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Eating behavior problems in children with epilepsy (6–16 years): A cross-sectional comparative study

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ABSTRACT

Objective: The aim of this study was to evaluate the risk of eating behavior disorders in children and adolescents aged 6–16 years diagnosed with epilepsy, and to compare this risk with that of healthy controls without epilepsy, in order to elucidate the impact of epilepsy on eating behaviors.

Materials and Methods: The study included 78 patients diagnosed with epilepsy who presented to the Pediatric Neurology Outpatient Clinic of Balıkesir University Health Practice and Research Hospital between November 1, 2024 and January 1, 2025, along with 62 age-matched healthy controls without epilepsy and with normal neurological examinations. The diagnosis of epilepsy was established according to the International League Against Epilepsy (ILAE) criteria. Both groups completed the validated Turkish version of the Children's Eating Behaviour Questionnaire (CEBQ). Clinical characteristics of the epilepsy group, including age, sex, seizure type, epilepsy duration, seizure frequency and antiseizure medications (ASMs) use, were also evaluated.

Results: No statistically significant differences in eating behavior scores were observed between children with epilepsy and healthy controls ($p > 0,05$). Furthermore, eating behavior was not significantly associated with epilepsy duration, seizure type or antiseizure medication use. When the epilepsy group was examined within itself, differences in eating behavior were observed between genders. ($p < 0,05$).

Conclusion: In this study, eating behaviors assessed by the CEBQ didn't different between children with epilepsy and healthy controls, nor were they associated with epilepsy-related clinical characteristics. However, gender-related changes in eating behavior have been observed in the epilepsy group. Large-scale studies are needed to investigate this situation.

1. Introduction

Epilepsy is a chronic disorder of the central nervous system characterized by recurrent seizures and is frequently observed during childhood and adolescence. Its global prevalence ranges between 0.6% and 1.2%, making it one of the leading causes of morbidity in pediatric neurology [1–3]. The diagnosis of epilepsy is established based on clinical history, physical and neurological examination, electroencephalography (EEG), and neuroimaging modalities [4]. Epilepsy should be evaluated across a broad clinical spectrum, as both the intrinsic nature of the disease and the adverse effects of antiseizure medications may substantially impair quality of life [5].

In 2022, the International League Against Epilepsy (ILAE) updated the classification and definitions of childhood-onset epilepsy syndromes, adopting a framework more closely aligned with clinical practice. In this updated classification, epilepsy syndromes are defined through an integrated assessment of age at onset, seizure types, electroencephalographic characteristics, genetic findings, and clinical course. Terminology such as "benign," which failed to reflect potential neurodevelopmental consequences, has been abandoned. Instead, concepts such as "self-limited epilepsies" and "developmental and/or epileptic encephalopathies" have been emphasized to better describe disease trajectory and possible cognitive outcomes. This contemporary ILAE approach highlights the importance of a comprehensive evaluation of children with epilepsy, extending beyond seizure control to encompass cognitive, behavioral, and developmental outcomes [6].

Children with epilepsy exhibit a higher prevalence of behavioral and psychiatric problems compared with their healthy peers [7]. Although

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various psychiatric and cognitive disorders frequently coexist with epilepsy, there is also growing recognition that these conditions themselves may confer an increased risk for epilepsy, suggesting a bidirectional relationship [8]. However, it remains unclear whether psychiatric and neurodevelopmental disorders predispose individuals to epilepsy or whether epilepsy itself increases vulnerability to these conditions [9]. In a population-based study, epilepsy was not found to independently trigger behavioral problems, and seizures alone were not sufficient to cause behavioral difficulties [10].

Eating disorders constitute a heterogeneous group of psychiatric conditions characterized by significant disturbances in eating behavior, leading to impaired energy intake or absorption and resulting in detrimental effects on both physical health and psychosocial functioning. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), this group includes anorexia nervosa, bulimia nervosa, binge-eating disorder, avoidant/restrictive food intake disorder, pica, rumination disorder, and other specified feeding or eating disorders [11]. These disorders are associated with distorted perceptions of body weight and shape, as well as maladaptive attitudes toward food [12]. Their etiology is multifactorial, involving genetic vulnerability, environmental and stress-related factors, and sociocultural influences [13].

