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ORIGINAL ARTICLE / ÖZGÜN MAKALE

EXAMINATION OF THE RELATIONSHIP OF SERUM ADIPOKINE HORMONE LEVELS WITH THE DEGREE AND SOME MARKERS OF OBESITY IN OBESE CHILDREN

OBEZ ÇOCUKLARDA SERUM ADİPOKİN HORMON DÜZEYLERİNİN OBEZİTE DERECELERİ VE BAZI BELİRTEÇLERİ İLE İLİŞKİSİNİN İNCELENMESİ

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ABSTRACT

Objective: *Obesity is defined as excessive increase in adipose tissue in the body. Adipose tissue also provides the secretion of adipokine hormones such as leptin, adiponectin, asprosin and apelin, which play an important role in metabolism and energy homeostasis. For this reason, changes in the levels of these adipokine hormones secreted from the adipose tissue, not the increase in adipose tissue alone, are effective in determining childhood obesity and explaining the pathological mechanisms. Evaluation of these polypeptides will be effective in the diagnosis of obesity and elucidating the pathological mechanisms associated with obesity.*

Material and Method: *In our study, it was conducted to reveal the relationship of obese children with some markers of obesity such as serum leptin, adiponectin, asprosin and apelin levels, and blood lipids, fasting blood glucose and insulin resistance index (HOMA-IR) values. 105 obese children and 38 normal weight children participated in our study. The demographic information of the children was determined by the face-to-face survey method. Leptin, adiponectin, asprosin and apelin levels were determined in morning fasting blood samples from obese and normal weight children with the help of commercial Elisa kit.*

Result and Discussion: *Leptin and adiponectin levels were not different in obese and normal weight children ($p>0.05$). Asprosin and apelin levels were found to be significantly higher in obese children compared to normal weight children ($p<0.001$). Positive correlations were observed between adipokine hormones in both obese and normal children. The ratios of leptin/asprosin, leptin/apelin, adiponectin/asprosin, adiponectin/apelin, asprosin/apelin were found to be statistically significantly lower in obese children when compared with normal weight children ($p<0.05$). In our study, high levels of asprosin and apelin, which have conflicting results in the literature, and positive correlations between adipokine hormones can be considered as important findings.*

Keywords: *Adiponectin, apelin, asprosin, childhood obesity, leptin*

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ÖZ

Amaç: *Obezite, vücuttaki yağ dokusunun aşırı artışı olarak tanımlanır. Yağ dokusu ayrıca metabolizma ve enerji homeostazında önemli rol oynayan leptin, adiponektin, asprosin ve apelin gibi adipokin hormonlarının salgılanmasını sağlar. Bu nedenle, sadece yağ dokusundaki artış değil, yağ dokusundan salgılanan bu adipokin hormonlarının düzeylerindeki değişiklikler çocukluk çağı obezitesini belirlemede ve patolojik mekanizmaları açıklamada etkilidir. Bu polipeptitlerin değerlendirilmesi obezitenin tanısında ve obeziteyle ilişkili patolojik mekanizmaların aydınlatılmasında etkili olacaktır.*

Gereç ve Yöntem: *Çalışmamız, obez çocukların serum leptin, adiponektin, asprosin ve apelin düzeyleri gibi bazı obezite belirteçleri ile kan lipitleri, açlık kan şekeri ve insülin direnci indeksi (HOMA-IR) değerleri arasındaki ilişkiyi ortaya koymak amacıyla yapıldı. Çalışmamıza 105 obez çocuk ve 38 normal kilolu çocuk dahil edildi. Çocukların demografik bilgileri yüz yüze anket yöntemi ile belirlendi. Leptin, adiponektin, asprosin ve apelin düzeyleri obez ve normal kilolu çocuklardan alınan sabah açlık kan örneklerinde ticari Elisa kiti yardımıyla tayin edildi.*

