Research Article | Internal Medicine

Associations of Dietary Fructose and Sucrose Intake with HbA1c and Anthropometric Measurements in Patients with Type 2 Diabetes Mellitus

Gulsah Kaner¹, Cagla Ayer^{1*}, Tuba Yalcin¹, Buse Bakir¹

Abstract

Aim. This study aimed to evaluate the associations of daily dietary intake of fructose and sucrose with HbA1c levels and anthropometric measurements in patients with Type 2 diabetes mellitus.

Methods. A total of 64 individuals were included in the study. Demographic characteristics, eating habits, frequency of fructose and sucrose source consumption, and dietary intakes were assessed through questioning. Anthropometric measurements, including body weight, height circumference (HtC), waist circumference (WC), hip circumference (HC), neck circumference (NC), wrist circumference (WrC), and mid-upper arm circumference (MUAC), were evaluated. The HbA1c level was categorized into two groups: good glycemic control (HbA1c \leq 7%) and poor (HbA1c > 7%) glycemic control.

Results. The mean daily fructose and sucrose intakes were 10.57 ± 8.28 g ($2.38 \pm 1.96\%$), and 29.21 ± 24.78 g ($6.29 \pm 5.35\%$), respectively. All the anthropometric measurements assessed were lower in the group with good glycemic control; however, only in case of BMI, MUAC, NC, and WrC, these differences were significant. Patients with good glycemic control were found to consume more fruit. Anthropometric measurements were not related to daily fructose intake, but positively associated with sucrose intake and HbA1c levels. Specifically, HbA1c levels were negatively associated with fiber intake and positively associated with sucrose intake. Moreover, HbA1c levels were positively affected by NC and consumption of non-alcoholic carbonated drinks, and negatively affected by WC and consumption of sugar-containing instant coffee.

Conclusions. The mean daily fructose intake among diabetics was found to be moderate and sucrose intake was consistent with the recommended range. The level of HbA1c was shown to be associated with all the anthropometric parameters assessed, and it was found that sugar-sweetened beverages could affect the level of HbA1c. However, further studies with larger sample sizes are needed to investigate these relationships more comprehensively.

Keywords

Diabetes Mellitus; Fructose; Anthropometric Measurements; Diet

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Introduction

Diabetes mellitus (DM) is a chronic disease that occurs because of the insufficiency and/or ineffectiveness of the hormone insulin produced by the pancreas and significantly affects patients' quality of life. Almost 463 million adults are estimated to have DM globally, and this number will reach 700 million by 2045. Most diabetic patients have Type 2 DM (T2DM), and an estimated 374 million people are at risk of T2DM [1]. A high-sugar diet consumption is thought to lead to an increase in the prevalence of obesity and DM worldwide [2, 3]. Sucrose, a disaccharide comprising glucose and fructose, has been associated, along with fructose, with T2DM development [4].

Fructose, one of the essential components of sugar consumption, is metabolized in the body by a specific mechanism. Unlike glucose, which is phosphorylated by hexokinase, fructose is phosphorylated by an enzyme called fructokinase. This pathway bypasses the phosphofructokinase step, which is one of the strictly regulated glycolytic checkpoints. Therefore, while glucose metabolism is regulated by phosphofructokinase with negative feedback, fructose can continuously enter the glycolytic pathway and cause insulin resistance in the liver by stimulating de novo lipogenesis in an uncontrolled manner [5, 6]. The lack of negative feedback of fructokinase causes rapid phosphorylation of all fructose entering the cell and depletion of adenosine triphosphate (ATP). Another characteristic feature of fructose metabolism is that the uric acid level rises with the activation of purine metabolism enzymes. Due to these metabolic properties, high fructose consumption is associated with various cardiometabolic risk factors, especially obesity and T2DM, and fructose is also referred to as a lipogenic monosaccharide [7].

Consumption of sugar-sweetened beverages (SSBs), which are significant sources of fructose and sucrose, is associated with increased risk of T2DM [8, 9]. In addition, it has been found that high fructose consumption may negatively affect peripheral insulin sensitivity [10] and positively correlate with body mass index (BMI), waist circumference (WC), and glycated hemoglobin (HbA1c) [7, 11–13]. In addition, excessive sucrose consumption was found to have a negative impact on the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) value [14, 15].

