The association between polyunsaturated fatty acids in breast milk and infant eczema and its relationship with infant gut microbiota

doi: 10.6133/apjcn.202601/PP.0009
Published online: January 2026

Running title: PUFAs in breast milk and infant eczema

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ABSTRACT

Background and Objectives: Current evidence on the relationship between breast milk fatty acids and infant eczema is limited. The present study aimed to investigate the association between polyunsaturated fatty acids (PUFAs) in breast milk and the incidence of infant eczema and its relationship with infant gut microbiota. **Methods and Study Design:** Twenty-five infants diagnosed with eczema and fifty healthy infants born during the same period were recruited at 1 month postpartum. A follow-up was conducted on healthy infants at 6 months postpartum to determine if any new-onset eczema occurred. Breast milk and infant feces were collected at each interview. **Results:** In the case-control study, after adjusting for confounding factors, C20:3n-3, C20:5n-3, total n-3 PUFAs, and total n-3 PUFAs/total n-6 PUFAs in breast milk were significantly inversely associated with infant eczema risk. The abundance of *Clostridium* and *Streptococcus* in the gut of infants with eczema were significantly lower than those in control group (p <0.05). C20:3n-3 and total n-3 PUFAs in breast milk were positively correlated with *Clostridium* abundance. In the follow-up study, the decreases of C20:3n-3 and total n-3 PUFAs in the breast milk of new-onset eczema group were greater than those of healthy group at 6 months postpartum. Moreover, the abundance change of *Clostridium* in infants with new-onset eczema was significantly greater than that in healthy group. **Conclusions:** C20:3n-3 and total n-3 PUFAs in breast milk were associated with decreased risk of infant eczema, and this association may be related to the abundance of *Clostridium* in infant's gut.

Key Words: infant eczema, breast milk, fatty acids, polyunsaturated fatty acids, gut microbiota

INTRODUCTION

Eczema (atopic dermatitis) is an allergic inflammatory skin condition characterized by intense pruritus and recurrent eczematous lesions.[1,2] According to the diagnostic criteria established by the International Study of Asthma and Allergies in Childhood (ISAAC), the globally reported prevalence of infant eczema in 2021 ranged from 13.5% to 41.9%,[3] with a prevalence of 30.48% documented in China.[4] Eczema often serves as the initial clinical manifestation of allergic diseases, frequently progressing to allergic rhinitis and asthma within subsequent years,[5,6] thereby posing potential risks to the long-term health of infants. However, the underlying mechanism of infant eczema has not been fully understood.

Several studies have indicated that the gut microbiota of infants may be related to the development of infant eczema by influencing functions such as the infant immune system and sensitization.[7-11] Studies have shown that *Bifidobacterium*, *Megasphaera*, *Haemophilus*, and *Streptococcus* are more abundant in healthy infant's gut microbiota; whereas *Escherichia/Shigella*, *Veillonella*, *Faecalibacterium*, *Lachnospiraceae incertae sedis*, and *Clostridium XIva* are more abundant in infants with eczema.[12] In addition, nutrients and bioactive compounds in breast milk are also closely related to eczema, including glycoproteins, oligosaccharides, and polyunsaturated fatty acids (PUFAs).[13] Our previous study found that the dysbiosis of the infant gut microbiota is associated with changes in the levels of C18:3n-3 and total n-3 PUFAs in breast milk.[14] Therefore, PUFAs in breast milk may influence infant eczema by affecting the infant gut microbiota. To our knowledge, there was only one previous study that explored the associations of PUFAs in breast milk, the gut microbiota of infants and allergic diseases in infants. This study found that arachidonic acid in breast milk may promote the secretion of serum IgE, catalyze the production of 2-series prostaglandins and 4-series leukotrienes, leading to the dysbiosis of the infant gut microbiota and thus the onset of atopic dermatitis.[15] However, whether the association between breast milk PUFAs and the risk of infant eczema is related to infant gut microbiota is still unknown.

The present study aimed to investigate the association between PUFAs in breast milk and infant eczema and its relationship with infant gut microbiota.

MATERIALS AND METHODS

Institutional review board statement

The research received ethical approval from the Ethics Committee of the Medical College of Qingdao University (QDU-HEC-2021101), and all participants provided informed written consent prior to their involvement.

Study design and participants

Figure 1 presents the flow diagram of the study design. In the case-control study, one-month-old infants diagnosed with eczema were recruited from the Affiliated Women and Children's Hospital of Qingdao University during the period from June to December 2023, while the control group was selected at a ratio of 1:2 from infants born during the same period. Infants were excluded if they fulfilled any of the following conditions: mixed-fed or formula-fed; preterm birth; multiple pregnancies; exposure to antibiotics or probiotics. A total of 75 mother-child pairs (25 in the eczema group and 50 in the control group) were included in the

study. Subsequently, infants in the control group were followed up until 6 months of age, and infants diagnosed with eczema at six months old were classified into the new-onset eczema group, while the rest were assigned to the healthy group. The basic information was collected, including maternal age, body mass index (BMI), education, parity, annual household income and delivery mode, as well as the infants' sex, birth weight, and birth length.

Outcome definition

Infants were assessed for eczema at 1 and 6 months of age using structured questionnaires and clinical evaluations of visible lesions performed by specialist allergy physicians. The diagnosis of eczema was based on the modified UK Working Party's diagnostic criteria for atopic dermatitis,16 which require a history of pruritus (manifested as itchy skin, scratching, or rubbing) together with at least two of the following: a history of generalized dry skin, or a history of rash affecting the flexural areas, cheeks, or extensor surfaces of the limbs.