Adolescence represents a critical developmental period marked by profound psychological and physiological changes during which self-concept and body image are shaped. The prevalence of eating disorders increases during this stage and is associated with adverse outcomes such as substance use, suicidal behaviors, academic difficulties, and social isolation [14]. In children and adolescents with epilepsy, several factors may contribute to the development of eating disorders. Certain antiseizure medications (ASMs) may induce appetite changes, weight gain, or metabolic side effects, thereby influencing eating behaviors. Additionally, difficulties in seizure control may disrupt perceived bodily autonomy and trigger maladaptive eating patterns [15,16].

The literature suggests that individuals with epilepsy are at an increased risk of developing eating disorders [17]. In Türkiye, Coşkun and colleagues did not identify a direct association between eating disorders and Rolandic epilepsy in children but emphasized the need for more comprehensive research in this area [18]. Overall, the relationship between epilepsy and eating disorders remains insufficiently understood. Adverse experiences during early adolescence have also been associated with binge-eating behaviors [19–21].

In light of this evidence, investigating eating behavior disturbances in children and adolescents with epilepsy is of considerable clinical importance for both early intervention and effective follow-up. The aim of the present study was to assess the risk of eating disorders in individuals aged 6–16 years with epilepsy and to compare these findings with those of a healthy control group, thereby contributing to a better understanding of epilepsy-specific characteristics.

2. Methods

This cross-sectional, comparative study was conducted between November 1, 2024 and January 1, 2025, at the Pediatric Neurology Outpatient Clinic of Balıkesir University Health Practice and Research Hospital. The study group consisted of 78 children aged 6–16 years who had been diagnosed with epilepsy according to the International League Against Epilepsy (ILAE) criteria, had no other chronic diseases, and voluntarily agreed to participate.

The control group included 62 age-matched healthy children without a diagnosis of epilepsy, with normal neurological examinations, no chronic diseases or regular medication use, who presented to the outpatient clinic with complaints such as headache, syncope, or vertigo, and who consented to participate.

Exclusion criteria for the control group were: history of psychiatric disorders, use of medication for chronic conditions other than epilepsy, presence of vascular or demyelinating diseases, history of acute symptomatic seizures, or refusal to participate in the study. Written informed consent was obtained from the parents of all participants.

Clinical data collected for children with epilepsy included age, sex, duration of epilepsy, seizure type, electroencephalography (EEG) findings, antiseizure medications (ASMs) use, and seizure frequency. Parents of all participants completed the Turkish version of the Children's Eating Behaviour Questionnaire (CEBQ), which has previously been validated for reliability and validity. Permission to use the questionnaire was obtained via e-mail, and parents were provided with detailed instructions prior to its administration.

2.1. Children's eating Behavior Questionnaire (CEBQ)

The Children's Eating Behavior Questionnaire (CEBQ), developed by Wardle et al., is a 35-item, parent-reported instrument designed to assess appetite- and eating-related behaviors in children. The questionnaire is rated on a 5-point Likert scale ranging from 1 (never) to 5 (always). It comprises eight subscales: food responsiveness, emotional overeating, enjoyment of food, desire to drink, satiety responsiveness, slowness in eating, emotional undereating, and food fussiness.

In the original validation study, these eight subscales were shown to explain between 50% and 80% of the total variance, and the internal consistency of the subscales was high, with Cronbach's alpha values ranging from 0.74 to 0.91[22]. The subscales are conceptually grouped into two broader dimensions: food approach behaviors (food responsiveness, emotional overeating, enjoyment of food, and desire to drink), reflecting increased interest in and appetite for food, and food avoidance behaviors (satiety responsiveness, slowness in eating, emotional undereating, and food fussiness), reflecting reduced appetite and avoidance of food [23].

Higher scores on the food approach subscales indicate greater interest in food and increased appetite, whereas higher scores on the food avoidance subscales are interpreted as greater food refusal and reduced appetite. The Turkish adaptation of the CEBQ was conducted by Yılmaz et al. in 2011, and the psychometric evaluation demonstrated acceptable reliability, with Cronbach's alpha coefficients ranging from 0.61 to 0.84 [24].

Ethical approval for the study was obtained from the Balıkesir University Health Sciences Non-Interventional Research Ethics Committee (Approval No: 2024/149, dated October 25, 2024).Data consent forms were obtained from the patients, parent(s), or legal representative(s) before study participation, in accordance with local regulations and the ethical principles that have their origin in the principles of the Declaration of Helsinki.

