CLINICAL PRACTICE & RESEARCH

ABSTRACT

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Is There a Relationship Between Food Addiction, Dietary Quality and Metabolic Parameters in Obese Adults?: A Cross-Sectional Study Example

Emre Duman¹ (D), Alev Keser² (D), Serkan Asil³ (D)

Objective: This cross-sectional study evaluated the association between food addiction, dietary quality, and metabolic parameters and determined food addiction prevalence in overweight and obese adults.

Materials and Methods: This study was conducted with 134 obese and overweight adults. Food addiction was evaluated with the Yale Food Addiction Scale (YFAS), and dietary quality was assessed with the Healthy Eating Index-2010 (HEI-2010). The HEI-2010 scores range from 0 to 100 (>80: good dietary quality, 51–80: needs improvement, <51: poor dietary quality).

Results: 19.4% of the participants had a food addiction. The ratio of those with poor dietary quality (61.5%) in participants with food addiction was higher than those without food addiction (52.8%) (p>0.05). The ratio of participants with high AST levels to participants with food addiction (34.6%) is higher than those without food addiction (17.6%) (p<0.05). Participants with out food addiction have lower cholesterol intake than participants with food addiction (p<0.05). YFAS symptom scores were positively correlated with AST, SBP, and DBP levels and negatively correlated with the age of the participants (p<0.05).

Conclusion: It was founded that a relationship may exist between food addiction, dietary quality, and metabolic parameters of obese and overweight adults. Especially in treating obesity, it is essential to make appropriate interventions to increase dietary quality.

Keywords: Healthy eating index, food addiction, healthy nutrition, body weight, obesity

INTRODUCTION

Although obesity prevalence is increasing in almost all countries today, this increase is expected to be higher in the coming years (1). The World Health Organization (WHO) states that the incidence of obesity has increased nearly three times in the last 40 years worldwide, and 13.0% of adults (\geq 18 years) were obese, and 39.0% were overweight in 2016 (2). This increase in obesity prevalence poses an essential threat to public health as it is a risk factor, especially for type 2 diabetes mellitus (T2DM), some types of cancer, coronary heart diseases (CHD), osteoarthritis, and many chronic diseases (3).

With obesity gaining a global dimension in every age group, more focus is placed on the risk factors that cause this problem. Accordingly, it is stated that an imbalance between energy intake and expenditure, metabolism, physical inactivity, genetics, food consumption, and healthy behaviour increase the risk of obesity (4). Recently, it has been suggested that obesity may develop similarly to substance dependence due to the consumption of tolerant food. Different mechanisms defined as "food addiction" may play a role in overeating some foods with high carbohydrate and fat content (5).

Food addiction is a chronically recurring problem caused by various key factors promoting the desire for food or nutrients and increasing pleasure, energy, or excitement. Significantly overweight and obese individuals are suggested to have a food addiction. Neurobiological studies reveal similarities that arise in the reward system for obesity and substance addiction (6). Nevertheless, it should be particularly noted that not all foods cause addictive behaviour. It is stated that the effects of foods containing high amounts of salt, oil, sugar, and food additives are pronounced to be more (7).

Despite the similarity between excessive food consumption and substance abuse, clinicians don't consider food addiction in evaluating patients or developing a treatment plan (5). However, food addiction in individuals may adversely affect dietary quality, defined as "nutritional adequacy" (8). High dietary quality is vital for improving health and reaching and maintaining the ideal body weight. Because dietary quality reflects the food variety, diet patterns, and preparation techniques (9), this research was planned and conducted on overweight and obese adults to assess the relationship between food addiction, metabolic parameters, and dietary quality.

MATERIALS and METHODS

Study Participants

This research was conducted between March 2018 and November 2018 at Ankara Gulhane Training and Research Hospital (AGTRH). Overweight and obese male and female participants aged 38–64 were included. The patient selection criteria are shown in Figure 1. The sample size was calculated using the Student-t test with a 0.05 significance level and 0.80 power. It was determined that a sample consisting of 134 people would be sufficient.

The participants in this cross-sectional study provided written informed consent. All ethical requirements were confirmed by the Ankara University Clinical Research Ethics Committee (date:22/11/2017, No:19-1187-17) and by the Provincial Health Directorate of Ankara Governorship (date:27/12/2017, No:75252626.604.01.02-E-9554) by the Declaration of Helsinki.

Data Collection

Height was measured using a standard stadiometer. The body weight (kg), body fat percentage (%), and lean body mass (kg) of participants were measured with a TANITA bio-analyzer (model: BC 545 N) in bare feet and after fasting for at least 8 hours. Body mass index (BMI) values were classified according to WHO criteria (overweight: 25–29.9 kg/m², obese class I: 30–34.9 kg/m², obese class II: 35–39.9 kg/m², obese class III: ≥40 kg/ m²) (10). Waist circumference (WC) was measured using a measuring tape 1 cm in width and made of a material that does not stretch. According to WC, male and female individuals are classified as "increased risk" (WC ≥94 cm, men; ≥80 cm, women) and "substantially increased risk" (WC ≥102 cm, men; ≥88 cm, women) (11). The cut point of the waist-to-height ratio (WHtR) was accepted as ≥0.5 (12).