Maternal dietary intakes

One month after childbirth, a semi-quantitative food frequency questionnaire was used to collect maternal dietary intake during the past month. The food classification included 11 major categories and 28 kinds of food, such as grains, beans, meats, aquatic products, eggs, dairy products, fungi and algae, vegetables, fruits, nuts, pastries, and beverages. The parturients were asked to recall the frequency and portion size of various foods consumed in the past month with the help of a food chart (once a week, 2-3 times a week, 4-6 times a week, once a day, twice a day, and three or more times a day).[17] Maternal energy, nutrient and fatty acid intakes were calculated using the *Chinese Food Composition Table (6th Edition)*.[18]

Sample collection

Breast milk was collected at one and six months after delivery. To reduce the risk of contamination, mothers were advised not to use any topical skincare products or ointments on the breast region within 24 hours before sample collection. All samples were expressed from one breast between 9 and 11 a.m. to reduce the impact of circadian rhythms. Before expression, the nipple area was cleaned thoroughly with a sterile saline solution. Both foremilk and hindmilk were completely extracted using a clinical-grade electric breast pump (Medela, UL 2601-1). After being gently inverted to achieve uniformity, about 10 mL of the sample was transferred into sterile tubes. Infant feces samples were collected on the same day as milk collection. Infant feces (about 1 gram) were collected using sterilized diapers and

promptly transferred into sterile containers by trained personnel. All specimens were placed in a portable insulated container with cooling packs and delivered to the laboratory within two hours, then stored at −80°C until analysis.

Determination of breast milk PUFAs composition

The PUFAs composition of breast milk (% in total fatty acids) was detected by gas chromatography equipped with an Agilent HP-88 column (60 m, 0.25 mm × 0.20 μm). A detailed exposition has been provided in our previous literature.14 Briefly, lipids were extracted from breast milk using chloroform/methanol (1:1, v/v). The isolated lipids were subsequently combined with 3 mL methanol containing 0.9 mol/L H2SO4 and 1 mL toluene. The mixture was incubated at 70°C for 2 hours to facilitate the formation of fatty acid methyl esters. The temperature of the sample inlet was maintained at 260 °C. The pressures of N2 and H2 were set at 50 kPa and 75 kPa, respectively. The temperature program was as follows: 0-2 min, 125 °C; 3-28.5 min, 145 °C; and 28.6-108.5 min, 220 °C.

16S rRNA gene amplicon sequencing

In a sterile environment, total genomic DNA was extracted from infant fecal samples using the Tiangen TGuide S96 kit (with bead-beating). After Qubit quantification, the V3-V4 region of the bacterial 16S rRNA was amplified with 338F (5'-ACTCCTACGGGAAGCAG-3') / 806R (5'-GGACTACHVGGGTWTCTAAT-3'), purified using the Omega DNA purification kit (Tiangen Biotech Co., Ltd., Beijing), quantified using Qsep-400 (BiOptic, Inc., New Taipei City, Taiwan, ROC), then paired-end 250 bp sequenced on Illumina Novaseq6000 (Beijing Biomarker).[19] Strict quality control was applied and QIIME2 pipeline was used to read. Based on the quality of single nucleotides, the raw data were primarily filtered using Trimmomatic (version 0.33). The primer sequences were identified and removed through Cutadapt (version 1.9.1) to obtain high-quality clean reads. Then, the clean reads were subjected to feature classification to output ASVs (amplicon sequence variants). The counts of the original ASV data were converted to relative abundances. ASVs with relative abundances less than 0.005% were discarded.

Statistical analysis

All statistical analyses were conducted using STAMP, R version 4.4.2 and SPSS 25. Continuous variables were tested for normal distribution, and significance for group differences was assessed using an unpaired t-test or Mann-Whitney U test. Categorical

variables were analyzed using the chi-square (χ^2) test. A logistic regression model was employed to analyze the relationship between PUFAs levels in breast milk (categorized into tertiles) and infantile eczema risk. The crude model did not adjust for confounding factors; Adjusted model adjusted for the mother's age, BMI, education, parity, delivery mode, adjusted for the annual household income, parental history of allergies, use of disinfectants, presence of pets, as well as the infant's gender, birth weight, and birth length. Alpha diversity analysis was conducted to assess the abundance and diversity of microbial communities, utilizing three indices: Chao1 index, Shannon index, and PD whole tree index. Beta diversity of gut microbiota was evaluated by principal coordinates analysis (PCoA) based on unweighted UniFrac distance. The abundance of gut bacteria in the eczema group and control group was compared using Welch's t-test. The correlations between the abundances of gut microbiota and PUFAs in breast milk were evaluated using Spearman's rank correlation analysis. Paired t-test or Wilcoxon matched-pairs signed-rank test was used to analyze the changes in breast milk PUFAs and infant gut bacteria over time. $p < 0.05$ was considered statistically significant.

RESULTS

Characteristics of mothers and infants

As shown in Table 1, no significant group differences were observed in the baseline characteristics, including maternal age, BMI, education, parity, delivery mode, annual household income, parental history of allergies, use of disinfectants, presence of pets, maternal nutrient intakes (energy, protein, fat, carbohydrate, total fatty acids, total saturated fatty acids, total monounsaturated fatty acids, total PUFAs) and infants' sex, birth weight and birth length ($p > 0.05$).

Association of PUFAs in breast milk and infant eczema

In the case-control study, we found that the levels of C20:3n-3, C20:5n-3, total n-3 PUFAs and total n-3 PUFAs/total n-6 PUFAs in breast milk were all significantly lower in the eczema group compared to the control group ($p < 0.05$, Figure 2). After adjustment for multiple confounders, C20:3n-3, C20:5n-3, total n-3 PUFAs, and total n-3 PUFAs/total n-6 PUFAs in breast milk were significantly negatively associated with the risk of infant eczema at 1 month postpartum. The corresponding ORs (95% CIs) were 0.10 (0.02, 0.55), 0.19 (0.04, 0.86), 0.13 (0.03, 0.69), and 0.08 (0.01, 0.52), respectively (Figure 3).

Table 2 shows the changes in four differential PUFAs levels in breast milk during the follow-up period in both the healthy group and the new-onset eczema group. No significant changes were observed in the four differential PUFAs in the healthy group (p >0.05). In the new-onset eczema group, C20:3n-3 and total n-3 PUFAs exhibited significant reductions across the follow-up period (p <0.05). In addition, the decreases of C20:3n-3 and total n-3 PUFAs in the breast milk of new-onset eczema group were significantly greater than those of the healthy group (p <0.05), which partly verified the results of the case-control study.