The link between added fructose (foods that contain high-fructose corn syrup) and DM has been extensively studied in the scientific literature. However, fewer studies have examined the relationship between daily dietary fructose intake and anthropometric measurements. Therefore, **the aim of this study** was to evaluate the associations of daily dietary intake of fructose and sucrose with HbA1c levels and anthropometric measurements in patients with T2DM. Our findings may provide a framework for understanding the relationship between daily dietary fructose and sucrose intake and anthropometric measurements for DM in the adult population.

Materials and Methods

Study Design

This cross-sectional study was carried out among participants with T2DM who applied to the diet polyclinic of a private hospital between October 2019 and March 2020 in Izmir, Turkey. The data were obtained by the researcher using a questionnaire form in face-to-face interviews. The questionnaire was prepared in line with the literature and was created in an understandable manner for the patients [16, 17]. The questionnaire comprehension was tested on 10 people, and any necessary adjustments were made before the questionnaire was finalized. The questionnaire form included questions about the socio-demographic characteristics, eating habits, and nutritional status of individuals.

Participants

A total of 64 participants (30 males, 34 females) were included in the study. Inclusion criteria were individuals at the age of 18-65 years having T2DM, residing in Izmir, Turkey, and being able to provide informed consent. This study excluded patients with Type 1 DM, those with mental, oncological, and hormonal diseases, as well as pregnant and lactating women.

Food Consumption Record and Frequency of Fructose Consumption

To determine the eating habits and nutritional status of patients, the form for the consumption frequency of fructose sources over the past month was taken by the researcher. The questions covered the frequency and amount of consuming nutritional sources containing natural and highfructose corn syrup. Furthermore, a daily food consumption record was taken from patients using the 24-hour recall method. A Picture Catalogue for Food and Nutrition was used to ensure that patients accurately indicated the amount of food they reported [18]. The energy and nutrient content of the daily diet was analyzed using the "Nutrition Information System Package Program (BeBiS)" developed for Turkey [19].

Anthropometric Measurements

Body weight, height, WC, hip circumference (HC), neck circumference (NC), wrist circumference (WrC), and midupper arm circumference (MUAC) were measured by a trained dietitian in accordance with the technique recommended by the World Health Organization (WHO). The patients' body weight was measured in the morning using a bioelectrical impedance analyzer (TANITA BC-532, Tokyo, Japan). Height was measured via a 0.01 cm sensitive stadiometer with the patient standing upright, feet positioned together, and head aligned in the Frankfort plane. WC was measured with a non-extensible measuring tape at the midpoint between the lowest rib and the crista iliaca, standing in front of the patient. HC was measured around the widest part of the hip with a non-flexible measuring tape, with the patient standing, arms hanging at the sides, and legs positioned next to each other while standing next to the patient. The waist-to-hip ratio (WHR) and the waist-to-height ratio (WHtR) were calculated. MUAC was measured using a flexible tape at the midpoint between the olecranon and acromion processes. The measure was taken in the patient's non-dominant arm. NC was measured just below Adam's apple (a laryngeal prominence). WrC was measured to the nearest 0.1 cm using a tape meter. The patient was asked to hold their wrist anterior surface up; the superior border of the tape measure was placed just distal to the prominences of radial and ulnar bones. WrC was measured without any tape pressure over it. Each of the anthropometric measurements was conducted twice, and the averages were recorded. BMI was calculated by dividing body weight (kg) by the square of height (m^2) [20].

Determination of HbA1c Level

HbA1c was studied by high-performance liquid chromatography (HPLC) method on a Arkray-Adams HA8180V analyzer (Minneapolis, USA). In the "Clinical Practice Guideline for Diagnosis, Treatment and Follow-up of Diabetes Mellitus and Its Complications" published by the Society of Endocrinology and Metabolism of Turkey in 2020, the glycemic control target of HbA1c in patients with DM was reported as $\leq 7\%$ (53 mmol/mol) [21]. With this guideline in mind, HbA1c levels were categorized into two groups: participants with HbA1c level of 7% and below (HbA1c $\leq 7\%$) were assigned to the group of good glycemic control, and participants with levels above 7% (HbA1c > 7%) were assigned to the group of poor glycemic control.