Figure 1. Flow chart of patient selection AGTRH: Ankara Gülhane Training and Research Hospital

Biochemical measurements of participants were made by the AGTRH Medical Biochemistry Laboratory and were evaluated according to the reference values of this laboratory. Serum triglyceride (TG, 50–200 mg/dL), total cholesterol (TC, <200 mg/dL), fasting blood glucose (FBG, 74–110 mg/dL), high-density lipoprotein cholesterol (HDL-C, 35–85 mg/dL), low-density lipoprotein cholesterol (LDL-C, <130 mg/dL), alanine aminotransferase (ALT, 10–28 U/L), aspartate aminotransferase (AST, 9–36 U/L) and uric acid (UA, 3.5–7.5 mg/dL) values were evaluated. Accordingly, biochemical parameters in the normal range were considered "low", and those above the reference range were considered "high".

	FAO (n=26)		NFO (n=108)		Total (n=134)		р
	n	%	n	%	n	%	
Gender							0.184ª
Male	9	34.6	53	49.1	62	46.3	
Female	17	65.4	55	50.9	72	53.7	
Dietary quality							0.421^{b}
Poor	16	61.5	57	52.8	73	54.5	
Needs improvement	10	38.5	51	47.2	61	45.5	
HEI-2010 score							0.832°
Mean±SD	48.0±11.70		48.6±12.19		48.5±12.05		
Median	46.3		48.2		47.7		
Min–Max	32.3-79.4		26.5-75.4		26.5-79.4		
Age (year)							0.196^{d}
Mean±SD	50.7±6.63		52.6±6.86		52.3±6.83		
Median	49.0		53.0		52.0		
Min–Max	41.	0–64.0	38.	0–64.0	38.	0–64.0	

FAO: Food-addicted overweight/obese (\ge 3 symptoms + satisfying clinical impairment/distress criteria); NFO: Non-food-addicted overweight/obese; HEI-2010: Healthy Eating Index-2010; SD: Standard deviation. Significance was calculated with a: Fisher's exact test; b: Pearson's Chi-squared (χ^2) test; c: Student-t-test; d: Mann Whitney U test



Figure 2. Relationship between YFAS symptom scores and (a) age (r=-0.179, p=0.039); (b) AST (r=0.217, p=0.012); (c) SBP (r=0.186, p=0.032); and (d) DBP (r=0.200, p=0.021)

YFAS: Yale Food Addiction Scale; AST: Aspartate transaminase; SBP: Systolic blood pressure; DBP: Diastolic blood pressure

The participant's systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements were made three times after 20 minutes of rest with the Omron M2 device. Values were recorded by taking an average of the last two to determine the result. The dietary intake of individuals was collected using a 24-hour dietary recall method and a 1-day food consumption record. Nutrition Information System BeBIS (Version 8.1) was used to calculate energy intake and nutrients from foods consumed throughout the day.

Dietary quality was evaluated with the Healthy Eating Index-2010 (HEI-2010). This version of the HEI-2010 comprises 12 components. The HEI-2010 scores between 0 and 100 (>80: good dietary quality, 51–80: needs improvement, <51: poor dietary quality) (13). Yale Food Addiction Scale (YFAS) was used to assess the food addiction of the patients. It is a scale consisting of 27 items questioning seven criteria for eating habits in the last 12 months to determine individuals' food addiction status. The first 18 questions in the scale are five-point Likert type. However, the answers to questions between 19 and 24 are either yes or no. Question 25 provides information on how often certain foods have been tried to reduce or give up in the past year, while questions 26 and 27 provide information on foods people have difficulty controlling. This scale was developed by Gearhardt et al. (14). Its Turkish validity and reliability were performed by Bayraktar et al. (15), and Cronbach's alpha value was found as 0.93. The number of symptoms of food addiction goes from 0 to 7. The degree of addiction is proportional to the number of symptoms. In determining food addiction, questions 15 and 16 are essential for the clinic, and those with a minimum score of 1 and those with many signs of 3 or more are defined as "food-addicted" (14, 15).

Statistical Analysis

The Statistical Package for the Social Sciences (Version 22) Software was used to conduct the statistical analyses. Results were expressed as the frequency (n), percentage (%), mean $(\vec{\chi})$,

