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The Psychological Determinants of Emotional and External Eating Behavior in a University Student Sample from Turkey*

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Psychological factors and adverse childhood events at an early age have been poorly investigated in relation to risky eating behavior and obesity. The importance of this relationship grows as these behaviors are becoming public health problems. The main objective of the present study was to examine the effects of interrelated psychological factors such as childhood negative life events, symptoms of depression and anxiety, and impulsivity on eating behaviors in a university student sample in Turkey. A total of 414 undergraduate students (60.4% women) in Turkey, completed the Turkish versions of the Dutch Eating Behavior Questionnaire (DEBQ) to assess emotional eating and external eating and the CDC-Kaiser Permanent Adverse Childhood Experiences (ACE), Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), and Barratt Impulsivity Scale-11 (BSI-11). Structural equation models (SEM) were used to evaluate whether depressive/anxiety symptoms could be a mediator between impulsivity and emotional eating/external eating. Childhood adverse events were both weakly correlated with eating behavior and impulsivity measures (r = .18,r = .275, p <.001, respectively). Depression and anxiety levels were found to be significantly associated with all variables except for the body mass index (BMI; r = .121–.395, p <.001). Body mass index (BMI) was also significantly correlated with emotional eating (r = .231, p <.001) and restrained eating (r = .226, p <.001). Impulsivity was positively and directly associated with symptoms of anxiety and depression and emotional and external eating (respectively β = .27, p <.001; β = .31, p <.001; β = .16, p = .006; β = .13, p = .047). The effect of adverse experiences on both emotional eating (β = .147; 95% CI [.087, .247]) and external eating (β = .091; 95% CI [.032, .168]) was found to be partially mediated by impulsivity and

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symptoms of anxiety and depression. The results supported the proposition that symptoms of depression and anxiety levels are mediators between impulsivity and emotional eating/external eating. This finding suggests that a through psychological assessment should be taken into consideration when evaluating the eating behavior of university students and prevention and treatment strategies applied for disordered eating behaviors in the future.

Keywords: Eating behavior, obesity, childhood adverse events, depression, anxiety, impulsivity

Highlights:

- Adverse childhood events correlated with impulsivity and emotional eating.
- Adverse childhood events had an indirect effect on symptoms of anxiety/depression through impulsivity in our model.
- Impulsivity positively and directly correlated with symptoms of anxiety/depression and emotional/external eating.
- Anxiety symptoms had a direct effect on emotional and external eating.
- Symptoms of anxiety and depression were mediators between impulsivity and emotional / external eating.

Individuals engage in eating behavior as a matter of survival, normally every single day. Specifically, choices have to be made about what to eat, when, and how much. In today's world, these choices have become more difficult for us compared to our ancestors since food might be regarded as more abundant, cheap, and available in a wide variety (Vögele & Gibson, 2010). From a psychological perspective, eating is fundamentally a rewarding behavior and is therefore intrinsically linked to emotions and mood. Healthy eating behavior means being able to enjoy that rewarding aspects of food without falling prey to a loss of control overeating (Vögele & Gibson, 2010).

Numerous factors are known to determine or guide eating behaviors automatically and implicitly (Cohen & Farley, 2010). Eating behaviors, a set of actions that establish the relationship of the individual with food, are essentially acquired during the early years of life; though, they can be altered during the adolescence period. It is known that the years of university education are a critical period that brings changes in eating behaviors of the young individuals. Poor eating habits are a major public health concern among young adults who transited to university life (Nelson et al., 2008), during which they are also exposed to stress, time, and peer pressure (e.g., Sajwani et al., 2009; Webb et al., 1998). University students skip meals and develop poor eating habits, consequently run the risk of incurring deficiencies in certain essential nutrients, developing eating disorders, and even suffering from obesity (Chin & Mohd, 2009; Savige et al., 2007). In addition to poor eating habits, rapid changes in physical growth and psychosocial development have placed young adults as nutritionally vulnerable group (Chin & Mohd, 2009; Savige et al., 2007).

Disordered eating acquired at a younger age can persist into adult life and lead to obesity and other chronic medical conditions with an increase in health problems, loss of workforce, decrease in quality of life, and economic losses (e.g., Goettler et al., 2017). Prevalence estimates of disordered eating in university students range from 11% to 17% in female and 4% in male students (e.g., Lipson & Sonneville, 2017). Disordered eating and obesity should be handled within the biopsychosocial model and defined as an illness that includes genetic factors, environmental factors, and psychological factors (Rosenbaum & White, 2016).

