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Intestinal Microbiota Changes in Individuals Living with HIV and the Effect of Antiretroviral Therapy

HIV ile Yaşayan Bireylerde Bağırsak Mikrobiyotası Değişiklikleri ve Antiretroviral Tedavinin Etkisi

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Abstract

Introduction: Human immunodeficiency virus (HIV) infection is a chronic infectious disease that can be effectively managed with highly active antiretroviral therapy (HAART). HIV infection is closely associated with gastrointestinal disorders, alterations in the intestinal microbiota, and chronic inflammation. This study aimed to investigate changes in the gut microbiota of HIV-infected and uninfected individuals living in Türkiye.

Materials and Methods: The study included 15 people living with HIV (PLWH) and 10 healthy controls. PLWH were evaluated at baseline and again at least three months after treatment initiation, using a paired longitudinal study design. Blood and fecal samples were collected from all participants. Intestinal microbiota composition was analyzed using 16S ribosomal ribonucleic acid gene sequencing.

Results: Significant differences in intestinal microbiota composition were observed between PLWH and healthy controls. PLWH exhibited significantly lower alpha diversity, along with alterations in the composition of Proteobacteria and Firmicutes. Differences in the relative abundance of several genera, including *Faecalibacterium, Prevotella,* and *Lactobacillus*, were also observed compared with healthy controls. Following HAART, partial changes in microbiota composition were noted, with *Phascolarctobacterium* and *Blautia* among the taxa showing differences relative to the pretreatment period.

Conclusion: HAART was associated with partial restoration of the gut microbiota composition in PLWH. However, microbiota alterations persisted despite treatment.

Keywords: Microbiota, human immunodeficiency virus, highly active antiretroviral therapy, dysbiosis

Öz

Giriş: İnsan immün yetmezlik virüsü (HIV) enfeksiyonu, yüksek etkili antiretroviral tedavi (HAART) ile etkin şekilde kontrol altına alınabilen kronik bir enfeksiyon hastalığıdır. HIV enfeksiyonu gastrointestinal hastalıklar, bağırsak mikrobiyotası değişiklikleri ve kronik enflamasyon ile yakından ilişkilidir. Bu çalışmada, Türkiye'de yaşayan HIV enfeksiyonu olan ve olmayan bireylerde bağırsak mikrobiyotasındaki değişikliklerin araştırılması amaçlanmıştır.

Gereç ve Yöntem: Çalışmaya 15 HIV ile yaşayan birey ve 10 sağlıklı gönüllü dahil edildi. HIV ile yaşayan bireyler başlangıçta ve tedavi başlandıktan en az üç ay sonra tekrar değerlendirildi. Tedavi öncesi ve sonrası örnekler aynı bireylerden alınmış olup eşleştirilmiş boylamsal bir tasarım benimsenmiştir. Tüm katılımcılardan kan ve dışkı örnekleri alındı. Bağırsak mikrobiyotası analizi için 16S ribozomal ribonükleik asit gen dizilemesi yöntemi kullanıldı.

Bulgular: HIV ile yaşayan bireyler ile sağlıklı kontroller arasında bağırsak mikrobiyotası açısından anlamlı farklılıklar saptandı. HIV ile yaşayan bireylerde alfa çeşitlilikte anlamlı azalma gözlenirken; Proteobacteria düzeylerinde değişimler ve Firmicutes kompozisyonunda farklılıklar saptandı.

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Ayrıca, *Faecalibacterium*, *Prevotella* ve *Lactobacillus* cinslerinin göreceli bolluğunda farklılıklar tespit edildi. HAART tedavisi sonrasında, tedavi öncesi döneme kıyasla *Phascolarctobacterium* ve *Blautia* cinslerinde farklılıklar ile karakterize kısmi mikrobiyota değişimleri gözlendi.

Sonuç: HAART tedavisi, HIV ile yaşayan bireylerde bağırsak mikrobiyotasının kısmen düzelmesi ile ilişkilidir. Bununla birlikte, tedaviye rağmen mikrobiyota değişikliklerinin tamamen normale dönmediği görülmüştür.

