. Nutr. Rev., 71: 168-179.

Al-Khudairy, L., S. Stranges, S. Kumar, N. Al-Daghri and K. Rees, 2013. Dietary factors and type 2 diabetes in the Middle East: what is the evidence for an association? A systematic review. Nutr., 5: 3871-3897.

Al-Lawati, J.A., A.J. Mohammed, H.Q. Al-Hinai and P. Jousilahti, 2003. Prevalence of the Metabolic Syndrome Among Omani Adults. Diabetes Care, 26: 1781-1785.

Al-Lawati, J.A., R. Mabry and A.J. Mohammed, 2008. Addressing the Threat of Chronic Diseases in Oman. Preventing Chronic Disease: Public Health Research, Practice and Policy, Centers for Disease Control and Prevention, USA., 5: 1-7.

Al Riyami, A., M.A.D. Elaty, M. Morsi, H. Al Kharusi, W. Al Shukaily and S. Jaju, 2012. Oman World Health Survey: Part 1-Methodology, Sociodemographic Profile and Epidemiology of Non-Communicable Diseases in Oman. Oman Med. J., 27: 425-443.

Ahluwalia, N., V.A. Andreeva, E. Kesse-Guyot and S. Hercberg, 2013. Dietary patterns, inflammation and the metabolic syndrome. Diabetes Metab., 39: 99-110.

Ali, A., B.S. Al-Belushi, M.I. Waly, M. Al-Moundhari and I.A. Burney, 2013a. Dietary and lifestyle factors and risk of non-Hodgkin's lymphoma in Oman. Asian Pacific J. Cancer Preven., 14: 841-848.

Ali, A., K.M. Al-Zuhaibi, M.I. Waly, A. Al-Alawi, J. Al-Sabahi and D. Sankar, 2013b. Chemical composition, fatty acids content and glycemic index of two different types of Omani halwa. Pak. J. Nutr., 12: 753-760.

Asfour, M.G., A. Lambourme, A. Sokiman, S. Al-Belushi, D. Al-Asfoor, A. Bold, H. Mahtab and H. King, 1995. High prevalence of diabetes mellitus and impaired glucose tolerance in the Sultanate of Oman: results of the 1991 national survey. Diabet Med., 12: 1122-1125.

Axis-Shield, PoC A.S. and Nyco Card HbAlc, 2003. P.O.Box 6863. Rodelgkka N0504, Oslo, Norway. http://www.axis-shield.com/.

Baumgartner, R.N., K.M.L. Koehler, L. Romero and P.J. Garry, 1996. Serum albumin is associated with skeletal muscle in elderly men and women. Am. J. Clin. Nutr., 64: 552-528.

Bolsinger, J., A. Pronczuk and K.C. Hayes, 2013. Dietary carbohydrate dictates development of Type 2 diabetes in the Nile rat. J. Nutr. Biochem., 24: 1945-1952.

Calton, E.K., A.P. James, P.K. Pannu and M.J. Soares, 2014. Certain dietary patterns are beneficial for the metabolic syndrome: reviewing the evidence. Nutr. Res., 34: 559-568.

Caton, S.J., M. Bielohuby, Y. Bai, L.J. Spangler, L. Burget, P. Pfluger, C. Reinel, M. Czisch, M. Reincke, S. Obici, E. Kienzle, M.H. Tschop and M. Bidlingmaier, 2012. Low-carbohydrate high-fat diets in combination with daily exercise in rats: effects on body weight regulation, body composition and exercise capacity. Physiol. Behav., 106: 185-192.

Chisholm, K.W. and K. O'Dea, 1987. Effect of short-term consumption of a high fat diet on glucose tolerance and insulin sensitivity in the rat. J. Nutr. Sci. Vitamin, 33: 377-390.

Cintra, D.E.C., A.G.V. Costa, M.D.C. Peluzio, S.L.P. Matta, M.T.C. Silva and N.M.B. Costa, 2006. Lipid profile of rats fed high-fat diets based on flaxseed, peanut, trout, or chicken skin. Nutr., 22: 197-205.

Davidson, J., C. Koro, B. Arondekar, B.H. Lee and D. Fedder, 2008. A retrospective analysis of the fasting plasma glucose and glycosylated hemoglobin and pharmacotherapy change patterns among type 2 diabetes mellitus patients. Clin. Ther., 30: 287-293.

