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Full Length Research Paper

Supplementary effects of vinegar on body weight and blood metabolites in healthy rats fed conventional diets and obese rats fed high-caloric diets

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The objective of this work was to evaluate the effects dietary of vinegar on body weight, and blood metabolites of healthy rats fed a conventional diet, and of obese rats fed a high-caloric diet. Twenty male Wistar rats were used. Ten of them were healthy ones (normal body condition) and were fed with conventional diet. The other 10 rats were obese and were fed with high-caloric diet. Plasma glucose, triglycerides, cholesterol, high- and low-density lipoproteins (HDL and LDL) concentrations were measured weekly from 0 to 4 weeks. Healthy and obese rats were randomly assigned to control (water) and oral vinegar (0.8 ml/kg body weight) supplementation groups. Vinegar reduced total gain and average daily weight gain in both healthy and obese rats. From 1 to 4 weeks, plasma glucose was reduced by vinegar supplementation only in obese rat. From 2 to 4 weeks, plasma triglycerides and total cholesterol were also reduced by vinegar only in obese rat. Vinegar did not affect plasma HDL and LDL. It is concluded that vinegar might serve as a protective measure to avoid excessive body weight gains and high plasma concentrations of glucose, triglycerides and cholesterol in obese patients fed high-caloric diets.

Key words: Vinegar, glucose, cholesterol, triglycerides, obesity.

INTRODUCTION

In the last decades, México has experienced a significant shift in socio-economic conditions and urbanization, with an impact on diet and sedentary life styles. A study revealed that 50% of Mexican television advertising targets children; touting high-calorie, processed foods that are known to contribute to increased body mass index (Ramirez-Ley et al., 2009). The highly processed, calorie-dense, nutrient-depleted diet frequently leads to exaggerated supraphysiological post-prandial spikes in blood glucose and lipids which induces immediate oxidant stress increasing in direct proportion to the increases in glucose and triglycerides after a meal (O'Keefe et al., 2008). These changes have been associated with the epidemiologic transition currently experienced in Mexican people, characterized by high prevalence of overweight and obesity (Barquera et al., 2007). A survey indicate that 30% of the total population in Mexico has acute or chronic obesity of which 26% is in children from 5 to 11 years old, 72% in women older than 20 years, and 67% in men (Barquera et al., 2007). Given the many costly consequences of epidemic obesity, the search for a reliable weight management treatment has taken on a new urgency. The first widely touted obesity treatments were acidic foods like vinegar (Mermel, 2004). Although, the effects of vinegar to decrease glycaemia have been inconsistent (Salbe et al., 2009), researchers have revealed that acetic acid of this functional food upregulates the expression of genes for fatty acid oxidation enzymes and thermogenic protein, thus suppressing the accumulation of body fat and liver lipids (Kondo et al., 2009), as well as increasing faecal bile acid

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excretion (Fushimi et al., 2006). The potential benefits of vinegar are in dependence of several factors, such as health status and ingestion of nutrients. For example, a recent study shows that the addition of vinegar reduces postprandial glycaemia in patients with type-2 diabetes only when it is added to a high-glycaemic index meal (Liatis et al., 2010). It was attributed to the fact that patients with diabetes exhibit (by definition) higher postprandial glucose excursions than healthy persons, which leads to a higher proportional glucose-lowering effect of any anti-hyperglycemic intervention (Liatis et al., 2010). However, evidences indicate that cider apple vinegar improved the serum lipid profile in both normal and diabetic rats by decreasing serum triglycerides, low density lipoprotein (LDL)-cholesterol and increasing high density lipoprotein (HDL)-cholesterol (Shishehbor et al., 2008). Using these approaches and because obesity also impairs glucose tolerance and hyperlipidemia (Oben et al., 2006), we hypothesized that vinegar should reduce blood glucose, cholesterol, and triglycerides in obese rats fed a high-caloric diet, but not in healthy rats fed a conventional diet. Therefore, the objective of this study was to evaluate the dietary effects of vinegar on body weight and blood metabolites of healthy rats fed with a conventional diet and of obese rats fed with a high-caloric diet.

