

Neonatal Meconium Microbiota and Its Possible Association with Allergy in Two-Year Follow-up: A Pilot Study

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Abstract

This study aimed to investigate microbiota diversity in the first faecal (meconium) samples of newborns and to examine their possible relationship with the development of allergy over time. Considering the effects of the microbiota on immune tolerance development, the gut-brain axis, and systemic immunity, it was evaluated whether early differences in diversity and composition would be determinants of allergy risk. To overcome the limitations of traditional culturing methods, Next-Generation Sequencing (NGS) is a high-throughput method used in microbiota research. This technology enables the elucidation of the complex structure of the intestinal ecosystem and the presence of difficult-to-culture microorganisms. In our study, the composition, diversity, and structure of the human intestinal microbiota were examined in detail by metagenome analysis of the V3-V4 regions of the 16S ribosomal RNA (rRNA) gene in 10 different faecal meconium samples. Bioinformatics analyses were performed using QIIME2, and alpha-diversity (Shannon, Chao1) and beta-diversity (Bray-Curtis, UniFrac) indices were calculated. The findings confirmed that the dominant phyla were Firmicutes and Bacteroidetes. In a case in which food allergy was detected after the introduction of complementary feeding, microbial diversity in meconium was lower than in other samples, and *Diaphorobacter*/*Clostridioides* ratios were higher than in other samples. Alpha diversity indices were lower in the individual who developed allergies. Beta-diversity analyses showed that the allergic individual clustered differently from the others. Our findings indicate that the gut microbiota in early life may contain essential biomarkers for the development of allergic diseases. Current literature suggests that the gut microbiota, which develops early in life, plays a critical role in immune system development, and that disruption of this balance (dysbiosis) may be an essential factor in the pathogenesis of allergic diseases. In this context, our study aims to contribute to the body of knowledge in this field by compiling current findings on how neonatal microbiota structure affects allergic predisposition.

Highlights

- -Newborn meconium microbiota contains potential biomarkers for the development of allergies later in life.
- -All the data obtained from our study includes data such as the first nutrition and microorganisms taken from the environment, as well as the microbiota passed from mother to baby.
- - It revealed a trend for lower alpha diversity (Shannon and Chao1, $p = 0.20$) and higher *Escherichia/Shigella* abundance ($p = 0.1612$) in the individual who developed allergy.

Introduction

The human gut microbiota plays a critical role not only in digestion but also in immune system development and training, immune tolerance, and the regulation of neurological processes. Microbial metabolites, such as short-chain fatty acids (SCFAs), help prevent allergic diseases by promoting the development of regulatory T cells. The human intestinal tract is a complex and dynamic ecosystem that

hosts approximately 10 times as many microorganisms as human cells, a community known as the microbiota.

