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Research Article

Perceived Stress Enhances Eating Disorders by Affecting Leptin, Ghrelin and Adiponectin Levels

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Abstract

Objectives: The aim of this study is to assess the relationship between stress and eating disorders in young adults, which may be the initial symptom or result of many diseases today.

Methods: SCOFF and PSS tests were applied to 313 women in order to assess eating disorders (ED) and stress disorder (SD). According to the results of the questionnaire tests, the participants were divided into subgroups according to their ED status and body mass index (BMI). Subgroups were compared to investigate the relationship between ED and SD. The effect of SD on adipocytokine levels was compared among the 4 subgroups.

Results: The ED rate was 46.6%, and the SD rate was 34.5%. The SD rate was observed to be higher in the ED(+) group when compared with the ED(-) group (54.1% vs. 17.4%, $p<0.001$). In all three groups (BMI<18.5 and ED(+), BMI>25 and ED(+), and 18.5≤BMI≤25 and ED(+)), SD risk was observed to be higher than in the 18.5≤BMI≤25 and ED(-) group (OR 8.56, 7.34, and 3.59; $p=0.001$, $p=0.002$, and $p=0.012$, respectively). Leptin levels were lower, and ghrelin and adiponectin levels were higher in the SD(+) subgroup compared with the SD(-) subgroup in the group with ED(+) and BMI<18.5. Leptin levels were higher, and ghrelin and adiponectin levels were lower in the SD(+) subgroup compared with the SD(-) subgroup in the group with ED(+) and BMI>25.

Conclusion: Perceived stress significantly influences leptin, ghrelin, and adiponectin levels and is associated with eating disorders in young adults.

Keywords: Adiponectin, Eating Disorders, Ghrelin, Leptin, Stress Disorder

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Stress is a term that is frequently used in various social, academic, and employment settings. It is well known that everyone needs a certain amount of motivating pressure to do their best. However, when pressure exceeds a person's ability to cope, it causes stress. Moreover, stress can create a cycle of distress and reduce the ability to cope, even in ordinary situations. Many stress factors are encountered frequently today. In today's competitive teaching and study environment, students are faced with more stress than ever before. The source of stress can be education, exams, peers, teachers, or parents.[1] College students frequently encounter stressful situations that can cause negative academic, emotional, and health consequences.[2] They are also subjected to a number of stressful causes, such as gradual overload, constant pressure to succeed, competition with colleagues, and, in some countries, financial burden and concerns about the future.[3] As all these can lead to psychopathology, the health of college students, especially healthcare students, has been the subject of increasing focus in recent years.[4,5] Voltmer et al.[6] reported a decrease in health quality and an increase in risk patterns, indicating the need for prevention and health promotion focusing on individual behaviour.

Eating disorders (ED) are important health problems that may occur as a result of the interaction of genetic, endocrinological, hypothalamic, and environmental factors such as stress. Stress factors can affect a person's eating habits. The change in eating habits paves the way for conditions called nutritional disorders. Nutritional disorders include Anorexia Nervosa (AN), Bulimia Nervosa (BN), binge ED, reactive binge eating disorders, evening eating syndrome, and nonspecific nutritional disorders.[7] Nutritional disorders cause insufficient and/or disproportionate intake of carbohydrate, protein, and fat, as well as insufficient intake of vitamins, minerals, and trace elements in the body. Lack of prodromic symptoms and diagnostic criteria in the early stages of ED often causes delayed diagnosis. In the later stages of malnutrition, it can lead to more serious diseases such as severe depression, epilepsy, hair loss, muscle loss, bone loss, dental caries, growth retardation, anemia, and gastrointestinal and cardiac problems. Intervention for malnutrition with early diagnosis can eliminate the risk of future negative consequences.[8]

The aim of this study was to determine the prevalence of eating disorders among college students at Celal Bayar University Faculty of Medicine and Faculty of Nursing and to investigate the relationship between eating disorders and perceived stress in order to contribute to the development of appropriate preventive strategies for improving public health. Also, this study aimed to evaluate the effects of stress disorder on metabolic parameters such as leptin, ghrelin, and adiponectin.