MATERIALS AND METHODS

This study was developed using the animal care guidelines specified by the Academical Committee of the Instituto de Investigación de Zonas Desérticas of the Universidad Autónoma de San Luis Potosí, according to regulations enacted by the federal government of México (NOM-062-ZOO-1999).

Rats and feeds

Twenty male Wistar rats 8 to 9 weeks of age with an average body weight (BW) of 152.5 g were installed in a room with inverted light-dark cycle (lights on at 19:00 h, lights off at 07:00 h) and mean temperature 25 ± 3°C. Rats were allowed free access to tap water and 20 g of a commercial rodent feed (Rodent Laboratory Chow 5001, Purina, St Louis, MO, USA) by day 10. Then rats were randomly divided into two groups. One group (healthy) fed with the same quantity and type of rodent feed previously described. It represented a daily estimated intake of 60 kcal of metabolizable energy (ME). The second group was fed the same diet, but it was enriched with 10% (as fed basis) of coconut oil. In the second group (obesity), each rat was daily fed an estimation of 120 kcal ME. This preliminary period lasted for 140 days. After this time, rats fed conventional rodent feed that averaged 308.4 g BW were considered as healthy rats, and rats fed enriched rodent feed that averaged 420 g BW were considered as obese rats (Yang et al., 2010). Then, the rats were individually housed in polypropylene metabolic cages and were fed with the corresponding feed (conventional or enriched rodent feed). Both healthy and obese rats were randomly assigned to two groups, control (water) and vinegar (0.8 ml/kg BW). The unfiltered and unpasteurized vinegar (Vizana Nutrition, Monclova, Coahuila, México) was made by an acetic fermentation of a wine made from golden apple, pineapple, honey, and sugarcane. According to the manufacturer, the vinegar contains 1% acetic acid. The vinegar dose was orally administered twice a day for 4 weeks via an esophageal tube. Sterile water was used to adjust the volume at the same volume the vinegar was administered to the rats.

Animal measurements and biochemical and pathological assessments

Body weights were recorded once a week for 6 weeks, and total gain and average daily weight gain were calculated. Weekly, in the mornings, approximately 3 h into the dark cycle and 3 h after vinegar administration, whole blood samples were drawn from the tail vein. Blood samples were transferred into micro-centrifuge tubes and were centrifuged at 3000 rpm for 30 min at 4°C (Centra CL3-R, Thermo IEC, San Antonio Tx USA). The plasma was removed and was stored at -18°C until biochemical eval uation. The plasma glucose, cholesterol, high-density lipoproteins cholesterol low-density lipoproteins cholesterol, and triglycerides concen-trations were measured by enzymatic methods (Sera Pack® Plus, Bayer, Argentina) using an ultraviolet-visible (UV-VIS) spectro-photometer (Agilent 8453, Palo Alto, CA, USA).

Because there are some evidences that vinegar can cause injury to the esophagus mucose membrane (Mohamed et al., 2001; Chung, 2002), after 5 weeks of experimental study, three rats per treatment were given an overdose of sodium pentobarbital (65 mg/kg BW; Euthanyl, Maple Leaf Foods, Cambridge, Ontario, Canada) and were perfused through the heart with saline (50 ml) and 50 ml of phosphate buffered paraformaldehyde (2%). Esophagus, stomach and duodenum were removed quickly from the carcass, rinsed (saline solution), and extended. Preparations were observed for detailed independent evaluation by two pathologists to look for possible inflammation and ulcers in mucosa. As a reference for these lesions, in three rats, 4 ml/kg ethyl alcohol and 200 mg/kg sodium salicylate were orally administered by two wk via an esophageal tube according to Nam et al. (2006).