Different results have been reported in studies on the first meconium samples in the literature. Kielenniva [1] reported that meconium microbiota were not associated with subsequent atopy and allergy development in a prospective cohort study of 212 infants in Finland. On the other hand, Wang [2] showed that the interaction between maternal microbiota and infant microbiota is especially decisive in the development of food allergy in his maternal-infant cohort study conducted in China. Therefore, there is heterogeneity in findings across studies conducted in different geographic contexts. Extensive reviews over the past few years show that the gut microbiota in early life plays a critical role in both immune system development and the pathogenesis of allergic diseases [3, 4]. In particular, mode of delivery, breastfeeding, antibiotic use and environmental exposures are the main factors shaping microbiota development. It is suggested that these differences may affect long-term immune system training [5]. During the first year, the microbiota is expected to mature rapidly and approach an adult-like state, but disruptions to this process may be a risk factor for allergic diseases [6, 7]. The hygiene hypothesis was first proposed by Strachan [8], who suggested that insufficient exposure to microorganisms during childhood may increase the risk of allergic diseases. More recent studies have also supported this hypothesis, showing that early loss of microbial diversity may inhibit the development of immune tolerance [9, 10]. The mode of birth also plays a critical role in this process. While beneficial species such as *Lactobacillus* and *Bifidobacterium* dominate in the microbiota of babies born with expected vaginal delivery in the early period, colonisation of skin and environmental bacteria is more pronounced in those born by cesarean section. Azad et al. [11] and Shao et al. [12] reported that the microbiota composition remains distinct for an extended period in infants born by cesarean section, which may increase the risk of future immunological diseases. Breast milk promotes the growth of beneficial bacteria, such as *Bifidobacterium*, particularly through oligosaccharides. It has been shown that potential pathogens, such as *Clostridium* and *Escherichia/Shigella*, may predominate among infants fed artificial formula. In addition, although breast milk supports beneficial bacteria through prebiotic oligosaccharides, dysbiosis is more common among infants fed formula [13, 14]. Recent cohort studies show that early-life differences in the microbiota are strongly associated with childhood allergic diseases. For example, it has been shown that the risk of atopic dermatitis increases over subsequent years among infants with low alpha-diversity in their first meconium samples [17]. Similarly, another study reported that early dysbiosis in the gut microbiota may be linked to autoimmunity and the development of type 1 diabetes [18]. Environmental factors also play a critical role in this process; factors such as urbanisation, a Western-type diet, pet exposure, and the number of siblings directly affect microbiota diversity [19, 20]. In addition, studies show that early antibiotic exposure delays microbiota maturation and increases the risk of allergy [21]. In recent intervention studies, it has been suggested that early administration of probiotics and prebiotics may modulate the immune system [22]. In light of these studies, the number of studies examining the allergy that may develop with the first stool (meconium) in our country is limited in the literature. In this context, our study contributes to the

literature as one of the first from our country to show that the microbiota at birth may be associated with allergic reactions in adulthood.

Methods

Sampling: In our study, meconium samples were taken from 10 newborns under sterile conditions within the first 48 hours after birth and stored at -80°C.

DNA isolation was performed using the PowerFecal Pro DNA Isolation Kit.

Sequencing: The V3–V4 regions of the 16S rRNA gene were amplified with specific primers and sequenced on the Illumina MiSeq/NovaSeq platforms.

Bioinformatics Analysis: Debugging was performed using the DADA2 algorithm in QIIME2, and taxonomic classification was performed using the SILVA v138.1 database. Shannon and Chao1 indices were used for alpha-diversity analysis, and Bray-Curtis, UniFrac, and PCoA methods were utilised for beta-diversity analysis.

Comparative Approach: Although the sample size was small, a different stool composition was observed in the meconium of the individual who developed an allergy in our study, suggesting that no individual would have developed an allergy.

Results

According to the information received from the patients, babies with meconium 5, 6, and 8 used formula as the first food after birth, while other babies (including baby 10) started on breast milk. All babies have formed meconium in the hours after their first feeding. Again, babies should be monitored regularly at 6, 12, 18, and 24 months, whether they have a chronic disease (diabetes, celiac, etc.) or have developed allergies. It has been reported that none of the babies have a chronic disease yet, but some food allergies develop with the transition to supplementary food in baby 10. The family continues to follow the physician's food list.

According to the laboratory findings, Firmicutes (45–63%) and Bacteroidota (25–49%) were the dominant phyla in meconium samples. Actinobacteria and Proteobacteria were detected at low rates. In the individual who developed an allergy (example 10), an imbalance in the Firmicutes/Bacteroidota ratio and an increase in the *Diaphorobacter* and *Clostridioides* genera were noted. In alpha-diversity analyses, the Shannon index ranged from 3.12 to 4.01, while the Chao1 index ranged from 405 to 512 (Fig. 1). These values were measured at the lowest level in the individual who developed an allergy. In the beta-diversity analysis, the allergic individual clustered separately from the other samples.

Box plots illustrate the differences in diversity between the samples. It is observed that indices are low among individuals who develop allergies. (Statistical comparison: Shannon $p = 0.2$, Chao1 $p = 0.2$).

It is observed that the individual shows a different clustering in the baby who develops an allergy (Fig. 2).

Firmicutes and Bacteroidota are the dominant phyla, and differences in proportions are observed between individuals (Fig. 3).