Psychological factors influencing eating behaviors include factors such as stress, depression, anxiety, boredom, and loneliness (e.g., Torres & Nowson, 2007). Obesity, associated with many disorders such as disorders of the cardiovascular system, of the endocrine system, of the locomotor system, is often accompanied by depression, stress and anxiety, reported trauma, and adverse events, as well as with psychopatological eating styles such as emotional eating, addictive eating behaviors, and binge eating (e.g., Torres & Nowson, 2007; Smyth et al., 2008). Unlike depressive disorders accompanied by the loss of appetite, depression with atypical characteristics is characterized by an increase in appetite that can later lead to weight gain and obesity (American Psychiatric Association, 2013). Emotional eating has been considered a marker of this depression subtype (e.g., Paans et al., 2018) because it shares the atypical feature of increased appetite in response to distress such as feelings of depression with this subtype of depression. Emotional eating can be described as a tendency to overeat in response to negative emotions. Individuals may engage in emotional eating as an attempt to cope with stress and other negative emotions. Yet, in the long-term, emotional eating is often regarded as a maladaptive emotion regulation strategy (e.g., Konttinen, 2020). In such cases, although the individual does not feel hungry physiologically, he/she exhibits an eating behavior (Taitz & Safer, 2012). In emotional eating, eating is used as a way to reduce the emotional intensity that cannot be clearly described when bored, idle or alone, and eating behavior is often displayed in response to emotions such as anger, sadness, and anxiety / irritability. An increasing number of prospective studies have shown that emotional eating predicts subsequent obesity in adulthood and these studies consistently support the idea that self-reported emotional eaters consume energy-dense foods more frequently and are at increased risk for developing obesity (e.g., Koenders & van Strien, 2011; Van Strien et al., 2016; Konttinen et al., 2019). Van Strien et al. (2016) reported that emotional eating acted as a mediator between depression and 5-year weight gain in Dutch mothers.

In addition to stress and depression, adverse life experiences during childhood such as emotional abuse, physical abuse, sexual abuse, sexual harassment, bullying by peers, witnessing domestic violence, and serious accidents that threatened the lives of subjects might be potential risk factors for obesity by means of emotional eating (Treuer et al., 2005; Evers et al., 2010; Alvarez et al., 2007). It is reported that dissociation might have played a mediating role between the presence of early trauma and the development of

obesity and eating disorders (e.g., Treuer et al., 2005). Subjects with adverse childhood experiences have a higher risk of developing maladaptive coping strategies, including stress-induced emotional eating (e.g., Evers et al., 2010). Perceived stress might explain the link between child abuse and the development of obesity in adulthood (Alvarez et al., 2007).

Impulsivity is a personality trait corresponding to a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others" (Moeller et al., 2001). Impulsivity has been found to be associated with overeating (e.g., Jansen et al., 2009) and eating disorders (e.g., Rosval et al., 2006; Waxman, 2009). It has been reported that increased impulsivity is associated with psychiatric comorbidities in obesity, which may affect the results after bariatric surgery (e.g., Sarwer et al., 2021). In recent years, with increasing obesity, bariatric surgery operations have also increased. Therefore, it is emphasized that psychiatric evaluation is important in determining the indications and contraindications for bariatric surgeries and predictors of postoperative success.

There is a scarcity of existing literature related to the relationship between eating behaviors and psychological factors in university students in Turkey. The main objective of the present study was to examine the effects of interrelated psychological factors such as childhood negative life events, depression, anxiety, and impulsivity on eating behavior. We hypothesized that individuals with psychological problems, specifically individuals with negative life events during their childhood, individuals with higher levels of symptoms of anxiety and depression, and impulsivity levels were more prone to developing emotional eating that could lead to obesity. We also hypothesized that individuals with higher levels of symptoms of anxiety and depression often had dysfunctional coping strategies and were more prone to developing abnormal eating behaviors, accompanied by periods of overeating or restrained eating in order to moderate their negative mood states. Our final hypothesis tested was whether eating behaviors were mediated by levels of impulsivity and symptoms of anxiety and depression.

Method

Participants

The study was approved by the Marmara University School of Medicine's Ethics Review Board. In Total, 414 undergraduate students from the Marmara University School of Medicine in Istanbul, Turkey took part in the study after giving informed consents. Participants completed a set of questionnaires below. Body Mass Indexes (BMIs) were calculated using the participants' actual weight/height.

There were 250 females (60.4%) and 164 males (39.6%) participants. Mean age was 22.58 ± 2.19, ranging from 18 to 32 years. The marital status of the participant was heavily skewed, with 98.8% single, followed by 1% married, and 0.2% divorced. In terms of income, 142 of participants (34.2%) stated their income was low, 163 (39.4%) had moderate level

of income, and 109 (26.3%) had high-income. The sociodemographic and clinical data was demonstrated in Table 1.

Table 1
Sociodemographic characteristics of the study participants

Variables	Mean ± *SD* / *n*(%)
Age	22.58 ± 2.20
Gender	
Male	164 (39.6)
Female	250 (60.4)
Marital Status	
Single	409 (98.8)
Married/cohabiting	4 (1)
Divorced	1 (0.2)
Total education time (year)	16.77 ± 1.84
Body Mass Index (BMI)	22.46 ± 3.67
Employment	
Unemployed	403 (97.3)
Part-time employee	11 (2.7)
Employee	0(0)
Income (Turkish Lira)	
Low (<1500)	142 (34.2)
Middle (1500−2000)	103 (24.7)
Middle-high (2000−3500)	60 (14.5)
High (> 3500)	109 (26.3)
Smoking	
None	340 (82.1)
Present	74 (17.9)
Alcohol using	
None	311 (75.1)
Present	103 (24.9)
Psychiatric treatment	
None	384 (92.8)
Present	30 (7.2)

Instruments

The following questionnaires were administered using in the paper-pencil form to the study participants in order to evaluate their eating behaviors and psychological factors such as childhood negative life events, symptoms of depression and anxiety, self-esteem, and impulsivity.