Integrate Integrate <thintegrate< th=""> <thintegrate< th=""> <th< th=""><th>· · ·</th><th colspan="2">FAO (n-26)</th><th colspan="2">NFO $(n=108)$</th><th colspan="2">Total (n-134)</th><th>n</th></th<></thintegrate<></thintegrate<>	· · ·	FAO (n-26)		NFO $(n=108)$		Total (n-134)		n
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$\begin{array}{c c c c c c c c c c c c c c c c c c c $			/0		/0		/0	
Overweight 8 30.8 32 29.6 40 29.8 Obese class I 8 30.8 40 37.0 48 35.8 Obese class II 6 23.1 32 29.6 38 28.3 Obese class II 4 15.3 4 3.8 8 6.1 WC (m) Substantially increased risk 23 11.5 14 13.0 17 12.7 Substantially increased risk 2 7.7 8 7.4 10 7.5 Substantially increased risk 2 7.7 8 7.4 10 7.5 Substantially increased risk 2 7.7 6 6.1.1 81 6.0.4 FIG (mg/dL) Optimal 16 61.5 54 50.0 64 47.8 . TC (mg/dL)	BMI (kg/m²)							0.148ª
Obese class II 6 23.1 32 29.6 38 28.3 Obese class III 4 15.3 4 3.8 8 6.1 WC (cm) 0.845° Increased risk 3 11.5 14 13.0 17 12.7 Substantially increased risk 3 11.5 14 13.0 17 7.7 Substantially increased risk 2 7.7 8 7.4 10 7.5 Substantially increased risk 2 7.7 8 7.4 10 7.5 Substantially increased risk 2 7.7 7 8 7.4 10 7.5 Substantially increased risk 2 7.7 8 7.4 10 7.5 Substantially increased risk 2 7.7 6 61.1 81 60.4 TC (mg/dL) 0 38.5 54 50.0 70 52.2 64 High 10 38.5 45 41.7 55 41.0 0.39° LD(c (mg/dL) 0 38.5 </td <td>Overweight</td> <td>8</td> <td>30.8</td> <td>32</td> <td>29.6</td> <td>40</td> <td>29.8</td> <td></td>	Overweight	8	30.8	32	29.6	40	29.8	
Obese class II 6 23.1 32 29.6 38 28.3 Obese class III 4 15.3 4 3.8 8 6.1 WC (cm)	Obese class I	8	30.8	40	37.0	48	35.8	
Obese class II 4 15.3 4 3.8 8 6.1 WC (cm)	Obese class II	6	23.1	32	29.6	38	28.3	
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Obese class III	4	15.3	4	3.8	8	6.1	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	WC (cm)							0.845 ^b
Substantially increased risk 23 88.5 94 87.0 117 87.3 WHR	Increased risk	3	11.5	14	13.0	17	12.7	
WHR 0.960* Increased risk 2 7.7 8 7.4 10 7.5 Substantialy increased risk 24 92.3 100 92.6 124 92.5 FBG (mg/dL) 0.749* 0.749* 0.749* 0.749* Optimal 11 42.3 42 38.9 53 39.6 High 15 57.7 66 61.1 81 60.4 TC (mg/dL) 0 38.5 54 50.0 70 52.2 High 10 38.5 54 50.0 64 47.8 Optimal 16 61.5 63 58.3 79 59.0 High 10 38.5 45 41.7 55 41.0 High 10 38.5 45 41.7 55 41.0 High 10 38.5 45 41.7 55 41.0 Low 2 7.7 16 14.8 18 13.4 Optimal 16 76.9 40.3 37	Substantially increased risk	23	88.5	94	87.0	117	87.3	
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $	WHtR							0.960 ^b
Substantially increased risk2492.310092.612492.5FBG (mg/dL) (0.749) (0.749) (0.749) (0.749) (0.749) Optimal1142.34238.95339.6High1557.76661.18160.4TC (mg/dL) (0.75) (0.75) (0.79) (0.79) Optimal1661.55450.07052.2High1038.55450.06447.8TG (mg/dL) (0.75) (0.77) 59.0 $(0.76)^{10}$ Uaw27.71614.81813.4Optimal2492.39285.211686.6LDLC (mg/dL) (0.77) 1614.81813.4 $(0.32)^{20}$ Optimal1973.16863.08764.9High726.94037.04735.1ALT (U/L) (0.71) $(0.75)^{20}$ $(0.47)^{20}$ $(0.47)^{20}$ Optimal1765.46156.57354.5High934.64743.56145.5AST (U/L) $(0.71)^{20}$ $(0.49)^{20}$ $(0.49)^{20}$ UA (mg/dL) $(0.74)^{20}$ $(0.49)^{20}$ $(0.49)^{20}$ Optimal1765.48982.410.679.1High934.61917.62820.9 $(0.49)^{20}$ Optimal16	Increased risk	2	7.7	8	7.4	10	7.5	
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Substantially increased risk	24	92.3	100	92.6	124	92.5	
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	FBG (mg/dL)							0.749ª