The abundance of dominant bacterial genera in each example is shown by a stacked bar chart (Fig. 4). Notably, the *Escherichia/Shigella* ratio is relatively high in individuals who develop allergies (Example 10). (Statistical comparison: *Escherichia/Shigella* $p = 0.1612$)

Abundance differences between specimens and genera were expressed by colour intensity. Distinct clusters are observed among individuals who develop allergies (Fig. 5).microbial community structure

The genus-level microbial community structure differed in terms of the relative contribution of individual taxa. In the circular phylogenetic representation, taxa with higher relative abundance were represented by proportionally weighted branches (Fig. 6).

Statistical Comparisons

- - Shannon index: $p = 0.2$
- - Chao1 index: $p = 0.2$
- - Abundance of *Escherichia/Shigella*: $p = 0.1612$

(Note: Due to the small sample size, the results should be interpreted with caution.)

Statistical evaluations, although limited by the small sample size, revealed trends in differences between individuals who developed allergies and those who did not. The allergic individual showed lower values in alpha diversity indices (Shannon and Chao1) ($p = 0.20$). This result is in line with Arrieta [3], which suggests that low diversity may increase the risk of allergic disease. In addition, the *Escherichia/Shigella* ratio was higher in the individual with allergies ($p = 0.1612$), suggesting potential dysbiosis in the gut microbiota. Although not statistically significant, these findings may indicate stronger associations when the sample size increases.

Discussion

The findings of our study suggest that differences in the diversity and composition of the neonatal meconium microbiota may serve as early biomarkers of the development of allergies in later life. Low alpha diversity and the increase in specific bacterial genera are particularly noteworthy.

Kielenniva [1] did not find such a relationship in her large-sample study and reported that meconium microbiota was not associated with atopic diseases. However, the low diversity observed in our study is consistent with the findings of Arrieta [3] and Hoskinson [4]. These studies have shown that delayed microbiota maturation in early life can increase the risk of asthma and allergies. Wang [2] emphasised that the maternal microbiota is particularly decisive and that fluctuations in Actinobacteria and

Firmicutes contribute to the development of allergies. In this context, our small-scale study provides an essential complementary data point that should be developed when evaluated alongside studies in different geographies.

The small sample size of our study and the presence of only one individual with allergies in the findings are among the limitations of our research. However, our results support the hypotheses regarding the relationship between the microbiota and allergy in early life. Reviews in the literature highlight that butyrate-producing bacteria (e.g., *Faecalibacterium prausnitzii*) are particularly important for gut health and immune tolerance. In our study, it is noteworthy that these species are found at relatively low abundance in individuals who develop allergies. In addition, the increase in species with known proinflammatory effects, such as *Ruminococcus gnavus*, in some samples may have initiated allergic processes in this patient. As emphasised in some studies, early disruptions in microbiota diversity may weaken epithelial barrier function and shift the immune response toward a Th2 profile (23).

In our study, the relationship between low alpha diversity and the development of allergies is consistent with these mechanisms. Furthermore, our genus-level analyses revealed a significant increase in *Escherichia/Shigella*, particularly among allergic individuals; this parallels prior literature linking these bacteria to mucosal inflammation and allergies. In the literature, Louis and Flint [15] emphasised the importance of butyrate-producing bacteria for epithelial integrity and immune homeostasis. In our study, the low abundance of these bacteria, combined with the low diversity observed in allergic individuals, may be clinically significant. Furthermore, Belkaid and Hand [16] have indicated that dysbiosis may trigger allergic and inflammatory responses by disrupting epithelial barrier functions. In this context, the increase in *Escherichia/Shigella* in our study may also stimulate the intestinal mucosal immune system.

Our findings are also consistent with extensive metagenomic analyses published in recent years. For example, another study [24] reported that the Firmicutes/Bacteroidota ratio in children with allergies changed, with the Clostridia class in particular decreasing. A similar trend was observed in our study. Furthermore, recent meta-analyses show that low microbial diversity increases the risk of both asthma and eczema [25]. Inadequate colonisation by beneficial bacteria, such as *Bifidobacterium* and *Lactobacillus*, in early life may adversely affect the development of immune tolerance [26]. Lee et al. [27] reported that *Escherichia coli* and related Enterobacteriaceae were more abundant in children with allergies, a finding that parallels the increase in *Escherichia/Shigella* observed in our study. These data strongly support the hypothesis that early-life microbiota composition may be a determinant of future allergic phenotypes.