Sonuç ve Tartışma: *Leptin ve adiponektin düzeyleri obez ve normal kilolu çocuklarda farklı değildi ($p>0,05$). Asprosin ve apelin düzeyleri obez çocuklarda normal kilolu çocuklara göre anlamlı olarak yüksek bulundu ($p<0,001$). Hem obez hem de normal kilolu çocuklarda adipokin hormonları arasında pozitif korelasyonlar gözlendi. Leptin/asprosin, leptin/apelin, adiponektin/asprosin, adiponektin/apelin, asprosin/apelin oranları obez çocuklarda normal kilolu çocuklara göre istatistiksel olarak anlamlı şekilde düşük bulundu ($p<0,05$). Çalışmamızda literatürde çelişkili sonuçlar bulunan asprosin ve apelin düzeylerinin yüksek olması ve adipokin hormonları arasındaki pozitif korelasyonlar önemli bulgular olarak değerlendirilebilir.*

Anahtar Kelimeler: *Adiponektin, apelin, asprosin, çocukluk çağı obezitesi, leptin*

INTRODUCTION

Childhood obesity is a growing global health problem all over the world. Obesity also cause an economic burden on the health system [1,2]. Children at risk need to be carefully identified and treated to effectively prevent obesity. The causes of childhood obesity are highly complicated and multifactorial. These factors include the genetic, environmental and ecological factors that can be associated with the family environment, the society in which they are raised, and particularly the school where they spend most of their days. Some endocrine disorders, insufficient sleep and some medications can also be considered as causes of obesity [1,3,4].

Obesity in children and adolescent is related with metabolic diseases such as psychological problems, obstructive sleep apnea, orthopedic disorders, polycystic ovarian diseases, type 2 diabetes, cardiovascular diseases and metabolic disorders [5]. Nowadays, the relationship of obesity with chronic diseases and the fact that the foundations of adult obesity are laid in childhood become substantial in the prevention, particularly in the diagnosis and treatment of childhood obesity.

By definition, obesity is defined as an overgrowth of adipose tissue, and adipose tissue is the main site where triacylglycerols are stored as energy [6,7]. However, adipose tissue does not only serve as a storage, but also secretes hormones called adipokines, which play an essential role in metabolism and energy homeostasis, such as leptin, adiponectin, apelin and asprosin [7,8]. These adipokines have a significant impact on obesity-related comorbidities and complications. Many studies have found increase plasma apelin and asprosin levels in obese individuals, and this increase has been associated with gain in adipose tissue [1,6,9,10]. These secretions of adipose tissue play a predispose role in diseases such as insulin resistance, metabolic syndrome and type 2 diabetes [8].

Understanding the pathophysiological relationship between the serum adipokine levels of obese individuals and obesity-related comorbidities may enable more accurate identification of high-risk patients. This definition comes to the fore more in child groups where the progression of obesity and obesity-related complications is important. Unlike the current data in this study on childhood obesity; not only one but several of the adipokines that may be associated with obesity have been evaluated together.

MATERIAL AND METHOD

Study Groups

In this study, the relationship between serum adipokine hormone levels and the degree of obesity and some markers in obese children was examined cross-sectionally. The study included 105 obese children (54 females and 51 males) between the ages of 10-17 who applied to the Pediatric Endocrinology Polyclinic of Keçiören Training and Research Hospital (KAEH) and 38 healthy and normal weight children (14 females and 24 males) who applied to the Healthy Child Polyclinic. The obese group is between the ages of 10-17 and has a BMI of ≥95 according to the WHO's 5-19 age and sex-specific references. Additionally, children were classified as I. degree obese (mild; 100-120% of Body Mass Index (BMI) corresponding to the 95th percentile), II. degree obese (moderate; 120-140% of BMI corresponding to the 95th percentile), III. degree obese (severe; BMI >140 corresponding to the 95th percentile) and this was considered as an inclusion criterion [11]. The control group included normal-weight children aged 10-17 years with a BMI between the 5th and 85th percentile according to the WHO (2007) 5-19 age and sex-specific references.

Local ethics committee approval (dated 11.05.2021 and numbered 2012-KAEK-15/2289) was obtained before sample collection for the study. Children and their families were informed verbally and in writing about the details of the study before they were included in the study. Along with that, consent was obtained from both parents and children by signing an informed consent form.

Blood samples were taken from the children participating in the study after at least 8 hours fasting. The blood sample was centrifuged and the serum part was separated and stored at -20°C.