Statistical Analysis

Data were analyzed via IBM SPSS 25.0. Categorical data are indicated in numbers and percentages, and numerical data - in mean and standard deviation values. The assumption of normality for continuous variables was checked using the Kolmogorov-Smirnov test (p > 0.05). Graphical (histograms, box plots, Q-Q-plots), and numerical (skewness and kurtosis indices) methods were used as well. The evaluations involved the use of the Chi-square test, t-test, Pearson correlation analysis and linear regression analysis (enter model) in independent groups. In the final stage of the linear regression model, a table was generated to display the statistically significant variables. The results were evaluated at the 95% confidence interval (CI) and the significance level of p < 0.05.

Results

The patients' demographic characteristics, their anthropometric measurements, variables of energy and some nutrient consumptions are shown in Table 1. A total of 58.8% of patients with good glycemic control were females and their mean age was 53.94 ± 10.93 years, while 53.3% of patients with poor glycemic control were males, and their mean age was 53.53 ± 11.05 years. The mean body weight was significantly higher in the group with poor glycemic control. All the anthropometric measurements assessed were lower in group with good glycemic control; however, only in case of BMI, MUAC, NC, and WrC, these differences were significant. The mean energy and fiber intake was higher in the group with good glycemic control, while the mean daily dietary fructose and sucrose consumptions were higher in the other group. However, there was no significant difference between the groups (Table 1).

Table 2 presents the data on the patients' consumption amounts of fructose sources as determined in the form for assessing the frequency of consuming high-fructose foods. Patients with poor glycemic control were found to consume significantly more sugary foods, non-alcoholic carbonated drinks, sugar-containing instant coffee, biscuits, cakes, cookies, and pastries. On the other hand, patients with good glycemic control were found to consume more fruit.

The relationship between patients' HbA1c levels, fructose and sucrose consumption and anthropometric measurements assessed is given in Table 3. HbA1c level was positively correlated with daily dietary sucrose intake, but not correlated with daily dietary fructose intake.

HbA1c level and sucrose intake were found to have a positive correlation with all the anthropometric measurements assessed. Additionally, fiber intake was negatively