High1557.76661.18160.4TC (mg/dL) 0 0 ptimal16 61.5 54 50.0 70 52.2 High10 38.5 54 50.0 64 47.8 0.766° TG (mg/dL) 0 38.5 54 50.0 64 47.8 0.766° Optimal16 61.5 63 58.3 79 59.0 0.766° High10 38.5 45 41.7 55 41.0 0.339° Low2 7.7 16 14.8 18 13.4 Optimal24 92.3 92 85.2 116 86.6 LDL-C (mg/dL) 0.32° 0.32° 0.32° Optimal19 73.1 68 63.0 87 64.9 High7 26.9 40 37.0 47 35.1 ALT (U/L) 0.342° $0.37.0$ 47 35.1 0.342° Optimal17 65.4 61 56.5 73 54.5 High9 34.6 47 43.5 61 45.5 AST (U/L) 0.045° 0.045° 0.045° Optimal17 65.4 89 82.4 106 79.1 High9 34.6 19 17.6 28 20.9 UA (mg/dL) 0 0.9 67.2 0.496°	Optimal	11	42.3	42	38.9	53	39.6	
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	High	15	57.7	66	61.1	81	60.4	
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	TC (mg/dL)							0.290ª
High1038.55450.06447.8TG (mg/dL)061.56358.37959.0Optimal1661.56358.37959.0High1038.54541.75541.0HDL-C (mg/dL)038.54541.75541.0Low27.71614.81813.4Optimal2492.39285.211686.6LDL-C (mg/dL)073.16863.08764.9High726.94037.04735.1ALT (U/L)034.64743.56145.5AST (U/L)034.64743.56145.5AST (U/L)00.461917.62820.9UA (mg/dL)065.4617468.59067.2	Optimal	16	61.5	54	50.0	70	52.2	
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	High	10	38.5	54	50.0	64	47.8	
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	TG (mg/dL)							0.766ª
High10 38.5 45 41.7 55 41.0 HDL-C (mg/dL)	Optimal	16	61.5	63	58.3	79	59.0	
HDL-C (mg/dL) 0.339° Low27.71614.81813.4Optimal2492.39285.211686.6LDL-C (mg/dL) 0.332° 08764.90.332^{\circ}Optimal1973.16863.08764.9High726.94037.04735.1ALT (U/L) 0.342° 0.342° 0.342° 0.342° Optimal1765.46156.57354.5High934.64743.56145.5Optimal1765.48982.410679.1UA (mg/dL) 0.496° 9067.2 0.496°	High	10	38.5	45	41.7	55	41.0	
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	HDL-C (mg/dL)							0.339ª
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Low	2	7.7	16	14.8	18	13.4	
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Optimal	24	92.3	92	85.2	116	86.6	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	LDL-C (mg/dL)							0.332ª
High726.94037.04735.1ALT (U/L)0.342°Optimal1765.46156.57354.5High934.64743.56145.5AST (U/L)0.045°*Optimal1765.48982.410679.1High934.61917.62820.9UA (mg/dL)0.496°Optimal1661.57468.59067.2	Optimal	19	73.1	68	63.0	87	64.9	
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	High	7	26.9	40	37.0	47	35.1	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	ALT (U/L)							0.342ª
High 9 34.6 47 43.5 61 45.5 AST (U/L) 0.045** Optimal 17 65.4 89 82.4 106 79.1 High 9 34.6 19 17.6 28 20.9 UA (mg/dL) 0.496 ^a	Optimal	17	65.4	61	56.5	73	54.5	
AST (U/L) 0.045** Optimal 17 65.4 89 82.4 106 79.1 High 9 34.6 19 17.6 28 20.9 UA (mg/dL) 0.496* Optimal 16 61.5 74 68.5 90 67.2	High	9	34.6	47	43.5	61	45.5	
Optimal 17 65.4 89 82.4 106 79.1 High 9 34.6 19 17.6 28 20.9 UA (mg/dL) 0.496ª	AST (U/L)							0.045ª*
High 9 34.6 19 17.6 28 20.9 UA (mg/dL) 0.496 ^a Optimal 16 61.5 74 68.5 90 67.2	Optimal	17	65.4	89	82.4	106	79.1	
UA (mg/dL) 0.496ª 0.496ª	High	9	34.6	19	17.6	28	20.9	
Optimal 16 61.5 74 68.5 90 67.2	UA (mg/dL)							0.496ª
opunda 10 01.0 /4 00.0 70 07.2	Optimal	16	61.5	74	68.5	90	67.2	
High 10 38.5 34 31.5 44 32.8	High	10	38.5	34	31.5	44	32.8	
SBP (mmHg) 0.799ª	SBP (mmHg)							0.799ª
Optimal 13 50.0 51 47.2 64 47.8	Optimal	13	50.0	51	47.2	64	47.8	
High 13 50.0 57 52.8 70 52.2	High	13	50.0	57	52.8	70	52.2	
DBP (mmHg) 0.990ª	DBP (mmHg)							0.990ª
Optimal 14 53.8 58 53.7 72 53.7	Optimal	14	53.8	58	53.7	72	53.7	
High 12 46.2 50 46.3 62 46.3	High	12	46.2	50	46.3	62	46.3	