2.2. Statistical analysis

The study data were analyzed using SPSS software (Statistical Package for the Social Sciences for Windows, version 22.0; SPSS Inc., Chicago, IL, USA). Descriptive statistics were presented as mean ± standard deviation for continuous variables and as frequencies and percentages for categorical variables. The normality of data distribution was assessed using the Kolmogorov–Smirnov test and visual inspection of histogram plots. Comparisons between groups were performed using the independent samples *t*-test (Student's *t*-test) for continuous variables that met the assumptions of parametric testing. When the assumptions of normality were not satisfied, the Mann–Whitney *U* test was applied for the analysis of continuous variables. A p value of less than 0.05 was considered statistically significant. To account for multiple comparisons across the eight CEBQ subscales, we applied Holm's step-down correction to control the Type I error rate. All p values obtained from the between-group comparisons of CEBQ scores were adjusted using the Holm method, and statistical significance was defined as an adjusted p value (pHolm) < 0.05 for multiple comparisons.

2.3. Post-hoc power analysis

A post-hoc power analysis was performed using G*Power software (version 3.1.9.4) to evaluate the statistical sensitivity of the study for the primary outcome. Based on the observed between-group difference in the Slowness in Eating subscale, which demonstrated a small effect size (Cohen's d ≈ 0.20), and assuming a two-tailed independent-samples *t* test with an alpha level of 0.05, the achieved power (1–β) for the comparison between patients with epilepsy (n = 78) and healthy controls (n = 62) was approximately 0.21–0.24. This indicates that the study had limited power to detect small effect sizes for the primary eating behavior outcome.

3. Results

This study included 78 patients diagnosed with epilepsy and 62 healthy volunteers. The mean age of the patients with epilepsy was 10.3 ± 3.7 years, while the mean age of the healthy controls was 10.3 ± 3.1 years. Among the patients with epilepsy, 38 were female (48.7%) and 40 were male (51.3%). In the control group, 32 participants were female (51.6%) and 30 were male (48.4%). There were no significant differences between the patient and control groups in terms of age or sex (Table 1).

The mean duration of epilepsy among the patients was 4.28 ± 2.0 years, and the mean duration of antiseizure medications (ASMs) use were 4.16 ± 1.9 years. Among the 65 patients receiving antiseizure treatment, analysis of medication types revealed that 52.3% (n = 34) were treated with levetiracetam, 30.8% (n = 20) with valproic acid, 13.8% (n = 9) with polytherapy. 1.5% (n = 1) with carbamazepine, and 1.5% (n = 1) with oxcarbazepine (Table 1).

The subscale scores of the Child Eating Behavior Questionnaire (CEBQ) were compared between patients with epilepsy and the control group. No statistically significant differences were observed between the epilepsy and control groups in terms of Food Responsiveness, Emotional Overeating, Enjoyment of Food, Desire to Drink, Satiety Responsiveness, Slowness in Eating, Emotional Undereating, and Food Fussiness subscale scores (all comparisons, $p > 0.05$) (Table 2).

Among the CEBQ subscales, Food Responsiveness, Emotional Overeating, Enjoyment of Food, and Desire to Drink are considered indicators of food approach behaviors, whereas Satiety Responsiveness, Slowness in Eating, Emotional Undereating, and Food Fussiness are regarded as indicators of food avoidance behaviors. In the present study, total food approach and food avoidance scores were similar between the epilepsy group and healthy controls ($p > 0.05$) (Table 2).

All participants in the study (patients with epilepsy and healthy controls) were categorized into four groups based on their mean scores for food approach and food avoidance behaviors: food approach vs. non–food approach, and food avoidance vs. non–food avoidance. Patients with epilepsy and healthy controls within the food approach and food avoidance groups were compared in terms of age and sex distribution. In both the food approach group and the food avoidance group, the epilepsy and healthy control subgroups were found to be comparable with respect to age (p = 0.914 and p = 0.771, respectively) and sex distribution (p = 0.832 and p = 0.634, respectively) (Table 3).