| Variables | | oA1c≤7% | | A1c>7% | | Total |
|--|--|--|--|--|--|---|
| | n | <u>%</u> | n nder | % | n | % |
| Males | 14 | 41.2 | 16 16 | 53.3 | 30 | 46.9 |
| Females | 20 | 58.8 | 14 | 46.7 | 34 | 53.1 |
| 1 cillales | 20 | | :0.33 | 40.7 | 57 | 55.1 |
| Age | 53 | .94±10.93 | 53. | 53±11.05 | 53 | .75±10.9 |
| (Mean±SD) | | p= | =0.88 | | | |
| Marital Status | | | | | | |
| Single | 12 | 35.3 | 6 | 20 | 18 | 28.1 |
| Married | 22 | 64.7 | 24 | 80 | 46 | 71.9 |
| | | Education | =0.17 nal Sta | atus | | |
| Illiterate | 1 | 2.9 | 0 | 0 | 1 | 1.6 |
| Primary school | 6 | 17.6 | 6 | 20 | 12 | 18.8 |
| High school | 12 | 35.3 | 15 | 50 | 27 | 42.1 |
| University | 15 | 44.1 | 9 | 30 | 24 | 37.5 |
| ChiveIsity | 15 | | =0.46 | 50 | 21 | 57.5 |
| | | Smo | king | | | |
| Yes | 9 | 26.5 | 10 | 33.3 | 19 | 29.7 |
| No | 16 | 47.1 | 10 | 33.3 | 26 | 40.6 |
| Quit | 9 | 26.5 | 10 | 33.3 | 19 | 29.7 |
| | | _ | 0.54 | | | |
| | | Alcohol co | | • | | |
| Yes | 15 | 44.1 | 10 | 33.3 | 25 | 39.1 |
| No | 19 | 55.9 | 20 =0.38 | 66.7 | 39 | 60.9 |
| DM duration | | | | | | |
| | 9 | .94±6.32 | 9. | 80 ± 6.45 | 9 | .88±6.33 |
| (Mean±SD) | | | | | | |
| (Mean±SD) | | | -0.93 | | | |
| | | p= | =0.93 quenc | y (months) | 1 | |
|] | | | | y (months) 36.7 | 21 | 32.8 |
| | Healt | p= h check fre | quenc | | | 32.8 67.2 |
|] ≤3 | Healt 10 | p= h check fre 29.4 70.6 | quenc 11 | 36.7 | 21 | |
|] ≤3 | Healt 10 | p= h check fre 29.4 70.6 | quenc 11 19 =0.54 | 36.7 63.3 | 21 | |
|] ≤3 | Healt 10 | p= h check fre 29.4 70.6 p= | quenc 11 19 =0.54 | 36.7 63.3 | 21 | |
| ≤3 >3 | Healt 10 24 | p= h check fre 29.4 70.6 p= Family | quenc 11 19 0.54 histor | 36.7 63.3 | 21 43 | 67.2 |
| ≤3 >3 Yes | Healt 10 24 32 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 | quenc 11 19 0.54 histor 25 | 36.7 63.3 y 83.3 | 21 43 57 | 67.2 89.1 |
| ≤3 >3 Yes | Healt 10 24 32 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 | quenc 11 19 0.54 histor 25 5 0.17 | 36.7 63.3 y 83.3 16.7 | 21 43 57 | 67.2 89.1 |
| ≤3 >3 Yes | Healt 10 24 32 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= | quenc 11 19 0.54 histor 25 5 0.17 | 36.7 63.3 y 83.3 16.7 | 21 43 57 | 67.2 89.1 |
| ≤3 >3 Yes No | Healt 10 24 32 2 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como | quenc 11 19 0.54 histor 25 5 0.17 rbidity | 36.7 63.3 7 83.3 16.7 | 21 43 57 7 | 67.2 89.1 10.9 |
| ≤3 >3 Yes No Yes | Healt 10 24 32 2 24 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 | quenc 11 19 0.54 histor 25 5 5 0.17 rbidity 20 | 36.7 63.3 7 83.3 16.7 7 66.7 | 21 43 57 7 44 | 67.2 89.1 10.9 68.8 |
| ≤3 >3 Yes No Yes | Healt 10 24 32 2 24 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 | 36.7 63.3 7 83.3 16.7 7 66.7 33.3 | 21 43 57 7 44 | 67.2 89.1 10.9 68.8 |
| ≤3 >3 Yes No Yes | Healt 10 24 32 2 24 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 | 36.7 63.3 7 83.3 16.7 7 66.7 33.3 | 21 43 57 7 44 | 67.2 89.1 10.9 68.8 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine | Healt 10 24 32 2 24 10 18 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 | quenc 11 19 :0.54 histor 25 :5 :0.17 rbidity 20 10 :0.74 diseas 16 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 8 80 | 21 43 57 7 44 20 34 | 67.2 89.1 10.9 68.8 31.2 53.1 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) | Healt 10 24 32 2 2 24 10 18 3 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 | 21 43 57 7 44 20 34 4 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal | Healt 10 24 32 2 24 10 18 3 2 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 5 | 21 43 57 7 44 20 34 4 3 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) | Healt 10 24 32 2 2 24 10 18 3 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 1 1 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 | 21 43 57 7 44 20 34 4 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal | Healt 10 24 32 2 24 10 18 3 2 1 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 p= | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 1 5 20,38 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 5 5 | 21 43 57 7 44 20 34 4 3 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal Respiratory | Healt 10 24 32 2 24 10 18 3 2 1 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 1 5 20,38 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 5 5 | 21 43 57 7 44 20 34 4 3 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal Respiratory Oral anti- | Healt 10 24 32 2 24 10 18 3 2 1 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 p= | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 1 5 20,38 | 36.7 63.3 7 7 83.3 16.7 7 66.7 33.3 5 80 10 5 5 | 21 43 57 7 44 20 34 4 3 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal Respiratory Oral anti- diabetics | Healt 10 24 32 2 24 10 18 3 2 1 M 33 | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 p= Iedical treat 97.1 | quenc 11 19 0.54 histor 25 5 -0.17 rbidity 20 10 -0.74 diseas 16 2 1 -0.98 tment 29 | 36.7 63.3 7 83.3 16.7 7 66.7 33.3 8 80 10 5 5 0f DM 96.7 | 21 43 57 7 44 20 34 4 3 2 62 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 3.1 96.8 |
| ≤3 >3 Yes No Yes No Cardiovascular Endocrine (thyroid) Gastrointestinal Respiratory Oral anti- | Healt 10 24 32 2 24 10 18 3 2 1 M | p= h check fre 29.4 70.6 p= Family 94.1 5.9 p= Como 70.6 29.4 p= Chronic 75 12.5 8.3 4.2 p= Iedical trea | quenc 11 19 0.54 histor 25 5 0.17 rbidity 20 10 0.74 diseas 16 2 1 1 5 5 10 20,74 diseas 16 2 1 1 5 2 10 10 10 10 10 10 10 10 10 10 | 36.7 63.3 7 83.3 16.7 7 66.7 33.3 5 80 10 5 5 0f DM | 21 43 57 7 44 20 34 4 3 2 | 67.2 89.1 10.9 68.8 31.2 53.1 6.2 4.6 3.1 |