In recent years, it has been demonstrated that neonatal meconium is not merely a reservoir of low-biomass bacteria, but also a metabolite pool reflecting prenatal exposures, and that it may influence the risk of allergic sensitisation through early microbiota maturation. In a cohort study of 950 infants, it was reported that as meconium metabolic richness and microbiota diversity in the first months increased, the risk of atopy at 1 year of age decreased; pathways associated with steroid, vitamin cofactor, and amino acid metabolism were particularly poorer in infants who developed atopy [28]. In our study, we

found evidence of atopy due to food allergies in patients with a low diversity index, consistent with these findings.

On the other hand, in a prospective study of 212 infants, no association was found between the bacterial composition of first-pass meconium and atopic eczema, wheezing, or cow's milk allergy through age 4 years (29). In another study of mother-infant dyads, the presence of *Holdemanella* in the mother's intestinal flora during the third trimester was associated with the absence of food allergy in the infant at 1 year of age. It has been reported that genera such as *Anaerotruncus*, *Roseburia* and *Ruminococcus*, which produce SCFA (especially butyrate) associated with a protective effect in infant flora, are periodically higher [30].

The microbiota diversity results we detected in 10 newborn samples in our study are consistent with two distinct lines of the literature. The first consists of studies emphasising the role of meconium as a metabolic niche. Petersen et al. [27] demonstrated that when the coordination between meconium metabolite richness and early-life microbiota maturation is disrupted, the risk of IgE-mediated atopy increases by 1 year of age. The second line of research argues that the bacterial composition of first-pass meconium may have limited predictive value for clinical outcomes. Kielenniva et al. [28] reported that meconium composition was not associated with future atopic findings in a cohort of 212 infants. Wang et al. [29] reported that mother-infant microbiota interactions are particularly decisive in the development of food allergy, and that SCFA-producing species may play a protective role. When interpreted alongside the low alpha diversity and increased *Escherichia/Shigella* observed in allergic individuals in our study, these findings from the literature suggest that early-life microbiota may influence allergy risk not only through bacterial composition but also through metabolic function and maternal determinants.

Conclusion

The neonatal meconium microbiota may contain biomarkers of the development of allergies in later life. Low diversity indices and increases in certain bacteria (e.g., *Diaphorobacter*) can be used to predict early allergy risk.

However, multicenter, longer-term, and larger-sample studies are needed to generalise these findings. From a clinical perspective, measures such as reducing cesarean delivery rates, promoting breastfeeding, and implementing probiotic interventions from the newborn period onward may help reduce the risk of allergies. In light of these studies, our study aims to determine whether the microbiota transmitted from mother to baby, which mainly forms after the first feeding, is associated with future allergies. In addition, statistical comparisons in our study revealed a tendency toward lower alpha diversity (Shannon and Chao1, $p = 0.20$) and higher *Escherichia/Shigella* abundance ($p = 0.1612$) in the individual who developed allergy. However, these differences did not reach significance due to the small sample size. With confirmation of these findings in larger cohorts, it is thought that meconium, the first faecal sample, may serve as a biomarker for predicting allergy risk in early life.

Declarations

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors have not declared any conflict of interest.

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Author Contribution

Ç.E.B.B. : methodology, validation, investigation, software, data curation, formal analysis, visualisation, project administration, writing original draft, and funding acquisition. T.N.K.B.: methodology, investigation, writing review and edition, project administration, validation. A.Y.: methodology, conceptualisation, resources, data curation, writing original draft, writing review and edition, data curation. All authors reviewed the manuscript.

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Data Availability

The data obtained in this study are archived in the NCBI Sequence Read Archive (SRA) under accession number PRJNA1432528.

