Associations between Emotional Eating and Metabolic Risk Factors at Adolescents with Obesity

Fatma Kubra Sayin[1,*], Muammer Buyukinan[2], Cigdem Damla Deniz[3] and Derya Arslan[4]

[1]*Department of Nutrition and Dietetics, University of Necmettin Erbakan, Health Science Faculty, Konya, Turkey*

[2]*Department of Pediatric Endocrinology and Metabolism, University of Health Sciences, Konya Training and Research Hospital, Konya, Turkey*

[3]*Department of Biochemistry, University of Health Sciences, Konya Training and Research Hospital, Konya, Turkey*

[4]*Department of Pediatric Cardiology, University of Health Sciences, Konya Training and Research Hospital, Konya, Turkey*

Abstract: *Purpose*: This study aimed to determine whether emotional eating (EE) and uncontrolled eating (UCE) scores affect the metabolic risk factors in obese adolescents.

Methods: A sample of 100 adolescents have BMI-SDS between 1.41 and 2.83 (aged 12-17 years) was selected. EE and UCE scores were estimated using the TFEQ21. The association of EE and UCE with anthropometric data, lipid profile, glucose profile, liver enzymes, and inflammation factors was assessed in boys and girls.

Results: Using Spearman rank correlation, EE scores significantly correlated with uric acid (r = 0.393 and P = 0.001), CRP (r = 0.273 and P = 0.017), TG (r = 0.317 and P = 0.001), TC (r = 0.258 and P = 0.019) and VAI (r = 0.276 and P = 0.034). Also UCE scores were showed positive correlation with CRP (r = 0.257 and P = 0.024).

Conclusion: In conclusion, interrelationships tend to exist between EE and triglyceride, uric acid, visceral adiposity index, and CRP levels among obese adolescents.

Keywords: Emotional eating, uncontrolled eating, obesity in adolescents, metabolic risk factors.

1. INTRODUCTION

Food selection and consumption is a complex process that is influenced by the relationship between many factors, such as genetic, psychological, physiological, economic, social, and cultural factors. One of the psychological factors affecting food selection and consumption is emotional eating behavior. The effects of different emotions on the eating behaviors of individuals were investigated. There are differences between individuals; it has been found that negative emotional conditions such as stress, anxiety, depression, anger increase food consumption, and disrupt eating habits [1]. Another type of psychological eating is uncontrolled eating behavior. Uncontrolled eating behavior is defined as the tendency to over food consumption because of losing control [2] Individuals with uncontrolled eating behavior often tend to eat against a negative external stimulus. Obesity is both a cause and a risk factor for the development of uncontrolled eating behavior. In fact, the study of Hays *et al*. (2002) [3] showed that the high BMI value was positively associated with uncontrolled eating behavior. Those who have uncontrolled eating behavior consume more fatty, salty, and high energy density foods [4]. The last type of psychological eating is cognitive restriction behavior. Cognitive restriction behavior was first defined as restricting food intake to support weight loss [2].

Psychological eating behaviors affect the nutrient consumption and preferences of individuals and should be considered, especially for the development of chronic diseases. In fact, the presence of chronic disease is associated with increased emotional eating behavior [5]. Hainer *et al*. (2006) [6] found that cognitive restriction and uncontrolled eating behaviors were related to cardiovascular diseases.

Adolescence is a critical period of development and vulnerability during which eating disorders can develop. Because adverse eating habits, such as intake of high energy-dense (fatty) foods, snacking, skipping breakfast, and eating less fruit and vegetables, seem to be more common among adolescents experiencing stress [7-10].

The risk of eating disorders is high in overweight adolescents. Therefore it is necessary to screen their

*Address correspondence to this author at the Yunus Emre Mahallesi, Beyşehir Caddesi, D block, No:281 Meram, Turkey;
E-mail: kubrakucur@hotmail.com

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psychological eating attitudes occasionally [11]. Clarification of the potential effect of psychological eating attitudes on metabolic risk factors may highlight intervention and prevention targets. So, the objective of the current study was to quantify the association between metabolic risk factors and emotional eating and uncontrolled eating scores derived using TFEQ21 among obese adolescents.

2. MATERIAL AND METHODS

2.1. Participants and Procedure

The research group of the study consisted of 100 (64 girls) overweight and obese adolescents who applied to Pediatric Endocrinology Clinic in Konya Education and Research Hospital between June 2017-December 2018. Patients were enrolled according to the following eligibility criteria: aged between 12 and 17 years, body mass index BMI SDS >1, and the ability to answer a self-reporting questionnaire. Exclusion criteria were as follows: the previous diagnosis of diabetes mellitus, known inflammatory disease, a history of malignant disease or pathologies, or drugs able to modify glucose metabolism. All children and one parent per each child gave written informed consent. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. Ethical approval was obtained from the Necmettin Erbakan University Medicinal Faculty ethics committee review board (Ethical No:2017/1067).