Sociodemographic Data Form

This form included demographic variables including age, gender, marital status, number of children, education, location, household members, occupation, and employment status. We also asked the number of glasses of raki (local hard alcohol drink) and wine consumed per week in that form.

The Dutch Eating Behavior Questionnaire (DEBQ)

The DEBQ is a 33 item-scale developed to evaluate emotional eating, external eating, and restrained eating behaviors (Van Strien et al., 1986). The DEBQ contains three subscales: emotional eating (DEBQ-E; 13 items; e.g., ‘‘Do you have a desire to eat when you are irritated’’?), restrained eating (DEBQ-R; 10 items; e.g., ‘‘Do you try to eat less at mealtimes than you would like to eat?’’), and external eating (DEBQ-X; 10 items; e.g., ‘‘If food smells and looks good, do you eat more than usual?’’). The DEBQ-E contains 13 items 4 of which describe eating in response to diffuse emotions and 9 of which describe eating in response to clearly labeled emotions. Response categories range from 1 (*never*) to 5 (*very often*). The DEBQ was found to be reliable in both obese and non-obese men and women (Van Strien et al., 1986). The DEBQ was adapted into Turkish by Bozan et al. (2011). Cronbach’s α coefficients of Turkish DEBQ was .90 for external eating, .92 for emotional eating, and .96 for restrained eating subscales (Bozan, Bas, & Asci, 2011). Cronbach’s α coefficient in this study was .85 for external eating, .97 for emotional eating, .90 for restrained eating subscales.

The CDC-Kaiser Permanente Adverse Childhood Experiences (ACE)

It is a 10-item self-report scale. It consists of 10 items, each measuring one type of childhood trauma. Each ‘yes’ answer is coded as 1 point. The total score ranges from 0 to 10. It has no cutoff value. The ACE scale was developed by the CDC and Kaiser Permanente in 1997 (Felitti et al., 1998). Adverse Childhood Experiences (ACEs) are categorized into three groups: abuse, neglect, and household challenges. Each category is further divided into subcategories. The ACE score is the total sum of the different categories of ACE reported by participants. Study findings showed a graded dose-response relationship between ACE and negative health and well-being outcomes. In other words, as the number of ACE increases so does the risk for negative outcomes. Cronbach’s alpha value of the total score of the Turkish ACE scale was .74 (Gündüz et al., 2018). Cronbach’s α coefficient in this study was .68.

Beck Depression Inventory (BDI)

The BDI is a self-report scale composed of 21 items and measures somatic, emotional, cognitive, and impulsive symptoms of depression. Each item takes a point between 0 and 3. The point that can be taken from inventory varies between 0 and 63, and high points indicate a rise in thedepressive mood. Overall scores for all questions is evaluated like this: a score between 10–16 shows low depression symptom, a score between 17–29 is middle depressive symptom, and a score between 30–63 is severe depressive symptom (Beck, Steer, & Carbin, 1988). The BDI has been adapted into Turkishby Hisli (1988). Split-half reliability of the Turkish BDI was found .74 and Cronbach’s alpha was .80 (Hisli, 1988). Cronbach’s α coefficient in this study was .90.

Beck Anxiety Inventory (BAI)

The BAI is a 21-item self-report questionnaire that lists symptoms of anxiety (Beck et al., 1988). The respondent is asked to rate how much each symptom has bothered him/her in the past week. The symptoms are rated on a four-point scale, ranging from *not at all* (0) to *severely* (4). The instrument has excellent internal consistency measure Cronbach’s alpha of .92 and high test–retest reliability (r =.75). Cronbach’s α coefficient of internal consistency of the Turkish BAI was .93 (Ulusoy et al. (1998). Cronbach’s α coefficient in this study was.90.

Barratt Impulsivity Scale-11 (BIS-11).

BIS-11 is a 30-item self-report questionnaire to assess impulsiveness as a trait, of which the metrics had presented evidence concerning its validity, reliability, and predictive

value in the study of validity and reliability (Barratt, 1959). BIS-11 is composed of 30 items scored on a Likert scale (ranging from never = 1 point to very frequently = 4 points). It assesses the three main dimensions of impulsive behavior: attentional (a lack of focus on the ongoing task), motor (acting without thinking), and non-planning impulsivity (orientation to the present rather than to the future). Cronbach's alpha coefficient for the total BIS-11 was .83 and test-retest reliability coefficient at one-month was .83 in a sample of adults (Stanford et al. 2009). BIS-11 has been adapted into Turkish and Cronbach's alpha coefficient for the total BIS-11 were .78 for undergraduate students and .81 for psychiatry patients, while test-retest reliability coefficient was found .83 for undergraduate students (Gulec et al. 2008). Cronbach's α coefficient in this study was .82.

