ORIGINAL ARTICLE

AHI EVRAN MEDICAL JOURNAL
E-ISSN: 2619 - 9203

Investigation of Changes in Lactic Acid Bacteria in the Gut Microbiota of Obese Children

Obez Çocukların Bağırsak Mikrobiyotasındaki Laktik Asit Bakterilerinin Değişiminin Araştırılması

Cihat ÖZTÜRK[1] Serdar FİDAN[2] Memiş BOLACALI[3] Elif SEVİM[4] Ali GÜNEŞ[2]

ÖZ

Amaç: Bu çalışmada, obez çocuklarda bağırsak mikrobiyotasındaki bakteri popülasyonlarının değişimlerini belirlemek ve obezitenin bağırsak mikrobiyota kompozisyonu üzerindeki etkilerini değerlendirmek amaçlanmıştır.
Araçlar ve Yöntem: Çalışmaya, KAEÜ Eğitim-Araştırma Hastanesi Çocuk Polikliniği'ne başvuran toplam 50 çocuk dahil edilmiştir. Çalışma grubunu, Dünya Sağlık Örgütü'nün vücut kitle indeksi (VKİ) kriterlerine göre obez olarak sınıflandırılan 25 çocuk ve kontrol grubunu VKİ'si normal olan 25 çocuk oluşturmuştur. Toplam bakteri, *Lactobacillus spp.*, *Bifidobacterium spp.*, *Enterococcus spp.*, *Staphylococcus spp.* ve Gram negatif bakteriler gibi hedef mikroorganizmalar kültür ve moleküler yöntemlerle analiz edilmiştir. İstatistiksel değerlendirme, bağırsak mikrobiyotasındaki popülasyon değişikliklerini tespit etmek amacıyla uygun analiz yöntemleriyle yapılmıştır.
Bulgular: Obez çocukların VKİ ve vücut ağırlıkları kontrol grubundan anlamlı derecede daha yüksek bulunmuştur ($p<0.001$). Obez çocuklarda total bakteri, *Enterococcus spp.*, *Lactobacillus spp.* ve Gram negatif bakteri oranlarında rakamsal bir artış gözlenirken, *Bifidobacterium spp.* ve *Staphylococcus spp.* oranlarında rakamsal bir azalma tespit edilmiştir. Yaş ile total bakteri sayısı arasında negatif yönlü düşük bir korelasyon ($r=-0.428$), yaş ile VKİ arasında ise pozitif yönlü bir korelasyon ($r=0.479$) bulunmuştur. Ayrıca, total bakteri ile *Enterococcus spp.* arasında ($r=0.558$) ve *Lactobacillus spp.* ile *Enterococcus spp.* arasında yüksek düzeyde pozitif korelasyonlar tespit edilmiştir ($r=0.730$).
Sonuç: Bu bulgular, mikrobiyotanın erken dönemde izlenmesi ve yönetiminin çocukluk çağı obezitesinin önlenmesi ve tedavisinde yeni fırsatlar sunabileceğini, ayrıca mikrobiyotanın düzenlenmesine yönelik müdahalelerin obezitenin önlenmesi ve yönetimi için umut vadeden yaklaşımlar olarak değerlendirilebileceğini göstermektedir.

Anahtar Kelimeler: bakteri; mikrobiyota; obezite; vücut kitle indeksi

ABSTRACT

Purpose: This study aimed to identify changes in bacterial populations within the gut microbiota of obese children and to evaluate the effects of obesity on gut microbiota composition.
Materials and Methods: The study included a total of 50 children admitted to the Pediatric Clinic of Kırşehir Ahi Evran University Training and Research Hospital. The study group consisted of 25 children classified as obese according to the body mass ındex (BMI) criteria of the World Health Organization (WHO), while the control group included 25 children with normal BMI. Target microorganisms such as total bacteria, *Lactobacillus spp.*, *Bifidobacterium spp.*, *Enterococcus spp.*, *Staphylococcus spp.*, and Gram-negative bacteria were analyzed using culture and molecular methods. Statistical analyses were performed to assess changes in the gut microbiota population.
Results: In obese children, numerical increases were observed in the total bacteria, *Enterococcus spp.*, *Lactobacillus spp.*, and Gram-negative bacteria, whereas numerical decreases were noted in *Bifidobacterium spp.* and *Staphylococcus spp.*. A negative correlation was found between age and total bacterial count, while a positive correlation was observed between age and BMI. Additionally, positive correlations were detected between total bacteria and *Enterococcus spp.*, also, between *Lactobacillus spp.* and *Enterococcus spp.*
Conclusion: These findings suggest that early monitoring and management of the gut microbiota could offer new opportunities in the prevention and treatment of childhood obesity, in addition, interventions to regulate the microbiota could also be considered as promising approaches for the prevention and management of obesity.