Infant eczema is associated with gut microbiota dysbiosis

In the case-control study, only 10 infants in the eczema group and 30 in the control group provided fecal samples. An analysis of maternal and infant baseline characteristics according to fecal sample availability (Supplementary Table 1) showed no statistically significant differences between the two groups (p >0.05). No significant differences were observed in the Chao1, Shannon, and PD whole tree indexes between the eczema group and the control group (Figure 4). Although analysis of similarities (ANOSIM) indicated that the bacterial microflora composition did not significantly differ between the two groups ($R^2 = 0.03$, $p = 0.064$), a trend of separation was observed for the red points (eczema group) and blue points (control group). A discernible separation between the two groups was evident along PCoA axis 1 and PCoA axis 2, which explained 19.30% and 15.49% of the total variation, respectively (Figure 5).

At the phylum level, *Actinomycetota*, *Bacillota* and *Pseudomonadota* were predominant colonizers in both control group and eczema group (Figure 6A). At the genus level, *Bifidobacterium* and *Klebsiella* were predominant colonizers in both groups (Figure 6B). At the phylum level, no differential bacteria were observed (p >0.05). At the genus level, the abundance of *Clostridium* and *Streptococcus* in the eczema group was significantly lower compared to the control group (p <0.05) (Figure 7).

Figure 8 presents a comparative analysis of the changes in the abundance of differential gut microbiota between the healthy group (n = 22) and the new-onset eczema group (n = 6) during the follow-up period. The change of *Clostridium* genus abundance in the new-onset eczema group was significantly greater than that in the healthy group (p <0.01, Figure 8A). This is similar to the findings of the case-control study, suggesting that the changes in the abundance of *Clostridium* in the infant gut are associated with the occurrence of eczema. No statistically significant difference was observed in *Streptococcus* genus abundance changes between the two groups (p >0.05, Figure 8B).

Infant gut microbiota composition is associated with the breast milk PUFAs

Spearman's correlation analysis was conducted to analyze the relationship between four differential PUFAs in breast milk and two differential infant gut bacteria. Levels of C20:3n-3 and total n-3 PUFAs were found to be positively correlated with the abundance of *Clostridium* (p <0.05) (Figure 9).

In the follow-up study, there was a significant positive correlation between the change of C20:3n-3 in breast milk and the change of *Clostridium* abundance in infant's gut (p <0.01) (Figure 10).

DISCUSSION

The present study found that C20:3n-3 and total n-3 PUFAs in breast milk were negatively associated with infant eczema risk, and this association might be related to the composition of gut microbiota in infants.

Many factors have been reported to be associated with the occurrence of infant eczema, such as genetics,[16] complementary food introduction,[20] and feeding patterns.[21] In addition, the composition of breast milk is also closely related to the development of infant eczema. There were a few studies on the relationship between the composition of fatty acids in breast milk and the occurrence of infant eczema with inconsistent results.[22] In the Dutch KOALA birth cohort study, higher concentrations of main n-3 PUFAs (combined C20:5n-3, C22:5n-3 and C22:6n-3) in mature breast milk were associated with a lower incidence of allergic sensitization at one year of age and a reduced risk of eczema at two years of age.[23] Another finding from The Childhood Asthma Study (CAS) in Perth, Western Australia, indicated that infants with non-atopic eczema had lower levels of n-3 PUFAs in their mothers' milk.[24] The results of our study are similar to the above-mentioned researches. However, the Melbourne Atopic Cohort Study (MACS) in Australia indicated that elevated levels of n-3 PUFAs in colostrum were associated with an increased risk of eczema persisting for up to 18 years.[25] These conflicting results may be attributed to differences in several factors, such as geographical location of the subjects, infant age, and feeding patterns. Interestingly, we first found that C20:3n-3 in breast milk had a significant association with a decreased risk of infant eczema. C20:3n-3 is an intermediate in the C20:5n-3 metabolic pathway,[26,27] and C20:5n-3 has been indicated to be related to the alleviation of allergic diseases.[28] The lack of a statistically significant relationship between C20:5n-3 and infant eczema in our study might be due to the relatively small sample size.

The underlying mechanism of PUFAs in breast milk negatively related to infant eczema remains unclear. The association between the infant gut microbiota and allergic diseases has attracted widespread attention in recent years. A case-control study conducted in China has shown that for infants with eczema, five bacterial genera are relatively abundant in the gut, including *Escherichia/Shigella*, *Veillonella*, *Faecalibacterium*, *Lachnospiraceae incertae sedis*, and *Clostridium XIva*.[12] Several studies, including our previous research, have proved that PUFAs in breast milk can influence the composition of the infant gut microbiota.[14,29,30] Therefore, we further analyzed the composition of the intestinal microbiota in infants and found that the relative abundance of Clostridium in infants with eczema was significantly lower than that in healthy infants and it was positively associated with the levels of C20:3n-3 and total n-3 PUFAs. A research conducted in Japan showed that the abundance of *Clostridium* in allergic infants at 1 month was lower than that in non-allergic infants, which is similar to the findings of our research.[9] On the contrary, two studies conducted in the Netherlands and Finland individually have shown that allergic infants have higher abundance of *Clostridium* in the gut microbiota.[10,31] *Clostridium* is an extremely heterogeneous and diverse genus of bacteria, comprising over 100 species.[32,33] Most of them are commensals in the environment or gut rather than pathogens,[34-36] with only a few species acting as opportunistic pathogens.[37] The divergent research results might be attributed to the functional heterogeneity among different *Clostridium* species. As regarding to the associations of PUFAs in breast milk, the gut microbiota and allergic diseases in infants, only one study conducted by Jiang and associates have pointed out that high levels of C20:4n-6 in breast milk significantly increased the abundance of *Escherichia* in the infant's intestinal tract, and subsequently induced skin allergy.[15] Large-sample prospective studies or intervention experiments are needed to verify the exact relationship in the future.