Ethics Approval

Ethics committee approval for this study was obtained from the Kafkas University Faculty of Medicine Ethics Committee with Decision No. 434 dated 29.05.2024. Our research was conducted in accordance with the Declaration of Helsinki. Participants' parents or guardians were informed about the study, and consent was obtained.

Informed Consent Statement

A statement was not required.

Conflicts of Interest

The authors declare no conflict of interest.

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Figures

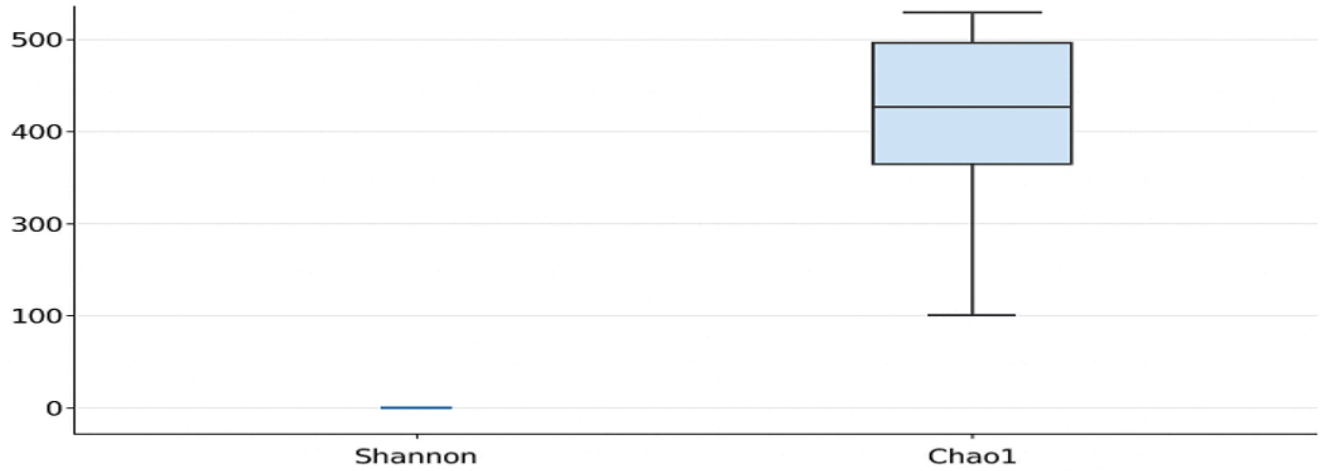


Figure 1

Alpha diversity indices (Shannon and Chao1).