Keywords: bacteria; body mass ındex; microbiota; obesity

Received: 23.01.2025; Accepted: 07.04.2025

[1]Department of Medical Microbiology, Faculty of Medicine, Kırşehir Ahi Evran University, Kırşehir, Türkiye.
[2]Department of Pediatrics, Faculty of Medicine, Kırşehir Ahi Evran University, Kırşehir, Türkiye.
[3]Department of Biostatistics and Medical Informatics, Faculty of Medicine, Kırşehir Ahi Evran University, Kırşehir, Türkiye.
[4]Department of Medical Biology, Faculty of Medicine, Kırşehir Ahi Evran University, Kırşehir, Türkiye.
Corresponding Author: Cihat Öztürk, Department of Medical Microbiology, Faculty of Medicine, Kırşehir Ahi Evran University, Kırşehir, Türkiye.
e-mail: cihat.ozturk@ahievran.edu.tr

How to cite: Öztürk C, Fidan S, Bolacalı M, Sevim E, Güneş A. Investigation of changes in lactic acid bacteria in the gut microbiota of obese children. Ahi Evran Med J. 2025;9(2):243-250. DOI: 10.46332/aemj.1625431

INTRODUCTION

Obesity is defined as a significant increase in fat mass, corresponding to a body mass index (BMI) >30 kg/m². Recognized as a rapidly growing global public health issue, obesity is responsible for more than 60% of deaths caused by diseases associated with a high BMI.[1] The high incidence of obesity or overweight in childhood is a risk factor for obesity in adulthood and has been associated with comorbidities such as diabetes mellitus, coronary diseases, respiratory disorders, and cancer. Due to its association with these diseases and its alarming rise in prevalence in recent years, obesity has emerged as a global pandemic and a severe public health concern.[2] According to the World Health Organization (WHO), as of 2023, the global obesity rate among children aged 5-19 years is 10.7%. This rate, which stood at 8.5% between 2000 and 2011, showed a significant increase to 14.8% between 2012 and 2023 a 1.5-fold rise.[3] In Turkey, it was reported in 2016 that one out of every four children aged 7-8 years was overweight or obese, with particularly notable increases in obesity rates among girls.[4] Furthermore, official data published by the Turkish Statistical Institute (TÜİK) in 2015 revealed that the obesity rate in Turkey was 18.5% in 2008, rising steadily to 24.5% in 2014. Although a partial decline was observed in 2022, one in five children was still reported to be obese.[5-7]

The human body harbors a unique community of microorganisms, including bacteria, fungi, viruses, and archaea, in addition to its own cells. The majority of this community, known as the microbiota, is composed of the gut microbiota, which hosts trillions of microorganisms. Studies on fecal samples from healthy, obese, or inflammatory bowel disease (IBD) individuals have revealed that the human gut contains at least 1.000 different bacterial species, with each individual harboring at least 160 species, 18 of which are shared across all individuals. While some evidence suggests that gut microbiota colonization may begin in the intrauterine period, it is widely accepted that it commences at birth, undergoes frequent changes during the first three years of life, and reaches maximum diversity in adolescence.[8]

The gut microbiota plays a crucial role in the production of metabolites and vitamins, the breakdown of indigestible nutrients, and the development of the immune system. It also regulates energy metabolism by producing active signaling molecules, such as short-chain fatty acids (SCFAs), which influence metabolic pathways, insulin sensitivity in adipose tissues, and peripheral organs. In newborns, colonization of the gut begins with exposure to the maternal vaginal and fecal flora, followed by contact with the microbiota of other family members and environmental microorganisms. During this initial phase, dominant bacteria such as *E. coli* and *Streptococcus*, along with facultative anaerobes, are replaced by obligate anaerobes like *Bifidobacterium spp.* and *Bacteroides* as the infant begins breastfeeding. With the transition to oral feeding, the microbiota evolves to resemble that of an adult.[9-13]

Lactobacillus species, which are commonly found in the gut microbiota of both humans and animals, are considered the most prominent and dominant genus of lactic acid bacteria (LAB). Often coexisting with *Bifidobacterium* species, these bacteria play a vital role in maintaining gut homeostasis.[14-18]