Table 1. Sociodemographic information and anthropometric measurements of participants.

Table 1 continues on the next page.

Associations of Dietary Fructose and Sucrose Intake with HbA1c and Anthropometric Measurements in Patients with Type 2 Diabetes Mellitus — 4/8

| Table 1 | (Continued). |
|---------|--------------|
|---------|--------------|

| Variables | HbA1c $\leq 7\%$ | | Total |
|--------------------------|--------------------|-------------------------------------|---------------------|
| | | arements (Mean | |
| Body weight | $77.85{\pm}10.61$ | $83.82 {\pm} 9.29$ | $80.65 {\pm} 10.38$ |
| (kg) | p=0 | .02 | |
| BMI (kg/m ²) | $27.40{\pm}2.03$ | 29.21±2.79 | 28.25 ± 2.56 |
| BMI (kg/m ⁻) | p=0 | | |
| WC (cm) | $109.94{\pm}17.83$ | $116.90{\pm}21.98$ | $113.20{\pm}20.03$ |
| we (cill) | p=0 | | |
| WHR | $1.18 {\pm} 0.39$ | 1.27 ± 0.45 | 1.22 ± 0.42 |
| WIIK | p=0 | .33 | |
| WHtR | $0.65 {\pm} 0.09$ | $0.69{\pm}0.14$ | 0.67±0.12 |
| WHIK | p=0 | .22 | |
| NC (and) | 35.97±3.22 | 38.23±3.33 | 37.03±3.44 |
| NC (cm) | p<(|).01 | |
| | 21.47±4.08 | 23.40±3.43 | 22.37±3.88 |
| WrC (cm) | p=0 | .04 | |
| | | 39.07±10.80 | 36.87±8.38 |
| MUAC (cm) | p=0 | .04 | |
| Dietary energy | y and some sel | ected nutrients (| Mean+SD) |
| | 1768.00 | 1708.55 | 1740.13 |
| Energy (kcal) | ± 592.63 | ± 91.34 | ± 547.79 |
| Energy (Real) | p=0 | | ±511.17 |
| | | $\frac{17.05\pm7.46}{17.05\pm7.46}$ | 18.92±8.23 |
| Fiber (g) | 20.50⊥0.05 p=0 | | 10.92±0.25 |
| | | $\frac{11.38\pm8.77}{11.38\pm8.77}$ | 10.57+8.28 |
| Fructose (g) | | | 10.37±0.20 |
| | p=0 36.30±29.03 | | 38.91±30.47 |
| Fructose (kcal) | | | 58.91±50.47 |
| | p=0 2.12+1.68 | $\frac{1.47}{2.67\pm2.23}$ | 2 20 + 1 00 |
| Fructose (%) | | | 2.38 ± 1.96 |
| | p=0 | | 20.21 / 24.50 |
| Sucrose (g) | | 32.50±27.13 | 29.21±24.78 |
| | p=0 | .32 | |
| Sucrose (kcal) | | 125.68±104.89 | 0 112.94±95.82 |
| | p=0 | | |
| Sucrose (%) | 5.57±4.68 | 7.11±5.99 | 6.29±5.35 |
| 2461000 (70) | p=0 | .25 | |

Notes: BMI – body mass index; WC – waist circumference; WHR – waist-to-hip ratio; WHtR – waist-to-height ratio; NC – neck circumference, WrC – wrist circumference; MUAC – mid upper arm circumference.

correlated with HbA1c levels and positively correlated with daily dietary fructose intake.

In the linear regression model created with significant variables, it was determined that HbA1c level was positively affected by NC and consumption of non-alcoholic carbonated drinks, and negatively affected by WC and consumption of sugar-containing instant coffee. Other variables in the model did not have a significant effect on the HbA1c value (Table 4).