Statistical analysis

There were four treatments that resulted from a factorial arrangement of treatment 2 × 2. Factor A was the body condition with two levels: health and obese. Factor B was the vinegar with two levels: 0 and 0.8 ml/kg BW. Therefore, the evaluated treatments were health -vinegar, health +vinegar, obese -vinegar, and obese +vinegar. The data was analyzed according to a completely randomized experimental design with five repetitions and repeated measurements (0 to 4 weeks) using the “MIXED” option of SAS (1999). The covariate structure that resulted in the lowest Akaike’s information criterion was first-order autoregressive. Initial BW was not included as a covariable in the model, because we expected higher BW in obese rats than healthy rats. When a two-way interaction was observed ($P < 0.05$), the SLICE option (SAS, 1999) was used to evaluate it. Significant differences were accepted when $P \leq 0.05$.

RESULTS

As expected, obese rats had higher final BW and average daily gain than healthy rats. Final BW in all rats was not affected by vinegar supplementation. However, vinegar supplementation reduced total gain and average daily weight gain in both healthy and obese rats as compared to rats not supplemented (Table 1).

With exception of plasma glucose, from 0 to 4 weeks,

Table 1. Effects of vinegar on BW of healthy and obese rats.

Weight	Healthy		Obese		SEM	Effect
	-Vinegar	+ Vinegar	- Vinegar	+ Vinegar		
Initial BW (g)	317	320	416	425	13.3	BC
Final BW (g)	335	330	449	448	16.8	BC
Total gain (g)	18	10	33	23	6.5	BC, V
Daily gain (g)	0.64	0.36	1.18	0.82	0.232	BC, V

NS, not significant (P > 0.05); BC, body condition (healthy, obese) effect; V, vinegar effect.

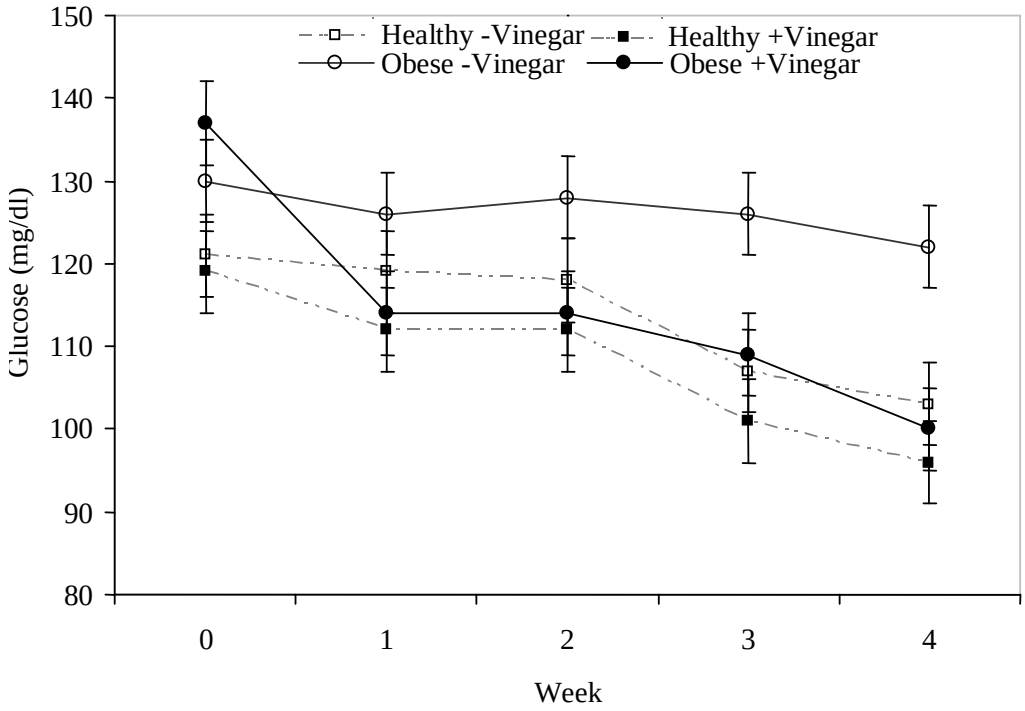

Figure 1. Plasma glucose concentrations in healthy and obese rats supplemented with vinegar.