Epilepsy-diagnosed cases were divided into two groups according to seizure type: focal and generalized seizures. Patients with focal and generalized seizures were evaluated in terms of eating behaviors. The mean score of the emotional undereating subscale was statistically significantly higher in patients with focal epilepsy compared with those with generalized epilepsy. However, this significance was no longer observed after applying the Holm–Bonferroni correction for multiple comparisons. Overall, no statistically significant differences were found between the groups in eating behavior scale scores (p > 0.05) (Table 4).

Of the patients diagnosed with epilepsy, 65 (83.3%) were receiving antiseizure medications (ASMs), whereas 13 (16.7%) were not. Patients with epilepsy who were receiving ASMs and those who were not were compared in terms of Child Eating Behavior Questionnaire subscale scores, food approach, and food avoidance indices. No statistically significant differences were found between the groups (Table 5).

The Child Eating Behavior Questionnaire subscale and total scores of patients with epilepsy receiving levetiracetam (n = 34) and valproic acid (n = 20) were compared. No statistically significant differences were found between the two treatment groups in terms of the food responsiveness, emotional overeating, enjoyment of food, desire to drink, satiety responsiveness, slowness in eating, emotional undereating, and food fussiness subscales, nor in the total food approach and food avoidance scores (Table 6).

In patients with epilepsy, correlation analyses were performed to examine the relationships between epilepsy duration and duration of ASMs use and the Child Eating Behavior Questionnaire subscales, food approach, and food avoidance scores. No statistically significant correlations were found between either duration variable and any of the subscale scores or the total food approach and food avoidance scores (Table 7).

In patients with epilepsy, significant differences were observed

Table 1
Demographic characteristics of the epilepsy and control groups.

Variable	Epilepsy (n = 78) Mean ± SD / n (%)	Controls (n = 62) Mean ± SD / n (%)	p-value
Age (years)	10.3 ± 3.7	10.3 ± 3.1	0.968
Sex			
Female	38 (48.7%)	32 (51.6%)	0.734
Male	40 (51.3%)	30 (48.4%)	
Weight (kg)	40.7 ± 18.9	39.1 ± 15.1	0.908
Weight percentile	55.7 ± 32.1	56.9 ± 32.9	0.734
Height (cm)	140.8 ± 20.5	138.7 ± 15.9	0.497
Height percentile	47.2 ± 29.7	46.3 ± 31.5	0.889
BMI	19.3 ± 4.7	18.7 ± 4.5	0.956
BMI percentile	60.8 ± 32.5	63.1 ± 31.1	0.656
Duration of epilepsy (years)	4.2 ± 2.0	–	
Duration of ASMs use (years)	4.1 ± 1.9	–	
ASMs use		–	
Yes	65 (83.3%)	–	
No	13 (16.7%)	–	
Type of ASMs			
Levetiracetam	34 (52.3%)		
Valproic acid	20 (30.8%)		
Carbamazepine	1 (1.5%)		
Oxcarbazepine	1 (1.5%)		
Combination therapy (polytherapy)	9 (13.8%)		
Seizure type			
Generalized	69 (49.3%)	–	
Focal	9 (6.4%)	–	

Mean: average; SD: standard deviation; *n*: number of participants; %: percentage; *p*: statistical significance value.

Table 2
Comparison of Child Eating Behavior Questionnaire (CEBQ) Subscale Scores Between Children With Epilepsy and Healthy Controls.

CEBQ Subscales	Epilepsy (n = 78) Mean ± SD	Controls (n = 62) Mean ± SD	*p*-value
Food Responsiveness	2.2 ± 0.8	2.4 ± 1.1	0.523
Emotional Overeating	1.8 ± 0.8	1.9 ± 0.8	0.301
Enjoyment of Food	3.4 ± 0.8	3.4 ± 0.9	0.703
Desire to Drink	2.7 ± 1.0	2.9 ± 1.0	0.267
Satiety Responsiveness	2.6 ± 0.6	2.8 ± 0.7	0.260
Slowness in Eating	2.3 ± 0.8	2.5 ± 1.1	0.555
Emotional Undereating	2.8 ± 0.8	2.7 ± 0.9	0.585
Food Fussiness	2.8 ± 0.9	2.8 ± 0.9	0.764
Food Approach Total	10.3 ± 2.7	10.7 ± 2.9	0.294
Food Avoidance Total	10.7 ± 2.1	11.0 ± 2.2	0.509

Mean, average; SD, standard deviation; *n*, number of participants; %, percentage; *p*, statistical significance value.