Discussion

The most notable finding of this study was the lack of association between anthropometric measurements and daily dietary fructose intake, but a positive association with daily dietary sucrose intake and HbA1c levels. In addition, HbA1c levels were negatively associated with fiber intake and positively associated with daily dietary sucrose intake.

Excessive consumption of sugars, especially fructose-

Table 2. Comparison of daily consumed amount of dietary fructose sources according to HbA1c levels.

| Foods | HbA1c $\leq 7\%$ | HbA1c >7% | Total | |
|---|-------------------|---------------------|-------------------|--|
| Sugar foods (alder) | $11.44{\pm}11.58$ | $27.20{\pm}28.57$ | 18.83 ± 22.56 | |
| Sugary foods, (g/day) | p<0 | .01 | | |
| | 195.01 | 163.71 | 180.34 | |
| Fruit, (g/day) | ± 116.15 | ± 105.67 | ± 111.60 | |
| | p=0. | 27 | | |
| Soft drinks (m.I. (day) | 37.72±41.05 | $60.36 {\pm} 66.62$ | 48.33±55.27 | |
| Soft drinks, (mL/day) | p=0. | 10 | | |
| Nau alaahalia aadha | 29.52 | 94.87 | 60.15 | |
| Non-alcoholic carbo- nated drinks, (mL/day | ±37.87 | ± 135.53 | ± 101.42 | |
| nated drinks, (inL/day | ′′ p<0 | .01 | | |
| Sugar-containing | $0.59{\pm}2.52$ | $23.17 {\pm} 62.67$ | 11.18 ± 44.05 | |
| instant coffee, (g/day) | p=0. | .04 | | |
| | 11.45 ± 14.43 | $12.12{\pm}18.88$ | 11.76±16.53 | |
| Chocolate, (g/day) | p=0. | 87 | | |
| Sweets (alder) | $0.00{\pm}0.00$ | $0.83 {\pm} 4.56$ | $0.39{\pm}50.66$ | |
| Sweets, (g/day) | p=0. | 29 | | |
| Biscuits, cakes, coo- | 22.73 ± 30.94 | $56.17 {\pm} 62.22$ | 38.41±50.66 | |
| kies, pastries, (g/day) | p<0 | .01 | | |
| Milky desserts, (g/day | 46.31±18.84 | 46.27±34.12 | 46.29±26.87 | |
| winky uessents, (g/ua) | p=0. | 99 | | |
| | | | | |

Table 3. Relationship between participants' HbA1clevels, fructose and sucrose consumption, and
anthropometric measurements.

| Variables | HbA1c (%) | | Fructose (g) | | Sucro | se (g) | | |
|--|-----------|--------|--------------|------|-------|--------|--|--|
| | r | р | r | р | r | р | | |
| Anthropometric measurements | | | | | | | | |
| Body weight (kg) | 0.39 | 0.01 | -0.21 | 0.09 | 0.29 | 0.02 | | |
| BMI (kg/m ²) | 0.45 | < 0.01 | -0.07 | 0.57 | 0.29 | 0.02 | | |
| WC (cm) | 0.38 | 0.01 | -0.18 | 0.16 | 0.48 | < 0.01 | | |
| WHR | 0.27 | 0.03 | -0.21 | 0.11 | 0.37 | < 0.01 | | |
| WHtR | 0.37 | 0.01 | -0.13 | 0.32 | 0.47 | < 0.01 | | |
| NC (cm) | 0.57 | < 0.01 | -0.18 | 0.15 | 0.32 | 0.01 | | |
| WrC (cm) | 0.41 | < 0.01 | -0.24 | 0.05 | 0.37 | < 0.01 | | |
| MUAC (cm) | 0.29 | 0.02 | 0.06 | 0.66 | 0.25 | 0.04 | | |
| Dietary energy and some nutrient consumption | | | | | | | | |
| Energy (kkal) | 0.08 | 0.52 | 0.07 | 0.59 | 0.47 | < 0.01 | | |
| Fiber (g) | -0.26 | 0.04 | 0.33 | 0.01 | 0.09 | 0.5 | | |
| Fructose (g) | 0 | 0.98 | | | | | | |
| Fructose (%) | -0.01 | 0.96 | | | | | | |
| Sucrose (g) | 0.29 | 0.02 | | | | | | |
| Sucrose (%) | 0.26 | 0.04 | | | | | | |