An increase in BMI is associated with significant changes in the composition of the intestinal microbiota. Dysbiosis, characterized by microbial imbalances and reduced functional diversity in the gut microbiota, leads to increased intestinal permeability and alterations in SCFA production. These changes disrupt glucose and lipid metabolism, trigger inflammation, and ultimately contribute to obesity. Low-grade inflammation, combined with impaired lipid and glucose homeostasis, alters the regulation of satiety. Physiological imbalances such as increased intestinal permeability, endotoxemia, elevated pro-inflammatory cytokines, excessive fat accumulation, insulin resistance, increased calorie intake, and metabolic syndrome risk are exacerbated by these changes.[19]

These findings highlight the complex interactions between gut microbiota and obesity, emphasizing the critical role of microbial balance in maintaining metabolic health.

There is ongoing debate about whether changes in the microbiota are a cause or a consequence of obesity. Most studies to date have focused on adult populations, with relatively few investigations conducted in obese children.[3] This study aims to identify bacterial population changes in the gut microbiota composition of obese children during childhood and evaluate the impact of obesity on the microbiota.

MATERIALS and METHODS

Study Group

This study was approved by Kırşehir Ahi Evran University Clinical Research Ethics Committee (Date: 01.06.2021 and Numbered 2021-10/11).

This study was conducted on a total of 50 children who visited the Pediatric Clinic of KAEÜ Training and Research Hospital and had not used antibiotics within the 30 days prior to sample collection. The study group consisted of 25 obese children (13 boys and 12 girls) aged between 7 and 17 years, and 25 healthy children (13 boys and 12 girls) within the same age range. Obese children were identified based on the World Health Organization's (WHO) BMI criteria as having BMI values ≥30 kg/m² for their age and gender, while the healthy group included children with BMI values within the range of 18.5–24.9 kg/m².

Anthropometric Measurements

Each child's body weight was measured to the nearest 0.1 kg using a calibrated scale while wearing light clothing. Height measurements were recorded to the nearest 0.1 cm in a standing position without shoes, using a vertical ruler. Body mass index (BMI) was calculated using the formula weight (kg)/height squared (m²). A BMI of ≥30 kg/m² was defined as obese.[20]

Collection of Fecal Samples

Families and children visiting Kırşehir Ahi Evran University Training and Research Hospital were informed about the study, asked for their voluntary participation, and written informed consent forms were obtained from participating families. Sterile sample containers were provided to the volunteers, and fecal samples were collected. The collected samples were transported to the laboratory as quickly as possible under a cold chain. In the laboratory, the fecal samples were promptly processed; portions were used for bacterial culturing, while the remaining samples were stored in sterile containers at -20°C for further analysis.

Bacterial Isolation from Fecal Samples

Fecal samples collected in sterile containers were promptly transported to the laboratory. One g portion of each sample was transferred into sterile 5 ml falcon tubes, and 3 ml of sterile physiological saline was added. The mixture was vortexed to achieve a homogeneous solution. Serial dilutions were prepared from 10^{-1} to 10^{-8}, and the following culture methods were employed:

Lactic Acid Bacteria (LAB): Cultured on man-rogosa-sharpe (MRS) agar. Plates were incubated anaerobically in jars with hydrogen and carbon dioxide-releasing gas packs at 37°C for 2 days.

- **Bifidobacteria:** Cultured on bifidobacterium agar (BA) under similar anaerobic conditions and incubated at 37°C for up to 5 days.

- ***Staphylococcus* spp.:** Cultured on mannitol salt agar (MSA) and incubated aerobically at 37°C for 24 hours.

- ***Enterococcus* spp.:** Cultured on bile esculin agar (BEA) and incubated aerobically at 37°C for 24 hours.

- **Gram-negative *Enterobacteriaceae* Family:** Cultured on eosin-methylene-blue (EMB) agar and incubated aerobically at 37°C for 24 hours.

- **Total Bacterial Count:** Cultured on plate count agar (PCA) and incubated aerobically at 37°C for 24 hours.