Table 3
Comparison of age and sex according to obesity-prone and underweight-prone status in patients with epilepsy and controls.

		Epilepsy (n = 31) Mean ± SD / n (%)	Control (n = 25) Mean ± SD / n (%)	p
Food Approach Group(n = 56)	Age (years)	10.4 ± 4.0	10.5 ± 2.9	0.914
	Female	14 (53.8%)	12 (46.2%)	0.832
	Male	17 (56.7%)	13 (43.3%)	
		Epilepsy (n = 40) Mean ± SD / n (%)	Control (n = 31) Mean ± SD / n (%)	
Food Avoidance Group (n = 71)	Age (years)	10.1 ± 3.8	9.8 ± 3.1	0.771
	Female	21 (60%)	14(40%)	0.634
	Male	19 (52.8%)	17 (47.2%)	

Mean: mean, SD: standard deviation, n: number of participants, %: percentage, p: level of statistical significance.

Table 4
Child Eating Behavior Questionnaire Scores in Patients with Focal and Generalized Epilepsy.

Child Eating Behavior Questionnaire Subscales	Focal Epilepsy (n = 9) Mean ± SD	Generalized Epilepsy (n = 69) Mean ± SD	p	Holm adjusted p
Food Responsiveness	2.3 ± 0.9	2.2 ± 0.8	0.735	1.000
Emotional Overeating	2.0 ± 1.1	1.8 ± 0.8	0.630	1.000
Enjoyment of Food	3.6 ± 0.9	3.4 ± 0.8	0.604	1.000
Desire to Drink	2.4 ± 1.2	2.7 ± 1.0	0.317	1.000
Satiety Responsiveness	2.5 ± 0.6	2.7 ± 0.6	0.590	1.000
Slowness in Eating	2.5 ± 1.0	2.3 ± 0.8	0.449	1.000
Emotional Undereating	3.4 ± 0.7	2.7 ± 0.8	0.034	0.340
Food Fussiness	2.8 ± 1.2	2.8 ± 0.9	0.924	0.924
Food Approach	10.4 ± 3.6	10.3 ± 2.6	0.833	1
Food Avoidance	11.4 ± 2.0	10.6 ± 2.1	0.315	1

Mean: mean; SD: standard deviation; n: number of participants; p: level of statistical significance. Holm-adjusted p values were calculated using the Holm–Bonferroni correction for multiple comparisons.

Table 5
Child Eating Behavior Questionnaire Scores in Patients With and Without Antiseizure Medications (ASMs).

Child Eating Behavior Questionnaire Subscales	With ASMs (n = 65) Mean ± SD	Without ASMs (n = 13) Mean ± SD	p
Food Responsiveness	2.2 ± 0.9	2.1 ± 0.5	0.946
Emotional Overeating	1.8 ± 0.8	1.9 ± 0.8	0.760
Enjoyment of Food	3.4 ± 0.8	3.6 ± 0.6	0.385
Desire to Drink	2.8 ± 1.1	2.4 ± 0.6	0.369
Satiety Responsiveness	2.7 ± 0.6	2.6 ± 0.6	0.617
Slowness in Eating	2.3 ± 0.8	2.4 ± 1.1	0.813
Emotional Undereating	2.8 ± 0.7	2.8 ± 1.1	0.898
Food Fussiness	2.8 ± 0.9	2.8 ± 1.1	0.957
Food Approach	10.3 ± 2.9	10.1 ± 1.7	0.989
Food Avoidance	10.7 ± 2.2	10.6 ± 1.8	0.896

Mean: mean; SD: standard deviation; Antiseizure Medications (ASMs); n: number of participants; p: level of statistical significance.

Table 6
Child Eating Behavior Questionnaire Scores in Patients With Epilepsy Receiving Levetiracetam and Valproic Acid.