2.2. Questionnaire

The data were collected by a two-part questionnaire. In the first part of the questionnaire, demographic information was collected that consisting of questions determining the age, gender, meal habits, breastfeeding status, and smoking habits. In the second part, the three-factor eating questionnaire TFEQ21 used that evaluates eating behavior through three factors with 21 items. The questionnaire covered the following three aspects of eating motivation: cognitive restraint (6 items), emotional eating (6 items), and uncontrolled eating (9 items). These factors measure cognitive, behavioral and emotional aspects in human eating attitudes: "Cognitive Restraint" (CR), understood as individuals' conscious efforts to control what they eat to keep or lose weight; "Uncontrolled eating" (UE), which expresses the tendency to eat excessively in response to the loss of control over the food itself; and "Emotional eating" (EE), understood as the need to overeat when individuals are unable to cope with emotionally negative situations. Our study mainly focused on the EE and UCE scores.

The scale developed by Stunkard and Messic (1985) [12] was adapted to Turkish by Karakuş *et al*. (2016) [13]. The first subscale assesses emotional eating (6 items, for example, item "I start to eat when I feel anxious") (α = 0.870). The second subscale measures uncontrolled eating (9 items, example item "Sometimes when I start eating, I just can't seem to stop") (α = 0.787). The third subscale measures cognitive restraint (6 items, for example, item "I deliberately take small helpings to control my weight") (α = 0.801). Items were measured using 4-point Likert scales that ranged from "definitely true" to "definitely false".

2.3. Anthropometry

Children's weights were measured using a calibrated scale, and height was measured with a standard stadiometer. Body mass index (BMI) was calculated as weight/height2 in kilograms per square meter. In addition, waist circumference was measured. The pubertal stage was graded according to the Tanner classification. BMI standard deviation scores (SDS) were calculated from the age and sex-specific normal anthropometric data, according to the standards established for Turkish children [14].

2.4. Blood Sample Analysis

Plasma glucose, total and high-density lipoprotein (HDL) cholesterol, triglycerides, and uric acid concentrations were measured by enzymatic methods with Beckman Kits using Beckman Coulter AU 5800 biochemical analyzer (Beckman Coulter, Inc., USA). LDL cholesterol was calculated by the Friedewald equation. Insulin levels were measured by a chemiluminescent method with Siemens immulite 2000XPi immunoassay analyzer (Siemens Inc.). Insulin resistance, estimated by the homeostatic model assessment-method (HOMA-IR) for glucose metabolism.

Hemogram analysis in Sysmex XN-1000 hematology analyzer (Sysmex Corporation, Kobe, Japan) was carried using semiconductor laser flow cytometry and nucleic acid fluorescence staining techniques. Vitamin D was analyzed by automatic immunoassay analysis with Abbott Kits (Abbott Laboratories, IL 60064, USA) on Architect plus i 2000 SR Analyzer. HbA1c was determined using high-performance liquid chromatography (Trinity Biotech

Premier 9210, USA). HOMA-IR index, calculated from the fasting glucose, and insulin concentrations according to the following formula (fasting insulin fasting glucose)/22.5.20. The visceral adiposity index (VAI) was calculated using the formulas proposed by Amato *et al*. [14] for men, VAI=[WC/36.58+(1.8BMI)] (TG/0.81) (1.52/HDL) and for women, VAI=[WC/ 39.68+(1.88BMI)](TG/1.03)(1.31/HDL).

2.5. Statistical Analysis

Descriptive statistics were initially calculated to examine sample demographics and means and standard deviations for study measures. The statistical significance of the differences between metabolic parameters and TFEQ21 between girls and boys was an estimated independent sample T-test. A correlation test was used to assess the relation of EE score, UCE scores with metabolic biomarkers in order to identify variables to control for in the regression models. A linear regression analysis was performed to estimate the association between EE scores and metabolic risk factors that have a correlation with EE scores. To quantify the association between EE with Uric acid, CRP, TG levels, we generated stepwise regression models. Analyses were adjusted for sex, age, BMI. The criterion for statistical significance was set at $P < 0.05$. All analyses were conducted using SPSS Statistics, version 16.

