Is Honey Really Better than Table Sugar in Body Weight Control? A Case of Study Based on the Obudu Honey and Refined Sugar Comparison

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Abstract

Background: The essential role of honey and table sugar, the world's choice sweeteners, in the global obesity epidemic and the misconceptions surrounding the preferences for the use of honey and sugar as sweeteners informed this investigation.

Aim: To determine whether the Obudu honey actually has a comparative advantage over table sugar in body weight regulation when used as food sweetener.

Methods: Thirty five male Wistar rats (100 g – 120 g) divided equally into five groups were fed with rat chow only (Normal control; NC), 15% sugar (S15%), 30% sugar (S30%), 12% honey (H12%) and 24% honey (H24%) respectively for 13 weeks; during which Fasting Blood Glucose (FBG), body weight and diet intakes were measured. The percent of sugar or honey included in the diet was based on average composition in common sweetened foods and beverages and also in equivalent calories.

Results: At the end of the study, measured parameters including diet intake, body weight gain, organs and White Adipose Tissue (WAT) weights, FBG, serum glucose, adipokines (adiponectin and leptin), and lipid profile of the honey-fed animals were not significantly different (p > 0.05) from the corresponding sugar-fed groups or the normal control. Also, histological examination of the WAT showed no marked effect of both sweeteners.

Conclusion: In short term honey sweetened diet may not exert any weight control advantage over refined sugar sweetened diet and vice versa.

Keywords: Body weight; White adipose tissues; Lipid profile; Adipokine; Honey; Table sugar

The consumption of excess table sugar has been reported to upset blood glucose homeostasis in the body [4]. Its rapid absorption from the digestive tract provides for a quick surge in blood glucose thereby stimulating a spike in insulin secretion which could lead to hypoglycemia [5]. This hypoglycemia caused by rapid fluctuations in blood glucose concentration in turn, stimulates hunger spasms and the desire to consume more calories thus, more fat storage [6]. Hence, several studies linking table sugar consumption with increased risk of medical conditions such as dental caries, obesity, cardiovascular disease, diabetes, gout, fatty liver disease and some cancers have been carried out. However, few topics in nutrition have evolved as much debate and controversy as the relationship between sugar consumption and etiology and pathogenesis of various health disorders. The potential interaction between the consumption of fructose-containing sugars and non-alcoholic fatty liver disease, weight gain, obesity and diabetes has been evaluated by a number of researchers; and these studies have linked the increased prevalence of these conditions largely to high sugar consumption. Consequently, an alternative natural sweetener, honey, has been recommended as a substitute for table sugar and it (honey) is today the World’s most commonly consumed sweetener [7].

Honey is a sweetener produced by bees particularly, Apis mellifera from the nectar of flowering plants [8] and serves as a reliable energy source for man and also for bees [9]. Although it is reported to contain at least 181 substances [10], approximately, 95% of honey dry matter is carbohydrates with the principal carbohydrates being glucose and fructose [11]. On average, honey is 1–1.5 times sweeter than refined sugar based on dry weight [12] and like refined sugar honey is naturally hygroscopic [13]. Several studies have reported the health benefits of honey, the reasons for which it is claimed to be a preferred sweetener to table sugar. These include improvement of glycaemic control in normal and diabetic rats [14]; reduction of body weight gain in experimental rats compared to sucrose [15]; reduction of blood lipids, homocysteine and C reactive protein (CRP) levels in normal and hyperlipidemic subjects. Other reports have also shown that honey stimulates insulin secretion, decrease blood glucose levels, elevates hemoglobin concentration and improves lipid profile [16].

However, calorimetric measurements have shown that natural honey is more calorie dense than refined sugar; A tablespoon full of refined sugar and of honey contains 49 and 68 calories, respectively; i.e. 1:1.5 calorie ratio [16]. Furthermore, the Glycemic Index (GI) of honey varies from 32 to 85 as against 68 ± 5 of refined sugar [7,17]; and diets with substantially high upper margins of glycemcic index (such as honey) may be detrimental to health and some metabolic pathologies like diabetes due to elevated and/or

Abbreviations

WAT: White Adipose Tissues; FBG: Fasting Blood Glucose.