After incubation, bacterial colonies were counted on the culture plates, and the results were recorded.[21]

Statistical Analysis

The data obtained from the study were analyzed using the "IBM SPSS Statistics 29.0" software package (IBM

Corp., Armonk, New York, USA). Descriptive characteristics of the children were expressed using "mean, standard deviation, median, minimum, and maximum values" for continuous variables. The normality of the data distribution was assessed through “skewness and kurtosis values, histograms, and Q-Q plot graphs,” and it was determined that the data followed a normal distribution. To examine the similarities and differences in descriptive characteristics between obese and control groups, the "Independent Sample t-Test" and the "Mann-Whitney U Test" were used for continuous variables. Relationships between quantitative variables were analyzed using Pearson or Spearman correlation analysis. Additionally, linear regression analysis was employed to evaluate the impact of some parameters on each other. A significance level of $p<0.05$ was considered statistically significant.[22]

RESULTS

The BMI and body weights of obese children were significantly higher than those of the control group ($p<0.001$). Although there were no statistically significant differences in the total bacterial count, *Bifidobacterium spp.*, and *Staphylococcus spp.* counts between the obese and control groups ($p>0.05$), these values were numerically lower in the obese group. Conversely, lactic acid bacteria, Gram-negative *Enterobacteriaceae* family, and *Enterococcus spp.* counts were numerically higher in the obese group compared to the control group, though these differences were not statistically significant ($p>0.05$) (Table 1).

Table 1. Various parameters of children in the obese and control groups.

Parameters	Control[a]	Obese[a]	Test Statistic; P-Value
Age[b]	12.24±2.62	12.16±2.82	t: 0.104 ; p:0.918
Height[c]	157.00 (130.00-178.00)	155.00 (63.00-178.00)	U: 284.500; p: 0.586
Weight[b]	49.04±15.02	83.60±21.26	t: 6.637; **p: <0.001**
BMI[b]	20.07±3.97	33.28±3.09	t: 13.134 ; **p: <0.001**
Total Bacterial Count[b]	8.78±1.06	8.63±0.94	t: 0.514 ; p: 0.610
Gram (-) Bacteria and Enterobacteriaceae[b]	6.00±0.95	6.09±0.96	t: 0.299 ; p: 0.766
Lactic Acid Bacteria[b]	6.56±1.14	6.61±1.04	t: 0.524 ; p: 0.890
Enterococcus spp. [b]	4.94±1.14	4.95±1.48	t: 0.025; p:0.980
Bifidobacterium[c]	5.66 (4.48-6.20)	5.48 (1.00-7.28)	U: 170.00 ; p: 0.999
Staphylococcus spp.[b]	3.76±1.07	3.57±1.10	t: 0.623 ; p: 0.537

a: Values are expressed on a logarithmic scale. **b:** Mean ± SEM. **c:** Median (Min-Max).
t: Independent Student T-test; **U:** Mann-Whitney U test. **SEM:** Standard Error of the Mean.

In obese children, a low-level positive correlation was observed between age and BMI (r=0.479), while a low-level negative correlation was found between age and total bacterial count (r=-0.428). A moderate positive correlation was identified between total bacterial count and *Enterococcus spp.* (r=0.558), and a high-level positive correlation was observed between lactic acid bacteria and *Enterococcus spp.* (r=0.730). Additionally, a moderate positive correlation was detected between *Enterococcus spp.* and *Bifidobacterium spp.* counts (r=0.671) (Table 2). The regression analysis demonstrates that *Enterococcus spp. count* significantly predicts total bacterial count, explaining 28% of its variation (R^2=0.311, AdjR^2=0.280, p=0.005) (Table 3). In the study, it was determined that 51% of the variation in lactic acid bacteria among obese children was attributed to *Enterococcus spp.* counts (R^2=0.533, AdjR^2=0.511, $p<0.001$) (Table 3). The regression analysis indicates that Enterococcus spp. counts explain 23.5% of the variation in Bifidobacterium spp. among obese children (AdjR^2=0.235, p=0.028) (Figure 1, Table 3).

Table 2. Correlations between parameters in obese children.

Parameters	BMI‡	Total Bacterial Count‡	Gram-negative Enterobacteriaceae‡	Lactic Acid Bacteria‡	*Enterococcus* spp. ‡	Bifidobacterium spp. #	*Staphylococcus* spp. ‡
Age	0.479*	-0.428*	-0.065	-0.329	-0.314	0.001	0.079
BMI		-0.275	0.075	-0.135	-0.155	-0.122	0.017
Total Bacterial Count			0.114	0.316	0.558**	0.079	0.364
Gram-negative *Enterobacteriaceae*				0.188	0.164	0.054	-0.077
Lactic Acid Bacteria					0.730**	0.263	-0.047
***Enterococcus* spp.**						0.671**	0.251
Bifidobacterium spp.							0.455

‡: Pearson correlation; #: Spearman correlation; *: p<0.05; **: p<0.01.