Methods

Patients and Study Design

A total of 313 volunteer students and research assistants aged between 18 and 30 years from the Faculties of Medicine and Nursing of Celal Bayar University were enrolled in this study between 2014 and 2016. Patients with conditions that may lead to malabsorption, including ulcerative colitis, Crohn's disease, celiac disease, cystic fibrosis, chronic pancreatitis, and chronic liver or kidney disease, were excluded. In addition, individuals with a history of gastrointestinal surgery, including gallbladder, esophagus, stomach, small intestine, large intestine, liver, or pancreatic resection, were not included in the study.

The study protocol was approved by the Ethics Committee of Celal Bayar University (Approval No: 232; Approval Date: June 04, 2014). All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to blood sampling.

All 313 individuals completed the SCOFF questionnaire and the Perceived Stress Scale (PSS) to evaluate eating disorders and perceived stress levels, respectively.[9,10] The questionnaires were translated into Turkish by the authors, and a preliminary validity assessment was conducted prior to the study.[11,12] Following the questionnaire assessments, anthropometric measurements, including height and weight, were recorded. Age, medical history, and the presence of any comorbid diseases were also documented.

Participants included in the study had no comorbid diseases and were not receiving hormonal therapy. According to the literature, a body mass index (BMI) between 18.5 and 25 kg/m^2 was accepted as the normal range.

Based on the SCOFF questionnaire results, participants were categorized according to eating disorder (ED) status (SCOFF ≥2 positive vs. SCOFF <2). In addition, participants were grouped according to BMI categories (BMI >25 kg/m^2, BMI <18.5 kg/m^2, or 18.5≤BMI≤25 kg/m^2). Comparisons were performed to evaluate the relationship between these parameters and stress levels according to PSS score levels among the subgroups.

For biochemical analyses, blood samples were obtained from a total of 120 volunteers distributed among the following subgroups: ED(+) and BMI <18.5 (n=25), ED(+) and BMI >25 (n=25), 18.5≤BMI≤25 and ED(+) (n=30), and 18.5≤BMI≤25 and ED(-) (n=40).

Serum Analysis

Blood samples were collected from volunteers into vacuum tubes without anticoagulants for biochemical analyses.

Samples were centrifuged at 300×g for 10 minutes, and serum fractions were separated. The obtained serum samples were stored at –80°C until batch analysis.

The following biochemical parameters were measured: fasting glucose, fasting insulin, cortisol, lipid profile, sodium (Na), potassium (K), chloride (Cl), calcium (Ca), phosphorus (P), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), blood urea nitrogen (BUN), creatinine, thyroid-stimulating hormone (TSH), free triiodothyronine (free T3), free thyroxine (free T4), anti-gliadin IgA, anti-gliadin IgG, tissue transglutaminase IgA, and hemogram levels. Insulin resistance was evaluated using the homeostatic model assessment for insulin resistance.

In accordance with the study by Fontbonne et al.[13], insulin resistance was defined as HOMA-IR values above 1.80 for women and 2.12 for men.

All biochemical parameters were analyzed at the Celal Bayar University Biochemistry Laboratory. Adiponectin, leptin, and ghrelin plasma levels were measured using enzyme-linked immunosorbent assay (ELISA) methods.

Adiponectin levels were determined using commercial ELISA kits (Assaypro Human Adiponectin ELISA Kit, Missouri, USA). The mean intra-assay coefficient of variation (CV) of the kit was 3.0%, while the inter-assay CV was 8.3%.

Leptin levels were measured using ELISA kits (DRG Instruments GmbH, Marburg, Germany). The analytical sensitivity of the assay was 1.0 ng/mL. The intra-assay CV values were 5.95% at 3.15 ng/mL and 6.91% at 24.62 ng/mL. The inter-assay CV values were 11.55% at 2.71 ng/mL and 8.66% at 26.15 ng/mL.

Ghrelin levels were measured using commercial ELISA kits (Human Ghrelin Total, EMD Millipore Corporation, Missouri, USA). The analytical sensitivity of the assay was 50 pg/mL. The intra-assay CV values were 1.26% at 384 pg/mL and 0.90% at 904 pg/mL. The inter-assay CV values were 7.81% at 384 pg/mL and 6.28%.