3. RESULTS

3.1. Characteristics of the Participants

One hundred (36% male) adolescents between the ages of 12 and 17 years and have BMI-SDS more than

Table 1: Participants' Demographic Characteristics (Mean and SD)

	All (n=100)	Boys (n =36)	Girls (n = 64)	p-value between boys and girls
Age (years)	14.44±1.75	14.4±1.61	14.46±1.84	NS
Weight (kg)	86.25±13.98	91.27±13.02	83.51±13.83	≤0.05
Height (cm)	164.54±8.40	167.11±10.39	161.35±6.39	≤0.05
BMI (kg/m2)	32.18±4.00	32.48±3.02	32.01±4.47	NS
BMI SDS	2.09±0.25	2.06±0.31	2.10±0.29	NS
ALT (IU/L)	27.54±22.19	39.54±28.90	20.87±13.62	≤0.001
AST (IU/L)	29.45±14.65	37.32±19.88	25.20±8.35	≤0.001
Uric acid (mg/dL)	5.99±1.40	6.88±1.22	5.58±1.52	≤0.001
CRP	6.13±3.11	8.40±4.48	5.22±3.98	≤0.05
TSH	2.80±1.54	2.97±1.76	2.71±1.41	NS
T4	1.23±0.16	1.23±0.15	1.23±0.16	NS
B12	331.71±106.71	333.21±106.17	330.65±107.82	NS
D VİT	14.79±10.08	16.83±7.82	13.69±11.02	NS
TG (mg/dL)	129.24±69.02	149±79.19	130±66.82	NS
TC (mg/dL)	168.98±41.64	166±46.77	170±38.83	NS
HDL-C (mg/dL)	40.19±7.35	39.74±7.62	40.44±7.24	NS
LDL-C (mg/dL)	106.89±30.04	105±31.85	107±29.22	NS
Insulin (mIU/L)	23.75±17.75	22.75±10.03	24.29±20.81	NS
Glukoz (mg/dL)	86.64±13.26	87.71±6.88	86.06±15.69	NS
HOMA-IR	5.11±3.99	4.97±2.25	5.58±5.84	NS
VAI	295.96±188.54	377.23±228.88	249.52±145.18	≤0.05
HbA1c	5.52±0.31	5.52±0.29	5.53±0.32	NS
PLO	128.33±39.38	125.12±36.32	129.23±41.14	NS
NLO	2.56±3.76	2.14±0.99	2.79±4.61	NS
UCE score	23.55±7.65	24.05±7.98	21.20±8.35	NS
EE scores	12.57±5.62	13.58±5.14	12.95±5.55	NS
CR score	15.29±5.25	14.75±4.87	15.58±5.52	NS
TFEQ21 Score	51.31±10.74	51.00±12.10	51.48±10.07	NS

Abbreviations: WC, waist circumference; HDL-C high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment: insulin resistance; UCE: uncontrolled eating; EE: emotional eating; CR: cognitive restraint.

one were prescreened for inclusion in the study. Characteristics of the participants and significant differences between boys and girls are shown in Table **1**. The mean of BMI-SDS in males was 2.06±0.31 and 2.10±0.29 in females. Mean of emotional eating scores (EE) was 13.58±5.14 in males and 12.95±5.55 in females, and uncontrolled eating scores (UCE) was 24.05±7.98 in males and 21.20±8.35 in females no significant differences were found between males and females. TFEQ21 score of two boys and girls 51.00±12.10 and 51.48±10.07, respectively. No significant differences between boys and girls were observed at EE, UCE, and cognitive restriction scores. As shown in Table **1**, levels of ALT (IU/L), AST (IU/L), uric acid (mg/dL) ($p < 0.001$), CRP and VAI ($p < 0.05$) were significantly higher in males than females.

3.2. Correlation of EE and UCE Levels with Metabolic Parameters

The findings of correlations are shown in Table **2** between metabolic risk factors and emotional eating and uncontrolled eating scores. Overall, positive correlations were found between EE and Uric acid (r = 0.393 and P = 0.001), CRP (r = 0.273 and P = 0.017), TG (r = 0.317 and P = 0.001), TC (r = 0.258 and P = 0.019), and VAI (r = 0.276 and P = 0.034). Also UCE scores were showed positive correlation with CRP (r = 0.257 and P = 0.024).

A linear regression analysis was calculated to examine associations between emotional eating scores and metabolic risk factors, which were had a correlation; variables are shown in Table **3**. EE behavior accounted for 15% (Adjusted $R^2 = 0.157$) of the variance in uric acid, and TG for 9% (Adjusted $R^2=0.091$).

4. DISCUSSION

Our study evaluated the effect of EE and UCE on metabolic risk factors of overweight and obese adolescents. For determining the EE and UCE levels Three-Factor Eating Questionnaire (TFEQ)21 used that evaluates eating behavior through three factors [16]. These factors measure cognitive, behavioral, and emotional aspects of human eating attitudes [16]. Emotional, uncontrolled, and restrictive eating is problematic eating behavior characterized by food intake independent of hunger but based on emotional state.