Gerber, P.A. and K. Berneis, 2012. Regulation of low-density lipoprotein sub-fractions by carbohydrates. Curr. Opin. Clin. Nutr. Metab. Care, 15: 381-385.

Guay, V., B. Lamarche, A. Charest, A.J. Tremblay and P. Couture, 2012. Effect of short-term low- and high-fat diets on low-density lipoprotein particle size in normolipidemic subjects. Metab., 61: 76-83.

Haring, R., S.E. Baumeister, W. Lieb, von B. Sarnowski, H. Volzke, S.B. Felix, M. Nauck and H. Wallaschofski, 2014. Glycated hemoglobin as a marker of subclinical atherosclerosis and cardiac remodeling among non-diabetic adults from the general population. Diabetes Res. Clin. Pract., 27, pii:S0168-8227(14)00218-6.doi:10.1016/j.diabres. 2014.05.004. [Epub ahead of print].

Hauner, H., A. Bechthold, H. Boeing, A. Bronstrup, A. Buyken, E. Leschik-Bonnet, J. Linseisen, M. Schulze, D. Strohm and G. Wolfram, 2012. German Nutrition SocietyEvidence-based guideline of the German Nutrition Society: carbohydrate intake and prevention of nutrition-related diseases. Ann. Nutr. Metab., 60: 1-58.

Heer, M. and S. Egert, 2014. Nutrients other than carbohydrates: their effects on glucose homeostasis in humans. Diabetes Metab. Res. Rev., 2014, 8 doi: 10.1002/dmrr.2533. [Epub ahead of print].

Jayapalan, S., H. Saboorian, J.W. Edmunds and H.M. Aukema, 2000. High Dietary Fat Intake Increases Renal Cyst Disease Progression in Han: SPRD-cy Rats. J. Nutr., 130: 2356-2360.

Johnson, R.K., L.J. Appel, M. Brands, B.V. Howard, M. Lefevre, R.H. Lustig, F. Sacks, L.M. Steffen and J. Wylie-Rosett, 2009. on behalf of the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism and the Council on Epidemiology and Prevention. Dietary Sugars Intake and Cardiovascular Health: A Scientific Statement From the American Heart Association. Circulation, 120: 1011-1020.

Jurgonski, A., J. Juœkiewicz and Z. Zdunczyk, 2014. A high-fat diet differentially affects the gut metabolism and blood lipids of rats depending on the type of dietary fat and carbohydrate. Nutr., 6: 616-626.

Kawamura, M. Jr, D.N. Ruskin, J.D. Geiger, D. Boison and S.A. Masino, 2014. Ketogenic Diet Sensitizes Glucose Control of Hippocampal Excitability. J. Lipid Res., 2014, 28. pii: jlr.M046755. [Epub ahead of print].

Kim, B.J., Y.H. Kim, D.H. Sinn, K.J. Kang, J.Y. Kim, D.K. Chang, H.J. Son, P.L. Rhee, J.J. Kim and J.C. Rhee, 2010. Clinical usefulness of glycosylated hemoglobin as a predictor of adenomatous polyps in the colorectum of middle-aged males. Cancer Causes and Control, 21: 939-944.

la Fleur, S.E., M.C. Luijendijk, van der E.M. Zwaal, M.A. Brans and R.A. Adan, 2013. The snacking rat as model of human obesity: effects of a free-choice high-fat high-sugar diet on meal patterns. Int. J. Obes. (Lond), 38: 643-649.

Leonardi, D.S., M.B. Feres, G.V. Portari, M.E. Zanuto, S. Zucoloto and A.A. Jordao, 2010. Low-carbohydrate and high-fat diets on the promotion of hepatic steatosis in rats. Exp. Clin. Endocrinol. Diabetes, 118: 724-729.

Lichtenstein, A.H. and U.S. Schwab, 2000. Relationship of dietary fat to glucose metabolism. Atherosclerosis, 150: 227-243.

Mensink, R.P., P.L. Zock, A.D.M. Kester and M.B. Katan, 2003. Effect of dietary fatty acid and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am. J. Clin. Nutr., 77: 1146-1155.

Munshi, R.P., S.G. Joshi and B.N. Rane, 2014. Development of an experimental diet model in rats to study hyperlipidemia and insulin resistance, markers for coronary heart disease. Ind. J. Pharmacol., 46: 270-276.