Child Eating Behavior Questionnaire Subscales	Levetiracetam (n = 34) Mean ± SD	Valproic Acid (n = 20) Mean ± SD	p
Food Responsiveness	2.3 ± 1.0	2.4 ± 0.9	0.403
Emotional Overeating	2.0 ± 0.9	2.0 ± 0.8	0.807
Enjoyment of Food	3.6 ± 0.8	3.4 ± 1.1	0.650
Desire to Drink	3.0 ± 1.1	2.8 ± 1.2	0.664
Satiety Responsiveness	2.7 ± 0.7	2.8 ± 0.7	0.458
Slowness in Eating	2.2 ± 0.7	2.8 ± 1.0	0.110
Emotional Undereating	2.9 ± 0.7	3.0 ± 0.9	0.850
Food Fussiness	2.8 ± 0.9	2.9 ± 1.0	0.814
Food Approach	10.8 ± 2.8	10.6 ± 3.4	0.661
Food Avoidance	10.6 ± 2.0	11.4 ± 2.6	0.223

Mean: mean; SD: standard deviation; n: number of participants; %: percentage; p: level of statistical significance.

between female and male patients in several Child Eating Behavior Questionnaire subscales. Enjoyment of food scores were higher in male patients compared with female patients (3.7 ± 0.8 vs. 3.2 ± 0.8; p = 0.011). In contrast, female patients had significantly higher scores for slowness in eating (2.6 ± 1.0 vs. 2.1 ± 0.7; p = 0.029) and emotional undereating (3.0 ± 0.8 vs. 2.7 ± 0.9; p = 0.037). In addition, the food avoidance index was significantly higher in female patients than in male patients (11.4 ± 2.2 vs. 10.2 ± 1.9; p = 0.035). No statistically significant sex-based differences were observed in the remaining subscales or in the food approach index (Table 8).

In the healthy control group, no statistically significant differences were found between girls and boys in any of the Child Eating Behavior Questionnaire subscales or in the food approach and food avoidance indices (Table 9).

The distribution of patients and healthy controls was compared according to BMI percentile groups. No statistically significant difference was found between patients and healthy volunteers in terms of their distribution across BMI percentile groups (p = 0.853) (Table 10).

4. Discussion

In this study, eating behaviors of children diagnosed with epilepsy were compared with those of their healthy peers using the Child Eating Behavior Questionnaire (CEBQ). According to our findings, no statistically significant differences were observed between patients with epilepsy and healthy controls in any of the eight CEBQ subscales or in the main dimensions of "food approach" and "food avoidance." In addition, no significant associations were demonstrated between eating behavior patterns and clinical variables such as epilepsy duration, duration of antiseizure medications (ASMs) use, seizure type, or the presence of ASMs treatment.

There were also no differences between the epilepsy group and healthy volunteers in terms of age, sex, or body mass index (BMI). Similarly, in the study by dos Passos et al., no differences were found with respect to age or sex; however, overweight children had higher scores on the "food approach" subscale and lower scores on the "food avoidance" subscale [25]. This study suggested that eating behaviors may parallel a child's weight status. In our study, although no differences in eating behavior were observed between the epilepsy and control groups, within the epilepsy group, female patients had significantly higher scores for slowness in eating and emotional undereating. In addition, the food avoidance index was higher in girls than in boys.

When evaluated by sex, no significant differences were found between the epilepsy and control groups overall. However, previous studies have reported that during adolescence, body image

Table 7
Results of Correlation Analyses Between Epilepsy Duration, Duration of Antiseizure Medications (ASMs) use, and Child Eating Behavior Questionnaire Subscales A) Epilepsy duration B) Duration of Antiseizure Medications (ASMs) use.

n = 78		FR	EO	EF	DD	SR	SE	EU	FF	FA	FV
Epilepsy duration	r	−0.139	−0.131	−0.082	−0.063	0.047	0.018	−0.067	−0.038	−0.082	0.012
	p	0.226	0.254	0.473	0.582	0.683	0.873	0.560	0.741	0.473	0.914
n = 65		**FR**	**EO**	**EF**	**DD**	**SR**	**SE**	**EU**	**FF**	**FA**	**FV**
ASMs duration	r	−0.165	−0.209	−0.193	−0.078	0.033	0.054	−0.093	0.017	−0.148	0.027
	p	0.190	0.095	0.123	0.538	0.796	0.669	0.462	0.891	0.241	0.828

r = Spearman correlation coefficient; p = two-tailed significance value.
FR: Food Responsiveness; EO: Emotional Overeating; EF: Enjoyment of Food; DD: Desire to Drink; SR: Satiety Responsiveness; SE: Slowness in Eating; EU: Emotional Undereating; FF: Food Fussiness; FA: Food Approach; FV: Food Avoidance.