Descriptive statistics of the scales used in the present study were shown in Table 2.

Table 2
Descriptive statistics of the study scales

SCALES	Cronbach's alpha	Mean	Std. Deviation	Minimum	Maximum
DEBQ-EmE	.971	33.35	14.93	13.00	65.00
DEBQ-RE	.905	24.09	8.13	10.00	49.00
DEBQ-ExE	.852	32.67	6.84	14.00	50.00
BDI	.909	10.96	8.99	.00	57.00
BAI	.904	10.12	9.31	.00	53.00
ACE-Q	.683	.94	1.44	.00	8.00
Barratt-A	.658	17.22	3.69	9.00	30.00
Barratt-M	.698	19.39	4.32	11.00	35.00
Barratt-NonP	.681	24.11	4.60	11.00	41.00

Note. DEBQ-EmE: Dutch Eating Behavior Questionnaire − Emotional Eating;DEBQ-RE: Dutch Eating Behavior Questionnaire – Restrained Eating; DEBQ-ExE: Dutch Eating Behavior Questionnaire − External Eating;BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; ACE-Q: CDC-Kaiser Permanente Adverse Childhood Experiences Questionnaire; Barratt-A: Barratt Impulsivity Scale − Attentional; Barratt-M: Barratt Impulsivity Scale − Motor; Barratt-NonP: Barratt Impulsivity Scale − Non-planning

Statistical Analysis

Data screening

Preliminary data exploration indicated that missing values were distributed completely at random (Little's MCAR test: $\chi 2 = 97.09$, $df = 96$, $p = .45$). Fewer than 2.5% of cases for each variable had missing values. Missing values were replaced with the mean values for age, weight and heights. For all subscales items' missing values were replaced with the median value. All scales were screened for deviation from normality. For any subscale, univariate outliers were identified according to the resistant labeling rule (Hoaglin, Iglewicz, & Tukey, 1986). The remaining outliers after logarithmic or square root transformation were replaced with the mean values corresponding to the absolute z score of 2.68. Distribution shape was reexamined after replacement of the outliers. Shapiro Wilk test results were not satisfactory except for that of External Eating ($p = .093$). The absolute skewness value of all study variables was between .026 and 1.448, and the kurtosis was between .034 and 1.107. Finally, the data was agreed to be normally distributed considering the histogram suggestions too.

Correlations were calculated to explore the associations of interest followed by path analysis using SPSS version 25.0 (IBM SPSS Statistics for Windows. Armonk, NY: IBM Corp.). Pearson product-moment was calculated of each pairwise association. Structural equation models, using Maximum Likelihood Estimation, were run to test hypothesized mediational effects and interactions among the latent variables: adverse childhood experiences (ACEs), impulsivity (BIS-11), anxiety (BAI), depression (BDI) and eating behaviors (Emotional Eating and External Eating from DEBQ) as an endogenous variable, using AMOS version 23.0 (Amos 23.0 User's Guide. Chicago: IBM SPSS). Parcel building method was carried out to enhance scale communality and to reduce random errors caused by the large number of observed items. Number of parcels were determined in balanced as suggested by Bandalos (2002) to prevent possible bias of inflated model fit and path coefficients. All of the scales whose structural validity was studied before are known to be unidimensional, except BISS-11. Factorial algorithm was used in construction in which the items were sequentially distributed to each parcel by the highest to the lowest factor loadings, alternating the direction of items-picking turns through the parcels. Domain representative type of parcels were generated for BISS-11 proposed by Graham et al. (2000) for multidimensional constructs.

Structural equation models (SEM) were applied for mediation analysis. We calculated the indirect effect of the specified paths with bootstrapping method using 5000 bootstrapped samples (iterations) from the data by random sampling recommended by Hayes (2009). Goodness of fit of the models was evaluated using multiple indices: normative chi-square statistics ($\chi^2/df < 3$), goodness of fit index (good if GFI > .90, best if above .95), comparative fit index (good if CFI > .90 best if above .95), Tucker-Lewis index (good if CFI > .90, best if above .95) root mean square error of approximation (RMSEA < .06), standardized root mean square residuals (SRMR < . 08) of which cut-off values were indicated by Hu and Bentler (1999). The statistically significance threshold was set at $p < .05$.