Statistical Analysis

All statistical analyses were performed using SPSS for Windows, version 15.0 (IBM Corp., Armonk, NY, USA). The distribution characteristics of continuous variables were evaluated using descriptive statistics and normality assessments. Continuous variables were expressed as mean±standard deviation (SD), while categorical variables were presented as frequency (n) and percentage (%). Comparisons between categorical variables were performed using the chi-square test. For comparisons between two independent groups, Student's t-test was applied for normally distributed variables. When the data did not meet the assumptions of normal distribution, the Spearman correlation test was used. For comparisons involving more than two groups, one-way analysis of variance (ANOVA) was applied where appropriate. All statistical tests were two-tailed, and a p value of <0.05 was considered statistically significant.

Results

The average age of the study population was 24.62 years (range, 18–30 years). According to the results of the SCOFF Test (Sick, Control, One Stone, Fat, Food) and the Perceived Stress Scale (PSS) questionnaire test, the rate of SD was 34.5% (n=108), and the rate of ED was 46.6% (n=146).

While 54.1% (n=79) of individuals in the ED group (n=146) had SD, the SD rate in the non-ED population was 17.4% (n=29) (p<0.001) (Table I). It was observed that individuals with BMI<18.5 or BMI>25 had more SD than individuals with normal BMI (64.3% vs. 35.7%, 61% vs. 39%, and 24.3% vs. 75.7%; p<0.001, p<0.001, and p<0.001, respectively) (Table 2).

Four subgroups were defined as BMI<18.5 and ED(+) (n=42), BMI>25 and ED(+) (n=23), 18.5≤BMI≤25 and ED(+) (n=63), and 18.5≤BMI≤25 and ED(-) (n=167) according to BMI levels and ED status. Subgroup analysis was performed for SD among the four subgroups. The 18.5≤BMI≤25 and ED(-) subgroup was determined as the control group. A comparison was made between these subgroups to assess the relationship between stress levels. It was observed that the stress rates were significantly higher in the three groups with eating disorders when compared with the control group (BMI<18.5, p=0.001 for the ED(+) group; BMI>25, p=0.002 for the ED(+) group; and 18.5≤BMI≤25, p=0.012 for the ED(+) group). It was determined that the risk of SD was higher in the BMI<18.5 and ED(+) group, BMI>25 and

Table 1. The relationship between eating disorders and stress disorder

Groups	SD (+)	SD (-)	p
ED (+)	79 (%54.1)	67 (%45.9)	<0.001
ED (-)	29 (%17.4)	138 (%82.6)	

BMI: Body mass index; ED: Eating disorders; SD: Stress disorder.

Table 2. SD rates according to BMI

	SD (-)	SD (+)	p
BMI<18.5	35,7%	64,3%	<0.001
18.5≤BMI≤25	75,7%	24,3%	<0.001
BMI>25	39%	61%	<0.001

BMI: Body mass index; SD: stress disorder.

ED(+) group, and 18.5≤BMI≤25 and ED(+) group than in the 18.5≤BMI≤25 and ED(-) subgroup (OR 8.56, 7.34, and 3.59, respectively). In addition, there was a significantly higher risk of stress in the BMI<18.5 or BMI>25 and ED(+) subgroups than in the 18.5≤BMI≤25 and ED(+) subgroup (p=0.014 and p=0.021) (Table 3).

Blood analysis was performed on a total of 120 volunteers among the people participating in the survey study. These volunteers were divided into four subgroups according to their BMI and ED status: BMI<18.5 and ED(+) (n=25), BMI>25 and ED(+) (n=25), 18.5≤BMI≤25 and ED(+) (n=30), and 18.5≤BMI≤25 and ED(-) (n=40). Fasting glucose, fasting insulin, HOMA-IR score, cortisol, lipid profile, Na, K, Cl, Ca, P, hemogram, AST, ALT, ALP, GGT, BUN, creatinine, TSH, free T3, free T4, anti-gliadin IgA, anti-gliadin IgG, tissue transglutaminase IgA, ACTH, cortisol, and HOMA values were compared between the four subgroups of 120 participants. There was no statistically significant difference in biochemical values between the groups. It was observed that SD(+) was higher in subgroups that had ED(+), similar to the general population participating in the survey study (p=0.002, p=0.019, p=0.002) (Table 4).