plasma triglycerides, cholesterol, HDL and LDL concentrations were higher in obese rats than in healthy rats. In plasma glucose concentrations, interaction among week, body condition, and vinegar level indicated that from 1 to 4 weeks sampling, this sugar was reduced by vinegar supplementation only in obese rats, but not in healthy rats (Figure 1). From 0 to 1 week, plasma triglycerides (Figure 2) and total cholesterol (Figure 3) were not affected by vinegar supplementation. However, from 2 to 4 weeks, plasma triglycerides and total cholesterol were lower in obese rats supplemented with vinegar than in obese rats not supplemented. Plasma triglycerides and total cholesterol of healthy rats were not affected by vinegar supplementation. Also, vinegar supplementation did not have any effect on plasma HDL (Figure 4) and LDL (Figure 5) concentrations of both healthy and obese rats.

As compared to induced-lesion model rats, there were no lesion, inflammation, and ulcers in esophagus, stomach, and duodenum of healthy and obese rats supplemented with vinegar.

DISCUSSION

Although, vinegar became one of the earliest widely touted obesity cures, the transient success of its use as weight management agent is still controversial (Mermel, 2004). The findings of this study show that vinegar reduced total weight and average daily weight gain in both healthy and obese rats. These results are in agreement with Kondo et al. (2009) findings, who indicated that acetic acid helps to reduce body weight of high-fat-fed mice by suppressing body fat.

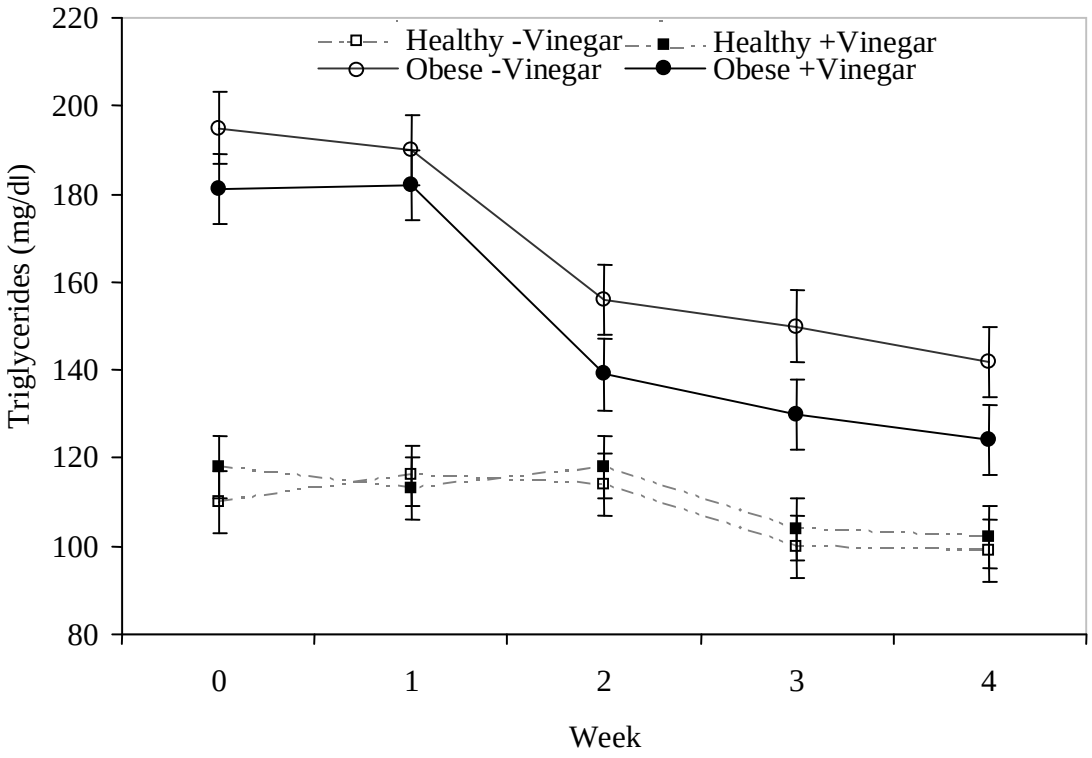

Figure 2. Plasma triglycerides concentrations in healthy and obese rats supplemented with vinegar.