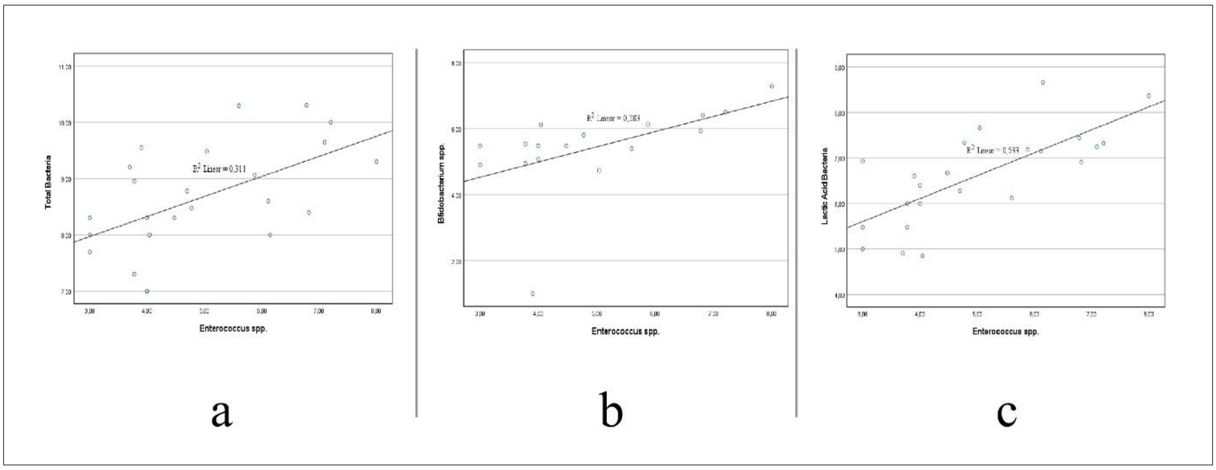

Figure 1. Graphical distribution of regression analysis for bacterial counts in obese children. a: Regression graph between total bacterial count and *Enterococcus spp.* b: Regression graph between lactic acid bacteria and *Enterococcus spp.* c: Regression graph between *Bifidobacterium spp.* and *Enterococcus spp.*

Table 3. Regression analysis results for total bacterial count in obese children.

		Total Bacterial Count			
Variables	**B**	**SE**	**β**	**t**	**p**
Constant	6.900	0.583	-	**11.828**	**<0.001**
Enterococcus spp. count	0.356	0.113	0.558	3.152	**<0.005**
R: 0.558	R^2: 0.311	AdjR^2: 0.280	F: 6.935	p: 0.005	
		Lactic Acid Bacteria			
Constant	4.089	0.536	-	**7.627**	<0.001
Enterococcus spp. count	0.504	0.103	0.730	4.895	<0.001
R: 0.730	R^2: 0.533	AdjR^2: 0.511	F: 23.960	p: <0.001	
		Bifidobacterium			
Variables	**B**	**SE**	**β**	**t**	**P**
Constant	3.142		-	**7.627**	**0.268**
Enterococcus spp. count	0.461		0.730	4.895	**0.028**
R: 0.532	R^2: 0.283	AdjR^2: 0.235	F: 5.925	p:0.028	

B: Unstandardized beta, SE: Standard error for the unstandardized beta, β: Standardized beta, t: t test statistic

In healthy children, a low-level positive correlation was observed between total bacterial count and *Gram-negative Enterobacteriaceae* count (r=0.477), as well as between lactic acid bacteria and *Enterococcus spp.* count (r=0.436). A moderate positive correlation was identified between total bacterial count and *Bifidobacterium spp.* count (r=0.586) and between *Gram-negative Enterobacteriaceae* count and *Bifidobacterium spp.* count (r=0.527). Additionally, a low-level positive correlation was found between *Enterococcus spp.* and *Staphylococcus spp.* counts (r=0.461) (Table 4, Table 5). Regression analysis demonstrated that *Bifidobacterium spp.* counts significantly predicted total bacterial count in the control group, accounting for 27.7% of the variation (*AdjR²*=0.277, *p*=0.01). Similarly, *Enterococcus spp.* counts were found to significantly predict lactic acid bacteria counts, explaining 14.8% of the variation (*AdjR²*=0.148, *p*=0.048). Furthermore, in the control group, 17.3% of the variation in *Enterococcus spp.* counts was attributed to *Staphylococcus spp.* counts (*R²*=0.212, *AdjR²*=0.173, *p*=0.031). The regression analysis demonstrates that Gram-negative *Enterobacteriaceae* counts significantly predict total bacterial count in the control group, accounting for 19.2% of the variation (*AdjR²*=0.192, *p*=0.018) (Table 5).