Anahtar Kelimeler: Mikrobiyota, insan immün yetmezlik virüsü, yüksek etkili antiretroviral tedavi, disbiyozis

Introduction

The gut microbiota is thought to play an important role in maintaining host immune homeostasis. Recent studies have shown that alterations in microbial diversity are associated with numerous health conditions, including inflammatory bowel disease, cardiovascular diseases, and metabolic disorders[1,2]. Human immunodeficiency virus (HIV) has evolved from a fatal disease into a chronic infectious disease that can be effectively managed with highly active antiretroviral therapy (HAART). However, despite HAART, persistent inflammation in people living with HIV (PLWH) has remained an important determinant of morbidity and mortality[3,4]. Despite viral load suppression and an increase in CD4+ T-lymphocyte counts, the literature indicates that chronic inflammation persists because of immune damage in the mucosal tissue[5].

The gut is one of the primary immunologic organs, harboring approximately 70% of the body's immune cells. HIV disrupts the integrity of the epithelial barrier by damaging the gut-associated lymphoid tissue during the early stages of infection, leading to the translocation of microorganisms and microbial products into the circulation. This condition, referred to as a "leaky gut," is considered one of the major causes of HIV-related systemic inflammation[6,7]. In particular, the loss of Th17 cells and the resulting impairment of barrier integrity exacerbate chronic inflammation by facilitating the translocation of microbial products such as lipopolysaccharides[8]. These immunologic changes associated with HIV directly affect the composition and distribution of the gut microbiota. Numerous studies have reported reduced bacterial diversity, particularly changes in the *Prevotella/Bacteroides* ratio, and an increase in facultative anaerobes belonging to the Proteobacteria phylum in PLWH[9,10]. These alterations are thought to trigger inflammatory responses and contribute to metabolic complications. For example, a reduction in *Bacteroides* species has been suggested to impair immune regulation[11].

In patients with HIV, despite virologic suppression achieved with antiretroviral therapy, these alterations in the gut microbiota have been reported to persist and contribute to ongoing inflammatory processes. The persistence of dysbiosis in the gut microbial ecosystem of HIV-positive individuals receiving HAART is considered one of the major contributors to immune activation and chronic inflammation[12,13]. In this context, inflammation in PLWH is thought to result not only from viral replication but also from microbial translocation and disruptions in the gut microbiota. Findings reported in the literature suggest that these alterations in the gut microbiota may play a central role in HIV pathogenesis and the persistence of inflammatory responses[14,15].

The aim of this study was to investigate changes in the intestinal microbiota and the effects of antiretroviral therapy on these changes in PLWH and to determine the relationship between microbiota composition and HIV ribonucleic acid (RNA) levels and CD4+/CD8+ lymphocyte counts, which are markers of inflammation.

Material and Methods

Case Selection

After applying the inclusion and exclusion criteria (Supplementary Material), a total of 25 participants were enrolled between December 2021 and May 2022 from the outpatient clinics of a tertiary care university hospital. The study comprised three groups: 10 healthy controls, 15 treatment-naive PLWH at baseline (untreated), and the same 15 PLWH re-evaluated at their 3 months follow-up visit after initiating antiretroviral therapy, representing a paired longitudinal design. The treated and untreated samples were therefore obtained from the same 15 individuals before and after HAART initiation, and this paired design was accounted for in the statistical analyses. The clinical characteristics of the participants are presented in Table 1.

The study population resided in a southeastern region of Türkiye with distinct socioeconomic and lifestyle characteristics. Although dietary habits were not formally assessed, these environmental factors may have influenced gut microbiota composition. Written informed consent was obtained from all participants. The study protocol was approved by the Dicle University Non-Interventional Research Ethics Committee (approval number: 251, dated: 22.04.2021).