FAO: Food-addicted overweight/obese (\geq 3 symptoms + satisfying clinical impairment/distress criteria); NFO: Non-food-addicted overweight/obese; BMI: Body mass index; WC: Waist circumference; WHtR: Waist-to-height ratio; FBG: Fasting blood glucose; TC: Total cholesterol; TG: Triglyceride; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; UA: Uric acid; SBP: Systolic blood pressure; DBP: Diastolic blood pressure. Significance was calculated with a: Fisher's exact test; b: Pearson's Chi-squared (χ^2) test; *: P<0.05

Table 3. Evaluation of energy, macro and micronutrient intakes of participants							
Energy and nutrients	FAO (n=26)		NFO (n=108)		Total (n=134)		р
	Median	IQR	Median	IQR	Median	IQR	
Energy (kcal)	1703.5	1176.5	1868.2	873.3	1849.1	908.2	0.766
Carbohydrate (E%)	52.0	15.3	53.0	16.8	53.0	16.0	0.613
Protein (E%)	16.0	5.3	16.0	5.0	16.0	5.0	0.965
Total fat (E%)	32.5	14.3	29.5	16.5	30.0	16.3	0.551
Cholesterol (mg)	314.9	354.9	212.6	312.2	251.9	322.7	0.045*
Fiber (g)	20.7	11.7	23.1	15.1	22.4	14.8	0.501
Vitamin A (mcg)	621.0	327.6	513.3	420.0	539.2	402.1	0.363
Vitamin E (mg)	13.0	11.1	12.9	8.6	12.9	9.2	0.982
Thiamine (mg)	0.9	0.8	0.9	0.6	0.9	0.6	0.496
Riboflavin (mg)	1.1	0.6	1.1	0.7	1.1	0.7	0.466
Pyridoxine (mg)	1.0	0.7	1.1	0.8	1.1	0.8	0.587
Niacin (mg)	14.6	66.0	14.5	19.0	14.5	19.2	0.884
Vitamin B ₁₂ (mcg)	2.4	2.0	2.8	2.8	2.8	2.7	0.542
Folate (mcg)	257.2	179.3	295.1	193.1	285.3	188.7	0.288
Vitamin C (mg)	85.8	95.0	67.7	70.5	71.2	71.9	0.419
Potassium (mg)	1950.2	1301.5	2087.2	1263.7	2035.9	1186.5	0.148
Calcium (mg)	741.5	547.3	829.7	536.5	798.3	528.9	0.665
Phosphorus (mg)	925.2	426.9	1076.6	530.5	1044.9	505.3	0.210
Iron (mg)	235.1	153.0	10.2	6.3	10.2	6.2	0.601
Zinc (mg)	10.2	5.3	10.7	8.0	10.3	7.6	0.696

FAO: Food-addicted overweight/obese; IQR: Interquartile range. E%=Percentage energy from carbohydrates, protein or fat. Significance was calculated with the Mann Whitney U test. *: P<0.05

standard deviation (SD), median, interquartile range (IQR), minimum (min), and maximum (max). The significance level was selected as p<0.05 in hypothesis testing. The study used the Kolmogorov-Smirnov test to evaluate normal data distribution. Whether there is, the Mann-Whitney U test determined a statistical difference between qualitative variables if usual distribution assumptions were not provided and Student's t-test if provided. The Chi-Square test was used to examine the relationship between two categorical variables. Spearman's and Pearson's correlation analyses evaluated the relationship between the two numeric variables.

RESULTS

The research was conducted with 134 participants, 62 men (46.3%) and 72 women (53.7%), who applied to the Cardiology Policlinic of AGTRH. The mean age of the individuals is 52.3 ± 6.83 years. Twenty-six (19.4%, data not shown) of the individuals were identified as "food-addicted" (M: 34.6%; F: 65.4%). It was determined that most (54.5%) of the overweight and obese individuals participating in the study had poor dietary quality. In contrast, the individual with good dietary quality did not have any. The rate of participants with food addiction and without food addiction having poor dietary quality is 61.5% and 52.8%, respectively (p>0.05) (Table 1).

According to food addiction, there was no statistical difference between BMI, WC, and WHtR (p>0.05). In participants with and without food-addicted, FBG values were high at 57.7% and 61.1%, and SBP values were high at 50.0% and 52.8%, respectively. The ratio of participants with high AST levels to participants with food-addicted overweight/obese (FAO) (34.6%) is higher than non-food-addicted overweight/obese (NFO) (17.6%) (p<0.05) (Table 2).

Food-addicted individuals had higher cholesterol intake than nonfood-addicted individuals (p<0.05). There was no statistical difference between the two groups in terms of other nutrients and energy intake (p>0.05) (Table 3). There was a significant positive difference between YFAS symptom scores and AST levels and a considerable negative difference between YFAS symptom scores and the ages of the individuals (p<0.05) (Table 4). The relationship between YFAS symptom scores and age, AST, SBP, and DBP levels is shown in Figure 2.

DISCUSSION

Food addiction was detected in 26 (19.4%, data not shown) individuals who participated in the study (M: 34.6%; F: 65.4%) (Table 1). It was concluded that there was no statistical difference between BMI, WC and WHtR according to food addiction (p>0.05) (Table 2). While no statistical difference was found between other biochemical parameters for food addiction, a significant difference was observed only between AST levels (p<0.05) (Table 2). In addition, when individuals' 1-day food consumption record was examined, no significant difference was found between energy, macro, and micronutrients according to food addiction. Only a significant difference was found between cholesterol consumption (p<0.05) (Table 3).

A statistically positive correlation existed between YFAS symptom scores and AST, SBP, and DBP levels (Table 4) (Fig. 2). In addition, there was a statistically negative correlation between YFAS symptom scores and age (p<0.05) (Table 4). This result is supported by Yang et al. (16) and Eichen et al. (17). Although this situation shows that auto-control can be achieved better with age, it reveals that chronic diseases that occur with age can cause changes in the nutritional habits of participants.