Adipocytokine levels were studied in these four subgroups, and the differences between parameters are shown in Table 5. Leptin levels were lower, and ghrelin and adiponectin levels were higher in the SD(+) subgroup when compared with the SD(-) subgroup in the group with ED(+) and BMI<18.5 (p<0.001, p=0.011, and p=0.001, respectively). Leptin levels were higher, and ghrelin and adiponectin levels were lower in the SD(+) subgroup compared with the SD(-) subgroup in the group with ED(+) and BMI>25 (p<0.001, p<0.001, and p<0.001, respectively). In the subgroup with normal BMI, no such relationship could be established in terms of SD (p>0.05) (Table 5).

Table 3. Distribution of study groups and stress rates

Subgroups	SD (+)	SD (-)	OR (Lower-upper)	p
BMI<18.5 and ED (+)	64.3%	25.7%	8.56 (4.05-18.09)	0.001
BMI>25 and ED (+)	61%	39%	7.34 (3.53-15.63)	0.002
18.5≤BMI≤25 and ED (+)	42.9%	57.1%	3.59 (1.88-6.77)	0.012
18.5≤BMI≤25 and ED (-)	17.4%	82.6%		

BMI: Body mass index; OR: Odds ratio; SD: Stress disorder.

Table 4. Distribution of volunteer participants who donated blood for serum adipocytokine among groups and SD rates

Subgroups	SD (+)	SD (-)	p
BMI<18.5 and ED (+) (n=25)	64.0%	36%	0.002
BMI>25 and ED (+) (n=25)	60.0%	40%	0.019
18.5≤BMI≤25 and ED (+) (n=30)	43.3%	56.7%	0.002
18.5≤BMI≤25 and ED (-) (n=40)	17.5%	82.5%	

BMI: Body mass index; SD: Stress disorder.

Discussion

Eating disorders are important health problems that can occur as a result of the interaction of genetic, endocrinological, hypothalamic, and environmental factors. The rate of ED investigated with the SCOFF test varies between 11.8% and 48.8% among the young population.[14] The rate of ED in the population included in our study was found to be 46.6%. In the analysis using PSS among college students, the rate of SD in women was found to be 48%.[15] In a study conducted with other stress scales, it was observed that the rate of students under intense stress among medical students was up to 52%.[16] In another study conducted among female students, this rate was reported to be 43%.[17] The rate of SD in the population included in our study was found to be 34.5%.

Fairburn et al.[18] highlighted that the inability to cope with stressful situations properly is an important factor for the emergence and continuation of ED symptoms. Psychological stress was evaluated with a scale by Darby et al.[19], and in this study, it was stated that SD was related to ED symptoms. Sassaroli et al.[20] put forward a hypothesis that stress is related to some cognitive factors that predispose to ED, and as a result of the study, they obtained data supporting this. Indeed, the empirical demonstration of this mechanism has been more reliably supported, showing that important stress is at the source of eating disorders. It has been shown that acquired stress affects eating habits and contributes to obesity in the literature.[21] In another study, it was stated that the risk of a quality-of-life score related to mental health is low in women with altered eating behaviour.[22] There are also studies showing that the desire to lose weight, which did not exist before, is revealed by stress and affects eating behaviour. A decrease in ED symptoms has been shown in participants when the stress factor disappears.[23] In a study comparing patients with obesity with and without ED, it was found that acquired stress scores were higher in those with ED.[24] These findings show that measures should be taken to reduce stress in individuals with ED. In our study, it was investigated whether psychosocial stress is related to ED symptoms. While 54.1% of those in the ED group had SD, the

Tablo 5. Relationship between stress and adipocytokine SD; Stress disorders

Group		Average	SD (+)	SD (-)	p
BMI<18.5	Leptin	3.83±2.56	2.41±1.25	6.36±2.37	<0.001
	Ghrelin	1261.57±481.11	1438.75±506.16	946.56±198.97	0.011
	Adiponectin	25.68±1.78	26.71±1.22	23.86±0.96	0.001
BMI>25	Leptin	24.06±9.13	28.7±8.86	17.11±3.29	<0.001
	Ghrelin	200.50±86.12	144.42±40.18	284.61±64.54	<0.001
	Adiponectin	19.49±1.99	18.26±0.94	21.32±1.72	<0.001
18.5≤BMI≤25 and ED (+)	Leptin	8.89±6.89	11.62± 9.76	6.8±2.0	0.104
	Ghrelin	389.38±163.95	379.88±217.22	396.64±114.90	0.804
	Adiponectin	21.94±1.54	21.90±0.94	21,96±1.90	0.524
18.5≤BMI≤25 and ED (-)	Leptin	8.17±1.82	8,26±1.32	8.16±1.93	0.895
	Ghrelin	454.43±132.00	403.35±134.88	465.26±130,89	0.265
	Adiponectin	21.29±0.79	21.45±0.79	21.26±0.80	0.565