Sample Collection

Two tubes of blood (approximately 5 mL each) were collected from PLWH at baseline and again from the same participants at the 3 months follow-up visit, whereas one tube of blood

(approximately 2.5 mL) was collected from healthy controls at a single time point. Fresh stool samples (approximately 5 g) were collected from all participants and transported to the laboratory under controlled conditions at 4 °C. Stool samples were mixed with phosphate-buffered saline containing 20% glycerol, frozen at −80 °C under nitrogen, and stored until analysis.

Measurement Methods

Bacterial genomic deoxyribonucleic acid (DNA) was isolated from fecal samples using commercially available kits with enzymatic lysis (lysozyme and achromopeptidase). Sample quality was confirmed by gel electrophoresis, spectrophotometry, and fluorometric methods. DNA samples for polymerase chain reaction (PCR) analysis were stored at −20 °C. The V3–V4 region of the 16S ribosomal RNA (rRNA) gene was amplified by amplicon PCR using universal primers. The resulting products were purified, and DNA concentrations were measured using fluorometric methods. The purified DNA libraries were pooled and sequenced using the Illumina MiSeq system. Taxonomic classification was performed using the SILVA 138 reference database, and sequences were denoised using the DADA2 plugin within QIIME2. Chimera removal was performed during the denoising step. Samples with fewer than 10,000 reads were excluded from downstream analyses. Data were normalized by rarefaction to the minimum sample depth before diversity analyses.

Statistical Analysis

Demographic and clinical data were evaluated based on variables including age, sex, HIV RNA levels, CD4+ and CD8+ lymphocyte counts, and antiretroviral therapy use. Mean, median, standard deviation, minimum, and maximum values were calculated for numerical variables. Frequency and percentage distributions were reported for categorical variables. Paired comparisons between untreated and treated samples were performed using the Wilcoxon signed-rank test or paired Student's t-test, as appropriate, based on the within-subject longitudinal study design. Comparisons between independent groups were performed using the Mann–Whitney U test or Student's t-test, as appropriate. The chi-square test or Fisher's exact test was used for categorical variables. Various bioinformatics methods were applied to analyze microbial diversity and composition. The Shannon, Simpson, and Chao1 diversity indices were calculated to assess microbial alpha diversity. Principal coordinates analysis (PCoA) and principal component analysis (PCA) were used for beta diversity analyses to visualize similarities and differences among the groups. Beta diversity was assessed using Bray–Curtis and Jaccard distance metrics. Krona plots, relative abundance analysis, and taxonomic assignment analyses were performed to characterize the microbial community composition. Differences in beta diversity between groups were statistically evaluated using PERMANOVA (999 permutations) based on Bray–Curtis and Jaccard distance metrics, and the results are reported as R^2 values with corresponding p values. Linear discriminant analysis effect size (LEfSe) analysis was used to identify taxa that discriminated between groups based on linear discriminant analysis (LDA) scores. All statistical analyses were performed using QIIME2, R, and SPSS version 26, and a p-value <0.05 was considered statistically significant.

Table 1. Demographic and clinical characteristics of the study groups.

Clinical data	Healthy controls (n = 10)	Untreated (n = 15)	Treated (n = 15)	p-value
Age (years)	32.3 ± 8.9	35.4 ± 9.1	35.7 ± 9.1	0.456
Gender (male/female)	8/2	13/2	13/2	0.376[a]
BMI (kg/m^2)	24.8 ± 2.6	25.1 ± 3.1	25.1 ± 3.1	0.346
Smoking	3 (30%)	5 (33.3%)	5 (33.3%)	0.247[a]
Alcohol consumption	2 (20%)	26.7%	26.7%	0.534[a]
Transmission route				N/A
Heterosexual	-	12 (80%)	12 (80%)	
Homosexual	-	2 (13.3%)	2 (13.3%)	
IV drug use	-	6.7%	1 (6.6%)	
HIV RNA (copies/mL)	-	588,754 ± 1,124,846	578 ± 1,418	p < 0.01
CD4+ T-cell (cells/µL)	-	359 ± 158	655 ± 296	p < 0.001
CD8+ T-cell (cells/µL)	-	1001 ± 179	882 ± 116	p < 0.001
CD4/CD8 ratio	-	0.36 ± 0.14	0.76 ± 0.29	p < 0.001

Data are presented as mean ± SD or n (%).