In terms of food addiction, distribution by gender is not a statistical difference (p>0.05) (Table 1). Pursey et al. (18) determined that food addiction prevalence was 19.9% and approximately two times more common in women (12.2%) than men (6.4%) in the meta-analysis study. The researchers stated that this difference between genders might be due to hormonal factors and dietary habits. Similar to our research results, the food addiction prevalence in overweight and obese participants was determined by Hauck et al. (19) at 17.2% and Schulte and Gearhardt (20) at 19.3%, respectively.

However, some data do not support our research results and determine the prevalence of food addiction as Meule et al. (21) 47% or Ceccarini et al. (22) 34.1% in overweight and obese individuals. It is thought that this situation may be related to the age, gender distribution, and obesity class of individuals. No statistical difference was found between BMI, WC, and WHtR classification according to food addiction in this study (p>0.05) (Table 2). This result is because the study participants were only overweight and obese. This situation is accepted as one of the limitations of the study.

HEI-2010 total score mean of participants is 48.5 ± 12.05 , and the mean score of HEI-2010 with and without food addiction is 48.0 ± 11.70 and 48.6 ± 12.19 , respectively (p>0.05) (Table 1). In addition to this, no statistical correlation was found between the YFAS symptom score and the HEI-2010 scores (p>0.05) (Table 4). Two studies reported no statistical difference in dietary quality scores between FAO and NFO individuals. In these studies, FAO individuals met a significant amount of their daily energy intake from unhealthy foods, compared to NFO individuals, while they met significantly lower from healthy foods (23, 24). In this study, it was determined that the amount of cholesterol intake of FAO individuals was significantly higher than NFO individuals (p<0.05) (Table 3).

As a result, obese individuals often have "poor" dietary quality or "needs improvement" dietary quality. The fact that all individuals participating in this study are overweight and obese may be why no significant difference was found in dietary quality between individuals with and without food addiction. Obesity is defined as one of the leading causes of T2DM (16). On the other hand, it is stated that 80% to 90% of individuals with T2DM are overweight and obese (25). It was found that participants with food addiction and without food addiction who participated in this study had high FBG values of 57.7% and 61.1%, respectively (Table 2).

However, according to food addiction, there was no statistical difference between FBG levels (p>0.05). In a study by Guzzardi et al.

Table 4. Correlation of age, HEI-2010 score, anthropometric measurements,
biochemical parameters and blood pressure with YFAS symptom scores

	Total $(n=134)$		
	10tal (II-134)		
	r	р	
Age (year)	-0.179	0.039 ^{b*}	
HEI-2010 score	-0.009	0.914ª	
Body weight (kg)	0.027	0.760ª	
Height (cm)	-0.039	0.652ª	
BMI (kg/m²)	-0.016	$0.854^{ m b}$	
WC (cm)	-0.053	0.544 ^b	
WHtR	-0.004	0.967 ^b	
Body fat percentage (%)	0.068	0.432ª	
Body muscle mass (kg)	-0.068	0.433 ^b	
FBG (mg/dL)	-0.001	0.990 ^b	
TC (mg/dL)	0.025	0.774ª	
TG (mg/dL)	-0.070	0.419^{b}	
HDL-C (mg/dL)	0.046	0.599ª	
LDL-C (mg/dL)	0.073	0.405^{b}	
ALT (U/L)	0.163	0.060 ^b	
AST (U/L)	0.217	0.012 ^{b*}	
UA (mg/dL)	-0.068	0.479 ^b	
SBP (mmHg)	0.186	0.032 ^{b*}	
DBP (mmHg)	0.200	0.021 ^{b*}	

HEI-2010: Healthy Eating Index-2010; BMI: Body mass index; WC: Waist circumference; WHtR: Waist-to-height ratio; FBG: Fasting blood glucose; TC: Total cholesterol; TG: Triglyceride; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; UA: Uric acid; SBP: Systolic blood pressure; DBP: Diastolic blood pressure. Significance was calculated with a: Pearson's correlation test and b: Spearman's correlation test. *: P<0.05

(26) with 36 overweight and obese individuals over the age of 18, no difference was found between the mean FBG of individuals with and without food-addicted (p>0.05). Central obesity progresses in parallel with insulin resistance and hyperinsulinemia. In addition, the antilipolytic activity of insulin is more evident in the fat cells in the abdomen (27). When the waist circumference of the individuals in both groups participating in the study was evaluated, it was determined that most were in the substantially increased risk group (FAO: 88.5%, NFO: 87.0%) (Table 2). This result suggests that insulin is functionally inadequate in both groups and that hyperandrogenism and cortisol hypersecretion may also contribute to decreased insulin sensitivity in the muscles and liver (27).