Introduction

Table sugar, the world's most popular sweetener, has been a component of human diets since ancient times. Table sugar enhances the taste and palatability of foods thereby increasing the overall quantity of such foods consumed at a time, compared to the non-sweetened. Its effect on neurotransmitters and pleasurable sites in the brain following previous exposure is also known to cause addiction to sugar consumption [1] and subsequently an absolute dependence or compulsive intake of excess sugar [2]. These combined factors of increased food consumption occasioned by dependence or compulsive intake of excess sugar [2]. These addiction to sugar consumption [1] and subsequently an absolute

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prolonged postprandial hyperglycemia [15]. Additionally, highly palatable caloric-dense diets can potentially induced obesity, regardless of the dietary source of the calories, as most of those calories are eventually stored as fat [18].

Therefore, it appears paradoxical that honey can be recommend as a dietary supplement for diabetic patients [19], but zero tolerance for table sugar for the same patients. Meanwhile, Sheriff et al. [20] have noted that honey consumption by diabetic patients can lead to hyperglycemia and that its continuous use under such circumstances might worsen an already precarious state. Moreover, Kadirevu and Gurtu [21] have shown that excessive honey consumption by normal individuals can actually enhance body weight gain, which is known to predispose to diabetes or worsen its prognosis. Indeed, there are uncertainties in the use of honey in the management of diabetes and energy related conditions, as this is highly debatable and controversial [22] of utmost concern is the fact that majority of its users are oblivious of any associated health risk, because of the over emphasis on its benefits, hence the tendency of abuse.

Consequently, the objective of this study was to evaluate and compare the impact of equivalent calories of honey and refined sugar used as sweeteners in male Wistar rats on related biochemical parameters of energy metabolism.

Materials and Methods

Equipment

Honey was obtained from the Obudu Cattle Ranch, Obanliku Local Government Area, Cross River State, Nigeria, whereas refined sugar was purchased from Golden Sugar Company (Aropa, Lagos State, Nigeria). Digital electronic analytical balance (3600 b Bench Scale from Avery Weigh-Tronix Company, Smethwick, England) and glucose sensor meter with its corresponding test strips i.e. Accu-Chek (Roche Diagnostics GmbH, Mannheim, Germany) were obtained for analysis. All chemicals used were of analytical grade.

Animals and Housing Condition

Thirty five male Wistar rats (Rattus norvegicus) weighing 100–120 g were obtained from the Department of Genetics & Biotechnology, University of Calabar and used in the experiment. They were housed in standard laboratory cages (room temperature about 28°C and humidity of 50–64%) and maintained on a 12:12 hour light/dark cycle in the animal house facility of the same Department, where the animal experiments were also carried out.

Experimental Protocol

The rats were randomly assigned to five groups of seven each with similar mean group weights (mean ± SD). Group 1 (control) was fed rat chow only; group 2 (sugar-fed I) was fed 15% sugar diet (w/w); group 3 (sugar-fed II) was fed 30% sugar diet (w/w); group 4 (honey-fed I) was fed 12% honey diet (w/w); and group 5 (honey-fed II) was fed 24% honey diet (w/w). The percent sugar and honey incorporation was based on average percent sweeterener composition of common sweetened foods and beverages (www.medicalnewstoday.com). The percentages of the sweeteners are calorically equivalent i.e. 15% sugar equivalent to 12% honey, while 30% sugar equivalent to 24% honey, given that a teaspoon full of sugar and honey is reported to contain 49 and 68 calories respectively [17]. The sweetened diets were compounded every other day to avoid spoilage. The animals were allowed access to known quantities of diets and drinking water ad libitum. Daily, the diet left in the feeding troughs were carefully collected and weighed. And diet intake per group per day was determined as the difference in weight between initial amount supplied and the leftover. Body weight and Fasting Blood Glucose (FBG) were measured every week throughout the 13-weeks feeding duration, with an electronic balance and glucometer, respectively. Animal use was approved by the Faculty Animal Research Ethics Committee, Faculty of Basic Medical Sciences (FAREC-FBMS) - Approval Number: 014B2117 and the research procedure conducted according to its Guidelines on the Care and Use of Animals for Scientific Research Purposes (ASRP).