[a]χ^2 or Fisher's exact test (untreated vs. treated HIV patients).

SD, standard deviation; HIV, human immunodeficiency virus; BMI, body mass index; RNA, ribonucleic acid.

PCR Amplification and Illumina Sequencing

Variable regions of the 16S rRNA gene were amplified by PCR to determine the composition of the intestinal microbiota. In this study, the V3–V4 region of the 16S rRNA gene was targeted to achieve high-resolution characterization of bacterial diversity. Commonly used primer pairs were selected for PCR amplification: forward primer 341F (CCTACGGGNGGCWGCAG) and reverse primer 805R (GACTACHVGGGTATCTAATCC). PCR products were prepared into libraries according to the manufacturer's instructions (Illumina, San Diego, CA, USA) and sequenced on the Illumina MiSeq platform using paired-end sequencing with a read length of 2 × 300 bp. The raw sequences underwent quality filtering, trimming, and chimera removal before downstream analysis and were subsequently processed using QIIME2 (Quantitative Insights Into Microbial Ecology, version 2022.2).

Results

Demographic Characteristics

A total of 15 PLWH and 10 healthy controls were included in the study. Among the participants, 84% (n = 21) were male and 16% (n = 4) were female. The mean age was 35.4 ± 9.1 years, with an age range of 21–50 years. Patients received bictegravir/emtricitabine/tenofovir alafenamide, dolutegravir/emtricitabine/tenofovir disoproxil fumarate, or dolutegravir/lamivudine regimens. Treatment adherence was 100%, and no long-term drug-related adverse effects were observed. Untreated PLWH had significantly higher viral loads than the HAART-treated group, in which complete virologic suppression was achieved in all patients ($p < 0.01$). HAART significantly increased $CD4^+$ T-cell counts and improved the CD4/CD8 ratio ($p < 0.001$ for both), with no significant differences observed among the three HAART regimens. Demographic data are summarized in Table 1.

Fecal Bacterial Diversity in Patients with HIV

Alpha diversity measures microbial richness and evenness within a sample, whereas beta diversity assesses differences in microbial community composition between samples. The Shannon index was used to assess alpha diversity. In this study, alpha diversity was significantly lower in the HIV group than in the healthy control group ($p = 0.034$). Two ordination methods, PCA and PCoA, were used to compare beta diversity. PCoA based on Bray–Curtis dissimilarity revealed partial separation between the groups, although considerable overlap was observed. PERMANOVA did not demonstrate a statistically significant difference in overall microbial community composition according to HIV status ($R^2 = 0.054$, $p = 0.35$) or antiretroviral treatment ($R^2 = 0.131$, $p = 0.055$), suggesting that community-level differences may be subtle and that the current sample size may have limited the statistical power to detect them. A comparative analysis of the three groups is shown in Figures 1 and 2.

Fecal Bacterial Composition in Patients with HIV

LEfSe analysis was used to identify the taxa that best discriminated between groups. LEfSe identified *Clostridium* as the taxon with the highest discriminatory score in the healthy control group, whereas *Veillonellaceae*, *Phascolarctobacterium*, *Alphaproteobacteria*, and *Spirochaetes* showed the highest LDA scores in the untreated HIV group. These findings reflect the taxa that best discriminated between groups and should not be interpreted as direct measures of directional changes in abundance. The genus-level composition and LEfSe results are presented in Figures 3 and 4.

We compared microbial community composition between patients with HIV and healthy controls. *Bacteroidetes*, *Firmicutes*, and *Proteobacteria* constituted the majority of the phylum-level microbiota. At the phylum level, the relative abundance of *Bacteroidetes* appeared higher in untreated PLWH than in healthy controls and treated patients, whereas *Firmicutes* exhibited the opposite pattern, with lower representation in the untreated group. The phylum-level composition is presented in Figure 5.