Various lipid disorders occur due to obesity. Especially in obese individuals, serum HDL-C levels decrease, while TG, TC, and LDL-C levels increase (28). In this study, although there was no statistical difference in lipid parameters between participants with and without food addiction (p>0.05), it was determined that dyslipidemia was expected (Table 2). Furthermore, no correlation was found between YFAS symptom scores and HDL-C, LDL-C, TG and TC (p>0.05) (Table 2). The results of this study are supported by Yang et al. (16) and Guzzardi et al. (26). It is known that AST value increases, especially with food consumption with high energy and fat content (29). In this study, the percentage of individuals with high AST values was found to be significantly higher in participants with food-addicted overweight/obese (34.6%) than in those non-food-addicted overweight/obese (17.6%)(p<0.05) (Table 2). This situation may be related to higher BMI (Table 2) and fat consumption (Table 3) of participants with food addiction than those without. In addition, it has been suggested that there is a strong link between high serum UA levels and obesity (30). The relationship between elevated serum and various mechanisms can explain UA levels and obesity. Excess body fat is associated with high UA production, impaired UA metabolism, and poor excretion due to insulin resistance, causing hyperuricemia. In addition, it is suggested that a high UA level is a risk factor for cardiovascular diseases and hypertension (30). This study found that half of the participants in both groups had high blood pressure (Table 2). Poor dietary quality score contributes to these impaired findings (Table 1).

Obesity is significantly associated with alterations in the brain's dopaminergic pathways in the reward system. Furthermore, the gut microbiome also plays an essential role in the etiology of obesity. In obese individuals, there is a decrease in bacterial diversity and changes in bacterial gene expression. These changes also cause changes in nutrient metabolism (6). These data suggest that the relationship between food addiction and the gut-microbiota-brain axis should be explored using a multi-omics approach.

Study Limitations and Strengths

This study has some significant limitations. The first is that this study did not include the control group of individuals with average body weight. This situation restricted the evaluation relationship between dietary quality and food addiction. Secondly, although the study's sample was planned to be composed of adults between the ages of 19-64, there were participants at least 38 years old in this study. The fact that individuals of a certain age have similar eating habits may impact the study results. For these reasons, it is vital to create a sample that includes individuals from different age groups and nutritional statuses to assess the effect of food addiction on human health and dietary habits. Thirdly, the income levels of the individuals were not guestioned. But, it is known that dietary guality is affected by the income level of individuals. Higher rates of obesity are observed in low-income populations, and low-cost, palatable, and potentially addictive foods make up a large portion of individuals' diets. Therefore, not investigating the relationship between income level and food addiction is a limitation of this study.

CONCLUSION

In recent years, food addiction has been thought to be an eating behaviour disorder in the etiology of obesity. Evaluation of food addiction can bring a new perspective to treating obesity and help develop effective methods. The frequency of food addiction among participants in the study was 19.4%, which suggests that approximately one in five overweight and obese individuals may have a food addiction. Therefore, nutrition plans and policies should be developed to reduce the consumption of high-carbo-hydrate, fatty and salty foods, reduce the risk of food addiction, and improve dietary quality. In addition, it was determined that

most participants needed better dietary quality, and there were no individuals with good dietary quality. In this context, to increase the dietary quality of obese and overweight individuals, it should be ensured that they increase their consumption of vegetables, fruits, milk, and dairy products, dark green leafy vegetables, legumes, and seafood, and reduce their consumption of readymade products that are empty calories sources, processed grains, and sodium. It is essential to provide nutrition education to gain healthy eating habits starting from childhood, ensure dietitian employment in primary health care centres, and cooperate with dieticians to evaluate the psychological aspects of food addiction.

Ethics Committee Approval: The Ankara University Clinical Research Ethics Committee granted approval for this study (date: 22.11.2017, number: 19-1187-17).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – ED; Design – ED; Supervision – AK; Resource – ED, AK; Materials – SA; Data Collection and/or Processing – ED, SA; Analysis and/or Interpretation – ED; Literature Search – ED; Writing – ED, AK; Critical Reviews – ED, AK, SA.

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REFERENCES

- Janssen F, Bardoutsos A, Vidra N. Obesity prevalence in the long-term future in 18 European countries and in the USA. Obesity Facts 2020; 13(5): 514–27. [CrossRef]
- World Health Organization (WHO). Obesity and overweight. Available from: URL: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. Accessed Jul 30, 2022.
- Nimptsch K, Pischon T. Body fatness, related biomarkers and cancer risk: An epidemiological perspective. Horm Mol Biol Clin Investig 2015; 22(2): 39–51. [CrossRef]
- Yajnik CS, Deshmukh US. Maternal nutrition, intrauterine programming and consequential risks in the offspring. Rev Endocr Metab Disord 2008; 9(3): 203–11. [CrossRef]
- Merlo LJ, Klingman C, Malasanos TH, Silverstein JH. Exploration of food addiction in pediatric patients: a preliminary investigation. J Addict Med 2009; 3(1): 26–32. [CrossRef]
- Dong TS, Mayer EA, Osadchiy V, Chang C, Katzka W, Lagishetty V, et al. A distinct brain-gut-microbiome profile exists for females with obesity and food addiction. Obesity 2020; 28(8): 1477–86. [CrossRef]
- Avena NM, Wang M, Gold MS. Implications of food addiction and drug use in obesity. Psychiatr Ann 2011; 41(10): 478–82. [CrossRef]
- Leroy JL, Ruel M, Frongillo EA, Harris J, Ballard TJ. Measuring the food access dimension of food security: a critical review and mapping of indicators. Food Nutr Bull 2015; 36(2): 167–95. [CrossRef]
- Ding M, Ellervik C, Huang T, Jensen MK, Curhan GC, Pasquale LR, et al. Diet quality and genetic association with body mass index: results from 3 observational studies. Am J Clin Nutr 2018; 108(6): 1291–300.
- World Health Organization (WHO). A healthy lifestyle WHO recommendations. Available from: URL: https://www.who.int/europe/ news-room/fact-sheets/item/a-healthy-lifestyle---who-recommendations. Accessed Jul 30, 2022.