The present study has several advantages. Firstly, all infants were exclusively breastfed, avoiding potential biases caused by different feeding patterns. Secondly, the results observed in the case-control study at one month postpartum were further verified in the follow-up study. Thirdly, confounding factors related to infant eczema were adjusted as much as possible, which ensured the reliability of the results. Nevertheless, there are several limitations in our study. Firstly, the sample size is relatively small. Future studies with larger sample sizes are needed to verify the current findings. Secondly, breast milk PUFAs were only assessed at two time points, which may not accurately represent their concentration throughout the entire lactation period. Thirdly, this study was a single-center research and caution should be exercised when generalizing the conclusions to other populations.

Conclusion

In conclusion, levels of C20:3n-3 and total n-3 PUFAs in breast milk are significantly negatively associated with the risk of infant eczema, and this association may be related to the abundance of *Clostridium* in the infant intestinal tract.

SUPPLEMENTARY MATERIALS

All supplementary tables and figures are available upon request from the editorial office, and are also accessible on the journal's webpage (apjcn.qdu.edu.cn).

ACKNOWLEDGEMENTS

We thank the study participants for their contribution. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

The authors declare no conflict of interest.

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Table 1. Characteristics of mothers and infants

Variables	Eczema group (n = 25)	Control group (n = 50)	*p*
Maternal characteristics			
Age, years	32.48 ± 3.63	32.48 ± 4.30	1.000
BMI, kg/m^2	23.91 ± 2.25	24.08 ± 2.81	0.801
Education, n			0.172
Associate degree or below	8 (32.0%)	9 (18.0%)	
Bachelor's degree or above	17 (68.0%)	41 (82.0%)	
Parity, n			0.324
Nullipara	12 (48.0%)	30 (60.0%)	
Multipara	13 (52.0%)	20 (40.0%)	
Delivery mode, n			0.065
Vaginal	19 (76.0%)	27 (54.0%)	
Cesarean	6 (24.0%)	23 (46.0%)	
Household characteristics			
Annual household income, n			0.585
< ¥150,000	8 (32.0%)	13 (26.0%)	
≥ ¥150,000	17 (68.0%)	37 (74.0%)	
Parental history of allergies, n			0.612
Yes	1 (4.0%)	1 (2.0%)	
No	24 (96.0%)	49 (98.0%)	
Use of disinfectants			0.509
Yes	13 (52.0%)	30 (60.0%)	
No	12 (48.0%)	20 (40.0%)	
Presence of pets			0.631
Yes	4 (16.0%)	6 (12.0%)	
No	21 (84.0%)	44 (88.0%)	
Maternal daily dietary intake intake during lactation			
Energy, kcal	1626.32 (1239.16, 2311.69)	1792.19 (1471.83, 2345.54)	0.276
Protein, g	68.01 (46.39, 80.31)	68.99 (55.20, 95.42)	0.261
Fat, g	74.59 (56.91, 91.21)	73.06 (49.65, 99.35)	0.744
Carbohydrate, g	173.87 (96.06, 244.66)	216.4 (139.05, 297.02)	0.083
Total FAs, g	71.33 (56.78, 93.45)	74.25 (45.04, 97.79)	0.711
Total SFAs, g	25.49 (17.86, 29.69)	23.80 (16.22, 30.45)	0.621
Total MUFAs, g	23.17 (17.33, 32.56)	25.52 (14.77, 34.48)	0.911
Total PUFAs, g	24.69 (17.21, 28.35)	20.40 (14.13, 29.05)	0.914
Infant characteristics			
Sex, n			0.870
Boy	14 (56.0%)	27 (54.0%)	
Girl	11 (44.0%)	23 (46.0%)	
Birth weight, g	3346.72 ± 353.23	3450.20 ± 407.82	0.283
Birth length, cm	50.00 (49.00, 50.00)	50.00 (50.00, 51.00)	0.065

BMI, body mass index; FAs, fatty acids; SFAs, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids.

Table 2. Changes in PUFAs concentrations in breast milk across the follow-up period

PUFAs (% in total fatty acids)	The first month	The sixth month	*p*	Variation in PUFAs	
	Median (IQR)			Median (IQR)	*p*
C20:3n-3					0.008
healthy group (n = 39)	1.12 (0.86, 1.38)	0.97 (0.83, 1.18)	0.056	-0.04 (-0.50, 0.09)	
new-onset eczema group (n = 8)	1.30 (0.95, 1.44)	0.51 (0.40, 0.83)	0.017	-0.71 (-0.95, -0.25)	
C20:5n-3					
healthy group (n = 39)	0.20 (0.14, 0.37)	0.24 (0.19, 0.29)	0.879	0.00 (-0.12, 0.11)	0.218
new-onset eczema group (n = 8)	0.26 (0.09, 0.33)	0.12 (0.09, 0.22)	0.123	-0.05 (-0.15, 0.02)	
Total n-3 PUFAs					
healthy group (n = 39)	2.46 (2.00, 3.00)	2.33 (2.08, 2.58)	0.163	-0.07 (-0.85, 0.31)	0.044
new-onset eczema group (n = 8)	2.76 (2.12, 3.02)	1.56 (1.31, 1.97)	0.050	-1.11 (-1.64, -0.32)	
Total n-3 PUFAs/Total n-6 PUFAs					
healthy group (n = 39)	0.11 (0.09, 0.14)	0.10 (0.08, 0.12)	0.247	-0.01 (-0.04, 0.02)	0.308
new-onset eczema group (n = 8)	0.13 (0.08, 0.13)	0.08 (0.06, 0.11)	0.161	-0.02 (-0.08, 0.01)	