Nicklas, T.A. and C.E. O'Neil, 2014. Fulgoni VL3rd., Snacking patterns, diet quality and cardiovascular risk factors in adults. BMC Public Health, 14: 388.

O'Dea, K., K. Traianedes, P. Ireland, M. Niall, J. Sadler, J. Hopper and M. De Luise, 1989. The effects of diet differing in fat, carbohydrate and fiber on carbohydrate and lipid metabolism in type II diabetes. J. Am. Dietetic Assoc., 89: 1076-1086.

Pedersen, O., C.R. Kahn, J.S. Flier and B.B. Kahn, 1991. High fat feeding causes insulin resistance and a marked decrease in the expression of glucose transporters (Glut 4) in fat cells of rats. Endocrinol., 129: 771-777.

Rajaie, S., L. Azadbakht, M. Khazaei, M. Sherbafchi and A. Esmaillzadeh, 2014. Moderate replacement of carbohydrates by dietary fats affects features of metabolic syndrome: a randomized crossover clinical trial. Nutr., 30: 61-68.

Sarwar, N., J. Danesh, G. Eiriksdottir, G. Sigurdsson, N. Wareham, S. Bingham, S.M. Boekholdt, K.T. Khaw and V. Gudnason, 2007. Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. Circulation, 115: 450-458.

Schneider, S., M. Jerusalem, J. Mente and F. De Bock, 2013. Sweets consumption of preschool children-extent, context and consumption patterns. Clin. Oral. Investig., 17: 1301-1309.

Schwab, U., L. Lauritzen, T. Tholstrup, T. Haldorssoni, U. Riserus, M. Uusitupa and W. Becker, 2014. Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing type 2 diabetes, cardiovascular diseases and cancer: a systematic review. Food Nutr. Res., 10: 58.

Shimakawa, T., J.H. Warram, M.G. Herrera-Acena and A.S. Kroleweski, 1993. Usual dietary intake and hemoglobin A1 level in patients with insulin-dependent diabetes. J. Am. Dietetic Assoc., 93: 1409-1415.

Sliem, H.A., S. Ahmed, K. Corder, J.W. Nicholas and U. Ekelund, 2012. Metabolic syndrome in the Middle East. Ind. J. Endori. Metab., 17: 73-78.

Siegel, K.R., S.A. Patel and M.K. Ali, 2014. Non-communicable diseases in South Asia: contemporary perspectives. Br. Med. Bull., 111: 31-44.

Snedecor, G. and G. Cochran, 1989. Statistical Methods, 8th Edition, Iowa State University Press.

Stradling, C., M. Hamid, K. Fisher, S. Taheri and G.N. Thomas, 2013. A review of dietary influences on cardiovascular health: part 1: the role of dietary nutrients. Cardiovasc Hematol Disord Drug Targets, 13: 208-230.

Tremblay, A.J., B. Lamarche, V. Guay, A. Charest, V. Lemelin and P. Couture, 2013. Short-term, high-fat diet increases the expression of key intestinal genes involved in lipoprotein metabolism in healthy men. Am. J. Clin. Nutr., 99: 54-61.

Vineis, P., S. Stringhini and M. Porta, 2014. The environmental roots of non-communicable diseases (NCDs) and the epigenetic impacts of globalization. Environ. Res., 133: 424-430.

Wagner, K.H. and H. Brath, 2012. A global view on the development of non-communicable diseases. Prev. Med., 54: 38-41.

Wang, Y., P.Y. Wang, L.Q. Qin, G. Davaasambuu, T. Kaneko, J. Xu, S. Murta, R. Katoh and A. Sato, 2003. The development of diabetes mellitus in wister rats kept on a high-fat/low-carbohydrate diet for long periods. Endocrine, 22: 85-92.

Wilkes, J.J., A. Bonen and R.C. Bell, 1998. A modified high-fat diet includes insulin resistance in rat skeletal muscle but not adipocytes. Am. J. Physio-Endocri. Metab., 275: 679-686.

Zaragoza-Hermans, N.M., 1974. Studies on the metabolic effects induced in the rat by a high-fat diet. Control of glucose metabolism in adipose tissue of fed and fasted rats. Eur. J. Biochem., 8: 579-582.