Anthropometric Measurements

Demographic information were determined by face-to-face survey method before the children accepted to the study. Then, body weight (kg), height (cm), waist circumference (cm), hip circumference (cm), BMI (kg/m²), neck circumference (cm), waist/hip ratio were taken in accordance with the specified methods. Fat percentages of obese children were measured by the Bioimpedance Current (BIA) method (Tanita).

Experimental Measurements

The amounts of serum leptin, adiponectin, asprosin and apelin (Human ELISA kits, SunRedBio, China) were determined with a commercial kit. According to these measurement results, apelin/adiponectin ratio, leptin/adiponectin ratio and asprosin/adiponectin ratios were evaluated. In addition, all children's fasting blood glucose, triglyceride (TG), total cholesterol (TC), LDL-C, HDL-C, insulin and HbA1C measurements were also made with routine laboratory tests.

Statistical Analysis

The data obtained as a result of the study are indicated as the mean (standard deviation). Data analysis was performed using IBM SPSS Statistics 22.0 software (IBM Corporation, Armonk, NY, USA). Whether the distributions of continuous variables were normal or not was determined by Kolmogorov-Smirnov test. Chi-square test was used for comparison of qualitative data in comparison of demographic data. While the mean differences between groups were compared using Student's t test, the Mann–Whitney U test was applied for the comparisons of the continuous variables, in which the parametrical test assumptions were not met. Spearman-correlation test was used to examine the relationship between the parameters for the non-normally distributed data. P values of less than 0.05 were regarded as statistically significant.

RESULT AND DISCUSSION

In the Kolmogorov-Smirnov test (significance level was taken as 0.05), the adipokine hormone levels of the obese and normal weight children in our study did not show a normal distribution. For this reason, non-parametric tests, which do not require the normality condition, were applied in the statistical analysis of the findings.

Table 1 shows the demographic information of obese and normal weight children. The study groups were compared statistically according to age, age range, gender, family obesity status, familial

chronic disease history and mode of delivery, and the difference between other criteria was found statistically significant ($p<0.05$). Waist circumference, neck circumference, hip circumference, BMI and waist/hip circumference ratios were compared and a significant difference was found between these anthropometric measurements ($p<0.05$) (Table 1).

Table 1. Main characteristics of obese and normal weight children

Variable	Obese Child (N= 105)	Normal Child (N=38)	P
Age, Mean (Standard Deviation)	13.6 (2.1)	12.8 (1.9)	0.031
Age Range			
≤11 age, N (%)	23 (21.9)	10 (26.3)	0.028
12 age, N (%)	11 (10.5)	10 (26.3)	
13 age, N (%)	16 (15.2)	8 (21.1)	
14 age, N (%)	16 (15.2)	5 (13.2)	
≥15, age N (%)	39 (37.1)	5 (13.2)	
Gender			
Girls, N (%)	54 (51.4)	14 (36.8)	--
Boys, N (%)	51 (48.6)	24 (63.2)	
Obesity Status of the Family			
Yes, N (%)	87 (82.9)	15 (39.5)	0.000
No, N (%)	18 (17.1)	23 (60.5)	
Family History of Chronic Disease			
Yes, N (%)	71 (67.6)	17 (44.7)	0.013
No, N (%)	34 (32.4)	21 (55.3)	
Mode of Delivery			
Vaginal Delivery, N (%)	56 (53.3)	23 (60.5)	0.566
Cesarian Section, N (%)	49 (46.7)	15 (39.5)	
Anthropometric Measurements			
Waist Circumference (cm)	98.1 (11.9)	69.8 (8.6)	0.000
Neck Circumference (cm)	37.2 (3.2)	30.7 (2.3)	0.000
Hip Circumference (cm)	112.9 (11)	83.7 (10)	0.000
Fat percentage (%)	37.3 (7)	--	--
BMI (kg/m^2)	31.5 (4.7)	19.4 (2.8)	0.000
Waist/Hip ratio	0.86 (0.075)	0.83 (0.072)	0.020
Obesity Degree			
1th Degree, N (%)	51 (48.6)	--	--
2th Degree, N (%)	45 (42.9)	--	--
3th Degree, N (%)	9 (8.6)	--	--

Table 2 includes routine biochemistry tests of obese and normal weight children. TC, TG, LDL-C, HDL-C, fasting blood glucose, insulin level and HOMA-IR values were compared. Mean TG and fasting insulin levels of obese children were found to be significantly higher than those of normal weight children ($p<0.05$). The HDL-C level of the normal-weight children was found to be significantly higher than the obese children ($p<0.05$). There was no significant difference between obese and normal weight children in terms of mean TC, LDL-C, fasting blood glucose, and HbA1c levels ($p>0.05$) (Table 2).