Table 4. Correlations between parameters in healthy children.

Parameters	**BMI**‡	**Total Bacterial**‡	**Gram-negative Enterobacteriaceae**‡	**Lactic Acid Bacteria**‡	***Enterococcus* spp.** ‡	**Bifidobacterium spp.** #	***Staphylococcus* spp.** ‡
Age	0.323	0.191	0.229	0.388	0.037	0.349	0.280
BMI		-0.065	0.107	0.177	-0.247	0.023	0.182
Total Bacteria			0.477*	0.349	0.309	0.586**	0.258
Gram-negative Enterobacteriaceae				0.371	0.125	0.527*	-0.112
Lactic Acid Bacteria					0.436*	0.319	0.356
***Enterococcus* spp.**						0.434	0.461*
Bifidobacterium spp.							0.478

‡: Pearson correlation; #: Spearman correlation; *: p<0.05; **: p<0.01.

Table 5. Regression analysis results for total bacterial count in the control group.

			***Bifidobacterium* spp.**		
Variables	**B**	**SE**	**β**	**t**	**p**
Constant	**5.543**	**1,271**	**-**	**4,360**	**<0,001**
Gram-negative Enterobacteriaceae count	0.533	0,209	0,477	2,546	**0,018**
R: 0.477	R^2: 0.228	AdjR^2: 0.192	F: 6.480	p: **0.018**	
			Total Bacteria		
Variables	**B**	**SE**	**β**	**t**	**p**
Constant	**2.645**	**2.223**	**-**	**1.190**	**0.249**
Bifidobacterium count	1.149	0.399	0.562	2.879	**0.010**
R: 0.562	R^2: 0.315	AdjR^2: 0.277	F: 8.290	p: **0.01**	
			Lactic Acid Bacteria		
Variables	**B**	**SE**	**β**	**t**	**p**
Constant	**4.334**	**1.048**	**-**	**4.133**	**<0.001**
Enterococcus spp. count	0.433	0.205	0.436	2.122	**0.048**
R: 0.436	R^2: 0.190	AdjR^2: 0.148	F: 8.290	p: **0.048**	
			Enterococcus spp.		
Variables	**B**	**SE**	**β**	**t**	**p**
Constant	**3.171**	**0.794**	**-**	**3.994**	**<0.001**
Staphylococcus spp. count	0.468	0.201	0.461	2.321	**0.031**
R: 0.461	R^2: 0.212	AdjR^2: 0.173	F: 5.388	p: **0.031**	

DISCUSSION

In this study, the gut microbiota compositions of obese and healthy children were compared, with particular attention drawn to the changes in *Enterococcus spp.*, lactic acid bacteria, and total bacterial counts in obese children. The findings contribute to a better understanding of the mechanisms associated with obesity through alterations in microbiota composition.

In obese children, the increase in total bacterial count was found to be driven by *Enterococcus spp.* (*28.0%, AdjR²: 0.280, p: 0.005*). This finding highlights the prominent role of *Enterococcus spp.* in microbiota alterations associated with obesity. Previous studies have reported that *Enterococcus spp.* may increase intestinal permeability, leading to endotoxemia and systemic inflammation.[23,24] Additionally, *Enterococcus spp.* may negatively impact energy metabolism by reducing the production of short-chain fatty acids.[25]

In obese children, the increase in lactic acid bacteria was found to be driven by *Enterococcus spp.* (*51%, AdjR²: 0.511, p<0.001*). Lactic acid bacteria are known for their anti-inflammatory effects and contributions to gut homeostasis. However, in the context of obesity, an increase in these bacteria may be indicative of dysbiosis in the gut microbiota. A study highlighted the critical role of lactic acid bacteria in immune system regulation but emphasized the complexity of their effects in obesity.[26] This complexity may be attributed to certain *Lactobacillus* species promoting dysbiosis within the gut microbiota, potentially contributing to the development of obesity.[27]

In obese children, part of the increase in *Bifidobacterium spp.* was found to be associated with *Enterococcus spp.* (*24%, AdjR²: 0.235, p: 0.028*). While *Bifidobacterium* species are generally considered indicators of a healthy microbiota, the observed increase in obese children may represent a compensatory mechanism aimed at restoring microbial balance. This aligns with evidence suggesting that *Bifidobacterium* reduces intestinal inflammation and regulates energy intake.[11] However, the metabolic effects of these bacteria in the context of obesity may vary between individuals, highlighting the complexity of their role in this condition.