Microbiota Changes Before and After Treatment

A total of 15 patients with HIV were re-sampled and evaluated at least 3 months after HAART initiation. This group was

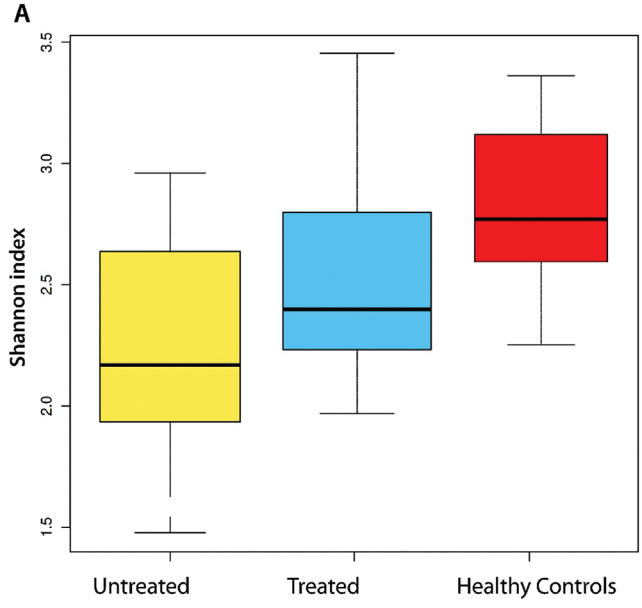

Figure 1. Alpha diversity of the gut microbiota in the study groups. Box-and-whisker plots showing the Shannon index in untreated HIV patients, treated HIV patients, and healthy controls.

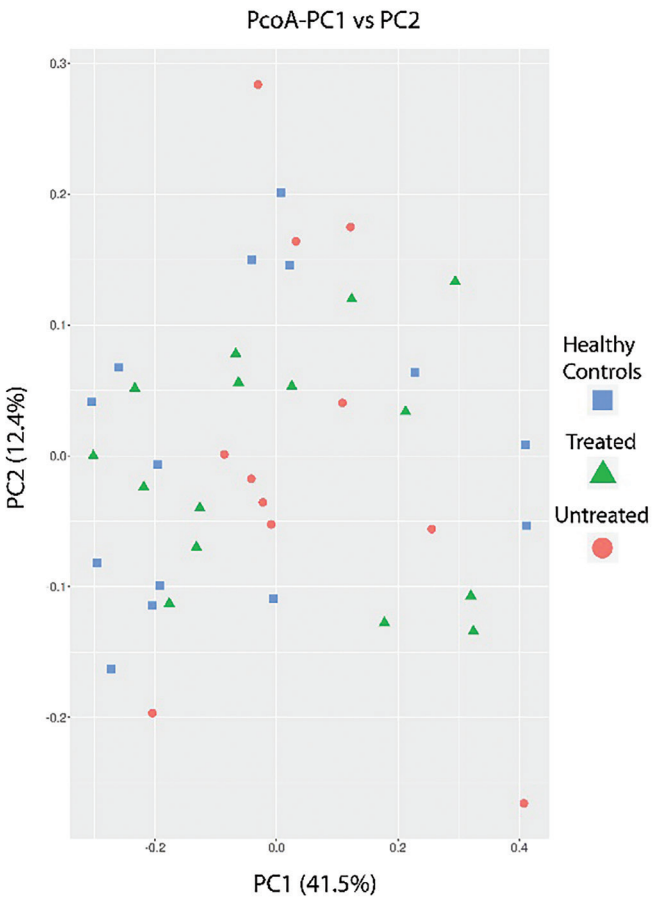

Figure 2. Beta diversity of the gut microbiota in the study groups. PCoA based on Bray–Curtis dissimilarity (PC1 vs. PC2). PC1 explained 41.5% and PC2 explained 12.4% of the total variance. PERMANOVA did not reveal statistically significant differences between groups according to HIV status (R2 = 0.054, p = 0.35) or antiretroviral treatment (R2 = 0.131, p = 0.055). PCoA, principal coordinates analysis.