Table 2: Correlations between Uncontrolled Eating, Emotional Eating Scores, and Metabolic Risk Factors

	Uncontrolled eating score		Emotional eating scores	
	r	p	r	p
Age (years)	0.069	0.45	0.098	0.341
Weight (kg)	0.078	0.398	0.136	0.103
BMI (kg/m2)	0.118	0.090	0.178	0.086
BMI SDS	0.105	0.079	0.203	0.078
ALT (IU/L)	0.128	0.263	0.179	0.087
AST (IU/L)	0.160	0.116	0.126	0.209
Uric acid (mg/dL)	0.179	0.088	0.393	0.001*
CRP	0.257	0.024*	0.273	0.017*
B12	0.068	0.256	0.156	0.130
D VİT	0.102	0.338	0.096	0.355
TG (mg/dL)	0.122	0.184	0.317	0.001*
TC (mg/dL)	0.067	0.127	0.258	0.019*
HDL-C (mg/dL)	0.105	0.240	0.037	0.487
LDL-C (mg/dL)	0.088	0.412	0.077	0.165
Insulin (mIU/L)	0.076	0.238	0.108	0.096
Glukoz (mg/dL)	0.080	0.362	0.188	0.085
HOMA-IR	0.091	0.278	0.212	0.036*
VAI	0.127	0.212	0.276	0.034*

Table 3: Multiple Linear Regression of Emotional Eating Scores and CRP, Uric Acid, TG Levels

	CRP			Uric acid			TG		
	OR(95%CI)	p	Adj R2	OR(95%CI)	p	Adj R2	OR(95%CI)	p	Adj R2
	0.281(0.083-0.479)	0.006	0.077	0.406(0.060-0.160)	0.000	0.157	0.317(1.058-5.858)	0.001	0.091
M1	0.245(0.081-0.484)	0.032	0.075	0.381(0.058-0.151)	0.000	0.285	0.308(1.267-6.064)	0.002	0.088
M2	0.225(0.074-0.477)	0.025	0.047	0.363(0.057-0.158)	0.014	0.163	0.306(0.962-5.886)	0.002	0.076
M3	0.301(0.027-0.426)	0.008	0.069	0.443(0.069-0.172)	0.000	0.167	0.310(0.654-5.62)	0.003	0.082

M1: Model adjusted for age and gender; M2: Model adjusted for BMI SDS; M3: Model adjusted for UCE.

In the current study, body composition variables were not shown a strong relationship with the EE and UCE factors. The lack of relationship between emotional eating and BMI of children is somewhat surprising given that previous studies have shown a relationship[17,18]. Similar to our results Carnell 2013 (19) and Löffler 2012 (20) have not reported a clear relationship between EE and being overweight in children. Eating in response to negative emotional states has been described as an obesogenic feature that contributes to weight gain in children (21,22). Despite this, the relationship between emotional eating and metabolic risk factors among obese children is unclear.

Moreover, we found that EE showed a correlation between VAI that is a novel index derived from BMI, WC, and lipid parameters. VAI is important for metabolic risk estimation because studies are showing that it is associated with visceral adiposity, low adiponectin level, impaired insulin, and glucose levels [23-25]. Also, in obese patients, visceral adipose tissue accumulation has been associated with increased production of free fatty acids, interleukin-6, tumor necrosis factor-α, CRP, and a decreased production of adiponectin [26].

Also, a significant relationship was revealed among the EE and uric acid levels. An increase in uric acid has been associated with obesity and all risk factors for atherosclerosis [27]. The mechanism by which the eating behaviors are able to induce an alteration of metabolic profile and the inflammatory profile is not known. But it may be explained by sweet eating, night eating, and craving for carbohydrates that may lead to increased waist circumference and visceral adiposity index, which may support the production of inflammatory molecules. Besides, subjects with higher EE scores were shown to have larger consumption of high-density food, such as cakes, biscuits, sweet high-fat foods, chocolate, crisps, and biscuits [28, 29] and a greater passion for sweet-and-fatty foods [30] compared with that of subjects with lower EE scores. Previous research has shown that EE is positively related to more frequent energy-dense snack consumption, for example, cakes, cookies, chocolate, ice-cream [31-34], and UCE is associated with higher energy and fat intake [35]. This means these problematic eating styles are associated with unhealthy dietary choices [31-33, 35].

In addition, the study is limited by the use of self-report measures completed by adolescents. Adolescents' reporting of their own emotional eating could be affected by difficulties understanding emotional eating questions and poor self-awareness.

5. CONCLUSIONS

In conclusion, the regressions conducted in our cohort confirmed the impact of EE predicting uric acid and TG. Also, EE has shown a positive correlation with CRP, uric acid, TG, TC, and VAI. The current study expands the actual knowledge about the effects of emotional eating behaviors on important metabolic and inflammatory alterations. Evaluating emotional eating situations of adolescents could represent an important clinical target for identifying a potential risk for cardiometabolic disease. However, further researches are needed to assess and evaluate whether early detection and intervention for any problematic eating situation could have a positive prognostic impact on the long-term metabolic risks of adolescents.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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