In this study, a positive correlation was identified between age and BMI (r=0.479), while a negative correlation was observed between age and total bacterial count (r=-0.428). The decrease in total bacterial count with age may be interpreted as a result of gut microbiota maturation and the influence of environmental factors.[10] Additionally, in obese children, the dominance of bacteria that enhance energy intake as age increases is thought to contribute to the rise in BMI.

In healthy children, *Bifidobacterium* was found to account for 28% of the variation in total bacterial count (*AdjR²: 0.277, p: 0.01*). This finding suggests that the microbiota in healthy individuals is diversely and stably balanced. A previous study reported that *Bifidobacterium* species, which regulate energy metabolism and suppress inflammation, are more dominant in the microbiota of healthy children, aligning with the results of this study.[28]

In obese children, a numerical increase in bacteria belonging to the Gram-negative *Enterobacteriaceae* family was observed. These bacteria can contribute to systemic inflammation and insulin resistance through the production of lipopolysaccharides (LPS).[29] In healthy children, the weak correlation observed between these bacteria and total bacterial count (r=0.477) suggests that a healthy microbiota can help regulate and control the abundance of these bacteria. This finding aligns with the literature, indicating that Gram-negative *Enterobacteriaceae* may be associated with complications of obesity in children.

Recently, this study highlights the complex and multifactorial nature of the relationship between childhood obesity and gut microbiota. In particular, bacterial groups such as *Enterococcus spp.*, lactic acid bacteria, and *Bifidobacterium* were found to play significant roles in the disruption of microbial balance and the modulation of metabolic processes in the context of obesity.[28] Modulating gut microbiota composition and targeting specific bacterial groups may offer innovative approaches for the treatment and management of obesity. Future research utilizing larger sample sizes and culture-independent methods (e.g., PCR and Next-Generation Sequencing) is crucial for identifying additional bacterial species associated with obesity and for developing microbiota-based therapeutic strategies.

Conflict of Interest

The authors declare that there is not any conflict of interest regarding the publication of this manuscript.

Acknowledgements:

This study was supported by Kırşehir Ahi Evran University Scientific Research Projects Directorate with the project number TIP.A4.21.014

Ethics Committee Permission

This study was approved by Kırşehir Ahi Evran University Clinical Research Ethics Committee (Date: 01.06.2021 and Numbered 2021-10/11).

Authors' Contributions

Concept/Design: AG, ES, SF. Data Collection and/or Processing: CÖ, SF. Data analysis and interpretation: CÖ, ES, SF, MB, AG. Literature Search: MB, CÖ, AG, ES. Drafting manuscript: MB, CÖ. Critical revision of manuscript: AG, ES, SF.

REFERENCES

1. Zsálig D, Berta A, Tóth V, et al. A Review of the Relationship between Gut Microbiome and Obesity. Appl. Sci. 2023;13(1):610.
2. Geng J, Ni Q, Sun W, Li L, Feng X. The links between gut microbiota and obesity and obesity related diseases. Biomed Pharmacother. 2022;147:112678.
3. Zhang X, Liu J, Ni Y, et al. Global Prevalence of Overweight and Obesity in Children and Adolescents: A Systematic Review and Meta-Analysis. JAMA Pediatr. 2024;178(8):800-813.
4. Demiray G, Yorulmaz F. Obesity Management with A Public Health Perspective. Value Health Sci. 2023; 13(1):147-155.
5. Tekin T, Çiçek B, Konyalıgil N. The Relatıonshıp Between Intestınal Mıcrobıota And Obesıty. J. Health Sci. 2018;27(1):95-99.
6. Türkiye İstatistik Kurumu (TÜİK). Stratejik Plan 2014–2018. Ankara: TÜİK Yayınları, Eylül 2014. Erişim adresi: https://www.tuik.gov.tr/Kurumsal/Stratejik_Plan. Access date 20 November, 2024.
7. TÜİK. Türkiye Sağlık Araştırması, (2022). Erişim Adresi: https://data.tuik.gov.tr/Bulten/Index?p=Turkiye-Saglik-Arastirmasi-2022-49747. Access date 20 November, 2024.
8. Çetinbaş S, Kemeriz F, Göker G, Biçer İ, Velioğlu YS. Human Microbiome: Nutritional and Health Effects. Akademik Gıda. 2017;15(4):409-415.
9. Turnbaugh PJ, Ley RE, Mahowald MA, Magrini V, Mardis ER, Gordon JI. An obesity-associated gut microbiome with increased capacity for energy harvest. Nature. 2006;444(7122):1027-1031.
10. Yatsunenko T, Rey FE, Manary MJ, et al. Human gut microbiome viewed across age and geography. Nature. 2012;486(7402):222-227.
11. Arrieta MC, Stiemsma LT, Amenyogbe N, Brown EM, Finlay B. The intestinal microbiome in early life: health and disease. Front Immunol. 2014;5:427.
12. Rodriguez JM, Murphy K, Stanton C, et al. The composition of the gut microbiota throughout life, with an emphasis on early life. Microb Ecol Health Dis. 2015;26(1):26050.
13. Milani C, Duranti S, Bottacini F, et al. The first microbial colonizers of the human gut: composition, activities, and health implications of the infant gut microbiota. Microbiol. Mol. Biol. 2017;81(4):10-1128.
14. Reuter G. The Lactobacillus and Bifidobacterium microflora of the human intestine: composition and