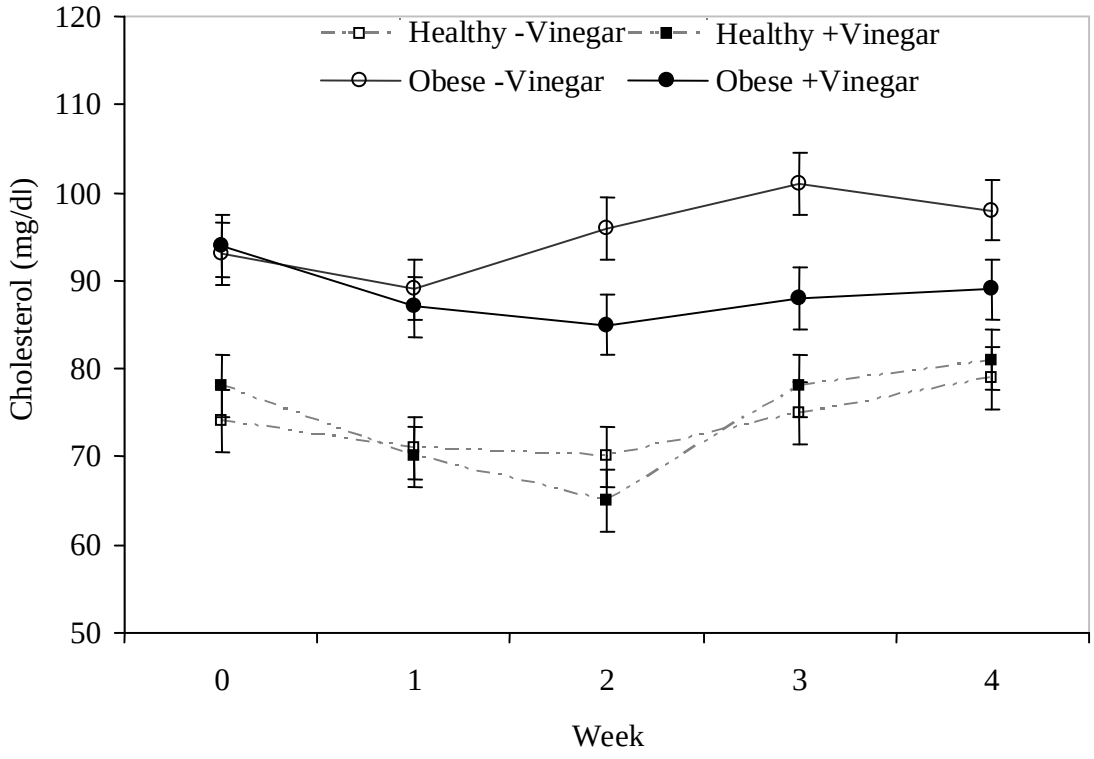

Figure 3. Plasma cholesterol concentrations in healthy and obese rats supplemented with vinegar.