Sample Collection

At the end of the study, final body weights and FBG were recorded and the rats euthanized. Whole blood was collected via cardiac puncture and emptied into sterilized non-heparinized tubes, from where serum was used for biochemical assays. Total WAT (White Adipose Tissue) pad (peri-renal and epididymal fats) and internal organs (liver, kidneys, heart and testes) were surgically removed at necropsy, blotted with Whatman filter paper and weighed with analytical balance. Relative total WAT fat or organ weights were calculated thus: w/W × 100 percent where, w is the wet weight of the fat pad or organ and W, the final body weight of the animal just before it was euthanized. Afterwards the WAT was suspended in 10% formal saline for tissue fixation, preparatory to histological processing.

Serum Biochemical Assays

Serum glucose concentration was determined using analytical kits from Randox Laboratories Ltd. (Ad more Diamond Road, Crumlin Co, Antrim, UK). Bio Vendor ELISA kits from Asheville, USA were used to evaluate adiponectin and leptin concentrations. Triacylglycerol (TG), Total Cholesterol (TCHOL), and high density lipoprotein cholesterol (HDL-C) were determined with analytical kits from Randox laboratories Ltd. Very low density lipoprotein – cholesterol (VLDL-C) and Low Density Lipoprotein – Cholesterol (LDL-C) were estimated by calculations.

Histopathological Studies

The fixed WAT, after embedding in paraffin wax, were sectioned with microtome (5 µm thickness) and stained on slides according to Harry’s Hematoxylin and Eosin staining procedures. Thereafter, the slides were mounted on Distrene, Plasticizer and Xylene (DPX) mountant, cover-slipped and viewed under a light microscope (Olympus CKX41, Olympus, Japan) and photomicrographs were taken (400×).

Statistical Analyses

All data were expressed as the mean ± SD. ANOVA was used to analyze the data, followed by LSD post hoc comparison of means. The SPSS software Version 20.0 (IBM SPSS Inc, Chicago, IL, USA) was used in the analysis. Differences were considered significant at p < 0.05.

Results

Time-Course Changes in Body Weight

Results of changes in body weight of the rats measured over the 13-weeks study period are shown in figure 1. From the result, there was an initial loss of body weight observed in the rats fed honey sweetened diets (i.e. H12% and H24%) within the first three weeks of feeding, due probably to non-adaptability to honey-based diet. However, by the fourth week, both the honey and sugar fed groups showed sequential increases in body weight gain, which were not statistically significant when compared to the control. Also, there was a correlation between the rats’ body weights and their diet intake which suggests that the body weight gain depended on the diet consumed.
Diet Intake

The diet consumption pattern within the 13-weeks study period was shown in figure 2. The incorporation of sugar and honey to the diets did not affect consumption rate as there were not significant differences in the weekly quantities of diets consumed between the test groups and control ($p > 0.05$). In general, the diet intake in the various groups increased progressively from the outset of the experiment though marginally, peaked at week 8 then, began to decline also progressively too, till the end of the study ($p > 0.05$).

Effect on Blood Glucose

Results of the weekly Fasting Blood Glucose (FBG) (Figure 3a) and serum glucose measured at the end of the experiment (Figure 3b) showed non-significant effects of the natural honey sweetened diets on blood glucose compared to the table sugar sweetened diets or the control ($p > 0.05$). The measured blood glucose of animals treated with both study diets, within and at the end of study, were within the normal control range.