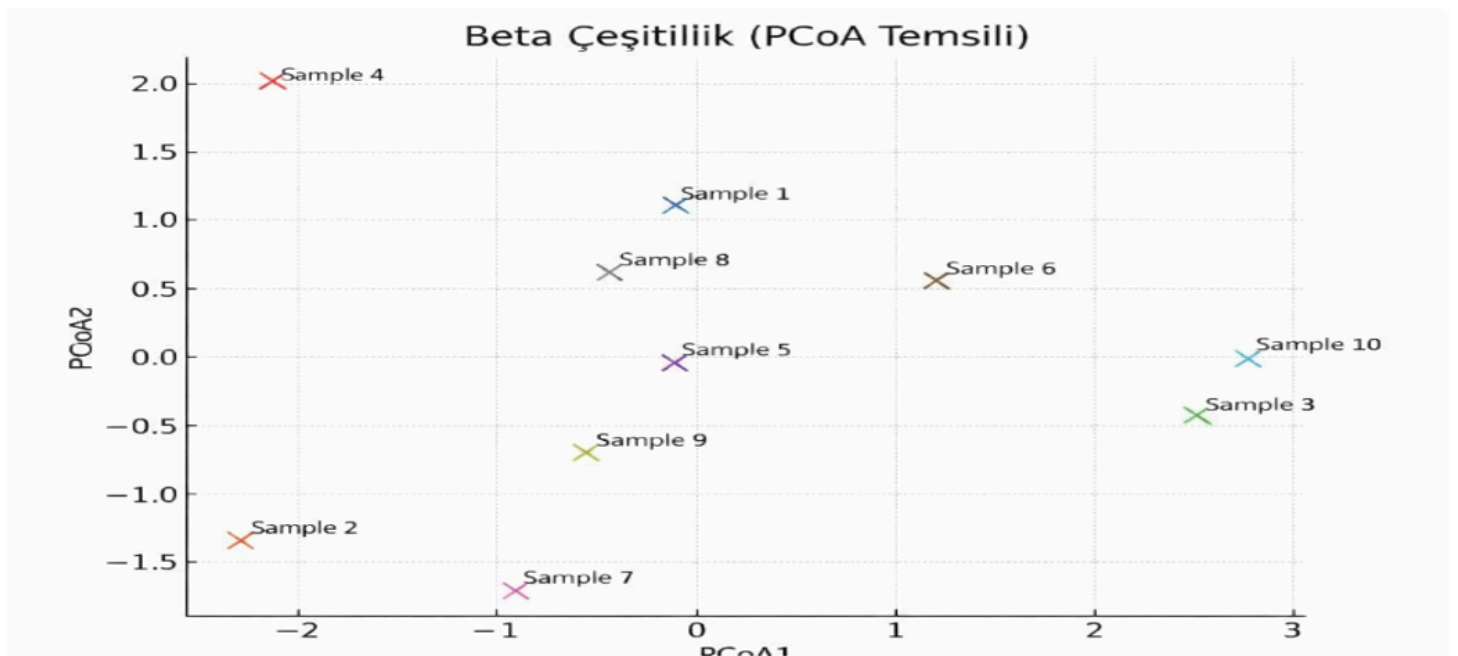


Figure 2

Beta diversity (PCoA representation).