PUFAs, polyunsaturated fatty acids

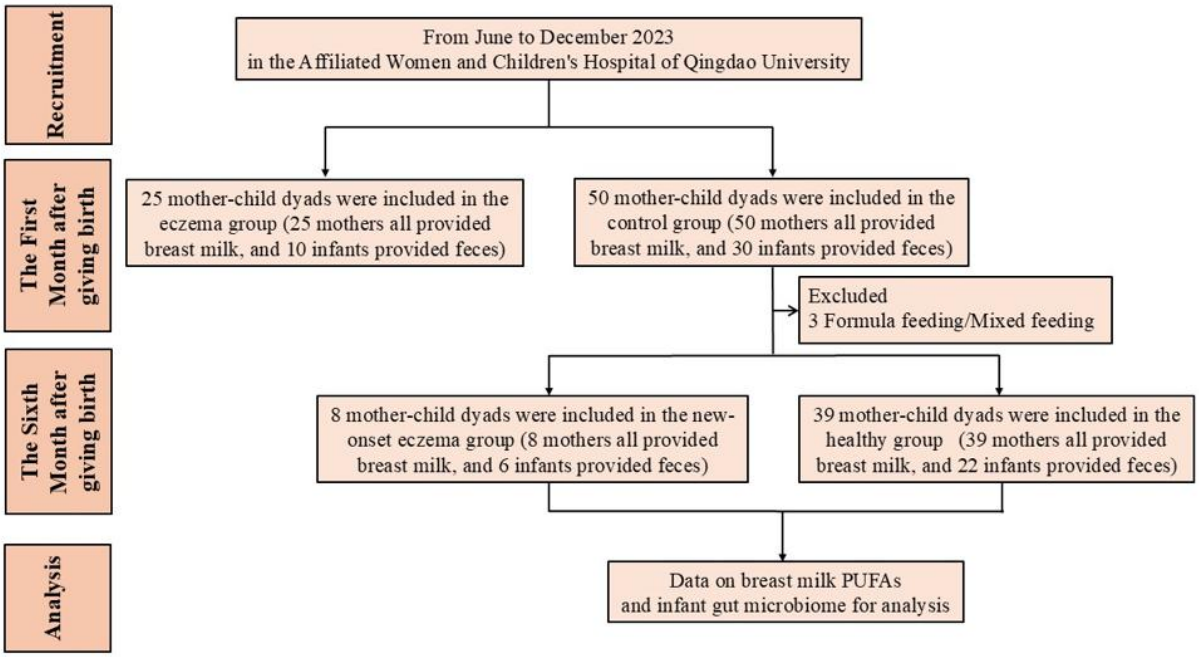

Figure 1. Flow diagram of study design. PUFAs, polyunsaturated fatty acids

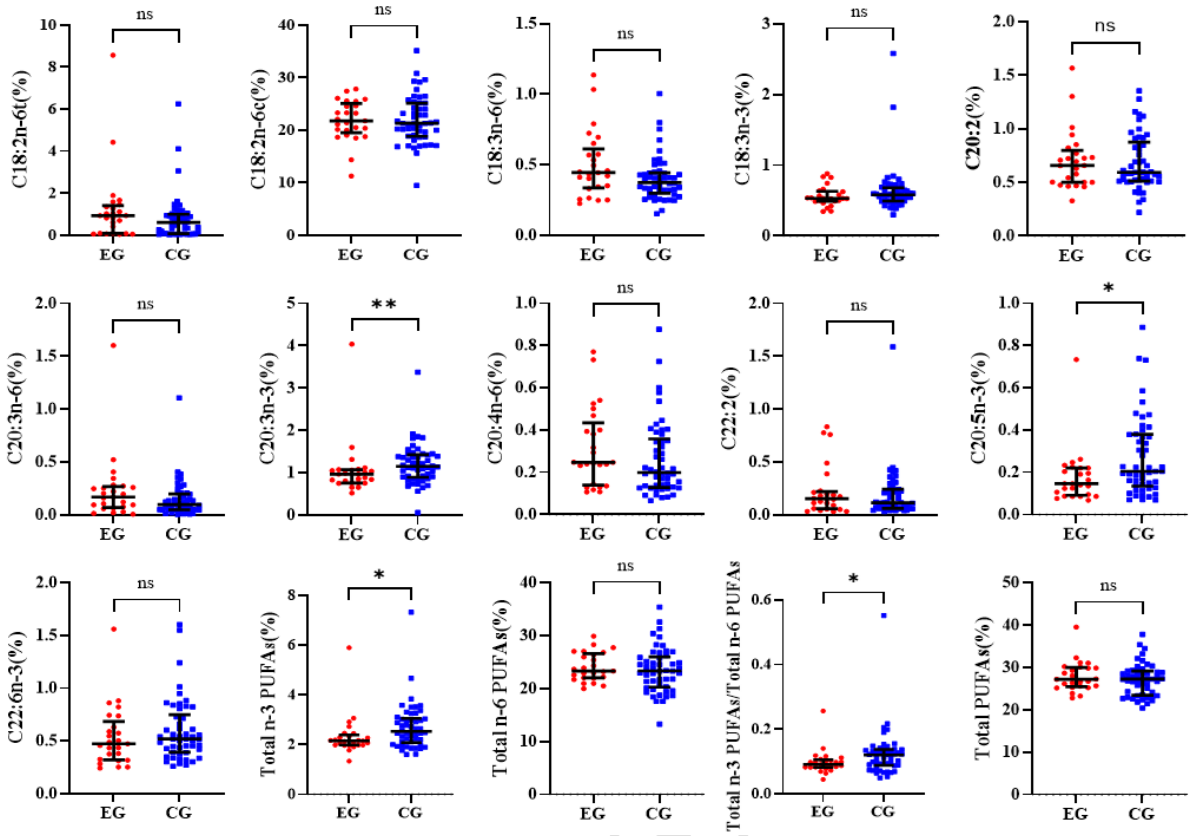

Figure 2. Comparative analysis of breast milk PUFAs levels between the eczema group (n = 25) and the control group (n = 50) at 1 month postpartum, presented as median and interquartile range. PUFAs, polyunsaturated fatty acids; t, trans; c, cis; EG, eczema group; CG, control group. *p < 0.05; **p < 0.01; ns, non-significant