Adipokine hormones such as leptin, adiponectin, asprosin, apelin, resistin, and visfatin secreted from adipose tissue [9,10,12,13] play an important role in metabolism and energy homeostasis [9]. Depending on these hormones, disorders such as insulin resistance, metabolic syndrome and diabetes may develop [10]. In determining childhood obesity and explaining the mechanisms, not only the increase in adipose tissue, but also the evaluation of the changes in the levels of these adipokines secreted from the adipose tissue will be effective. In our study, serum leptin, adiponectin, asprosin and apelin levels and their ratios to each other were evaluated in obese and normal weight children, and the correlations between them and their relationship with the degree of obesity and some of its markers were

examined. When the hormone levels of children were evaluated, leptin and adiponectin levels did not show a statistically significant difference between obese and normal weight children (p>0.05) (Table 3).

Table 2. Routine biochemistry tests of obese and normal weight children

Parameter	**Obese Child (N= 105)**	**Normal Weight Child (N=38)**	**P**
	Mean (SD)		
Total Cholesterol (mg/dl)	158.1 (45.8)	155.7 (35)	0.776
Triglyceride (mg/dl)	141.4 (92.5)	87.1 (50.5)	0.000
HDL-C	41.8 (12.3)	52.3 (11)	0.000
LDL-C	90.6 (31.1)	85.9 (29.5)	0.422
Glucose (mg/dl)	90.5 (23.6)	92.1 (7.9)	0.545
Insulin	19.8 (13.5)	7.9 (3.3)	0.000
HbA1c (%)	5.1 (1.2)	5 (0.4)	0.503
HOMA-IR	4.99 (3.8)	1.8 (0.7)	0.000

Table 3. Adipokine hormone levels and rates of hormone levels in obese and normal weight children

Adipokine Levels	**Obese Child* Mean (SD)**	**Normal Weight Child** Mean (SD)**	**P**
Leptin (ng/l)	4.25 (4.65)	3.59 (1.83)	0.265
Adiponectin (ng/l)	10.46 (12.01)	11.31 (9.62)	0.443
Asprosin (ng/l)	22.58 (18.33)	11.15 (11.17)	**0.000**
Apelin (ng/l)	19.36 (14.55)	10.66 (13.94)	**0.001**

*N= 105, **N=38

Leptin is associated with fat storage, which reflects energy status in the body [14]. Obese individuals with high fat content have high circulating leptin levels. However, when leptin promotes satiety, it was thought that obese individuals might develop a type of central leptin resistance that impairs the satiety response [15]. In our study, leptin levels did not show a significant difference between obese and normal weight children. However, the slight increase in leptin levels observed in obese children can be considered as a reflection of the development of leptin resistance in childhood. In a study conducted; obese children have been reported to have higher serum leptin levels than controls [16]. In our study, a negative correlation was observed between serum leptin levels and TC and LDL-C levels in obese children (p=0.008, p=0.002). Unlike our study, according to a study that reported that leptin levels may be determinative in the evaluation of blood lipids, leptin levels were found to be higher in obese children, and a positive correlation was reported between leptin levels and TG and LDL-C levels, and a negative correlation between HDL-C levels [17]. However, the effect of leptin on blood lipids is still unclear.

Adiponectin can be considered as a therapeutic approach to reduce the burden of chronic diseases such as obesity, diabetes and heart diseases [18]. In our study, although there was no significant difference between the adiponectin levels of obese and normal weight children, it was observed to be lower in obese children. Other studies have also reported lower adiponectin levels in obese children compared to controls [16,19]. In our study, adiponectin levels were observed to be lower in obese children aged 11 years and younger. In addition, we found a positive correlation between serum adiponectin levels and HOMA-IR values in children with normal weight (p=0.005). This may suggest that there may be a mechanism to prevent the development of obesity in children.