Results

Correlation Analyses

Correlations were computed among BMI, ACE-Q, three subscales of DEBQ, three subscales and total scores of BIS-11, BDI, BAI, and BMI. The results suggest that 42 out of 55 correlations were statistically significant ($r = .10 - .81$.), most of which were mild to moderate. Analysis revealed that childhood adverse events were associated with one out of the three subscales of eating behavior (i.e., emotional eating: $r = .10$, $p < .05$). Childhood adverse events were correlated slightly stronger with impulsivity measurements ($r = .186 - .275$, $p_{all} < .001$) than those with eating behavior measurements. Depression and anxiety levels were found to be significantly associated with all variables except BMI. BMI was significantly correlated with emotional eating and restrained eating. Another finding to be noted is that restrained eating behavior was not found to be significantly correlated with any measurements of impulsivity. All of the bivariate correlation results were shown in Table 3.

Table 3.
*Bivariate correlation*s

	1	2	3	4	5	6	7	8	9	10
1.BMI										
2.DEBQ-EmE	.231**									
3.DEBQ-RE	.226**	.236**								
4.DEBQ-ExE	.065	.315**	-.106*							
5.BDI	.024	.236**	.121*	.134**						
6.BAI	-.002	.246**	.169**	.195**	.602**					
7.ACE-Q	.105*	.098*	.029	.086	.329**	.277**				
8.Barratt-A	.115*	.184**	.006	.152**	.395**	.341**	.275**			
9.Barratt-M	.058	.118*	-.036	.142**	.168**	.181**	.208**	.525**		
10.Barratt-NonP	-.044	.145**	-.053	.061	.251**	.200**	.186**	.471**	.441**	
11.Barratt-Total	.046	.183**	-.037	.143**	.328**	.291**	.272**	.798**	.813**	.809**

Note. * Correlation is significant at the.05 level (2-tailed); ** Correlation is significant at the .01 level (2 tailed). BMI: Body Mass Index; DEBQ-EmE: Dutch Eating Behavior Questionnaire – Emotional Eating; DEBQ-RE: Dutch Eating Behavior Questionnaire – Restrained Eating; DEBQ-ExE: Dutch Eating Behavior Questionnaire – External Eating; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; ACE-Q: CDC-Kaiser Permanente Adverse Childhood Experiences Questionnaire; Barratt-A: Barratt Impulsivity Scale – Attentional; Barratt-M:Barratt Impulsivity Scale – Motor; Barratt-NonP: Barratt Impulsivity Scale – Non-planning.

Measurement Model

Six latent variables (adverse childhood events, impulsivity, anxiety, depression, emotional eating, and external eating) and twenty-one observed variables corresponding to parcels were included in the measurement model. Confirmatory Factor Analysis was run to evaluate whether the latent variables were adequately operationalized through their respective indicators or not. Although fit indices of non-modified model were acceptable, better fit to data was achieved by including two error covariances as suggested by modification indices in accordance with the theoretical background (as seen in Figure 1): $\chi^2/df = 1.714$; RMSEA = .04; CFI = .98; GFI = .93; TLI = .97; SRMR = .03. All factor loadings of indicators on latent variables were found to be statistically significant ($p < .001$), ranging from .72 to .98. Covariances between latent variables were also found to be statistically significant ($p < .001$).

SEM Model

The initial model contained direct pathways from adverse childhood events to both eating behaviors in addition to the final model shown in Figure 1. The preliminary results of the first structural equation modelling (SEM) procedure revealed that goodness of fit was improved with adding covariance between error terms of “e24” and “e30”: $\chi^2/df = 2.343$; RMSEA = .06; CFI = .96; GFI = .91; TLI = .96; SRMR = .08. However, direct paths from childhood to both of emotional eating and external eating were poor and non-significant. Even if there was no remarkable improvement in the fit indices, we decided

to remove the above-mentioned pathways to make the model consistent with the hypothetical basement. The fit indices of the trimmed model were χ^2/df = 2.316; RMSEA = .05; CFI = .96; GFI = .92; TLI = .96; SRMR = .08, indicating good fit.