Effect on Relative Organ Weights

Figure 4 entails the relative mean weights (g) of heart (H), kidneys (K), liver (L) and testes (T) of the test and control rats. There was no significant difference between the groups fed the sweetened diets and the control group in terms of their relative kidneys and hearts weights. The liver weights of animals fed the highest percent honey diet (H24% group) were found to be greater than the control ($p < 0.05$), but not the equivalent sugar group. Furthermore, the relative weights of testes of the honey-fed groups (H12% and H24%) were reduced compared to the corresponding sugar-fed groups (S15% and S30%) and control ($p < 0.05$).

Effect on White Adipose Tissue Weights

The total White Adipose Tissue (WAT) weight comprising of the sum of perirenal and epididymal fat pads, measured at the end of the 13-week study are shown in figure 5. Both the absolute (ABF) and relative (REF) total WAT weights of the animal groups fed sugar based diets did not differ significantly from the corresponding honey based groups or the control group ($p > 0.05$).

Effect on Serum Lipid Profile

The data of serum lipid profile assay of the test and control rats are presented in figure 6. There was no significant difference in lipid profile i.e. total cholesterol (TC), low density lipoprotein
cholesterol (LDL-C), triacylglycerol (TG), very low density lipoprotein cholesterol (VLDL-C) and high density lipoprotein cholesterol (HDL-C) concentrations of the groups administered sugar-based diet relative to the corresponding honey diet treated groups or normal control group ($p > 0.05$).

**Effect on Selected Serum Adipokines**

Taken together, the data obtained from the present study show that neither the honey nor sugar-sweetened diets significantly altered the concentrations of the measured serum adipokines (adiponectin and leptin) either comparing the equivalent energy groups or with the normal control (Figure 7a,b), implying a non-significant impact on energy storage in adipose tissue within the 3-month study period.

**Effect on White Adipose Tissues Histology**

The histology of the white adipose tissues was also studied (Figure 8). In sections, the fat cells appear empty and the nuclei are located at the periphery. Control group (NC) showed numerous adipocytes with well defined cell membranes. The nuclei were demonstrated at the periphery and connective tissue cells and fibroblasts were embedded amongst the adipocytes. Adipose of the 15% sugar fed group (S15%) showed numerous adipocytes as well, but with cell membranes were not clearly outlined. The connective tissue substance was however increased compared to the control group section. The 30% sugar fed photomicrograph (S30%) showed numerous but enlarged adipocytes (larger than any other group), suggesting an increased fat storage; however, the fibroblast and connective tissue substances were similar to those in group S15% section. Also, numerous adipocytes were observed in the adipose of the 12% honey fed rats (H12%). The adipocytes were densely packed with intact connective tissue and fibroblasts. In the highest percent honey fed group (H24%) it was evidently found smaller sized, but numerous adipocytes compared to the other groups. These vacuole-like fat cells or tiny pockets may imply...
more lipid deposits/droplets and hyperplastic differentiation of the adipose prior to more fat storage.