Notes: BMI – body mass index; WC – waist circumference; WHR – waist-to-hip ratio; WHtR – waist-to-height ratio; NC – neck circumference, WrC – wrist circumference; MUAC – mid upper arm circumference.

containing sugars, has been suggested to be associated with various chronic diseases such as obesity, cancer, and T2DM [22, 23]. In the literature, Livesey *et al.* classified daily fructose intake into three different groups [24]: fructose intake between 0-50 g/day was classified as moderate, 50-100 g/day as high, and 100-150 g/day as very high. In this study, the mean daily fructose intake (10.57 \pm 8.28 g/day) was found to be moderate, the ratio of energy derived from sucrose (6.29 \pm 5.35%) was consistent

| Variables | В | Standard error | CI (95%) | p value |
|--|--------|----------------|-------------------|---------|
| Constant | 1.091 | 2.22 | | |
| Age | -0.012 | 0.01 | (-0.03)-(0.01) | 0.22 |
| Gender | 0.313 | 0.37 | (-0.44)- (1.07) | 0.41 |
| Body weight (kg) | 0.025 | 0.03 | (-0.03)- (0.08) | 0.34 |
| BMI (kg/m ²) | 0.027 | 0.05 | (-0.08)-(0.13) | 0.61 |
| WC (cm) | -0.074 | 0.03 | (-0.14)-(-0.01) | 0.03 |
| WHR | 0.884 | 1.08 | (-1.29)-(3.06) | 0.42 |
| WHtR | 9.121 | 5.12 | (-1.18)-(19.42) | 0.08 |
| NC (cm) | 0.114 | 0.03 | (0.05)-(0.18) | < 0.01 |
| WrC (cm) | 0.016 | 0.03 | (-0.04)- (0.07) | 0.56 |
| MUAC (cm) | 0.008 | 0.01 | (-0.01)-(0.03) | 0.43 |
| Sugary foods (g/day) | 0.007 | 0.01 | (-0.003)-(0.016) | 0.15 |
| Non-alcoholic carbonated drinks (mL/day) | 0.003 | 0.00 | (-0.00)-(0.01) | 0.04 |
| Biscuits, cakes, cookies, pastries (g/day) | 0.003 | 0.00 | (-0.00)- (0.01) | 0.30 |
| Sugar-containing instant coffee (g/day) | -0.005 | 0.00 | (0.00)- (0.01) | 0.04 |
| Fiber (g) | -0.009 | 0.01 | (-0.03)-(0.01) | 0.34 |
| Sucrose (g) | -0.002 | 0.00 | (-0.01)-(0.00) | 0.48 |

| Table 4. Effect | t of some | variables | on HbA1c | levels | (Linear | regression | analys | is). |
|-----------------|-----------|-----------|----------|--------|---------|------------|--------|------|
|-----------------|-----------|-----------|----------|--------|---------|------------|--------|------|

Notes: B – Regression Coefficient; CI – confidence interval; BMI – body mass index; WC – waist circumference;

WHR - waist-to-hip ratio; WHtR - waist-to-height ratio; NC - neck circumference, WrC - wrist circumference;

MUAC – mid upper arm circumference.

with the WHO recommended sugar intake for children and adults [25]. Similar to the present study, a study conducted on 156 individuals with T2DM suggested that the average fructose intake of participants (males: 13.2 ± 12.09 g/day, females: 13.6 ± 11.10 g/day) was moderate [16]. In our study, it was observed, although not statistically significant, that diabetics with glycemic good control $(195.01 \pm 116.15 \text{ g})$ consumed more fruit, which are fructose sources, than those with poor glycemic control $(163.71 \pm 105.67 \text{ g})$. In addition, a positive relationship was found between dietary fructose intake and fiber intake. Moreover, HbA1c level was not correlated with daily dietary fructose intake. The relationships observed in this study suggest that consuming fructose from natural sources is critical in terms of metabolic control of DM.