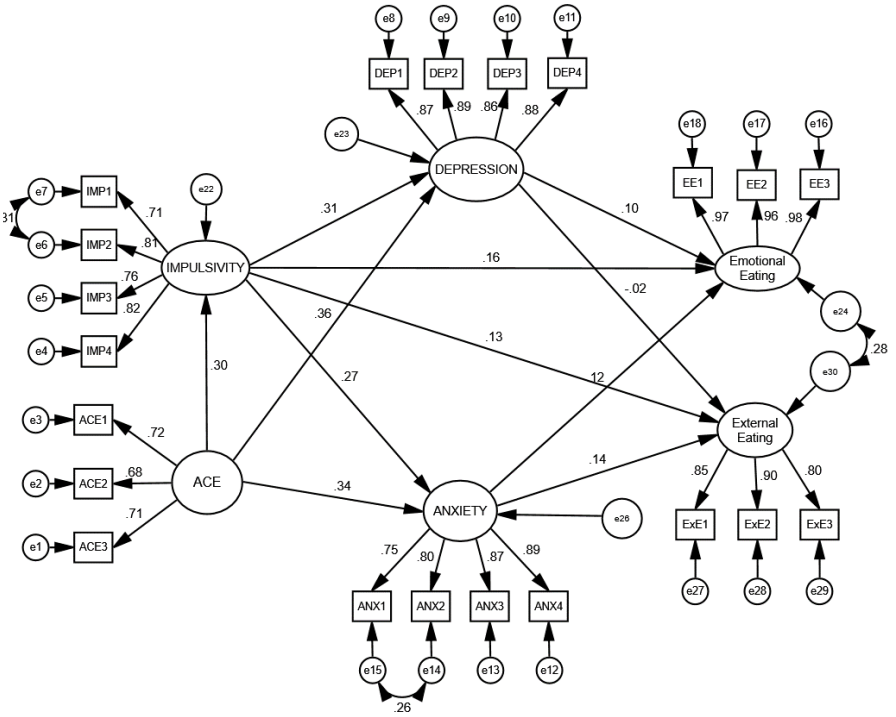
The SEM results indicated that all paths proposed in the hypothetical model, of which the coefficients were mild to moderate, were statistically significant (p <.001 and p <.05), except for depression to both external eating and emotional eating. Impulsivity, a key variable of the model, was positively and directly associated with anxiety, depression, emotional eating and external eating (respectively β = .27, p < .001; β = .31, p < .001; β = .16, p = .006; β = .13, p = .047). Anxiety had a significant direct effect on both external eating (β = .14, p = .018) and emotional eating (β = .12, p = .03), which makes a unique contribution to model in that depression could not remain as another affective component. The paths from adverse childhood events to impulsivity, anxiety and depression were the strongest in the model (respectively, β = .30, β = .34, β = .36; p_{all} < .001). The SEM results were displayed in Table 4 and Figure 1.

Table 4
Structural equation model (SEM) path coefficients and significance levels

Endogenous Variable	Exogenous Variable	β (Std.)	β	S.E.	C.R.	*p*
ACE	Impulsivity	.304	.353	.073	4.86	<.001
ACE	Anxiety	.339	.427	.078	5.493	<.001
ACE	Depression	.357	.441	.074	5.937	<.001
Impulsivity	Depression	.311	.330	.056	5.856	<.001
Impulsivity	Anxiety	.269	.291	.059	4.920	<.001
Impulsivity	Emotional Eating	.164	.195	.071	2.722	.006
Impulsivity	External Eating	.128	.132	.066	1.988	.047
Depression	Emotional Eating	.102	.114	.062	1.825	.068
Depression	External Eating	-.016	-.015	.058	-.265	.791
Anxiety	Emotional Eating	.120	.131	.061	2.165	.030
Anxiety	External Eating	.141	.134	.056	2.373	.018

Note. ACE: Adverse Childhood Events

Figure 1
Standardized path estimates of the final SEM representing interrelationship of adverse childhood experiences, impulsivity, depression, anxiety, emotional and external eating



The bootstrapping results revealed a number of significant indirect relationships. ACE had a significant indirect effect on depression (β = .094; CI 95% [.052, .154]) and anxiety (β = .082; CI 95% [.044, .137]) through impulsivity. Depression and anxiety were found to be partial mediators in relationship of impulsivity and emotional eating (β = .064; CI 95% [.022, .131]), but not in that of external eating. When examining the full model, the effects of ACE on external eating (β = .091; CI 95% [.032, .168]) and emotional eating (β=.147; CI 95% [.087, .247]) were found to be partially mediated by multiple mediators: impulsivity, anxiety, and depression. Table 5 shows the results of standardized bootstrapped estimates of direct and indirect relationships.

Table 5
Bootstrapped standardized coefficients of direct and indirect effects with 95% confidence interval

Model Pathways	Direct Effect	95% CI of DE		Indirect Effect	95% CI of IE	
		lower	upper		lower	upper
ACE→IMP→DEP	.357	.245	.555	.094	.052	.154
ACE →IMP →ANX	.339	.170	.534	.082	.044	.137
ACE→IMP→DEP+ANX→Em. Eating	-	-	-	.147	.087	.247
ACE →IMP →DEP+ANX →Ext. Eating	-	-	-	.091	.032	.168
IMP→DEP+ANX→Em. Eating	.164	.038	.282	.064	.022	.131
IMP→DEP+ANX→Ext. Eating	.128	-.016	.261	.033	-.005	.095

Note. ACE: Adverse Childhood Events; DEP: Depression; ANX: Anxiety; Em. Eating: Emotional Eating; Ext. Eating: External Eating.

Figure 1 presents the associated path diagram for the path analysis for mediation testing. Significant direct effects on eating behaviors included positive associations of impulsivity (β = .164, p = .006) and anxiety (β = .120, p = .030) for emotional eating and impulsivity (β = .128, p = .047) and anxiety (β = .141, p = .018) for external eating.