Asprosin is a fasting-related adipokine hormone that regulates glucose release from the liver [20]. Asprosin has been shown to be significantly associated with glucose and insulin release in the liver during fasting. It has been reported that increased asprosin levels may be a risk factor for insulin resistance. It is also thought that reducing asprosin levels may prevent hyperinsulinemia associated with metabolic syndrome [21]. Consistent with hepatic glucose release during fasting, serum asprosin levels increase with fasting and rapidly decrease with re-intake of food. Studies on asprosin suggest that

asprosin may be a potential target for the treatment of diabetes and obesity [20]. In our study, serum asprosin levels were found to be significantly higher in obese children than in normal-weight children. When both obese and normal weight children were evaluated together, it was observed that serum asprosin levels showed a positive correlation with insulin levels and HOMA-IR values (R=0.200; p=0.027 and R=0.225; p=0.013, respectively). A positive correlation was observed between serum asprosin level and TC in normal weight children (p=0.035). High levels of asprosin, a hormone that regulates glucose secretion from the liver and is released during fasting, in obese children can be considered not only as a result of obesity but also as a cause. In a study by Wang et al. [20] serum asprosin levels were found to be significantly higher in obese children compared to controls. In addition, asprosin levels were found to be higher in children with insulin resistance. According to these results; it has been reported that serum asprosin levels may be a new marker for predicting obesity and obesity-related diseases [20]. In another study, it was reported that asprosin levels were significantly higher in obese children compared to controls and that asprosin may be a predictor of obesity [21]. However, Long et al. [8] reported that obese children had lower asprosin levels compared to normal weight controls. Although it is accepted that these low asprosin levels will cause weight loss, it has been stated that this may be related to compensatory adaptations in energy metabolism. It has also been reported that this may be related to the degree of obesity [8]. However, in our study, no significant relationship was found between the degree of obesity and adipokine hormone levels.

Apelin, synthesized from adipose tissue, is one of the newly discovered bioactive peptides. Apelin plays an important role in feeding mechanisms by stimulating the secretion of cholecystokinin and in lowering blood pressure through the nitric oxide mechanism. Apelin synthesis is stimulated by insulin. It is known that plasma levels may increase especially in association with insulin resistance and hyperinsulinemia [22]. Apelin secretion decreases with fasting and increases with food intake similar to insulin. Apelin is effective in regulating fluid balance, blood pressure, heart contraction and stimulating the release of ACTH by the pituitary gland. Many studies have shown that there may be a relationship between serum apelin levels and metabolic diseases [23]. In our study, positive correlations were observed between apelin, insulin and HOMA-IR in normal weight children (p=0.011 and p=0.016, respectively). At the same time, a positive correlation was observed between apelin, TC and LDL-C in normal-weight children in our study (p=0.020, p=0.007, respectively). Studies show that there may be a relationship between serum apelin levels and metabolic diseases [9,22]. In our study, serum apelin levels were found to be significantly higher in obese children, supporting these findings. The fact that apelin is an insulin-stimulated hormone [22] and the association between obesity and hyperinsulinemia, insulin resistance, and diabetes [4] suggests that apelin may be elevated as a result of obesity. In a study, serum apelin levels were found to be significantly higher in obese children compared to controls, and it was stated that this may play a role in childhood obesity [9]. Similarly, in another study, serum apelin levels were found to be significantly higher in obese children compared to controls. In addition, a positive correlation was observed between serum apelin level and fasting blood glucose, serum insulin, HOMA-IR, TG and TC values in obese children. According to these results, it has been reported that serum apelin levels may play a role in the development of obesity-related complications such as insulin resistance, hypertension and metabolic syndrome in children [22]. Increased serum apelin levels with adipose tissue increase may play a role in the pathological mechanism of obesity. In this case, apelin can be used in clinical applications as a biomarker or a therapeutic target. However, in another study; It has been reported that obese children have lower plasma apelin levels than controls, and there is a negative correlation between plasma apelin levels and BMI, insulin levels, and HOMA-IR values [19]. The findings of studies investigating the level and role of apelin in obesity are inconsistent and more studies are needed.