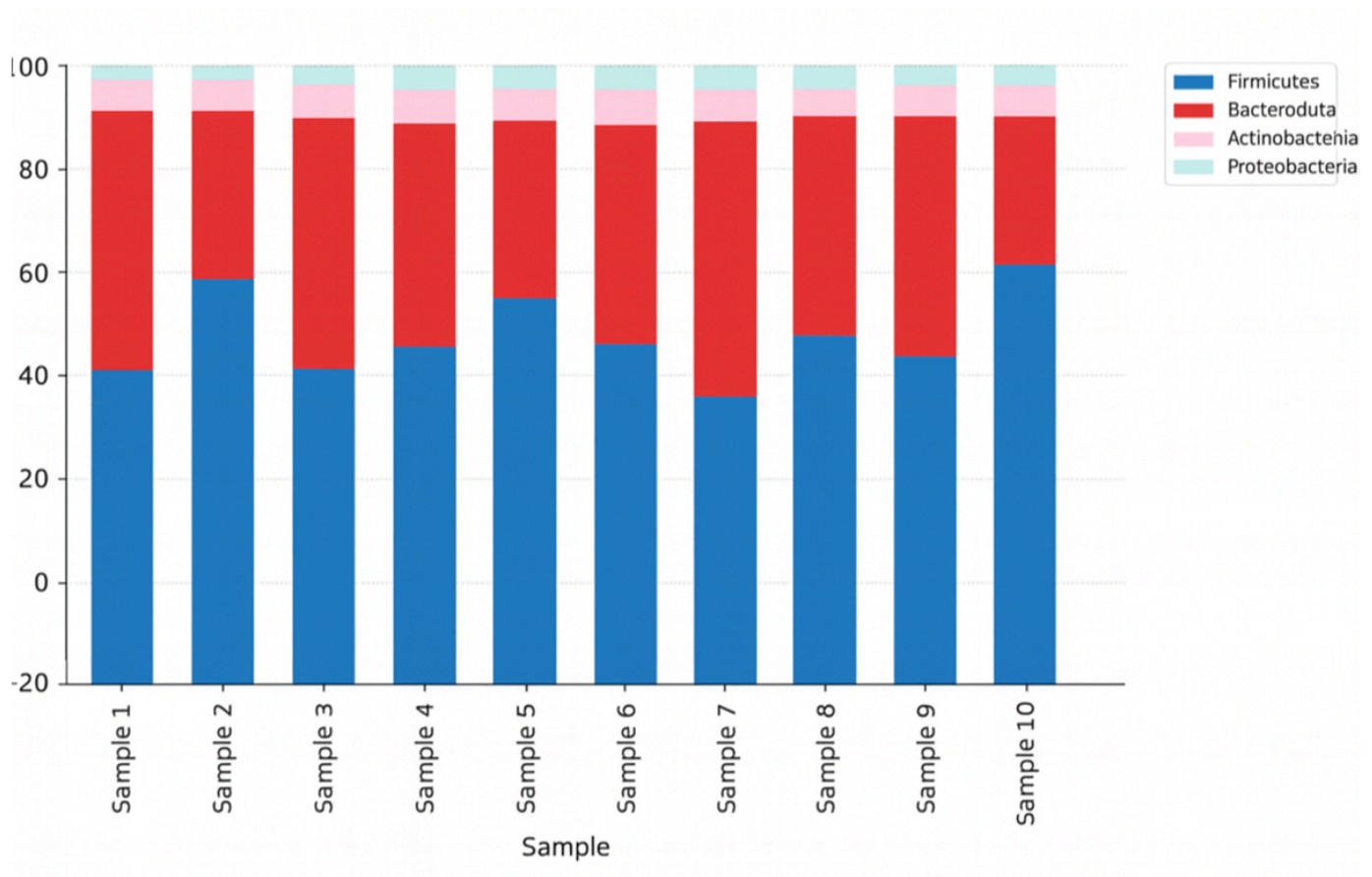


Figure 3

Phylum distribution in meconium samples.

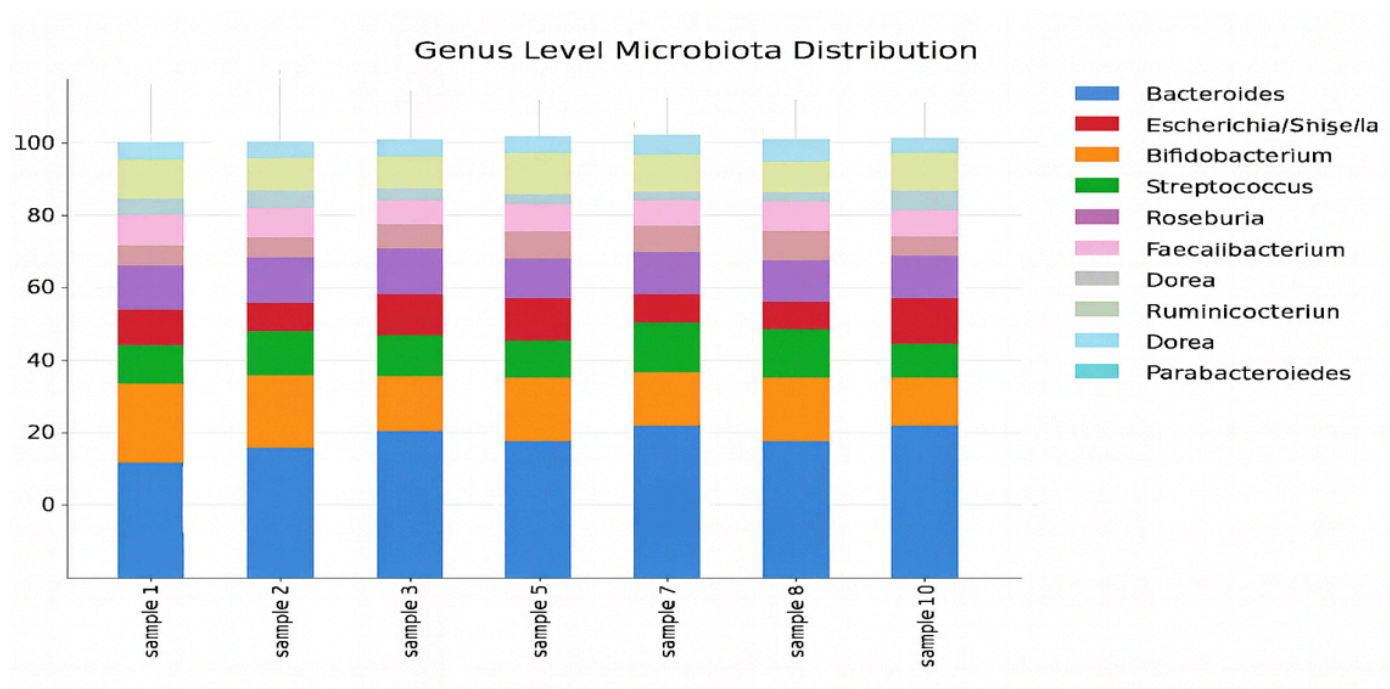


Figure 4

Microbiota distribution at the genus level.