Discussion

The potential for use of the honey and table sugar as sweeteners and their influence on body weight control in male wistar rats were studied. Honey and sugar both, well-known food sweeteners [19,23] were compared with 10% honey supplemented diets and fed to experimental rats for 13 weeks. Within this 3-month period, measured animals’ body weights showed sequential and progressive gain across the treatment groups, with the progressive weight gain not significantly different among the groups. This implies that the two sweeteners, honey and table sugar were not different in their effect on body weight gain, especially that body weight gain was found to be dependent on the amount of diet consumed - a strong positive correlation between body weight gain and diet intake. Earlier reports associated to the effect of honey-based diets on body weight have been inconsistent; one of the reasons for which this study was carried out. Whereas a previous study [22] showed that 20% honey-based diet fed to Sprague-Dawley rats for 13 weeks caused a significant increase in total body weight at the end of study compared to the normal control group; Chepulis & Starkey [15] reported a significant reduction in body weight gain of 10% honey-sweetened diet fed to Sprague-Dawley rats compared to rats given 7.9% sucrose-based diet for six weeks. The primary sweetening agent in table sugar is sucrose, which comprise of the monosaccharides, glucose and fructose [24]. Also, the effective sweeteners in honey are glucose and fructose [25]. The glucose and fructose, when consumed in equal amount should contribute equally to the energy reserve of the cell, hence body weight, irrespective of their sources [3] have explained earlier that the body metabolizes carbohydrates in a similar way regardless of their source, whether it is from wholly natural source (e.g. honey) or processed source (e.g. refined sugar). It is therefore understandable that both honey and sugar fed in equal amounts of energy may have contributed equally to energy reserve of the animals, which was reflected as identical effect on body weight. The observed non disparity in effect of the sweeteners on body weight is also strengthened by the fact that the amount of diet consumed by the study groups did not vary significantly and coupled with the facts that neither of the measured appetite regulating hormones (i.e. adiponectin nor leptin) concentrations was significantly affected by any of the sweeteners.

The adiponectins including adiponectin and leptin function to modulate the dietary intake of the animals and are crucial for metabolism of their adipose cells [26]. The present data was further corroborated by the observed non-significant difference in total WAT weights across the groups. In general, as the adipocytes get larger in size, there is a corresponding increase in leptin secretion [24] and decrease in adiponectin secretions [27]; which was not the observation in the current study indicating that, neither the honey nor sugar significantly influenced adiposity or adipose metabolism of the rats, regardless of their percentage supplementation. The reduction in leptin concentration of the higher percent sweetened diet, which was significant in the 50% group only, may involve altered serum insulin action possibly provoked by the rich-sugar S30% diet. Insulin increase in blood is known to enhance leptin uptake, by the brain in non-diabetic states [28] hence a decrease in peripheral blood. Furthermore, Vasselli [29] stated unequivocally that the rate of leptin transport can be modified by factors such as glucose and insulin. However, insulin and blood glucose measurements in future studies would provide a better understanding. The zero effect of the sweeteners on the histology of the WAT further supports this idea.

Fasting Blood Glucose (FBG) concentrations measured, did not significantly differ between the test groups and/or the normal control. This agrees with [30] whose work showed that honey-fed rats were normoglycaemic during 13 weeks of administration of 20% honey supplemented diet. Given that the level of FBG in an organism is a function of the amount of food ingested in conjunction with other factors [27] and the changes in diet intake during the study were concomitantly non-significant. It follows then that in the short term there may be no grounds for preferring honey over refined sugar with respect to glycemic control. Serum glucose measured at end of the 13-week diet treatment affirms, hence further strengthens.

In a previous study, honey treatment was shown to significantly reduce the levels of TC, TG, and LDL-C [10] where as another indicated an increased propensity to raise triacylglycerol/fat levels in cells [31], and refined sugar was associated with hyperlipedemia [11]. However, the present study showed that the sweetened diets exerted no remarkable effect on the serum lipid profile. Evidently, imbalance in the serum lipid profile of an organism is symptomatic of derangement in lipid metabolism influenced by the amount of energy substrates available to the cell [32]. The observed effect of the sweeteners on the lipid profile therefore follows from the non-striking impact on blood glucose and adipose tissue hormones.

Conclusion

Overall, data obtained from the present study, provide no sufficient biochemical grounds for preferring honey over table sugar as a sweetening agent in foods, regarding body weight control and adipose tissue energy metabolism, at least in rat under laboratory conditions. These findings are however subject to further research.

Contributions of authors

EUE and IJA conceived and designed the investigation; MAE and GEE carried out the feeding experiments, HCO and DU carried out the laboratory analysis. EUE, IJA and GEE supervised and directed the study and IJA, MAE and ABU drafted and proof-read the manuscript. All authors read and approved the manuscript.

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

References