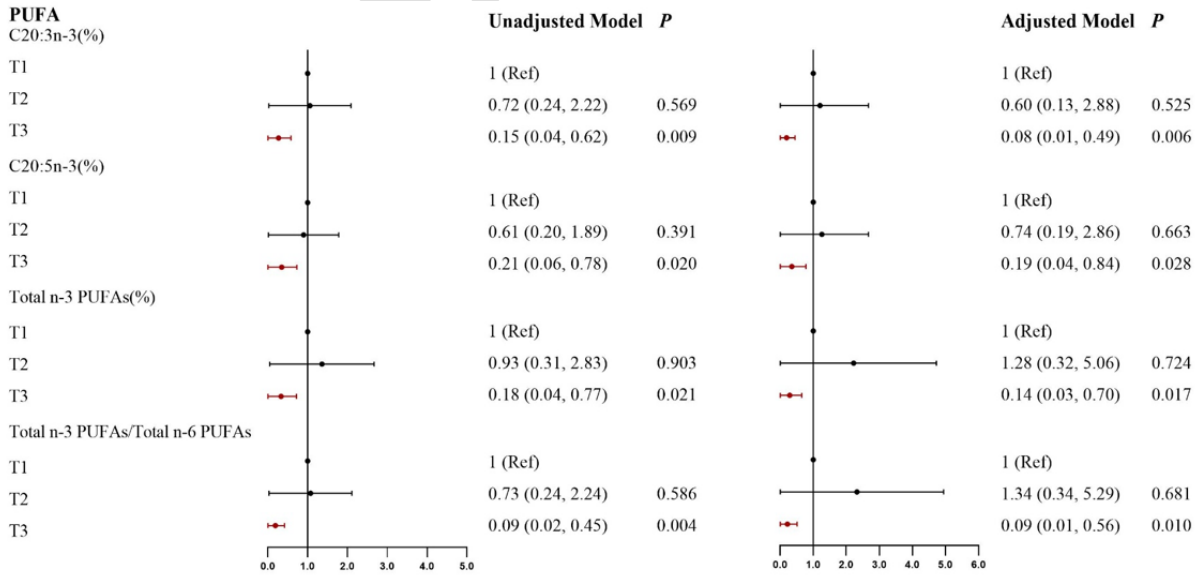

Figure 3. Association between maternal breast milk PUFAs and infant eczema risk at 1 month postpartum (n = 75). PUFAs, polyunsaturated fatty acids; Crude model: not adjusted; Adjusted model was adjusted for the mother's age, BMI, education, parity, delivery mode, annual household income, parental history of allergies, use of disinfectants, presence of pets and the infant's gender, birth weight, and birth length

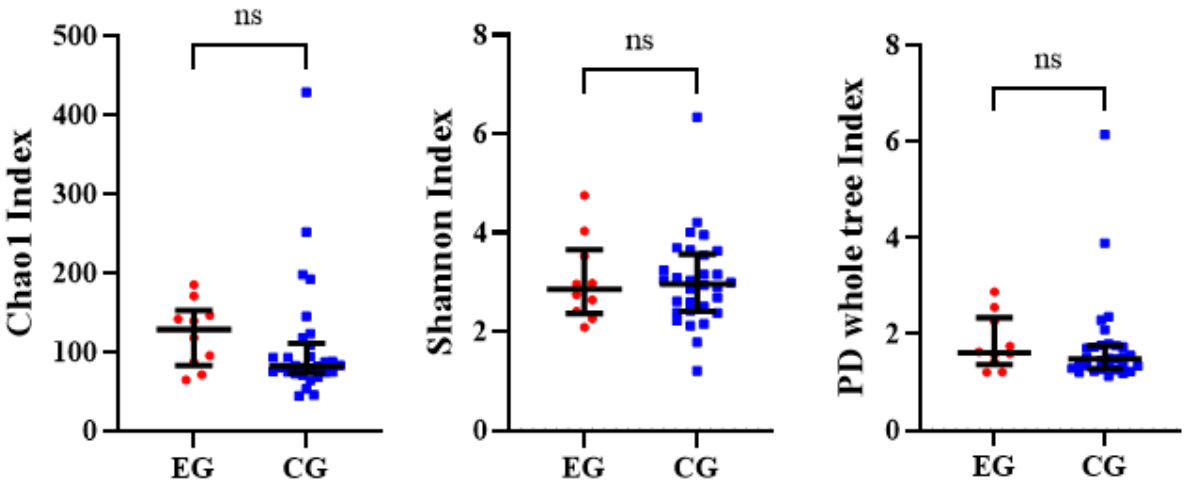

Figure 4. α-diversity (at the ASV level) of infant gut microbiota at 1 month postpartum (eczema group, n = 10; control group, n = 30). ns, non-significant; EG, eczema group; CG, control group