Discussion

The purpose of this study was to determine the effects of interrelated psychological factors such as childhood negative life events, depression, anxiety, and impulsivity on eating behavior. In this study, depressive symptoms were not associated with higher BMI, while significant associations were found between depressive symptoms and emotional eating, as well as between emotional eating and BMI. Moreover, according to the results of the mediation analysis, impulsivity was found to be a key variable of the model and was positively and directly associated with symptoms of anxiety, depression, external and emotional eating. Anxiety had a significant direct effect on both external eating and emotional eating. The paths from adverse childhood events to impulsivity, anxiety, and depression were the strongest in the model. While adverse childhood events (ACE) had a significant indirect effect on depression and anxiety through impulsivity; the effect of ACE on both of emotional eating and external eating was found to be partially mediated by multiple mediators (impulsivity, anxiety, and depression). Childhood adverse events were correlated with impulsivity measurements. On the other hand; symptoms of depression and anxiety levels were found to be correlated with all variables except BMI. Impulsivity was correlated with anxiety, depression, emotional eating, and external eating. Finally, the effect of ACE on emotional eating and external eating was mediated by impulsivity, anxiety, and depression.

Despite the finding that adverse childhood events significantly correlated with BMI, levels of symptoms of anxiety and depression were not correlated with BMI. This lack of association might be due to selection of participants with

narrow range of BMI scores from a non-clinical university student population. BMI was also significantly correlated with emotional eating and restrained eating. The association between emotional eating and BMI found in the present study is consistent with previous studies, where the emotion– and stress-related eating was measured in university students (e.g., Lazarevich et al., 2015; Ozier et al., 2008). Similarly, depression and anxiety levels of the study participants were significantly correlated with their emotional eating, restrained eating, and external eating. These results supported our hypothesis that depressed and anxious individuals often had dysfunctional coping strategies and would be prone to developing disordered eating, accompanied by periods of overeating or restrained eating in order to manage their negative mood states. Food can often be considered a natural reward or a source of satisfaction, while coping with negative emotions in the absence of alternative productive behaviors (Lazarevich et al., 2015). Childhood adverse events were correlated slightly stronger with impulsivity measures than those with eating behavior measurements with only significant correlation with emotional eating. Attentional, motor, and non-planning impulsivity levels of the study participants were significantly correlated with their emotional eating and external eating. We found that restrained eating behavior was not significantly correlated with any of the measures of impulsivity. This was consistent with the concept that some dimensions of impulsivity have been implicated in overeating and more specifically in exaggerated food consumption with a loss of control (Espel, Muratore, & Lowe, 2017). High impulsivity was reported to be associated with bulimia nervosa and the inverse correlation was found in anorexia nervosa (Cassin & Von Ranson, 2005).

Our structural equation modelling findings indicated that all paths proposed in the hypothetical model were statistically significant, except for depression to external eating and emotional eating. Impulsivity was a key variable of the model and positively and directly correlated with anxiety, depression, emotional eating, and external eating. Anxiety had a significant direct effect on both external eating and emotional eating, which made a unique contribution to the model. Although Lazarevich et al. (2016) reported that emotional eating acted as a mediator between depression and future body weight gain, in our study, depression did not have a significant direct effect on emotional and external eating. Lack of this association might be due to emotional or external eating characteristics of the study population, which is not a clinical population. Selection of participants with extreme emotional eating scores could have been a better strategy to identify emotional eating behavior. Another possible explanation of this finding might be the lower statistical power due to sample size. The paths from adverse childhood events to impulsivity, anxiety and depression were the strongest in the model. The bootstrapping results revealed a number of significant indirect relationships. ACE had a significant indirect effect on depression and anxiety through impulsivity. Depression and anxiety were found to be partial mediators in the relationship between impulsivity and emotional eating, but not in that of external eating. When examining the full model, the effect of ACE on both emotional eating and external eating was

found to be partially mediated by multiple mediators: impulsivity, anxiety, depression. The emotional eating mediation effect identified in the present study is consistent with previous studies that reported that emotional eating acted as a mediator between depression and future body weight gain (Clum et al., 2014, Goldschmidt et al., 2014).

Individual path coefficients of the final SEM representing interrelationship of adverse childhood experiences, impulsivity, depression, anxiety, emotional and external eating revealed a non-zero predictive relationship between impulsivity and both emotional eating and external eating through depression and anxiety. In other words; impulsivity only has a potential influence on emotional eating and external eating if it facilitates emotional/ external eating with depression and anxiety pathway. It does not have a direct effect but only an indirect one. Similarly, adverse childhood events only exert a potential influence on depression and anxiety if they facilitate them with impulsivity. There is no single pathway between impulsivity and emotional eating and external eating; however, behavior-specific psychological factors, such as depression and anxiety, significantly mediated the effects of impulsivity on eating behaviors, consistent with previously reported associations (Lazarevich et al. 2016; Van Strien et al. 2016).

Our findings emphasize the importance of relating emotional overeating with negative mood characteristics such as anxiety, depression, and impulsivity. In addition, the results agree with previous studies where a moderator effect of self-reported emotional eating on the relationship between distress and actual food intake has been reported (Konttinen et al., 2010; Mooreville et al.,2014; Van Strien et al., 2012; Van Strien et al.,2009). It is crucial to gain a thorough understanding of the relationship between emotions (depression and anxiety) and disordered eating in order to prevent not only obesity but also eating disorders and other related medical conditions, especially in young individuals (Eddy et al., 2007; Goossens et al., 2007).