The ratios of hormones were compared with each other, leptin/asprosin, leptin/apelin, adiponectin/asprosin, adiponectin/apelin and asprosin/apelin levels in obese children, it was found to be significantly lower ($p<0.05$) to normal weight children (Figure 1). Studies have calculated that the leptin/adiponectin ratio is higher in obese individuals than in normal-weight individuals [24,25]. In our study, although not statistically significant, the leptin/adiponectin ratio was found to be slightly higher in obese children. However, in our study, the ratios of all hormones were evaluated for the first time, and significant differences were observed between the ratios of leptin/asprosin, leptin/apelin,

adiponectin/asprosin, adiponectin/apelin, asprosin/apelin in obese children compared to normal-weight children. In one study, it was reported that the leptin/adiponectin ratio in obese children was more strongly associated with insulin resistance and cardiometabolic comorbidities than leptin and adiponectin separately [24]. In another study; It has been reported that the leptin/adiponectin ratio may be a better predictor of insulin sensitivity than leptin and adiponectin levels [25]. According to these results, the evaluation of the ratios of these interrelated hormones may reveal stronger results. In our study, positive correlations were observed between leptin and adiponectin and asprosin, between adiponectin and asprosin, and between asprosin and apelin in obese children. In normal weight children, positive correlations were observed between adiponectin and asprosin and between asprosin and apelin. In this case, the positive correlations between these hormones may be effective on the complex mechanism of obesity. Especially the fact that these hormones increase and decrease together can be effective in increasing or decreasing food intake. Our study was the first to examine the relationships between these adipokine hormones. However, considering the relationship between these hormones, more research is needed.

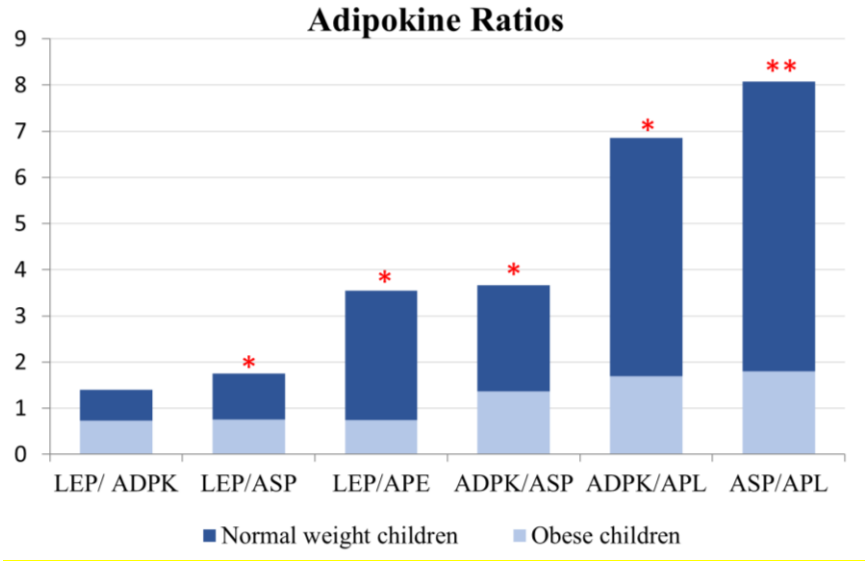

Figure 1. Adipokine hormone ratios in obese and normal weight children (* p<0.001, ** p<0.05)