In addition to being a disease itself, obesity is a critical risk factor for many other diseases, especially metabolic disorders such as T2DM [26]. Obesity is generally evaluated using BMI. However, an increase in anthropometric measurements such as WC, WHR, and WHtR is associated with the risk of DM [26, 27]. The existing literature has indicated that consumption of sucrose can trigger obesity as well. Higgins et al. [28] revealed a significant increase in body weight associated with sucrose consumption. Furthermore, the EPIC-Norfolk study [29] identified a positive correlation between sucrose consumption and BMI. Our findings align with previous research, reinforcing the link between sucrose intake and obesity. A positive correlation was found between sucrose consumption and all the anthropometric measurements assessed. In addition, anthropometric measurements were not related to daily fructose intake.

HbA1c level is considered as a more accurate measure for monitoring the glycemic control of individuals with DM than fasting blood sugar [9]. In this study, individuals who had been diagnosed with DM for approximately ten years were categorized into two groups: good glycemic control (HbA1c \leq 7) and poor glycemic control (HbA1c > 7), based on their HbA1c levels. HbA1c level was found to have a positive correlation with all anthropometric measurements and was positively affected by NC. Similar to our results, Kamarli *et al.* found a positive correlation between NC and HbA1c levels [30]. The use of anthropometric measurements in the clinical management of DM can potentially provide benefits.

The literature suggests that an increase in dietary fructose intake markedly increases insulin levels and causes insulin resistance [31]. A study of Jalilvand *et al.* [7] showed that eight weeks of low fructose diet resulted in significant improvement in fasting blood glucose and HbA1c levels in individuals with DM. A study conducted across eight European countries revealed an association between the consumption of SSBs and the risk of developing T2DM [32]. In a study by Dornas *et al.* [33], fructose- or glucose-sweetened beverages were added to the diet, and fructose-sweetened beverages were found to reduce insulin sensitivity by the end of 8 weeks. In the present study, HbA1c level was positively correlated with daily dietary sucrose intake. Daily fructose and sucrose intakes were higher in participants with poor glycemic control.

Fructose and sucrose are widely used as sweeteners in the food industry, especially in the production of soft beverages [23]. A study of Malik *et al.* [34] demonstrated an association between the consumption of SSBs, including soft beverages, sugar-containing instant coffee, ready-todrink fruit juices, etc., and the incidence of T2DM. SSBs are the main source of dietary fructose. However, other sources, including confectionary, fruit, fruit juices, etc., contribute significantly to total fructose consumption as well [35]. A study by Cantoral *et al.* suggested a link between SSB intake and metabolic alterations [36]. Similarly, Buziau *et al.* investigated the metabolic effects of fructose from different food sources and found that fructose from SSBs and juice, but not fructose from fruit, was associated with higher intrahepatic lipid contents [37]. In parallel, HbA1c levels were positively affected by consumption of non-alcoholic carbonated drinks. In our study, patients with poor glycemic control were found to consume significantly more sugary foods, non-alcoholic carbonated drinks, sugar-containing instant coffee, biscuits, cakes, cookies, and pastries.

Limitations

This study has some limitations that should be considered. Firstly, the cross-sectional design limited the ability to make causal inferences. Secondly, the study was terminated in March 2020 due to the COVID-19 pandemic and the onset of restrictions, resulting in a smaller sample size of 64 diabetic patients instead of the intended study population. Thirdly, data were obtained through self-reporting, which introduces the possibility of false reporting and recall bias due to the nature of the study. Lastly, the study sample consisted of patients from a single private hospital in Izmir, limiting the generalizability of the findings.

Conclusions

This study found that the mean daily fructose intake among diabetic individuals was moderate, and their sucrose intake was within the recommended range. Anthropometric parameters showed a positive correlation with sucrose intake and HbA1c levels, while demonstrating a negative correlation with daily fructose intake. Furthermore, HbA1c levels were negatively associated with fiber intake and positively associated with sucrose intake. However, it is important to note that further research is needed to investigate these associations in larger sample sizes.

Ethical Statement

For this study, Ethics Committee Approval No. 423, dated September 26, 2019, was obtained from the Non-Invasive Clinical Trials Ethics Committee of Izmir Katip Celebi University.

Informed Consent

Each individual was informed about the study, and the patient consent form was read and signed by those who wanted to participate.

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Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of Interest

This article is an extended version of an oral presentation at the congress of the 57th National Congress on Diabetes, Metabolism and Nutrition in Bodrum/TURKEY, on 1-5 June 2021. The authors declare that no conflicts exist.

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