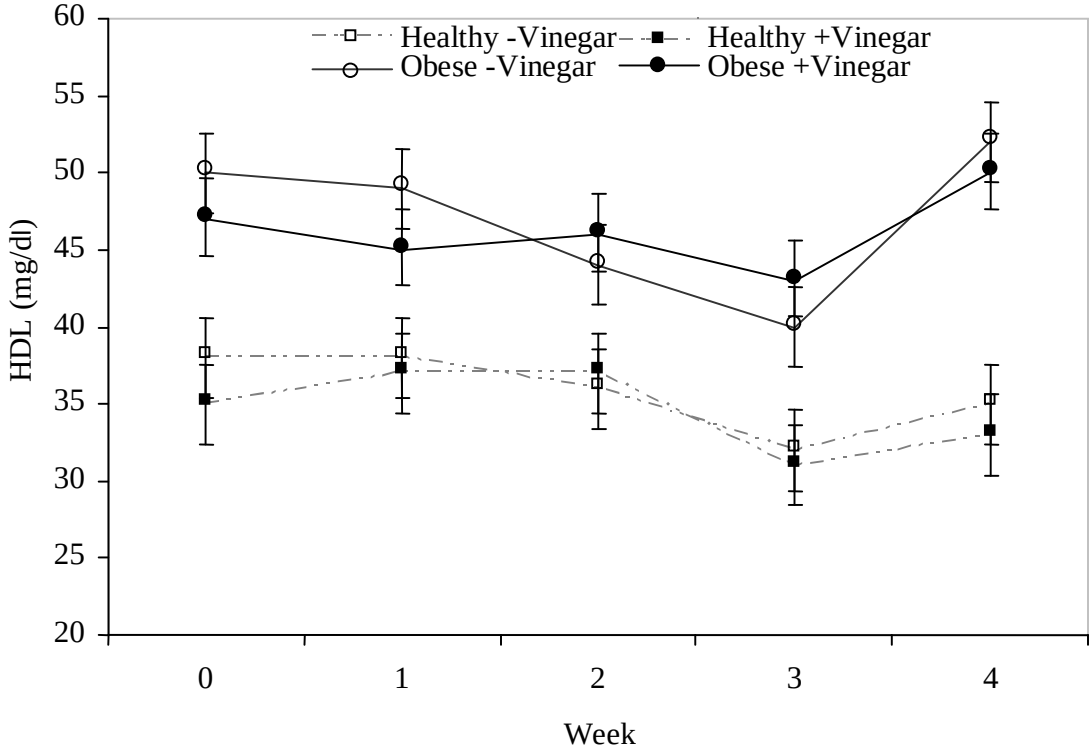

Figure 4. Plasma high-density lipoproteins (HDL) concentrations in healthy and obese rats supplemented with vinegar.

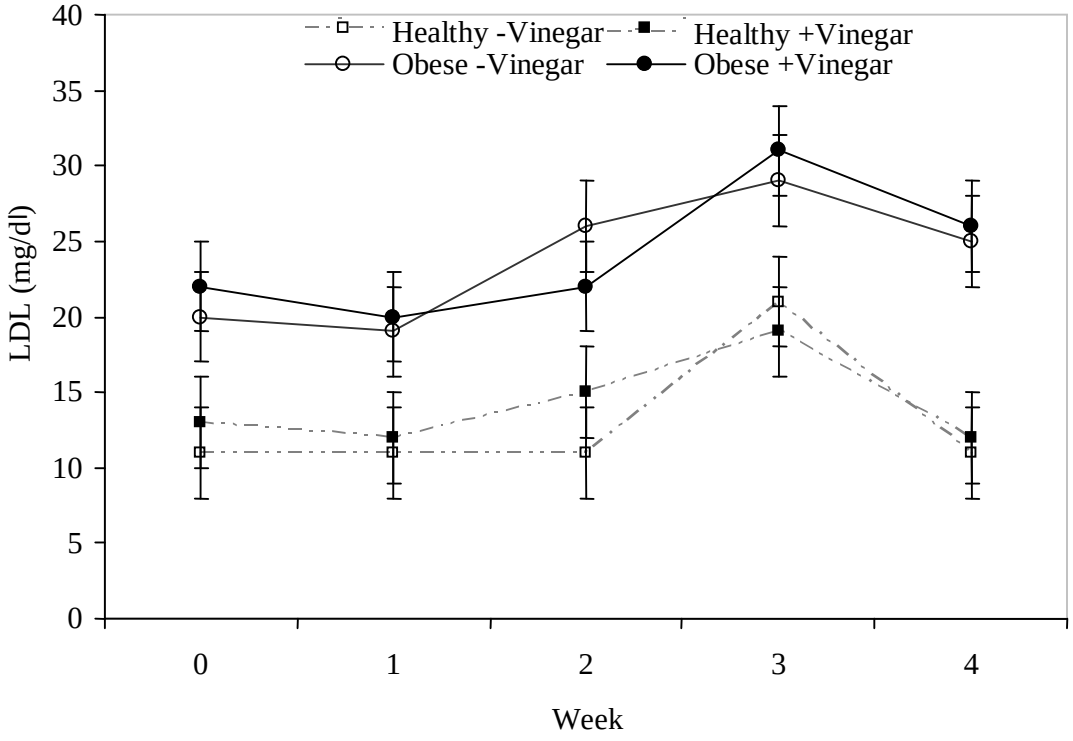

Figure 5. Plasma low-density lipoproteins (LDL) concentrations in healthy and obese rats supplemented with vinegar.