compared with healthy controls and the treatment-naive HIV group. Posttreatment alpha diversity showed an increasing trend compared with the treatment-naive group, although this difference did not reach statistical significance, likely owing to the limited sample size. Changes were particularly observed in taxa such as *Firmicutes* and *Elusimicrobia*. At the taxonomic level, *Ruminococcaceae* was among the taxa showing differences between the groups. LEfSe analysis further identified *Ruminococcaceae*, *Lachnospiraceae*, and *Oscillospira* as the taxa with the highest discriminatory scores in the treated group compared with untreated PLWH, suggesting compositional shifts following HAART initiation. Despite effective HAART, the fecal microbiota did not appear to fully return to a profile comparable to that of healthy controls. Details are shown in Figures 3 and 5.

Discussion

To our knowledge, this is one of the first studies in Türkiye to compare the intestinal microbiota of PLWH and healthy controls. This study showed that microbial diversity was reduced in the HIV group. At the same time, although diversity partially improved following the initiation of HAART, the improvement appeared to be incomplete.

Thanks to HAART, HIV infection is now considered a manageable chronic disease, and the life expectancy of PLWH is approaching that of the general population. However, quality of life is influenced by many factors, including age, sex, socioeconomic status, $CD4^+$ T-lymphocyte count, and HIV RNA level[16,17]. In recent years, chronic inflammation has been recognized as one of the most important determinants of morbidity and mortality. In this process, the gut microbiota plays a critical role. HIV-induced immune activation and inflammation accelerate immune aging, leading to the earlier onset of comorbidities. Damage to the intestinal epithelial barrier, especially during the early stage of infection, increases intestinal permeability, promotes microbial translocation, and contributes to disease progression by exacerbating systemic inflammation[18,19].

Many studies have reported that alpha diversity decreases in treatment-naive PLWH. Some studies have also shown an increase in microbial diversity after HAART. In our study, a decrease in alpha diversity was observed, consistent with previous reports, and partial improvement was observed after treatment. Unexpectedly, some taxa generally considered beneficial were less represented across all groups. One possible explanation for this observation is the regional dietary and environmental context of our study population; however, because dietary intake was not formally assessed, this interpretation remains speculative and warrants further investigation. In addition, LEfSe analysis was used to identify taxa that discriminated between groups; however, these findings do not directly indicate the magnitude or direction of changes in taxonomic abundance and should therefore be interpreted cautiously[20].

In our study, changes in the intestinal microbiota of PLWH before and after treatment were evaluated, and the effects of treatment on microbial diversity and composition were analyzed. Changes in the relative representation of Firmicutes were observed after treatment, suggesting that the effects of antiretroviral therapy on the gut microbiota may be limited. Previous studies have suggested that dysbiosis of the gut microbiota in PLWH is associated with changes in Actinobacteria and that an increase in this group may be linked to proinflammatory responses[21,22]. These findings suggest that