When compared by gender, asprosin and apelin levels in obese girls were found to be significantly higher than in normal-weight girls. Whereas obese and normal weight children were evaluated separately, asprosin hormone levels in normal weight girls [22.39 (15.79) ng/l] were found to be significantly lower than those in normal weight boys [6.98 (8.09) ng/l] (p<0.001), and apelin hormone levels in obese girls [21.63 (14.76) ng/l] were significantly higher than those in obese boys [3.58 (5.33) ng/l] (p<0.005). Asprosin and apelin levels in girls in the obese group were observed to be higher than in girls in the normal weight group. However, in the obese group, asprosin levels did not differ depending on gender, while it was found to be higher in boys in the normal weight group. Apelin levels were found to be higher in obese girls. In our study, when obese and normal weight children were considered together, apelin levels were found to be higher in girls than in boys (p<0.05). In a study, apelin levels were found to be significantly higher in girls compared to controls, similar to our study [9]. Another study reported lower serum asprosin levels in boys than girls and a negative association between asprosin and male gender in all groups [26]. The relationship between apelin and asprosin levels and gender differences has not been clearly investigated and explained. In a study, it was reported that apelin and adiponectin levels were independent of gender differences. However, significant differences in apelin hormones in the pubertal period and adiponectin in the pre- and post-pubertal periods suggested that there may be a relationship between sex hormones and adipokine hormone levels [19]. In another study, it was reported that the hormone levels of adipokines secreted from adipose tissue may increase

in girls because of hormonal differences during adolescence, because more adipose tissue and non-fatty tissue increase in boys [27].

When there was no statistically significant difference in hormone levels between the groups in terms of obesity degree, adiponectin and apelin levels showed a linear increase, while asprosin levels showed a linear decrease ($p>0.05$).

When the correlations between adipokine hormones were examined in obese children, positive correlations were observed between leptin and adiponectin and asprosin, between adiponectin and asprosin, and between asprosin and apelin (Table 4). Considering the correlations between adipokine hormones in normal weight children, positive correlations were observed between adiponectin and asprosin and apelin, and between asprosin and apelin (Table 4). When obese and normal weight children were evaluated together, positive correlations were observed between adiponectin and leptin, asprosin and apelin, and between asprosin and apelin (Table 4).

Tablo 4. The relationship between adipokine hormone relationship in obese and normal weight children

			R/P*	
		Leptin	**Adiponectin**	**Asprosin**
Obese Child	**Adiponectin**	**0.354 (0.000)**	--	
	Asprosin	**0.227 (0.035)**	**0.411 (0.000)**	--
	Apelin	-0.045 (0.670)	0.174 (0.098)	**0.254 (0.023)**
Normal Weight Child	**Adiponectin**	0.206 (0.275)	--	
	Asprosin	0.181 (0.339)	**0.379 (0.021)**	--
	Apelin	0.222 (0.446)	**0.628 (0.003)**	**0.603 (0.005)**
Total	**Adiponectin**	**0.319 (0.000)**	--	
	Asprosin	0.118 (0.205)	**0.400 (0.000)**	**--**
	Apelin	-0.107 (0.276)	**0.192 (0.043)**	**0.394 (0.000)**

*R/P: Correlation coefficient/Significance

Although there are conflicting results in the literature, high asprosin and apelin levels in obese children can be considered as important findings in our study. High levels of asprosin, an adipokine that increases food intake, may lead to the development of obesity. It could also be a therapeutic approach for the treatment of obesity. High apelin, a hormone that increases with food intake and insulin secretion, may be a result of obesity. However, more comprehensive studies are needed on these issues.

The study had two main limitations. One of these is that the number of children in the study and control groups was not equal, which limited the homogeneous evaluation of the data obtained. The other is the lack of standard measurements of adipokine hormone levels, which makes evaluation difficult.

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AUTHORSHIP CONTRIBUTIONS

Concept: M.D., S.Y.A.; Design: M.D., D.M.T., S.Y.A.; Control: M.D., S.Y.A.; Sources: S.Y.A.; Materials: D.M.T., A.D.B., E.F., F.M.K., S.Y.A.; Data Collection and/or Processing: D.M.T., A.D.B., E.F., F.M.K.; Analysis and/or Interpretation: M.D., A.D.B., E.F., F.M.K., S.Y.A.; Literature Review: M.D., S.Y.A.; Manuscript Writing: M.D., D.M.T., A.D.B., E.F., F.M.K., S.Y.A.; Critical Review: S.Y.A.; Other: -

CONFLICT OF INTEREST

The authors declare that there is no real, potential, or perceived conflict of interest for this article.

ETHICS COMMITTEE APPROVAL

The study protocol was approved by the Keçiören Clinical Research Ethics Committee (date: 11 May 2021, number: 15/2289).

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