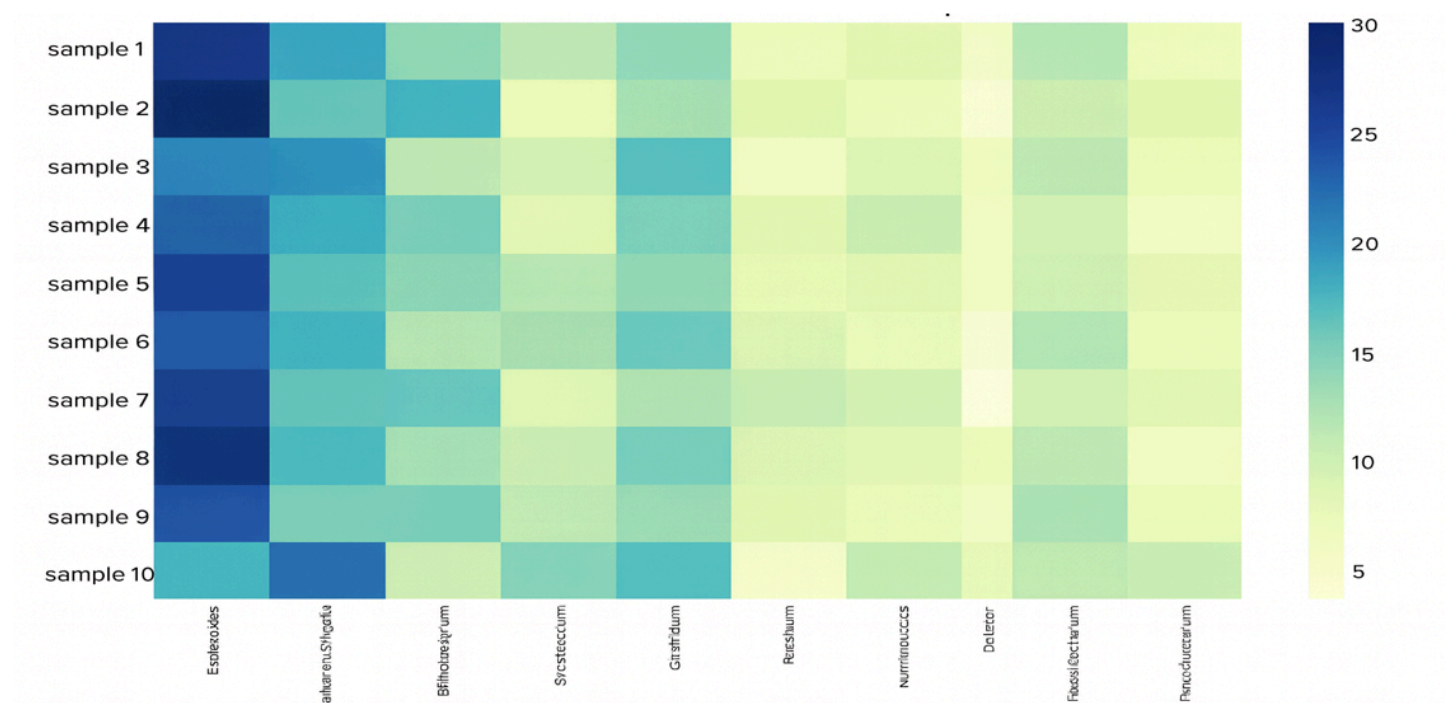


Figure 5

Microbiota distribution (heatmap) at the genus level.