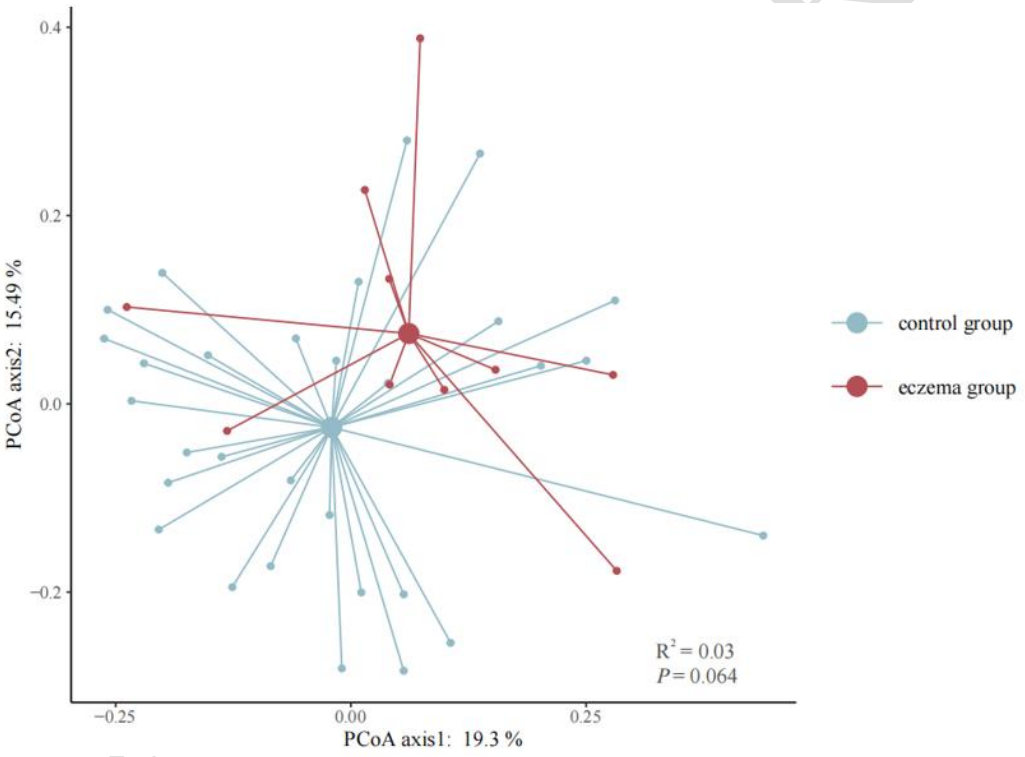

Figure 5. β-diversity of infant gut microbiota (ASV level) in the eczema group (n = 10) and control group (n = 30) at 1 month postpartum

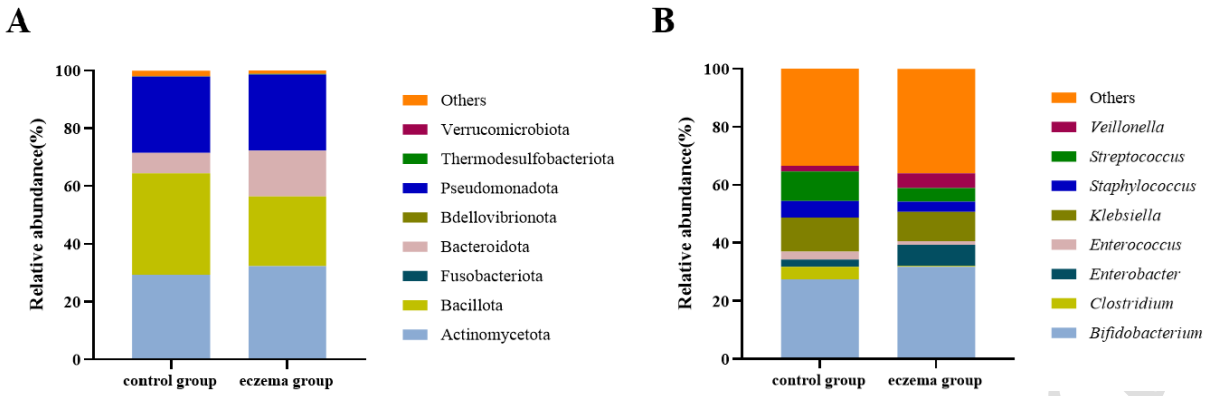

Figure 6. Infant gut microbial composition at the phylum (A) and genus (B) levels in the eczema group (n = 10) and control group (n = 30) at 1 month postpartum

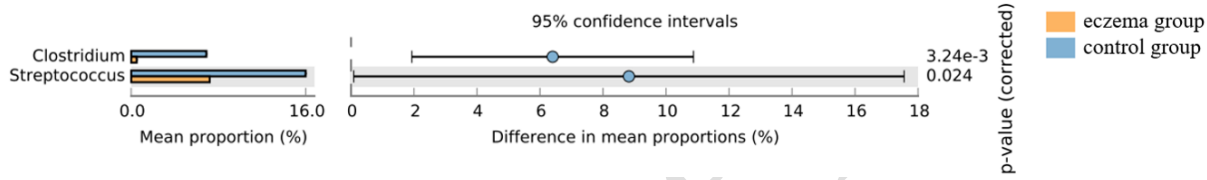

Figure 7. Differential infant gut microbiota between the eczema group (n = 10) and control group (n = 30) at 1 month postpartum (at the genus level)

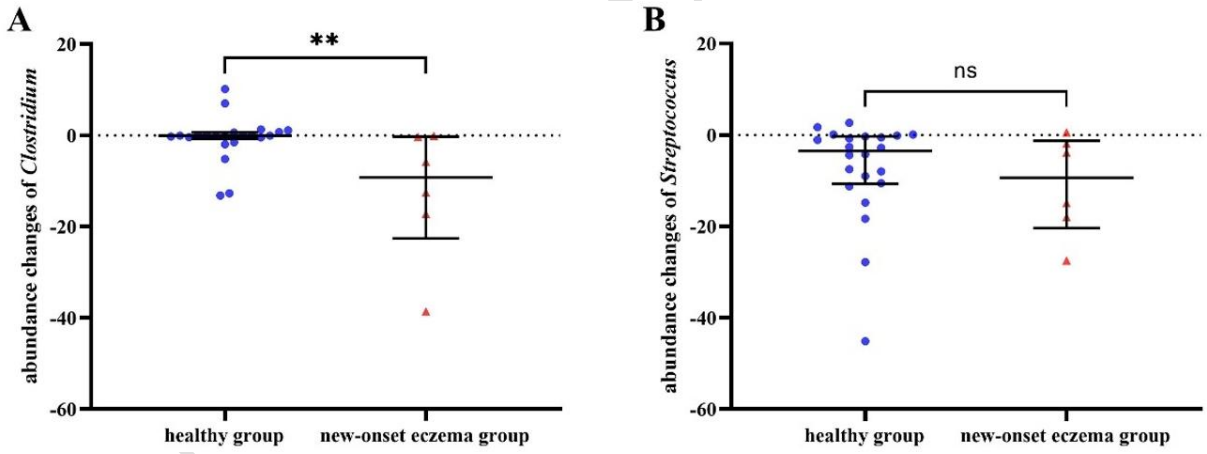

Figure 8. Comparative analysis of changes in the abundance of infant's gut microbiota during the follow-up period. **p < 0.01; ns, non-significant