BMI: Body mass index; ED: Eating disorders; SD: Stress disorder.

rate of SD in the non-ED population was found to be 17.4%. As a result, it was seen that the presence of SD was a factor affecting the development of ED. This situation can be explained by the fact that intense anxiety, perfectionism, and low self-esteem, which can be seen in stress disorders, can trigger eating disorders by directing the individual's attention to restriction or an increase in eating. In conclusion, early detection of individuals with stress disorders and medical intervention to this effect are important in terms of preventing eating disorders.

In a study, it was observed that overweight or underweight individuals were in similar psychological conditions, and they were also under more stress than individuals with normal BMI.[25] Looking at the distribution of BMI in stressed individuals in our study, it was observed that BMI was generally lower or higher than normal in stressed individuals. However, it has been found that the BMIs of individuals without stress disorder are mostly normal. Similarly, there are studies in the literature claiming that stress experience triggers eating disorders and affects BMI in a low or high way.[26] Stress disorder may develop in individuals who are thin or fat due to thoughts such as being withdrawn due to their external appearance and feeling inadequate. We think that, by organizing diet and exercise programs for these people, stress disorder can be prevented. Eating behaviour and metabolic parameters are a reflection of acquired stress.

Our study is one of the most comprehensive studies conducted to determine the relationship between leptin, adiponectin, ghrelin, and anthropometric values of ED and SD according to the recent literature. As a result of our study, a direct relationship was found between eating disorders and leptin, ghrelin, and adiponectin among women aged between 18 and 30. When the individuals with a BMI above 25 and those with eating disorders who have positive stress disorder are compared with those who are negative, in those with SD(+), it was observed that leptin levels were significantly high, and ghrelin and adiponectin levels were low. Leptin levels were lower, and ghrelin and adiponectin levels were higher in the SD(+) subgroup compared with the SD(-) subgroup in the group with ED(+) and BMI<18.5. No such relationship was found in those with normal BMI. Psychological stress can affect adipocytokine levels, leading to changes in eating habits and nutritional disorders. In today's societies, it is thought that stress can contribute to obesity by increasing the concentration of leptin. In a study, it was observed that leptin increased in individuals with high stress perception, and this was found to be statistically significant.[27] Declining leptin is an important indicator of energy deficiency, and it is thought to be a mechanism developed by the body against a decrease in energy consumption or production capacity. While leptin was positively correlated with fat mass, it was found to be negatively correlated with ghrelin.[28] Conversely, in low-weight participants, increased ghrelin and decreased leptin levels are present. Low body weight develops due to increased metabolic rate and decreased appetite. Higher ghrelin levels have been found in women with intense stress exposure. Here, it is thought that stress disorder may be caused by the increase in ghrelin by activating the hypothalamo-pituitary-adrenal axis and sympathetic nervous system.[29] There is also a relation between adiponectin levels and psychological stress. It is known that, in premenopausal and postmenopausal women, changes in levels of adiponectin cause changes in eating behaviour and BMI.[30]

Conclusion

In conclusion, as a result of our study, a direct relation was found between eating disorders and leptin, ghrelin, and adiponectin in women aged 18–30 years. However, it was found that stress both contributes to eating disorders and affects adipocytokine levels. This study was conducted with 313 volunteers and only in a limited age group in the female population. It is necessary to expand the study population and conduct further research. The root causes and effects of eating disorders and stress disorder, which are among the most important problems that concern especially the young age group, should be investigated in more extended studies.

Disclosures

Ethics Committee Approval: Ethical approval was obtained from Celal Bayar University Faculty of Medicine Ethics Committee (04.06.2014/232).

Informed Consent: Written informed consent was obtained from all participants prior to blood sampling.

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