succession. Curr Issues Intest Microbiol. 2001;2(2):43-53.
15. Ouwehand A. A review of dose-responses of probiotics in human studies. Beneficial Microbes. 2017;8(2): 143-151.
16. Rada V, Machova M, Huk J, Marounek M, Dušková. Microflora in the honeybee digestive tract: counts, characteristics and sensitivity to veterinary drugs. Apidologie. 1997;28(6):357-365.
17. Killer J, Dubná S, Sedláček I, Švec P. Lactobacillus apis sp. nov., from the stomach of honeybees (Apis mellifera), having an in vitro inhibitory effect on the causative agents of American and European foulbrood. Int. J. Syst. Evol. Microbiol. 2014;64(1):152-157.
18. Sharifpour MF, Mardani K, Ownagh A. Molecular identification and phylogenetic analysis of Lactobacillus and Bifidobacterium spp. isolated from gut of honeybees (Apis mellifera) from West Azerbaijan, Iran. Vet Res Forum. 2016;7(4):287-294.
19. Durmaz B. Relationship Between Obesity and Gut Microbiota. Turk Hij Den Biyol Derg. 2019;76(3): 353-360.
20. Özkarabulut AH. Beden Kitle İndeksi (BKI) ile belirlenmiş zayıf ve obez bireylerde barsak mikrobiyota profilinin karşılaştırılması. 2020.
21. Sepp E, Loivukene K, Julge K, Voor T, Mikelsaar M. The association of gut microbiota with body weight and body mass index in preschool children of Estonia. Microb Ecol Health Dis. 2013;24(1):19231.
22. Ignacio A, Fernandes MR, Rodrigues VA, et al. Correlation between body mass index and faecal microbiota from children. Clin Microbiol Infect. 2016;22(3): 1-258.
23. Ridaura VK, Faith JJ, Rey FE, et al. Gut microbiota from twins discordant for obesity modulate metabolism in mice. Science. 2013;341(6150):1241214.
24. Cani PD, Bibiloni R, Knauf C, et al. Changes in gut microbiota control metabolic endotoxemia-induced inflammation in high-fat diet–induced obesity and diabetes in mice. Diabetes. 2008;57(6):1470-1481.
25. Blaut M, Clavel T. Metabolic diversity of the intestinal microbiota: implications for health and disease. J Nutr. 2007;137(3):751-755.
26. Heeney DD, Gareau MG, Marco ML. Intestinal Lactobacillus in health and disease, a driver or just along for the ride? Curr Opin Biotechnol. 2018;49:140-147.
27. Million M, Maraninchi M, Henry M, et al. Obesity-associated gut microbiota is enriched in Lactobacillus reuteri and depleted in Bifidobacterium animalis and Methanobrevibacter smithii. Int J Obes (Lond). 2012;36(6):817-825.
28. Faith JJ, Rey FE, O'Donnell D, et al. Creating and characterizing communities of human gut microbes in gnotobiotic mice. ISME J. 2010;4(9):1094-1098.
29. Cani PD, Possemiers S, Van de Wiele T, et al. Changes in gut microbiota control inflammation in obese mice through a mechanism involving GLP-2-driven improvement of gut permeability. Gut. 2009;58(8):1091-1103.