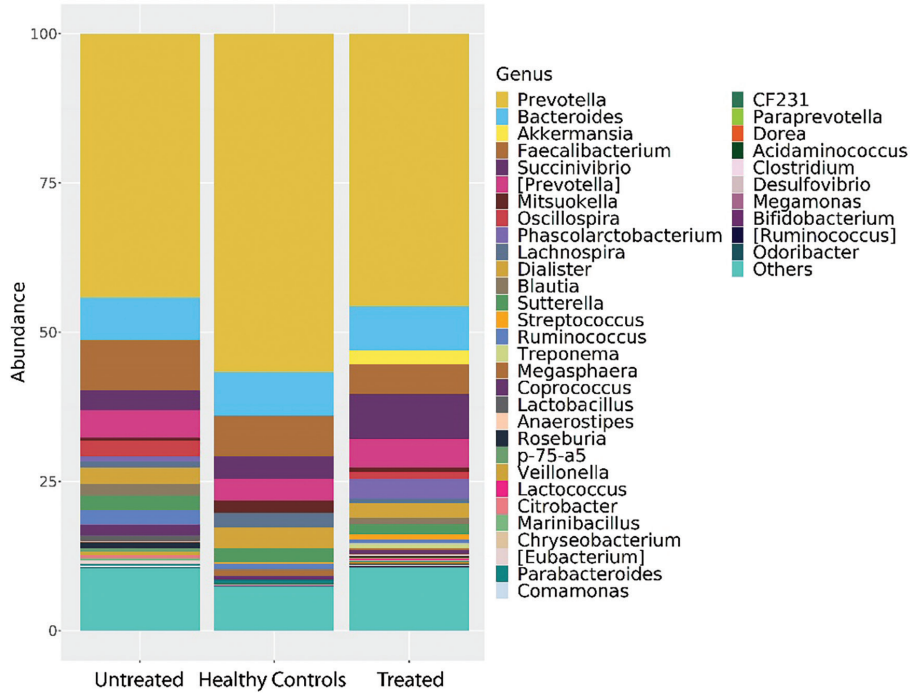

Figure 3. Relative abundance of the gut microbiota at the genus level.

Stacked bar plot showing the most abundant genera in untreated HIV patients, healthy controls, and treated HIV patients.

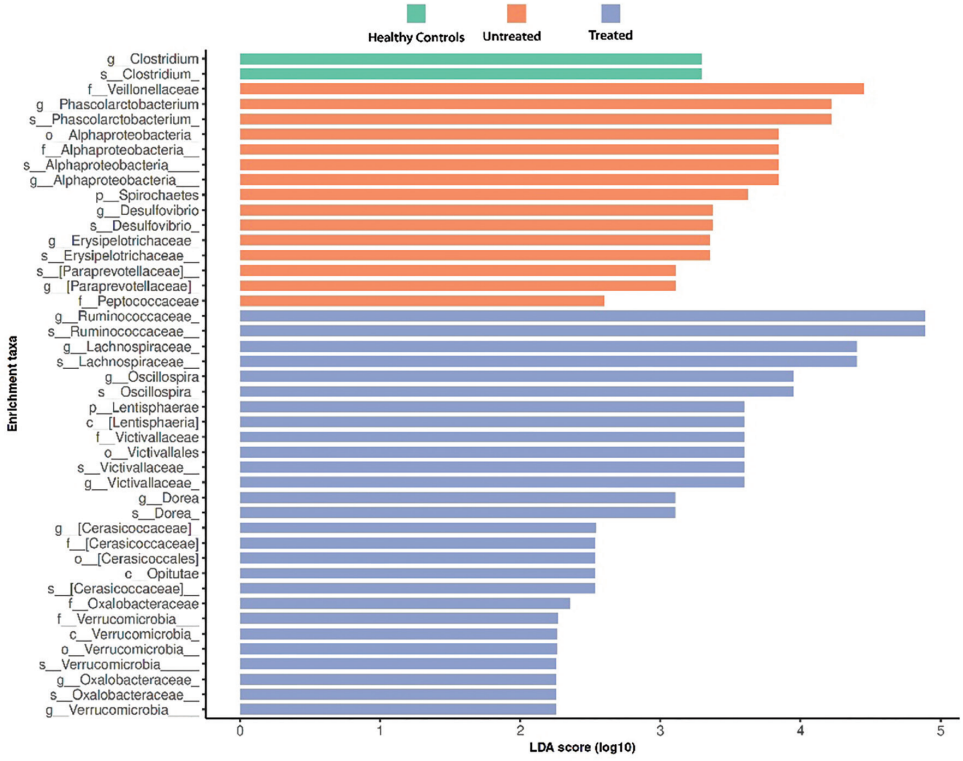

Figure 4. Differentially abundant taxa identified by LEfSe. Linear discriminant analysis (LDA) scores (>2.0, log10). The taxa shown exhibited the highest discriminatory power between groups based on LDA scores; however, these findings do not necessarily reflect the direction or magnitude of differences in abundance.

LEfSe, linear discriminant analysis effect size.