Recent evidences indicate that vinegar reduced blood glucose in humans (Mitrou et al., 2010). The mechanisms by which vinegar reduces postprandial blood glucose are still obscure. Previous studies (Hlebowicz et al., 2007) have shown that vinegar delays gastric emptying. Moreover, acetic acid has been shown to suppress disaccharidase activity (Owaga et al., 2000) and to enhance glycogen repletion in liver and muscle (Fushimi et al., 2001). However, as found in this study, the positive effects of vinegar on reduction of plasma glucose of healthy subjects are not usually evident (Ebihara and Nakajima, 1988; Mettler et al., 2009; Salbe et al., 2009). It has also been shown that the addition of vinegar to a high-glycaemic load reduces glycaemia in healthy individuals (Johnston and Buller, 2005; Östman et al., 2005). The mechanism, however, remains unclear and although it has been suggested that delayed gastric emptying might be responsible for the hypoglycemic effect of vinegar (Liatis et al., 2010), the results of relevant studies are uncertain (Brighenti et al., 1995; Liljeberg and Björck, 1998).

The fact that obese rats had higher plasma triglycerides and cholesterol than those healthy are expected finding. Dietary cholesterol has been reported to stimulate hepatic triacylglycerol biosynthesis by the reduction of fatty acid oxidation, leading to an increase in plasma triacylglycerol concentration (Fungwe et al., 1993, 1994). There are several explanations of why vinegar or acetic acid had higher impacts in rats fed high-energy or high-fat diets which were given by several authors. Fushimi et al. (2006) indicate that dietary acetic acid reduces serum cholesterol and triacylglycerol concentrations in rats fed a cholesterol-rich diet through inhibition of metabolic pathways of cholesterogenesis and lipogenesis in the liver, together with a concomitant enhancement of fatty acid oxidation and a stimulation of faecal bile acid excretion. Kondo et al. (2009) explained that acetic acid upregulates the expression of genes for fatty acid oxidation enzymes and thermogenic protein, such as acetyl-CoA oxidase, carnitine palmitoyl transferase-1, and uncoupling protein-2 by α2 5´-activated protein kinase mediation in the liver, thus suppressing the accumulation of body fat and liver lipids.

The high decrease of plasma triglycerides and cholesterol concentrations observed in this study might be attributed to the fact that obese rats exhibit higher concentrations of these blood metabolites than healthy rats, which leads to a higher proportion of glucose, cholesterol or triglycerides, rather than to the lowering effect of any anti-hyperglycaemic, anti-hypercholesterolemic or anti-hypertriglyceridemic interventions, as found in patients with type II diabetes (Liatis et al., 2010).

Vinegar supplementation did not affect plasma HDL or LDL concentrations. This could be due to the fact that the diets did not induce a high-glycaemic index, as evidenced by Shishehbor et al. (2008). These researchers found out that cider apple vinegar lowered serum LDL and increased serum HDL in normal rats as a result of the lowering effect of vinegar/or acetic acid on the glycemic index (Ostman et al., 2005). In addition, cider apple vinegar may improve lipoprotein patters not only by lowering the glycemic index, but also by its polyphenolic compounds (Shishehbor et al., 2008). Unfortunately, concentrations of polyphenols compounds in the vinegar to confirm these findings were not determined.

No lesion was detected in esophagus, stomach and duodenum, because vinegar used in this study contains less than 1% of acetic acid. Only high percentage (>5%) of acetic acid and high dose (1.02 ml/kg BW) of vinegar have induced histopathological alterations in stomach and duodenum (Mohamed et al., 2001).

It is concluded that vinegar supplementation might serve as a protective measure to avoid excessive body weight gains and concentrations in plasma glucose, triglycerides, and cholesterol in obese patient fed high-caloric diets.

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