Table 8
Child Eating Behavior Questionnaire Scores by Sex in Patients With Epilepsy.

Child Eating Behavior Questionnaire Subscales	Female (n = 38) Mean ± SD	Male (n = 40) Mean ± SD	p
Food Responsiveness	2.3 ± 0.9	2.2 ± 0.8	0.880
Emotional Overeating	2.0 ± 0.9	1.7 ± 0.7	0.217
Enjoyment of Food	3.2 ± 0.8	3.7 ± 0.8	0.011
Desire to Drink	2.7 ± 1.0	2.8 ± 1.1	0.721
Satiety Responsiveness	2.8 ± 0.7	2.6 ± 0.6	0.086
Slowness in Eating	2.6 ± 1.0	2.1 ± 0.7	0.029
Emotional Undereating	3.0 ± 0.8	2.7 ± 0.9	0.037
Food Fussiness	2.9 ± 1.0	2.8 ± 1.0	0.789
Food Approach	10.2 ± 2.8	10.5 ± 2.7	0.786
Food Avoidance	11.4 ± 2.2	10.2 ± 1.9	0.035

Mean: mean; SD: standard deviation; n: number of participants; p: level of statistical significance.

concerns—particularly among girls—may influence eating behaviors, with weight control behaviors and dietary restriction being more common [26,27]. In a study conducted in adolescents with epilepsy, eating disorders were reported to be more frequent in females and associated with lower BMI, while no association was found with epilepsy duration or seizure type [28]. Similarly, in our study, food avoidance was more pronounced in girls with epilepsy compared with boys. These findings suggest that the effect of sex on eating behavior may be related to multiple interacting factors.

Studies conducted in neurological disorders other than epilepsy have also yielded similar findings. In children with Tourette syndrome, food approach and emotional overeating behaviors were found to be higher; however, no differences were observed between groups in terms of BMI and no significant associations were identified between CEBQ subscales and tic severity [29]. In a study of children with Dravet syndrome, slowness in eating assessed using the CEBQ was reported to be more prominent compared with healthy controls [30]. These findings suggest that epilepsy subtypes and accompanying neurodevelopmental features may differentially influence eating behaviors.

Studies from Türkiye have also reported heterogeneous CEBQ results. In a large sample of healthy children, food fussiness and slowness in eating were more prominent in the preschool period, whereas desire to drink was higher in boys [31]. In a study by Coşkun et al. using the CEBQ in children with epilepsy, no significant relationship was found between BMI and eating behaviors [18]. This finding is consistent with the results of our study.

It has been suggested that epilepsy may influence appetite, weight changes, and eating behaviors in children, potentially related both to seizures themselves and to the central and metabolic effects of ASMs. In addition to their endocrine and metabolic effects, ASMs may indirectly affect nutrient absorption, leading to different outcomes in eating behaviors [32,33]. Valproic acid (VPA), in particular, is known to be associated with weight gain and appetite changes. Some studies have shown increased serum ghrelin levels in children receiving VPA [32], whereas others have reported decreased ghrelin levels [34].

In a review examining the effects of ASMs on appetite and weight in children aged 0–18 years, VPA was reported to be the medication most frequently associated with increased appetite and weight gain, while levetiracetam was noted to have minimal effects on appetite [35]. In our study, no significant differences were observed in CEBQ subscale scores either between patients receiving and not receiving ASMs treatment or between those treated with levetiracetam and VPA. This finding is consistent with a similar study reported from Türkiye [18]. These results suggest that changes in appetite and weight are not solely attributable to ASMs use but rather arise from the interaction of multiple biological, psychosocial, and environmental factors.

There are also studies in the literature emphasizing the effects of parental attitudes and polytherapy on eating behaviors. Balcı et al. reported higher enjoyment of food in children with epilepsy, while slowness in eating was more pronounced in those receiving polytherapy [36]. In a study of SCN1A-related epilepsies, children receiving more than three ASMs were reported to have more eating and gastrointestinal problems, and a reduction in seizure frequency was associated with improvement in eating problems [37].

The high prevalence of psychiatric comorbidities in children with epilepsy is another important factor when evaluating eating behaviors. Epilepsy has been frequently reported to co-occur with psychiatric disorders, particularly depression and anxiety [38]. In a study of adults

Table 9
Child Eating Behavior Questionnaire Scores by Sex in Healthy Controls.