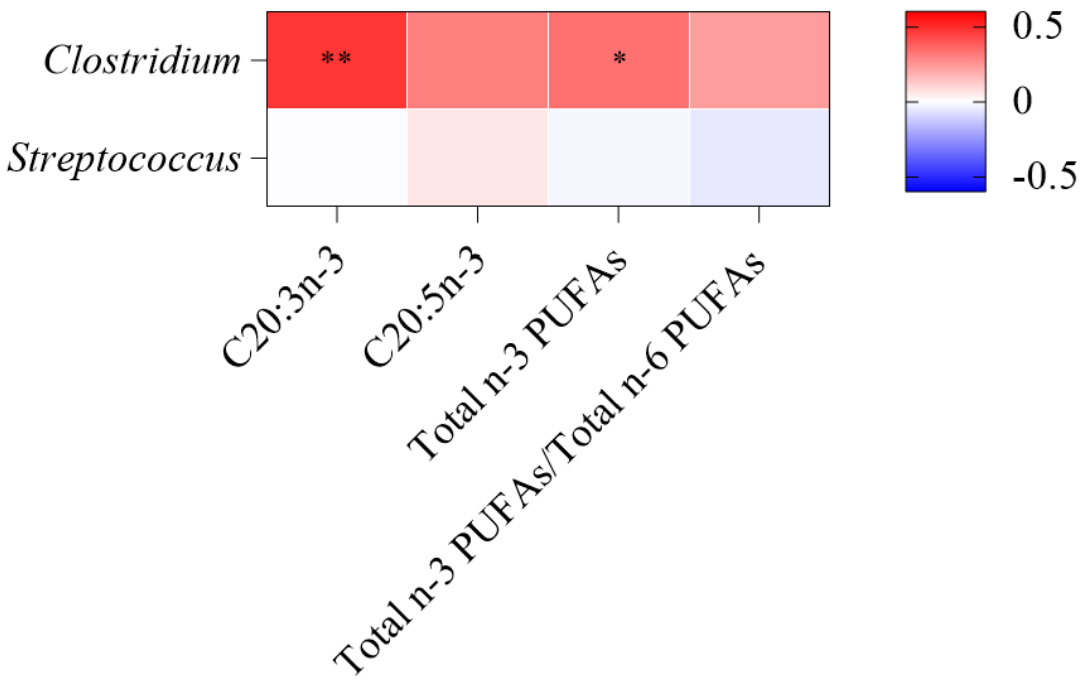

Figure 9. Correlation analysis between levels of differential PUFAs in breast milk and abundances of differential gut microbiota of infants (n = 40) at 1 month postpartum. PUFAs, polyunsaturated fatty acids; *p < 0.05; **p < 0.01

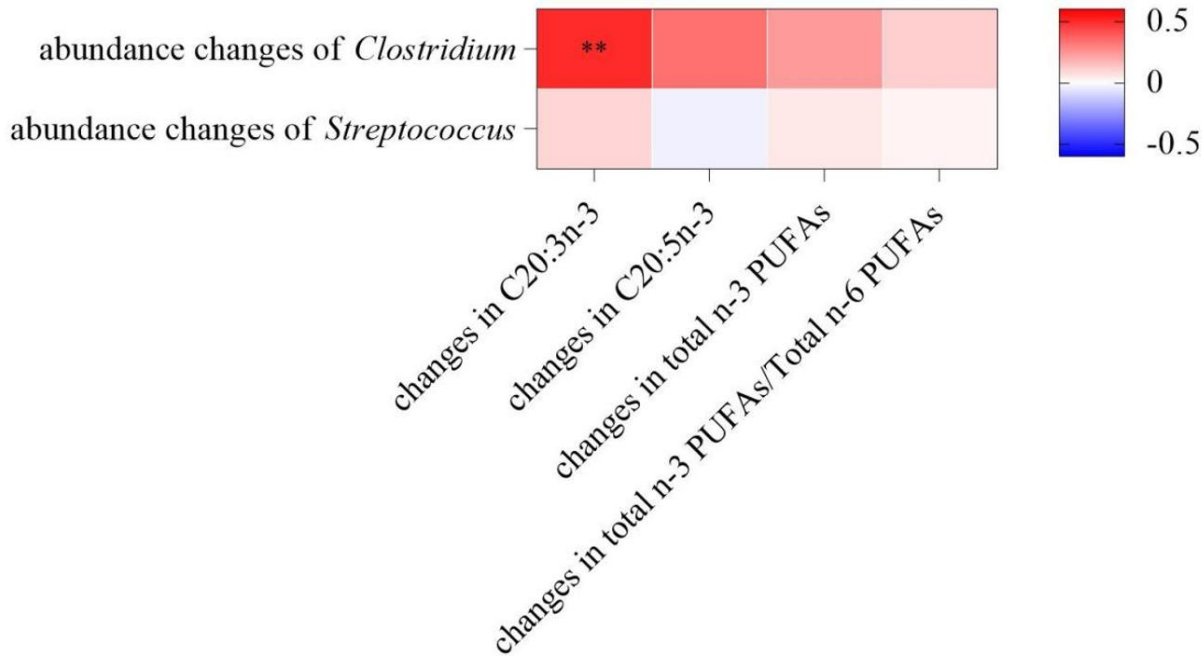

Figure 10. Correlation analysis between changes in differential PUFAs in breast milk and abundance changes in differential gut microbiota in infants (n = 28). PUFAs, polyunsaturated fatty acids; **p < 0.01.