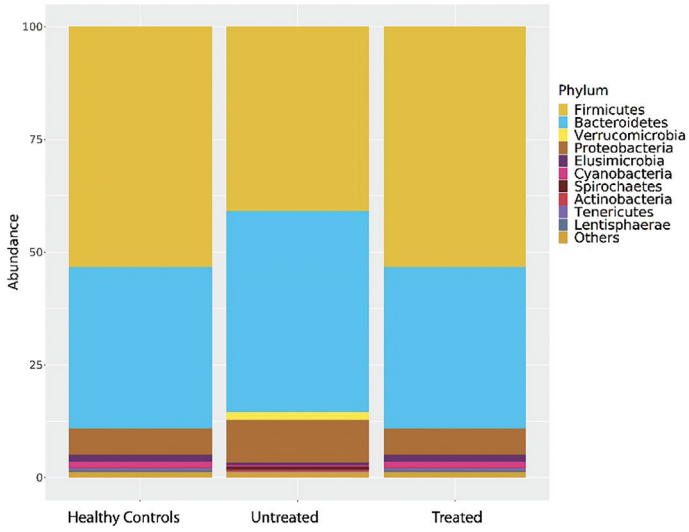

Figure 5. Relative abundance of the major bacterial phyla. Stacked bar plot showing phylum-level composition in healthy controls, untreated HIV patients, and treated HIV patients.

although antiretroviral therapy may have a partially beneficial effect on gut microbiota composition, complete restoration within a short follow-up period appears unlikely. Future long-term follow-up studies will be important for understanding how antiretroviral therapy promotes recovery of the gut microbiota and how different treatment regimens affect the microbiota[23].

Study Limitations

Several limitations of this study warrant consideration. The relatively small sample size is the primary limitation, as it restricts both the statistical power and the generalizability of the findings. This limitation is particularly relevant in microbiome studies, where substantial interindividual variability typically requires larger cohorts to detect significant differences. Consistent with this, PERMANOVA analysis did not reveal statistically significant differences in beta diversity according to HIV status ($R^2 = 0.054$, $p = 0.35$), which likely reflects insufficient statistical power rather than the true absence of community-level differences. The single-center design may also limit the representativeness of the findings, as the study population was drawn from a specific socioeconomic and geographic setting. The three-month follow-up period, although sufficient to assess early changes in the microbiota following HAART initiation, does not permit conclusions regarding long-term microbiota recovery. Potential confounding factors, including dietary habits, lifestyle, and environmental exposures, were not systematically assessed, and their contribution to the observed microbiota patterns cannot be excluded. In addition, data on HIV transmission routes were insufficient to evaluate route-specific differences in the microbiota. These limitations should be carefully considered when interpreting the findings, and larger multicenter studies with longer follow-up periods are warranted.

Conclusion

In conclusion, HIV infection is associated with alterations in the gut microbiota, including reduced microbial diversity, which may contribute to inflammation, immune dysregulation, and disease progression. Although antiretroviral therapy can partially correct dysbiosis, the microbial structure and function may not be fully restored. Therefore, the gut microbiota may play an important role not only in immune regulation but also in metabolic processes and long-term health outcomes. Future long-term, multicenter studies, particularly those involving regional cohorts with distinct environmental characteristics, are needed to better understand gut microbiota dynamics in PLWH and to evaluate whether microbiota-targeted interventions may provide additional benefits for this population.

Ethics

Ethics Committee Approval: The study protocol was approved by the Dicle University Non-Interventional Research Ethics Committee (approval number: 251, dated: 22.04.2021).

Informed Consent: Written informed consent was obtained from all participants.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Y.D., Concept: M.K.Ç., Design: Y.D., Data Collection or Processing: Y.D., Analysis or Interpretation: Y.D., E.S.T., Literature Search: Y.D., Writing: Y.D.

Conflict of Interest: No conflict of interest was declared by the authors.

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Supplementary Material: https://d2v96fxpocvxx.cloudfront.net/a7b689d1-7b5b-481e-8a29-9bd45e97266c/content-images/f3e65afd-bf2d-4bf7-bc2b-4b75a78c1890.pdf

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