- weist Llin vo. Vala faad addistion aasla 2.0
- World Health Organization (WHO). Waist circumference and waist-Hip ratio: Report of a WHO expert consultation. Available from: URL: https:// apps.who.int/iris/rest/bitstreams/53079/retrieve. Accessed Jul 30, 2022.
- 12. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. Int J Food Sci Nutr 2005; 56(5): 303–7. [CrossRef]
- Guenther PM, Reedy J, Krebs-Smith SM. Development of the healthy eating index-2005. J Am Diet Assoc 2008; 108(11): 1896–901.
- Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale food addiction scale. Appetite 2009; 52(2): 430–6. [CrossRef]
- Bayraktar F, Erkman F, Kurtulus E. Adaptation study of Yale food addiction scale. Bull Clin Psychopharmacol 2012; 22(1): 38.
- Yang F, Liu A, Li Y, Lai Y, Wang G, Sun C, et al. Food addiction in patients with newly diagnosed type 2 diabetes in Northeast China. Front Endocrinol (Lausanne) 2017; 8: 218. [CrossRef]
- Eichen DM, Lent MR, Goldbacher E, Foster GD. Exploration of "food addiction" in overweight and obese treatment-seeking adults. Appetite 2013; 67: 22–4. [CrossRef]
- Pursey KM, Stanwell P, Gearhardt AN, Collins CE, Burrows TL. The prevalence of food addiction as assessed by the Yale food addiction scale: A systematic review. Nutrients 2014; 6(10): 4552–90. [CrossRef]
- Hauck C, Weiß A, Schulte EM, Meule A, Ellrott T. Prevalence of 'food addiction' as measured with the Yale food addiction scale 2.0 in a representative German sample and its association with sex, age and weight categories. Obes Facts 2017; 10(1): 12–24. [CrossRef]
- Schulte EM, Gearhardt AN. Associations of food addiction in a sample recruited to be nationally representative of the United States. Eur Eat Disord Rev 2018; 26(2): 112–9. [CrossRef]
- 21. Meule A, Müller A, Gearhardt AN, Blechert J. German version of the

Yale food addiction scale 2.0: Prevalence and correlates of "food addiction" in students and obese individuals. Appetite 2017; 115: 54–61.

- Ceccarini M, Manzoni GM, Castelnuovo G, Molinari E. An evaluation of the Italian version of the Yale food addiction scale in obese adult inpatients engaged in a 1-month-weight-loss treatment. J Med Food 2015; 18(11): 1281–7. [CrossRef]
- Pursey KM, Collins CE, Stanwell P, Burrows TL. Foods and dietary profiles associated with 'food addiction' in young adults. Addict Behav Rep 2015; 2: 41–8. [CrossRef]
- Skinner JA, Garg ML, Dayas CV, Burrows TL. Is weight status associated with peripheral levels of oxytocin? A pilot study in healthy women. Physiol Behav 2019; 212: 112684. [CrossRef]
- Dursun ÜD, Kasım İ, Sümer S, Aksoy H, Kahveci R, Koç EM, et al. Diabetes mellitus prevalence among residents of state nursing homes in Ankara: Original Research. J Tepecik Edu Res Hos 2016; 26(3): 191–6. [CrossRef]
- 26. Guzzardi MA, Garelli S, Agostini A, Filidei E, Fanelli F, Giorgetti A, et al. Food addiction distinguishes an overweight phenotype that can be reversed by low calorie diet. Eur Eat Disord Rev 2018; 26(6): 657–70.
- 27. Day C, Bailey CJ. Obesity in the pathogenesis of type 2 diabetes. Br J Diabetes Vasc 2011; 11(2): 55–61. [CrossRef]
- Klop B, Elte JWF, Cabezas MC. Dyslipidemia in obesity: mechanisms and potential targets. Nutrients 2013; 5(4): 1218–40. [CrossRef]
- Purkins L, Love ER, Eve MD, Wooldridge CL, Cowan C, Smart TS, et al. The influence of diet upon liver function tests and serum lipids in healthy male volunteers resident in a Phase I unit. Br J Clin Pharmacol 2004; 57(2): 199–208. [CrossRef]
- Li F, Chen S, Qiu X, Wu J, Tan M, Wang M. Serum uric acid levels and metabolic indices in an obese population: A cross-sectional study. Diabetes Metab Syndr Obes 2021; 14: 627-35. [CrossRef]