Another crucial finding of the study is that it provided evidence of the mediating role of depressive/anxiety symptoms in the relationship between impulsivity and emotional/external eating in a university population in Turkey with recently increasing obesity rates. Efforts to reduce obesity and eating behaviors will require integrated biopsychosocial approaches that include mental health professionals (i.e., psychiatrists, psychotherapists), registered dietitian nutritionists, health coaches, and other forms of social support. These professionals and peer specialists must “be on the same page” on underlying drivers of disordered eating and detection of vulnerable individuals and any personalized treatment strategies.

The present study has certain limitations. It was conducted in a specific non-clinical population or at-risk population, thus it is difficult to generalize the results to other population groups. Therefore, future studies need to replicate this study among adolescents, patients with eating disorders, and the population of the elderly. Additionally, it has been proposed that selection of participants with extreme emotional eating score is a better strategy to identify emotional

eating behavior. Cross-sectional design and not testing other psychopathological constructs are also limitations.

In summary, the present study shows that the relationship between higher levels of impulsivity and increased emotional/external eating was mediated by levels of anxiety and depressive symptoms. It was also shown that the effect of adverse experiences on both emotional eating and external eating was partially mediated by impulsivity and symptoms of anxiety and depression. The results contribute to a theoretical understanding of the role of general personality traits such as impulsivity in disordered eating and identify potential targets for intervention. Teaching effective coping strategies along with nutrition education is particularly important at the adolescent age, and universities can provide a good environment for developing health education programs in young individuals.

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Psihološke determinante emocionalnih i eksternih prehrambenih ponašanja na uzorku studenata iz Turske

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Psihološki faktori i štetni događaji iz detinjstva (eng. Adverse childhood events) su slabo proučavani u kontekstu rizičnih prehrambenih ponašanja i gojaznosti. Važnost ove veze raste sa činjenicom da ova ponašanja postaju problem javnog zdravlja. Glavni cilj ove studije je bio da se ispita efekat povezanih psiholoških faktora kao što su štetni događaji iz detinjstva, simptomi depresije i anksioznosti i impulsivnost na prehrambena ponašanja na uzorku studenata iz Turske. Ukupno 414 studenata osnovnih studija (60,4% žena) iz Turske je popunilo tursku verziju Holandskog upitnika prehrambenih ponašanja (eng. Dutch Eating Behavior Questionnaire; DEBQ) radi procene emocionalnog i eksternog jedenja, kao i CDC-Kajzer Permanente Upitnik štetnih iskustava iz detnjstva (CDC-Kaiser Permanente Adverse Childhood Experiences; ACE), Bekov invetar depresivnosti (BDI), Bekov inventar anksioznosti (BAI), i Baratovu skalu impulsivnosti (BSI-11). Strukturalno modeliranje (SEM) je korišćeno radi procene da li depresivni/anksiozni simptomi mogu biti medijator veze između impulsivnosti i emocionalnog/eksternog jedenja. Štetna iskustva iz detinjstva su slabo korelirala sa merama prehrambenih ponašanja i impulsivnosti (r = 0,18, r = 0,275, p < 0,001, respectively). Nivoi anksioznosti i depresivnosti su korelirali značajno sa svim varijablama sem sa indeksom telesne mase (BMI; r = 0,121 – 0,395, p < .001). Indeks telesne mase (BMI) je bio značajno povezan sa emocionalnim jedenjem (r = 0,231, p < 0,001) i sa eksternim jedenjem (eng. restrained eating; r = 0,226, p < 0,001). Impulsivnost je bila pozitivno i direktno povezana sa simptomima anksioznosti i depresivnosti, kao i sa emocionalnim i eksternim jedenjem (redom: β = 0,27, p < 0,001; β = 0,31, p < 0,001; β = 0,16, p = 0,006; β = 0,13, p = 0,047). Veza štetnih iskustava iz detinjstava i emocionalnog (β = 0,147; 95% CI (0,087. 0,247)) i eksternog (β = 0,091; 95% CI (0,032, 0,168)) jedenja bila je delimično posredovana impulsivnošću i simptomima anksioznosti i depresivnosti. Rezultati su govorili u prilog tezi da su simptomi depresivnosti i nivo anksioznosti medijatori veze između impulsivnosti i emocionalnog/eksternog jedenja. Ovaj nalaz sugeriše da prilikom evaluacije prehrambenih ponašanja studenata i razmatranja strategija koje će biti primenjene za rešavanje problema neurednih prehrambenih navika, u budućnosti treba, kao opciju razmatrati i temeljnu psihološku procenu pacijenta.

Ključne reči: prehrambena ponašanja, gojaznost, štetna iskustva iz detinjstva, depresija, anksioznost, impulsivnost

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