Child Eating Behavior Questionnaire Subscales	Female (n = 32) Mean ± SD	Male (n = 30) Mean ± SD	p
Food Responsiveness	2.4 ± 1.1	2.4 ± 1.1	0.994
Emotional Overeating	1.9 ± 0.9	2.0 ± 0.7	0.537
Enjoyment of Food	3.4 ± 0.9	3.4 ± 1.0	0.930
Desire to Drink	2.8 ± 1.1	3.1 ± 0.9	0.156
Satiety Responsiveness	2.9 ± 0.7	2.8 ± 0.8	0.865
Slowness in Eating	2.6 ± 0.9	2.6 ± 1.3	0.625
Emotional Undereating	2.8 ± 1.0	2.8 ± 0.9	0.728
Food Fussiness	2.8 ± 1.0	2.8 ± 1.0	0.760
Food Approach	10.5 ± 3.0	10.9 ± 2.9	0.588
Food Avoidance	11.0 ± 2.5	11.0 ± 1.9	0.878

Mean: mean; SD: standard deviation; n: number of participants; p: level of statistical significance.

Table 10
Distribution of Patients and Healthy Controls Across BMI Percentile Groups.

	<3rd percentile (n = 7)	>3rd–<97th percentile (n = 123)	≥97th percentile (n = 10)	p
Epilepsy	3 (42.9%)	69 (56.1%)	6 (60.0%)	0.853
Controls	4 (57.1%)	54 (43.9%)	4 (40.0%)	
Total	7 (100%)	123 (100%)	10 (100%)	

n = number; % = column percentage.

with epilepsy, increased appetite and increased BMI were observed together, with only a weak association with sex [39].

In subgroup analyses based on seizure type, emotional undereating scores were higher in patients with focal epilepsy compared with those with generalized epilepsy; however, this difference lost statistical significance after Holm correction for multiple comparisons. This finding suggests that the initially observed difference may have been incidental and highlights the increased risk of Type I error associated with multiple comparisons. Similarly, analyses based on the main dimensions of food approach and food avoidance revealed no significant differences between epilepsy and control groups with respect to age or sex.

When our findings are combined, they show that children with epilepsy do not exhibit significantly different eating behavior patterns compared to the healthy group. This suggests that epilepsy itself may not be the primary determinant of eating disorders and that previously reported changes may be more closely related to accompanying psychiatric conditions, medication burden, or specific epilepsy syndromes rather than epilepsy itself. The observation of gender differences in eating behaviors in the epilepsy group highlights the potential impact of gender-specific factors that may require closer clinical attention, particularly during adolescence.

5. Conclusion and Recommendations

The eating behaviors of children and adolescents with epilepsy should be carefully examined, particularly in the context of potential interactions between neurological disease, treatment-related factors, and psychosocial effects. Although it has been reported that antiseizure medications affect appetite and body weight in some patients, current findings suggest that epilepsy alone may not sufficiently explain changes in eating behavior in the absence of psychiatric comorbidities. Well-designed prospective studies incorporating psychiatric assessments are needed to better elucidate the complex relationships between epilepsy, treatment, comorbid conditions, and eating behavior, and to inform targeted interventions aimed at improving quality of life.

Limitations.

The post-hoc power analysis suggested limited sensitivity for detecting small between-group differences in Eating Behavior Questionnaire subscale scores. As the observed effect sizes were generally small, nonsignificant findings should be interpreted cautiously, since subtle but potentially clinically meaningful differences may have remained undetected. Moreover, the use of multiple-comparison adjustments may have further reduced statistical power. Given the well-established association between epilepsy and psychiatric comorbidities that may independently affect eating behavior, the lack of direct assessment of conditions such as anxiety, depression, ADHD, and autism spectrum disorder represents an important limitation of this study. Future studies with larger cohorts and a priori power calculations targeting small effect sizes are needed to better characterize eating behavior patterns in patients with epilepsy.

CRediT authorship contribution statement

Özlem Kemer Aycan: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Methodology, Investigation. **Hilal Aydin:** Project administration, Methodology, Funding acquisition, Formal analysis, Data curation. **Özge Demircan Tulacı:** Software, Methodology, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yebeh.2026.110987.

Data availability

Data will be made available on request.

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